ABNORMAL PSYCHOLOGY



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Abnormal Psychology 2e

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CHAPTER OVERVIEW

1: Understanding Abnormal Behavior

The textbook starts by defining mental disorders, exploring how they have been conceptualized and treated throughout history. It introduces the major classification systems, including the DSM (Diagnostic and Statistical Manual of Mental Disorders) and the ICD (International Classification of Diseases), and discusses the impact of stigma on individuals with mental illnesses.

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1.1: Why It Matters- Understanding Mental Disorders

Why learn about abnormal psychology?



Figure **1.1.1**: Mental illnesses are common in the United States. Nearly one in five U.S. adults live with a mental illness (51.5 million in 2019), of which there were an estimated 13.1 million adults, or 5.2% of the population aged 18 or older with a serious mental illness. A 2015 study showed that 45% of the American homeless had some form of mental illness, and 25% had a more serious mental illness at some point during their lives.

Welcome to the study of abnormal psychology, sometimes referred to as psychopathology, clinical psychology, or the study of mental illness. In this module, we will define what psychological disorders are, how they are diagnosed and classified, and how assessments are used to identify them. There are many myths surrounding mental illness, so let's begin by addressing a few of them.

Mental Health Myths

Myth: Mental health problems don't affect me.

Fact: Mental health problems are actually very common. In 2020:

- Over one in five American adults experienced a mental health issue.
- Nearly one in 10 adults experienced a period of major depression and 17.0% of the U.S. population aged 12 to 17.
- One in 25 Americans lived with a serious mental illness, such as schizophrenia, bipolar disorder, or major depression.

Suicide is consistently considered to be about the 10th leading cause of death in the United States. It accounts for the loss of more than 45,000 American lives each year, more than double the number of lives lost to homicide. Learn more about mental health problems at mentalhealth.gov.

Myth: Children do not experience mental health problems.

Fact: Even very young children may show early warning signs of mental health concerns. These mental health problems are often clinically diagnosable, and can be a product of the interaction of biological, psychological, and social factors.

Half of all mental health disorders show first signs before a person turns 14 years old, and three-quarters of mental health disorders begin before age 24.

Unfortunately, less than 20% of children and adolescents with diagnosable mental health problems receive the treatment they need. Early mental health support can help a child before problems interfere with other developmental needs.

Myth: People with mental health problems are violent and unpredictable.

Fact: The vast majority of people with mental health problems are no more likely to be violent than anyone else. Most people with mental illness are not violent and only three to 5% of violent acts can be attributed to individuals living with a serious mental illness. In fact, people with severe mental illnesses are over 10 times more likely to be victims of violent crime than the general

population. You probably know someone with a mental health problem and don't even realize it because many people with mental health problems are highly active and productive members of our communities.

Myth: People with mental health needs, even those who are managing their mental illness, cannot tolerate the stress of holding down a job.

Fact: People with mental health problems are just as productive as other employees. Employers who hire people with mental health problems report good attendance and punctuality as well as motivation, good work, and job tenure on par with or greater than other employees.

When employees with mental health problems receive effective treatment, it can result in

- lower total medical costs,
- increased productivity,
- lower absenteeism, and
- decreased disability costs.

Myth: Personality weakness or character flaws cause mental health problems. People with mental health problems can snap out of it if they try hard enough.

Fact: Mental health problems have nothing to do with being lazy or weak and many people need help to get better. Many factors contribute to mental health problems, including the following:

- biological factors, such as genes, physical illness, injury, or brain chemistry
- life experiences, such as trauma or a history of abuse
- family history of mental health problems

People with mental health problems can get better and many recover completely. Learn more about mental health recovery at mentalhealth.gov.

Myth: There is no hope for people with mental health problems. Once a friend or family member develops mental health problems, he or she will never recover.

Fact: Studies show that people with mental health problems get better and many recover completely. Recovery refers to the process in which people are able to live, work, learn, and participate fully in their communities. There are more treatments, services, and community support systems than ever before, and they work.

Myth: Therapy and self-help are a waste of time. Why bother when you can just take a pill?

Fact: Treatment for mental health problems varies depending on the individual and could include medication, therapy, or both. Many individuals work with a support system during the healing and recovery process.

Myth: I can't do anything for a person with a mental health problem.

Fact: Friends and loved ones can make a big difference. Only 44% of adults with diagnosable mental health problems and less than 20% of children and adolescents receive needed treatment. Friends and family can be important influences to help someone get the treatment and services they need by

- reaching out and letting them know you are available to help;
- helping them access mental health services;
- learning and sharing the facts about mental health, especially if you hear something that isn't true;
- treating them with respect, just as you would anyone else; and
- refusing to define them by their diagnosis or using labels such as "crazy."

Myth: Prevention doesn't work. It is impossible to prevent mental illnesses.

Fact: Prevention of mental, emotional, and behavioral disorders focuses on addressing known risk factors such as exposure to trauma that can affect the chances that children, youth, and young adults will develop mental health problems. Promoting the social-emotional well-being of children and youth leads to the following:

- higher overall productivity

- better educational outcomes
- lower crime rates
- stronger economies
- lower health care costs
- improved quality of life
- increased lifespan
- improved family life

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1.2: Introduction to Mental Disorders

What you'll learn to do: define mental disorders and explain how they are classified



Mental disorders are conditions characterized by abnormal thoughts, feelings, and behaviors. Although challenging, it is essential for psychologists and mental health professionals to agree on what experiences and actions constitute the presence of a mental disorder. Inner experiences and behaviors that are atypical or violate social norms could signify the presence of a disorder; however, each of these criteria alone is inadequate. It is generally accepted that a mental disorder is defined by significant disturbances in thoughts, feelings, and behaviors. These disturbances must reflect some kind of dysfunction (biological, psychological, or developmental), must cause significant impairment in one's life, and must not reflect culturally expected reactions to certain life events.

In this section, you will learn about how disorders are defined, and also how they are diagnosed and classified. The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) is the most commonly used classification manual that clinicians use to read about the criteria, prevalence, risk factors, and details of over 200 disorders.

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1.3: What Are Mental Disorders?

Learning Objectives

- Describe how psychological disorders are defined as well as the inherent difficulties in doing so

A **mental disorder** is a condition characterized by thoughts, feelings, and behaviors that create **dysfunction.** Dysfunction means a disruption of a person's ability to live their life productively or in a way that impairs their relationships, ability to think clearly, communicate with others, hold a job, or deal with stressful events. **Psychopathology** is the study of psychological disorders, including their symptoms, **etiology** (i.e., their causes), and treatment. The term *psychopathology* can also refer to the manifestation of a psychological disorder.

Although consensus can be difficult, it is important for mental health professionals to agree on what kinds of thoughts, feelings, and behaviors are truly abnormal in the sense that they genuinely indicate the presence of psychopathology. The person who washes his hands 40 times per day and the person who claims to hear the voices of demons exhibit behaviors and inner experiences that most persons would regard as abnormal. Consider the nervousness a person may feel when talking to an attractive love interest or the loneliness and longing for home a freshman may experience during their first semester of college—these feelings may not be regularly present, but they fall in the range of normal reaction. If these feelings persist, however, they may turn into feelings of anxiety or depression. One way to think about mental disorder symptoms is that they are *more persistent and extreme* variations of normal experiences. So, what kinds of thoughts, feelings, and behaviors represent a true psychological disorder? Psychologists work to distinguish psychological disorders from inner experiences and behaviors that are merely situational or unconventional.

Definition of a Psychological Disorder

Perhaps the simplest approach to conceptualizing psychological disorders is to label behaviors, thoughts, and inner experiences that are atypical, distressful, dysfunctional, and sometimes even dangerous as signs of a disorder. For example, if you ask a classmate for a date and you are rejected, you probably would feel a little dejected. Such feelings would be normal. If you felt extremely depressed—so much so that you lost interest in activities, had difficulty eating or sleeping, felt utterly worthless, and contemplated suicide—your feelings would be **atypical**, would deviate from the norm, and could signify the presence of a psychological disorder because they are more intense and persistent than normal feelings of rejection or disappointment. Just because something is atypical, however, does not necessarily mean it is disordered.

For example, only about 4% of people in the United States have red hair, so red hair is considered an atypical characteristic (Figure 1), but it is not considered disordered; it's just unusual. And it is less unusual in Scotland, where approximately 13% of the population has red hair ("DNA Project Aims," 2012). As you will learn, some disorders, although not exactly typical, are far from atypical, and the rates in which they appear in the population are surprisingly high.



(a) (b) (c)

Figure **1.3.1**: Red hair is considered unusual, but not abnormal. (a) Isla Fischer, (b) Prince Harry, and (c) Marcia Cross are three natural redheads. (credit a: modification of work by Richard Goldschmidt; credit b: modification of work by Glyn Lowe; credit c: modification of work by Kirk Weaver)

If we can agree that merely being atypical is an insufficient criterion for having a psychological disorder, is it reasonable to consider behavior or inner experiences that differ from widely expected cultural values or expectations as disordered? Using this criterion, a woman who walks around a subway platform wearing a heavy winter coat in July while screaming obscenities at

strangers may be considered as exhibiting symptoms of a psychological disorder. Her actions and clothes violate socially accepted rules governing appropriate dress and behavior—these characteristics are atypical.

Cultural Expectations

Violating cultural expectations is not, in and of itself, a satisfactory means of identifying the presence of a psychological disorder. Since behavior varies from one **culture** to another, what may be expected and considered appropriate in one culture may not be viewed as such in other cultures. Culture can be defined as socially shared beliefs, values, norms, expectations, and practices within a group, community, or society at large.[1] For example, returning a stranger's smile is expected in the United States because a pervasive social norm dictates that we reciprocate friendly gestures. A person who refuses to acknowledge such gestures might be considered socially awkward—perhaps even disordered—for violating this expectation. However, such expectations are not universally shared. Cultural expectations in Japan involve showing reserve, restraint, and a concern for maintaining privacy around strangers. Japanese people are generally unresponsive to smiles from strangers (Patterson et al., 2007). Eye contact provides another example. In the United States and Europe, eye contact with others typically signifies honesty and attention. However, most Latin American, Asian, and African cultures interpret direct eye contact as rude, confrontational, and aggressive (Pazain, 2010). Thus, someone who makes eye contact with you could be considered appropriate and respectful or brazen and offensive, depending on your culture (Figure 1.3.2).



Figure 1.3.2: Eye contact is one of many social gestures that vary from culture to culture. (credit: Joi Ito)

Hallucinations (seeing or hearing things that are not physically present) in Western societies are a violation of cultural expectations, and a person who reports such inner experiences is readily labeled as psychologically disordered. In other cultures, visions that, for example, pertain to future events may be regarded as normal experiences that are positively valued (Bourguignon, 1970). Finally, it is important to recognize that cultural norms change over time: what might be considered typical in a society at one time may no longer be viewed this way later, similar to how fashion trends from one era may elicit quizzical looks decades later—imagine how legwarmers or the big hair of the 1980s would go over on your campus today.

American Psychiatric Association (APA) Definition

According to the American Psychiatric Association (APA) (2013), a psychological disorder is a condition that is said to consist of the following:

- **There are significant disturbances in thoughts, feelings, and behaviors**. A person must experience inner states (e.g., thoughts and/or feelings) and exhibit behaviors that are clearly disturbed—that is, unusual, but in a negative, self-defeating way. Often, such disturbances are troubling to those around the individual who experiences them. For example, an individual who is uncontrollably preoccupied by thoughts of germs spends hours each day bathing, has inner experiences, and displays behaviors that most would consider atypical and negative (disturbed) and that would likely be troubling to family members.
- **The disturbances reflect some kind of biological, psychological, or developmental dysfunction**. Disturbed patterns of inner experiences and behaviors should reflect some flaw (dysfunction) in the internal biological, psychological, and developmental mechanisms that lead to normal, healthy psychological functioning. For example, the hallucinations observed in schizophrenia could be a sign of brain abnormalities.
- **The disturbances lead to significant distress or disability in one's life**. A person's inner experiences and behaviors are considered to reflect a psychological disorder if they cause the person considerable distress, or greatly impair his ability to function as a normal individual (often referred to as functional impairment, or occupational and social impairment). As an illustration, a person's fear of social situations might be so distressing that it causes the person to avoid all social situations (e.g., preventing that person from being able to attend class or apply for a job).

- **The disturbances do not reflect expected or culturally approved responses to certain events**. Disturbances in thoughts, feelings, and behaviors must be socially unacceptable responses to certain events that often happen in life. For example, it is perfectly natural (and expected) that a person would experience great sadness and might wish to be left alone following the death of a close family member. Because such reactions are in some ways culturally expected, the individual would not be assumed to signify a mental disorder.

One simple way to remember the criteria in defining psychological disorders are the four D's: *deviance*, *dysfunction*, *distress*, and *danger* (and possibly even a fifth D for the *duration*).[2]

Some believe that there is no essential criterion or set of criteria that can definitively distinguish all cases of disorder from nondisorder (Lilienfeld & Marino, 1999). In truth, no single approach to defining a psychological disorder is adequate by itself, nor is there universal agreement on where the boundary is between disordered and not disordered. From time to time, we all experience anxiety, unwanted thoughts, and moments of sadness; our behavior at other times may not make much sense to ourselves or to others. These inner experiences and behaviors can vary in their intensity, but are only considered disordered when they are highly disturbing to us and/or others, suggest a dysfunction in normal mental functioning, and are associated with significant distress or disability in social or occupational activities.

Think It Over

- Identify a behavior that is considered unusual or abnormal in your own culture that would be considered normal and expected in another culture.

Glossary

atypical: describes behaviors, thoughts, or feelings that deviate from the norm

culture: socially shared beliefs, values, norms, expectations, and practices within a group, community, or society at large

etiology: cause or causes of a psychological disorder

dysfunction: disruption of a person's ability to live their life productively or that impair their relationships, ability to think clearly, communicate with others, hold a job or deal with stressful events

mental disorder: a condition characterized by thoughts, feelings, and behaviors that create dysfunction

psychopathology: study of psychological disorders, including their symptoms, causes, and treatment; manifestation of a mental disorder

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1. Betancourt, H., & López, S. R. (1993). The study of culture, ethnicity, and race in American psychology. *American Psychologist*, 48, 629–637. ↵
2. Davis, T. (2009). "Conceptualizing Psychiatric Disorders Using 'Four D's' of Diagnoses," 5. ↵

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1.4: Mental Health Treatment in the Past

Learning Objectives

- Describe etiological theories (supernatural, somatogenic, and psychogenic) used to explain mental illness up through the Middle Ages
- Explain modern views on mental illness, including the rationale and impact of deinstitutionalization

Lacking scientific information or ways to think about or approach situations, people in the past turned to other explanations for what caused mental disorders. It was common for them to believe that people with psychological disorders, or those exhibiting strange behavior, were possessed by demons or were being punished by God or gods for their sins. Following that logic, treatment involved forcing those afflicted to take part in exorcisms, suffer imprisonment, or even face execution.

Wilhelm Griesinger (1817–1868), is remembered for initiating reforms in the treatment of the mentally ill as well as introducing changes to the existing asylum system. Griesinger believed in the integration of the mentally ill into society and proposed that short-term hospitalization be combined with the close cooperation of natural support systems. He also provided valuable insights on the nature of **psychopathic** or antisocial behavior (characterized by persistent antisocial behavior, impaired empathy and remorse, and bold, disinhibited, and egotistical traits). Griesinger wrote, “Psychiatry has undergone a transformation in its relation to the rest of medicine. . . . This transformation rests principally on the realization that patients with so-called ‘mental illnesses’ are really individuals with illnesses of the nerves and brain.”[1] Much later, asylums were built to house the mentally ill, but the patients received little to no treatment, and many of the methods used were cruel. During the 1800s, Philippe Pinel and Dorothea Dix argued for more humane treatment of people with psychological disorders.

In the mid-1960s, the deinstitutionalization movement gained support and asylums were closed, enabling people with mental illness to return home and receive treatment in their own communities. Some did go to their family homes, but many became homeless due to a lack of resources and support mechanisms. It is important to note that even though we have a more scientific understanding of the causes of mental disorders today, there is still much we do not know and supernatural explanations are still sometimes used even now to explain abnormal behaviors. These explanations, such as demonic possession or even that a full moon can increase the intensity of mental illness symptoms, combined with other stereotypes and misunderstandings, are potent contributors to **stigma**, meaning prejudicial attitudes and discrimination against those who experience mental disorders.



Figure 1.4.1: This painting by Francisco Goya, called *The Madhouse*, depicts a mental asylum and its inhabitants in the early 1800s. It portrays those with psychological disorders as victims.

Today, instead of asylums, there are psychiatric hospitals run by state governments and local community hospitals, with an emphasis on short-term stays. However, most people suffering from mental illness are not hospitalized. A person suffering symptoms could speak with a primary care physician, who most likely would refer him to a psychiatrist or to someone who specializes in therapy. The person can receive outpatient mental health services from a variety of sources, including psychologists, psychiatrists, marriage and family therapists, school counselors, clinical social workers, and religious personnel. These therapy sessions would be covered through insurance, government funds, or private (self) pay.

Ancient Views on Mental Illness

Throughout history there have been three general theories of the **etiology** of mental illness: supernatural, somatogenic, and psychogenic. **Supernatural theories** attribute mental illness to possession by evil or demonic spirits, displeasure of gods, eclipses, planetary gravitation, curses, and sin. **Somatogenic theories** (from the Greek word *soma* meaning body, therefore biological causes) identify disturbances in physical functioning resulting from either illness, genetic inheritance, brain damage, or imbalance. **Psychogenic theories** focus on traumatic or stressful experiences, maladaptive learned associations, and cognitions, or distorted perceptions. Etiological theories of mental illness determine the care and treatment mentally ill individuals receive. As we will see below, an individual believed to be possessed by the devil will be viewed and treated differently from an individual believed to be suffering from an excess of yellow bile. Their treatments will also differ, from exorcism to blood-letting. The theories, however, remain the same. They coexist as well as recycle over time.

Trephination, as a treatment, is an example of possibly the earliest supernatural explanation for mental illness (Figure **1.4.2**). Examination of prehistoric skulls and cave art from as early as 6500 BC has identified surgical drilling of holes in skulls to treat head injuries and epilepsy as well as to allow the evil spirits trapped within the skull, that were presumed to be causing the symptoms of mental disorder, to be released (Restak, 2000). Trephination ultimately fell out of favor as a treatment for psychological disorders.



Figure **1.4.2**: Evidence of the ancient practice of trephination.

Around 2700 BC, Chinese medicine's concept of complementary positive and negative bodily forces (yin and yang) attributed mental (and physical) illness to an imbalance between these forces. As such, a harmonious life that allowed for the proper balance of yin and yang and movement of vital air was essential (Tseng, 1973) to health and a good life.

Mesopotamian and Egyptian papyri from 1900 BC describe women suffering from mental illness resulting from a wandering uterus (later named **hysteria** by the Greeks): the uterus could become dislodged and attached to parts of the body like the liver or chest cavity, preventing their proper functioning or producing varied and sometimes painful symptoms. As a result, the Egyptians, and later the Greeks, also employed a somatogenic treatment of strong-smelling substances to guide the uterus back to its proper location (pleasant odors to lure and unpleasant ones to dispel).

Throughout classical antiquity, we see a return to supernatural theories of demonic possession or godly displeasure to account for abnormal behavior that was beyond the person's control. Temple attendance with religious healing ceremonies and incantations to the gods were employed to assist in the healing process. Hebrews saw madness as punishment from God so treatment consisted of confessing sins and repenting. Physicians were also believed to be able to comfort and cure madness, however.

Greek physicians rejected supernatural explanations of mental disorders. It was around 400 BC that Hippocrates (460–370 BC) attempted to separate superstition and religion from medicine by systematizing the belief that a deficiency in or especially an excess of one of the four essential bodily fluids (i.e., humors)—blood, yellow bile, black bile, and phlegm—was responsible for physical and mental illness. For example, someone who was too temperamental suffered from too much blood and thus blood-letting would be the necessary treatment. Hippocrates classified mental illness into one of four categories—epilepsy, mania, melancholia, and brain fever—and like other prominent physicians and philosophers of his time, he did not believe mental illness was shameful or that mentally ill individuals should be held accountable for their behavior. Mentally ill individuals were cared for at home by family members and the state shared no responsibility for their care. **Humorism** remained a recurrent somatogenic theory up until the 19th century.

While Greek physician Galen (AD 130–201) rejected the notion of a uterus having an animistic soul, he agreed with the notion that an imbalance of the four bodily fluids could cause mental illness. He also opened the door for psychogenic explanations for mental illness by allowing for the experience of psychological stress as a potential cause of abnormality. Galen's psychogenic theories were ignored for centuries, however, as physicians attributed mental illness to physical causes throughout most of the millennium.

Mental Health during the Middle Ages

By the late Middle Ages, economic and political turmoil threatened the power of the Roman Catholic Church. Between the 11th and 15th centuries, supernatural theories of mental disorders again dominated Europe, fueled by natural disasters like plagues and famines that laypeople interpreted as brought about by the devil. Superstition, astrology, and alchemy took hold, and common treatments included prayer rites, relic touching, confessions, and atonement. Beginning in the 13th century, the mentally ill, especially women, began to be persecuted as witches who were possessed. At the height of the witch hunts during the 15th through 17th centuries, with the Protestant Reformation having plunged Europe into religious strife, two Dominican monks wrote the *Malleus Maleficarum* (1486) as the ultimate manual to guide witch hunts. Johann Weyer and Reginald Scot tried to convince people in the mid-to late-16th century that accused witches were actually women with mental illnesses and that mental illness was not due to demonic possession but to faulty metabolism and disease, but the Church's Inquisition banned both of their writings. Witch-hunting did not decline until the 17th and 18th centuries, after more than 100,000 presumed witches had been burned at the stake (Schoeneman, 1977; Zilboorg & Henry, 1941).

Also during medieval times, a person with mental illness might be considered to be possessed by demons. If someone was considered to be possessed, there were several forms of treatment to release spirits from the individual. The most common treatment was exorcism, often conducted by priests or other religious figures: incantations and prayers were said over the person's body, and they may have been given some medicinal drinks. Trephining was also practiced during this time—a small hole was made in the afflicted individual's skull to release spirits from the body. Most people treated in this manner died. In addition to exorcism and trephining, other practices involved execution or imprisonment of people with psychological disorders. Still others were left to be homeless beggars. Generally speaking, most people who exhibited strange behaviors were greatly misunderstood and treated cruelly. The prevailing theory of psychopathology in earlier history was the idea that mental illness was the result of demonic possession by either an evil spirit or an evil god because early beliefs incorrectly attributed all unexplainable phenomena to deities deemed either good or evil.

Supernatural Perspectives of Psychological Disorders

Convents throughout Europe in the 16th and 17th centuries reported hundreds of nuns falling into a state of frenzy in which the afflicted foamed at the mouth, screamed and convulsed, sexually propositioned priests, and confessed to having carnal relations with devils or Christ. Although today these cases would suggest serious mental illness, at the time, these events were routinely explained as possession by devilish forces (Waller, 2009a). Similarly, grievous fits by young girls are believed to have precipitated the witch panic in New England late in the 17th century (Demos, 1983). Such beliefs in supernatural causes of mental illness are still held in some societies today; for example, beliefs that supernatural forces cause mental illness are common in some cultures in modern-day Nigeria (Aghukwa, 2012).

? Dig Deeper: Dancing Mania



Figure **1.4.3**: In *The Extraction of the Stone of Madness*, a 15th century painting by Hieronymus Bosch, a practitioner is using a tool to extract an object (the supposed "stone of madness") from the head of an afflicted person.

Between the 11th and 17th centuries, a curious epidemic swept across Western Europe. Groups of people would suddenly begin to dance with wild abandon. This compulsion to dance—referred to as dancing mania—sometimes gripped thousands of people at a time (Figure **1.4.4**). Historical accounts indicate that those afflicted would sometimes dance with bruised and bloody feet for days or weeks, screaming of terrible visions and begging priests and monks to save their souls (Waller, 2009b). What caused dancing mania is not known, but several explanations have been proposed, including spider venom and ergot poisoning ("Dancing Mania," 2011).



Figure **1.4.4**: Although the cause of dancing mania, depicted in this painting, was unclear, the behavior was attributed to supernatural forces.

Historian John Waller (2009a, 2009b) has provided a comprehensive and convincing explanation of dancing mania that suggests the phenomenon was attributable to a combination of three factors: psychological distress, social contagion, and belief in supernatural forces. Waller argued that various disasters of the time (such as famine, plagues, and floods) produced high levels of psychological distress that could increase the likelihood of succumbing to an involuntary trance state. Waller indicated that anthropological studies and accounts of possession rituals show that people are more likely to enter a trance state if they expect it to happen, and that entranced individuals behave in a ritualistic manner, their thoughts and behavior shaped by the spiritual beliefs of their culture. Thus, during periods of extreme physical and mental distress, all it took were a few people—believing themselves to have been afflicted with a dancing curse—to slip into a spontaneous trance and then act out the part of one who is cursed by dancing for days on end.

From the Middle Ages to the Twentieth Century

By the 18th century, people who were considered odd and unusual were placed in asylums. **Asylums** were the first institutions created for the specific purpose of housing people with psychological disorders, but the focus was ostracizing them from society rather than treating their disorders. Often these people were kept in windowless dungeons, beaten, chained to their beds, and had little to no contact with caregivers.

In the late 1700s, a French physician, Philippe Pinel, argued for the more humane treatment of the mentally ill. Pinel suggested that they be unchained and talked to, and that's just what he did for patients at La Salpêtrière in Paris in 1795. Patients benefited from this more humane treatment, and many were able to leave the hospital.



Figure **1.4.5**: This painting by Tony Robert-Fleury depicts Dr. Philippe Pinel ordering the removal of chains from patients at the Salpêtrière asylum in Paris.

In the 19th century, Dorothea Dix led reform efforts for mental health care in the United States. She investigated how those who are mentally ill and poor were cared for, and she discovered an underfunded and unregulated system that perpetuated abuse of this population (Tiffany, 1891). Horrified by her findings, Dix began lobbying various state legislatures and the U.S. Congress for change (Tiffany, 1891). Her efforts led to the creation of the first mental asylums in the United States.



Figure **1.4.6**: Dorothea Dix was a social reformer who became an advocate for the indigent insane and was instrumental in creating the first American mental asylum. She did this by relentlessly lobbying state legislatures and Congress to set up and fund such institutions.

Despite reformers' efforts, however, a typical asylum was filthy, offered very little treatment, and often kept people for decades. At Willard Psychiatric Center in upstate New York, for example, one treatment was to submerge patients in cold baths for long periods of time. Electroshock treatment was also used, and the way the treatment was administered often broke patients' backs; in 1943, doctors at Willard administered 1,443 shock treatments (Willard Psychiatric Center, 2009). (Electroshock is now called electroconvulsive treatment, and the therapy is still used, but with safeguards and under anesthesia. A brief application of electric stimulus is used to produce a generalized seizure. Controversy continues over its effectiveness versus the side effects.) Many of the wards and rooms were so cold that a glass of water would be frozen by morning (Willard Psychiatric Center, 2009). Willard's doors were not closed until 1995. Conditions like these remained commonplace until well into the 20th century.

European psychiatry in the late 18th century and throughout the 19th century struggled between somatogenic and psychogenic explanations of mental illness, particularly hysteria, which caused physical symptoms such as blindness or paralysis with no apparent physiological explanation. Franz Anton Mesmer (1734–1815), influenced by contemporary discoveries in electricity, attributed hysterical symptoms to imbalances in a universal magnetic fluid found in individuals, rather than to a wandering uterus (Forrest, 1999). James Braid (1795–1860) shifted this belief in **mesmerism** to one in hypnosis, thereby proposing a psychogenic treatment for the removal of symptoms. At the time, famed Salpetriere Hospital neurologist Jean-Martin Charcot (1825–1893), and Ambroise Auguste Liébault (1823–1904) and Hyppolyte Bernheim (1840–1919) of the Nancy School in France, were engaged in a bitter battle over the best explanation for hysteria, with Charcot maintaining that the hypnotic suggestibility underlying hysteria was a neurological condition while Liébault and Bernheim believed it to be a general trait that varied in the population. Josef Breuer (1842–1925) and Sigmund Freud (1856–1939) would resolve this dispute in favor of a psychogenic explanation for mental illness by treating hysteria through hypnosis, which eventually led to the cathartic method that became the precursor for psychoanalysis during the first half of the 20th century.

Emil Kraepelin (1856–1926) was a German psychiatrist. H.J. Eysenck's *Encyclopedia of Psychology* identifies him as the founder of modern scientific psychiatry, psychopharmacology, and psychiatric genetics. Kraepelin believed the chief origin of psychiatric disease to be biological and genetic malfunction. His theories dominated psychiatry at the start of the 20th century and, despite the later psychodynamic influence of Sigmund Freud and his disciples, enjoyed a revival at century's end. Kraepelin announced that he had found a new way of looking at mental illness, referring to the traditional view as "symptomatic" and to his view as "clinical." This turned out to be his paradigm-setting synthesis of the hundreds of mental disorders classified by the 19th century, grouping diseases together based on classification of **syndrome—**common *patterns* of symptoms over time—rather than by simple similarity of major symptoms in the manner of his predecessors. Drawing on his long-term research, and using the criteria of course, outcome, and prognosis, he developed the concept of **dementia praecox**, which he defined as the "sub-acute development of a peculiar simple condition of mental weakness occurring at a youthful age." This led Kraepelin to name what we now know as schizophrenia, dementia praecox (the dementia part signifying the irreversible mental decline). As a colleague of Alois Alzheimer, he was a co-discoverer of *Alzheimer's disease* (thought to be a brain disease leading to dementia), and his laboratory discovered its pathological basis. This may have led to the **medical model of illness.**

As we will discuss later in this course, in psychology, the term *medical model* refers to the assumption that psychopathology is the result of one's biology, that is to say, a physical/organic problem in brain structures, neurotransmitters, genetics, the endocrine

system, etc., as with traumatic brain injury, Alzheimer's disease, or Down's syndrome. One aspect of such a model is that an ill patient should not be held responsible for the condition. The patient should not be blamed or stigmatized for the illness. The medical model is useful in many situations as a guide for diagnosis, prognosis, and research. However, for most mental disorders, exclusive reliance on the medical model leads to an incomplete understanding, and, frequently, to incomplete or ineffective treatment interventions.

? Link to Learning

View this timeline showing the history of mental institutions in the United States.

Glossary

asylum: institution created for the specific purpose of housing people with mental disorders

deinstitutionalization: process of closing large asylums and integrating people back into the community where they can be treated locally

dementia praecox: a "premature dementia" or "precocious madness"

etiology: the causal description of all of the factors that contribute to the development of a disorder or illness

humorism (or humoralism): a belief held by ancient Greek and Roman physicians (and until the 19th century) that an excess or deficiency in any of the four bodily fluids, or humors—blood, black bile, yellow bile, and phlegm—directly affected their health and temperament

hysteria: term used by the ancient Greeks and Egyptians to describe a disorder believed to be caused by a woman's uterus wandering throughout the body and interfering with other organs (today referred to as conversion disorder, in which psychological problems are expressed in physical form)

mesmerism: derived from Franz Anton Mesmer in the late 18th century, an early version of hypnotism in which Mesmer claimed that hysterical symptoms could be treated through animal magnetism emanating from Mesmer's body and permeating the universe (and later through magnets); later explained in terms of high suggestibility in individuals

psychogenesis: developing from psychological origins

psychopathic: a personality disorder characterized by persistent antisocial behavior; impaired empathy and remorse; and bold, disinhibited, and egotistical traits

somatogenesis: developing from physical/bodily origins

supernatural: developing from origins beyond the visible observable universe

stigma: social attitudes and prejudice against persons who are different that devalue the person or make them feel disgraced or ashamed and that can lead to exclusion or discrimination against others

syndrome: common patterns of symptoms over time

trephination: the drilling of a hole in the skull, presumably as a way of treating psychological disorders

1. Quoted in Edward Shorter (1997). A History of Psychiatry: From the Era of the Asylum to the Age of Prozac. Wiley. p. 76. ISBN 978-0-471-15749-6. ↵

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1.5: Mental Health Treatment Today

Learning Objectives

- Describe the ways in which mental health services are delivered today, including the distinction between voluntary and involuntary treatment

According to the U.S. Department of Health and Human Services (2017), 18.9% of U.S. adults experienced mental illness in 2017. For teens (ages 13–18), the rate is similar to that of adults, and for children ages eight through 15, current estimates suggest that approximately 13% experience mental illness in a given year (National Institute of Mental Health [NIMH], 2017).

With many different treatment options available, approximately how many people receive mental health treatment per year? According to the Substance Abuse and Mental Health Services Administration (SAMHSA), in 2017, 14.8% of adults received treatment for a mental health issue (NIMH, 2017). These percentages, shown in Figure 1, reflect the number of adults who received care in inpatient and outpatient settings and/or used prescription medication for psychological disorders.

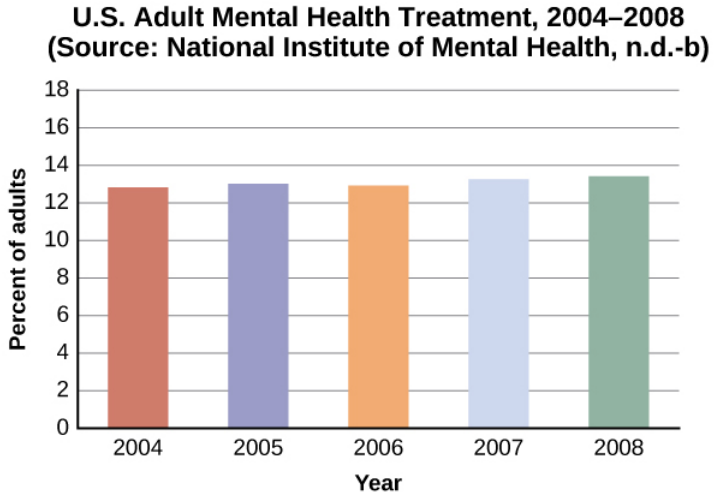


Figure **1.5.1**: The percentage of adults who received mental health treatment in 2004–2008 is shown. Adults seeking treatment increased slightly from 2004 to 2008.

Children and adolescents also receive mental health services. The Centers for Disease Control and Prevention's National Health and Nutrition Examination Survey (NHANES) found that approximately half (50.6%) of children with mental disorders had received treatment for their disorder within the past year (NIMH, n.d.-c). However, there were some differences between treatment rates by category of disorder (Figure 2). For example, children with anxiety disorders were least likely to have received treatment in the past year, while children with ADHD or a conduct disorder were more likely to receive treatment. Can you think of some possible reasons for these differences in receiving treatment?

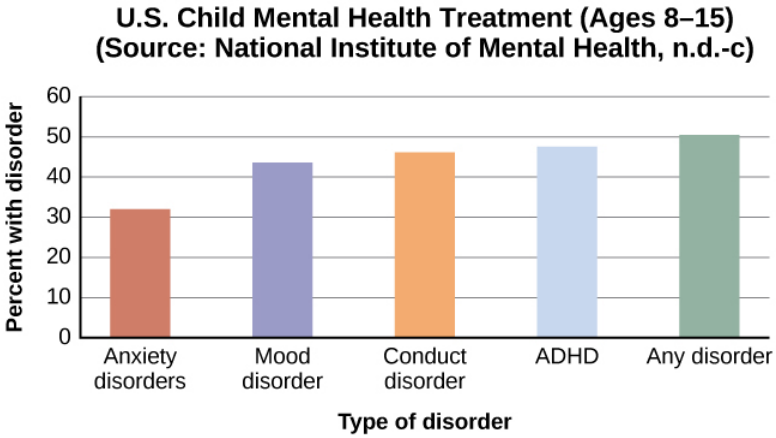


Figure **1.5.2**: About one-third to one-half of U.S. adolescents (ages eight through 15) with mental disorders receive treatment, with behavior-related disorders more likely to be treated.

Considering the many forms of treatment for mental health disorders available today, how did these forms of treatment emerge? We already learned about some historical views regarding mental illness (with some questionable approaches in light of modern understanding of mental illness). Now we will examine what treatment for mental health looks like in the more recent past.

As seen in the previous section, psychoanalysis was the dominant psychogenic treatment for mental illness during the first half of the 20th century, providing the launching pad for the more than 400 different schools of psychotherapy found today (Magnavita, 2006). Most of these schools cluster around broader behavioral, cognitive, cognitive-behavioral, psychodynamic, and client-centered approaches to psychotherapy applied in individual, marital, family, or group formats. Negligible differences have been found among all these approaches, however; their efficacy in treating mental illness is due to factors shared among all of the approaches (not particular elements specific to each approach): the therapist-patient alliance, the therapist's allegiance to the therapy, therapist competence, and placebo effects (Luborsky et al., 2002; Messer & Wampold, 2002).

In contrast, the leading somatogenic treatment for mental illness can be found in the establishment of the first psychotropic medications in the mid-20th century. Restraints, electroconvulsive therapy, and lobotomies continued to be employed in American state institutions until the 1970s, but they quickly made way for a burgeoning pharmaceutical industry that has viewed and treated mental illness as a chemical imbalance in the brain.

Both etiological theories coexist today in what the psychological discipline holds as the **biopsychosocial** model of explaining human behavior. While individuals may be born with a genetic predisposition for a certain psychological disorder, certain psychological stressors need to be present for them to develop the disorder. Sociocultural factors such as sociopolitical or economic unrest, poor living conditions, or problematic interpersonal relationships are also viewed as contributing factors. However, much as we want to believe that we are above the treatments described above, or that the present is always the most enlightened time, let us not forget that our thinking today continues to reflect the same underlying somatogenic and psychogenic theories of mental illness discussed throughout this cursory 9,000-year history.

Medicalizing Mental Illness

Starting in 1954 and gaining popularity in the 1960s, antipsychotic medications were introduced. These medications proved a tremendous help in controlling the symptoms of certain mental disorders, such as psychosis. Psychosis was a common diagnosis of individuals in mental hospitals, and it was often evidenced by symptoms like hallucinations and delusions, indicating a loss of contact with reality. Then in 1963, Congress passed and John F. Kennedy signed the Mental Retardation Facilities and Community Mental Health Centers Construction Act, which provided federal support and funding for community mental health centers (National Institutes of Health, 2013). This legislation changed how mental health services were delivered in the United States. It started the process of deinstitutionalization, the closing of large asylums, by providing for people to stay in their communities and be treated locally. In 1955, there were 558,239 severely mentally ill patients institutionalized at public hospitals (Torrey, 1997). By 1994, adjusted for the population, there were 92% fewer hospitalized individuals (Torrey, 1997).

Treatment Today

Today, there are community mental health centers across the nation. They are located in neighborhoods near the homes of clients, and they provide large numbers of people with mental health services of various kinds and for many kinds of problems. Unfortunately, part of what occurred with deinstitutionalization was that those released from institutions were supposed to go to newly created centers, but the system was not set up effectively. Centers were underfunded; staff was not trained to handle severe illnesses such as schizophrenia; there was high staff burnout; and no provision was made for the other services people needed, such as housing, food, and job training. Without these supports, those people released under deinstitutionalization often ended up homeless. Even today, a large portion of the homeless population is considered to be mentally ill (Figure **1.5.3**). Statistics show that 26% of homeless adults living in shelters experience mental illness (U.S. Department of Housing and Urban Development [HUD], 2011).



(a) (b)

Figure **1.5.3**: (a) Of the homeless individuals in U.S. shelters, about one-quarter have a severe mental illness (HUD, 2011). (b) Correctional institutions also report a high number of individuals living with mental illness. (credit a: modification of work by C.G.P. Grey; credit b: modification of work by Bart Everson)

Another group of the mentally ill population is involved in the corrections system. According to a 2006 special report by the Bureau of Justice Statistics (BJS), approximately 705,600 mentally ill adults were incarcerated in the state prison system, and another 78,800 were incarcerated in the federal prison system. A further 479,000 were in local jails. According to the study, “People with mental illnesses are overrepresented in probation and parole populations at estimated rates ranging from two to four times the general population” (Prins & Draper, 2009, p. 23). The Treatment Advocacy Center reported that the growing number of mentally ill inmates has placed a burden on the correctional system (Torrey et al., 2014).

Today, instead of asylums, there are psychiatric hospitals run by state governments and local community hospitals focused on short-term care. In all types of hospitals, the emphasis is on short-term stays, with the average length of stay being less than two weeks and often only several days. This is partly due to the very high cost of psychiatric hospitalization, which can be about $800 to $1000 per night (Stensland, Watson, & Grazier, 2012). Therefore, insurance coverage often limits the length of time a person can be hospitalized for treatment. Usually, individuals are hospitalized only if they are an imminent threat to themselves or others.

Most people suffering from mental illnesses are not hospitalized. If someone is feeling very depressed, complains of hearing voices, or feels anxious all the time, he or she might seek psychological treatment. A friend, spouse, or parent might refer someone for treatment. The individual might go see his primary care physician first and then be referred to a mental health practitioner.

Some people seek treatment because they are involved with the state’s child protective services—that is, their children have been removed from their care due to abuse or neglect. The parents might be referred to psychiatric or substance abuse facilities and the children would likely receive treatment for trauma. If the parents are interested in and capable of becoming better parents, the goal of treatment might be family reunification. For other children whose parents are unable to change—for example, the parent or parents are heavily addicted to drugs and refuse to enter treatment—the goal of therapy might be to help the children adjust to foster care and/or adoption (Figure **1.5.4**).



Figure **1.5.4**: Therapy with children may involve play. (credit: "LizMarie_AK"/Flick4)

Some people seek therapy because the criminal justice system referred them or required them to go. For some individuals, for example, attending weekly counseling sessions might be a condition of parole. If an individual is mandated to attend therapy, she is seeking services involuntarily. **Involuntary treatment** refers to therapy that is not the individual's choice. Other individuals might voluntarily seek treatment. **Voluntary treatment** means the person chooses to attend therapy to obtain relief from symptoms.

Psychological treatment can occur in a variety of places. An individual might go to a community mental health center or a practitioner in private or community practice. A child might see a school counselor, school psychologist, or school social worker. An incarcerated person might receive group therapy in prison. There are many different types of treatment providers, and licensing requirements vary from state to state. Besides psychologists and psychiatrists, there are clinical social workers, marriage and family therapists, and trained religious personnel who also perform counseling and therapy.

A range of funding sources pay for mental health treatment: health insurance, government, and private pay. In the past, even when people had health insurance, the coverage would not always pay for mental health services. This changed with the Mental Health Parity and Addiction Equity Act of 2008, which requires group health plans and insurers to make sure there is parity of mental health services (U.S. Department of Labor, n.d.). This means that co-pays, total number of visits, and deductibles for mental health and substance abuse treatment need to be equal to and cannot be more restrictive or harsher than those for physical illnesses and medical/surgical problems.

Finding treatment sources is also not always easy: there may be limited options, especially in rural areas and low-income urban areas; waiting lists; poor quality of care available for indigent patients; and financial obstacles such as co-pays, deductibles, and time off from work. Over 85% of the l,669 federally designated mental health professional shortage areas are rural; often primary care physicians and law enforcement are the first-line mental health providers (Ivey, Scheffler, & Zazzali, 1998), although they do not have the specialized training of a mental health professional, who often would be better equipped to provide care. Availability, accessibility, and acceptability (the stigma attached to mental illness) are all problems in rural areas. Approximately two-thirds of those with symptoms receive no care at all (U.S. Department of Health and Human Services, 2005; Wagenfeld, Murray, Mohatt, & DeBruiynb, 1994). At the end of 2013, the U.S. Department of Agriculture announced an investment of $50 million to help improve access and treatment for mental health problems as part of the Obama administration's effort to strengthen rural communities.

Glossary

biopsychosocial model: model in which the interaction of biological, psychological, and sociocultural factors is seen as influencing the development of the individual

involuntary treatment: therapy that is mandated by the courts or other systems

voluntary treatment: therapy that a person chooses to attend in order to obtain relief from his or her symptoms

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1.6: Introduction to Classifying Mental Disorders

What you'll learn to do: describe how mental disorders are identified and classified



Why is it important to classify abnormal behavior? Classification is important because it allows scientists to identify and group patterns of abnormal behavior. For example, in the case of depression, researchers might be able to identify common factors that help explain the origins of depression. Classification systems used in abnormal psychology aim to ease understanding and communication between clinicians and researchers by standardizing the language used around disorders. These systems also allow for consistent and reliable research about the diagnosis, prognosis, and treatment of disorders. The most popular classification system in the United States is the *DSM-5*, but the *ICD-11* is another popular system used internationally.[1]

Important decisions are made on the basis of classification, such as deciding on which therapy to choose or which drug to administer. Classification is not without its criticism, however. It has the potential to dehumanize by labeling, but it does allow clinicians to better predict behavior—schizophrenia, for example, follows a more or less predictable course.

This section reviews the classification and assessment of abnormal behavior, beginning with the DSM-5.

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1.7: Diagnosing and Classifying Mental Disorders

Learning Objectives

- Describe the basic features of the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5) and how it is used to classify disorders
- Outline the major disorder categories of the DSM-5

A first step in the study of mental disorders is carefully and systematically discerning significant signs and symptoms. How do mental health professionals ascertain whether or not a person's inner states and behaviors truly represent a psychological disorder? Arriving at a proper **diagnosis**—that is, appropriately identifying and labeling a set of defined symptoms—is absolutely crucial. This process enables professionals to use a common language with others in the field and aids in communication about the disorder with the patient, colleagues, and the public. A proper diagnosis is an essential element to guide proper and successful treatment. For these reasons, classification systems that organize psychological disorders systematically are necessary.

Diagnostic and Statistical Manual of Mental Disorders (DSM-5)

Although a number of classification systems have been developed over time, the one that is used by most mental health professionals in the United States is the ***Diagnostic and Statistical Manual of Mental Disorders*** **(DSM-5)**, published by the American Psychiatric Association in 2013. Additions and revisions were made in March 2022, so the most current edition is called the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision* (DSM-5-TR). (Note that the American Psychiatric Association differs from the American Psychological Association; both are abbreviated APA.) This textbook includes the updates from the DSM-5-TR, though we typically continue to reference the diagnostic manual simply as the DSM-5.

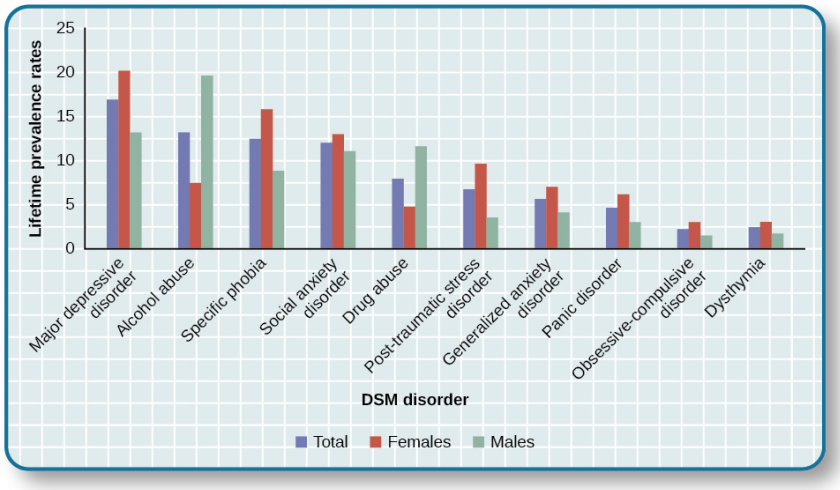


Figure **1.7.1**: The graph shows the breakdown of psychological disorders, comparing the percentage prevalence among adult males and adult females in the United States. Because the data is from 2007, the categories shown here are from the *DSM-4*, which has been supplanted by the DSM-5. Most categories remain the same; however, alcohol abuse now falls under a broader alcohol use disorder category.

The first edition of the *DSM*, published in 1952, classified psychological disorders according to a format developed by the U.S. Army during World War II (Clegg, 2012). In the years since, the DSM has undergone numerous revisions and editions. The DSM-5 includes many categories of disorders (e.g., anxiety disorders, depressive disorders, and dissociative disorders). Each disorder is described in detail, including an overview of the disorder (diagnostic features), specific symptoms required for diagnosis (diagnostic criteria), **prevalence** information (what percent of the population is thought to be afflicted with the disorder), and risk factors associated with the disorder. Figure **1.7.1** shows lifetime prevalence rates—the percentage of people in a population who develop a disorder in their lifetime—of various psychological disorders among U.S. adults. These data were based on a national sample of 9,282 U.S. residents (National Comorbidity Survey, 2007).

Table **1.7.1**. *DSM* Disorder Lifetime Prevalence Rates

DSM Disorder	Total	Females	Males
Major Depressive Disorder	17%	20%	13%
Alcohol Abuse	13%	7%	20%
Specific Phobia	13%	16%	8%
Social Anxiety Disorder	12%	13%	11%
Drug Abuse	8%	5%	12%
Post-Traumatic Stress Disorder	7%	10%	3%
Generalized Anxiety Disorder	6%	7%	4%
Panic Disorder	5%	6%	3%
Obsessive-Compulsive Disorder	3%	3%	2%
Dysthymia	3%	3%	2%

More recent data shows that the most prevalent disorders in any given time (not over a lifetime) are anxiety disorders, as shown in the following chart.[1]

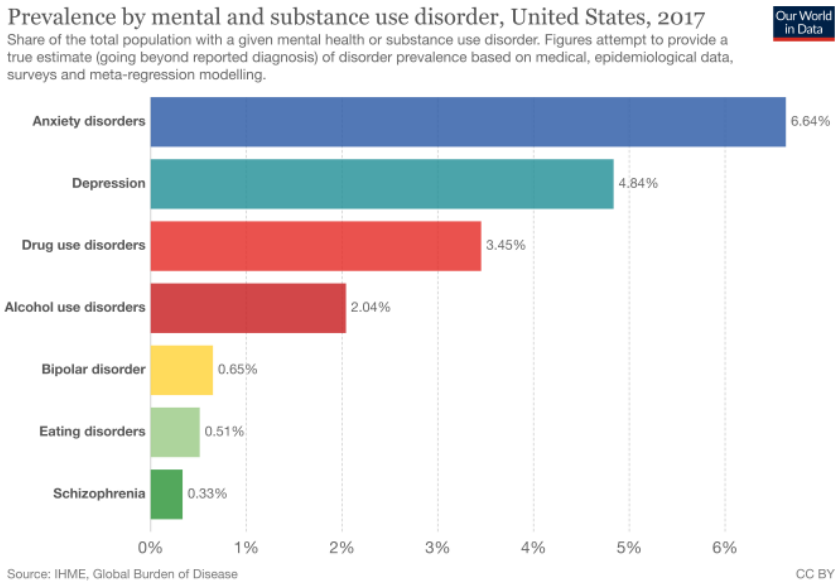


Figure **1.7.2**: The prevalence of mental and substance use disorders in the United States.

The DSM-5 also provides information about **comorbidity**; the co-occurrence of two disorders. For example, the DSM-5 mentions that 41% of people with obsessive-compulsive disorder (OCD) also meet the diagnostic criteria for major depressive disorder (Figure **1.7.2**). Drug use is highly comorbid with other mental illnesses; six out of 10 people who have a substance use disorder also suffer from another form of mental illness (National Institute on Drug Abuse [NIDA], 2007).

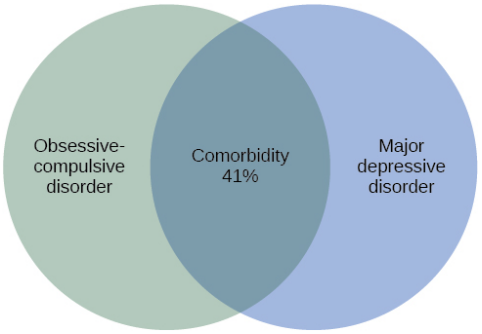


Figure **1.7.3**: Obsessive-compulsive disorder and major depressive disorder frequently occur in the same person.

? Comorbidity

Co-occurrence and comorbidity of psychological disorders are quite common, and some of the most pervasive comorbidities involve substance use disorders that co-occur with psychological disorders. Indeed, some estimates suggest that around a quarter of people who suffer from the most severe cases of mental illness exhibit substance use disorder as well. Conversely, around 10 % of individuals seeking treatment for substance use disorder have serious mental illnesses. Observations such as these have important implications for treatment options that are available. When people with a mental illness are also habitual drug users, their symptoms can be exacerbated and resistant to treatment. Furthermore, it is not always clear whether the symptoms are due to drug use, the mental illness, or a combination of the two. Therefore, it is recommended that behavior is observed in situations in which the individual has ceased using drugs and is no longer experiencing withdrawal from the drug in order to make the most accurate diagnosis (NIDA, 2018).

Obviously, substance use disorders are not the only possible comorbidities. In fact, some of the most common psychological disorders tend to co-occur. For instance, more than half of individuals who have a primary diagnosis of depressive disorder are estimated to exhibit some sort of anxiety disorder. The reverse is also true for those diagnosed with a primary diagnosis of an anxiety disorder. Further, anxiety disorders and major depression have a high rate of comorbidity with several other psychological disorders (Al-Asadi, Klein, & Meyer, 2015).

The DSM has changed considerably in the half-century since it was originally published. The first two editions of the *DSM*, for example, listed homosexuality as a disorder; however, in 1973, the APA voted to remove it from the manual (Silverstein, 2009). While the *DSM-3* did not list homosexuality as a disorder, it introduced a new diagnosis, ego-dystonic homosexuality, which emphasized homosexual arousal that the patient viewed as interfering with desired heterosexual relationships and causing distress for the individual. This new diagnosis was considered by many as a compromise to appease those who viewed homosexuality as a mental illness. Other professionals questioned how appropriate it was to have a separate diagnosis that described the content of an individual's distress. In 1986, the diagnosis was removed from the *DSM-3-R* (Herek, 2012).

✔ Watch It

This video provides an overview of some of the history related to the development and evolution of the *DSM*.



You can view the transcript for "We Were Super Wrong About Mental Illness: The DSM's Origin Story" here (opens in new window).

Additionally, beginning with the *DSM-3* in 1980, mental disorders have been described in much greater detail, and the number of diagnosable conditions has grown steadily, as has the size of the manual itself. DSM-1 included 106 diagnoses and was 130 total pages, whereas *DSM-3* included more than twice as many diagnoses (265) and was nearly seven times its size (886 total pages) (Mayes & Horowitz, 2005). Although DSM-5 is longer than *DSM-4*, the volume includes only 237 disorders, a decrease from the 297 disorders that were listed in *DSM-4*. The latest edition, DSM-5, includes revisions in the organization and naming of categories and in the diagnostic criteria for various disorders (Regier, Kuhl, & Kupfer, 2012), while emphasizing careful consideration of the importance of gender and cultural difference in the expression of various symptoms (Fisher, 2010).

Some believe that establishing new diagnoses might over pathologize the human condition by turning common human problems into mental illnesses (The Associated Press, 2013). Indeed, the finding that nearly half of all Americans will meet the criteria for a *DSM* disorder at some point in their life (Kessler et al., 2005) likely fuels much of this skepticism. The DSM-5 is also criticized on the grounds that its diagnostic criteria have been loosened, thereby threatening to "turn our current diagnostic inflation into diagnostic hyperinflation" (Frances, 2012, para. 22). For example, *DSM-4* specified that the symptoms of major depressive disorder must not be attributable to normal bereavement (loss of a loved one). The DSM-5, however, has removed this bereavement exclusion, essentially meaning that grief and sadness after a loved one's death can constitute major depressive disorder.

Categories in the DSM

The DSM-5 is divided into 22 chapters that include sets of related disorders. This organization is evident in every chapter so that related disorders appear closer to each other, and psychological and biological diseases often relate to each other. However, if an illness that is primarily medical is not specified in DSM-5, clinicians may use the current *ICD* diagnoses to specify the condition.

Link to Learning

View the DSM-5 Table of Contents here. Note that the overall outline is the same in the DSM-5-TR, though the contents and some of the language have changed slightly. For example, "dysthymia" is no longer used to describe "persistent depressive disorder," the terminology for "intellectual disability" has been replaced with "intellectual development disorder" and "conversion disorder" is better known as "functional neurological symptom disorder." A new disorder, prolonged grief disorder, was added to the section on trauma- and stressor-related disorders.

The current organization of the DSM-5 begins with neurodevelopmental disorders and then proceeds through **internalizing problems** (depression, anxiety, social anxiety, somatic complaints, post-traumatic symptoms, and obsession-compulsion) to **externalizing problems** (disruptive, impulse-control, conduct disorders and substance use, etc.). [2]

We have organized this course according to the DSM-5 and devote time in each of the modules to discuss the main features of mental disorders from each of the DSM-5 categories. Throughout these modules, you will learn the basic diagnostic criteria, the

etiology (causes), epidemiology (prevalence), and treatment options for each category of disorders. In this way, you can gain a basic understanding of each category of mental disorders, including all of the following:

1. neurodevelopmental disorders
2. schizophrenia spectrum and other psychotic disorders
3. bipolar and related disorders
4. depressive disorders
5. anxiety disorders
6. obsessive-compulsive and related disorders
7. trauma- and stressor-related disorders
8. dissociative disorders
9. somatic symptom and related disorders
10. feeding and eating disorders
11. elimination disorders
12. sleep-wake disorders
13. sexual dysfunctions
14. gender dysphoria
15. disruptive, impulse-control, and conduct disorders
16. substance-related and addictive disorders
17. neurocognitive disorders
18. personality disorders
19. paraphilic disorders
20. Other mental disorders[3]

Overview of the Major Disorder categories

In this course, the major disorders categories begin with **anxiety disorders**. Any anxiety or fear that interferes with normal functioning may be classified as an anxiety disorder. Commonly recognized categories include specific phobias: a specific unrealistic fear; social anxiety disorder: extreme fear and avoidance of social situations; panic disorder: suddenly overwhelmed by panic even though there is no apparent reason to be frightened; agoraphobia: an intense fear and avoidance of situations in which it might be difficult to escape; and generalized anxiety disorder: a relatively continuous state of tension, apprehension, and dread.

Another module deals with obsessive-compulsive and related disorders and trauma- and stressor-related disorders. While similar to anxiety disorders, obsessive-compulsive disorders and posttraumatic stress disorders now have their own distinct categories of classification within the DSM-5 because symptoms of anxiety are not necessarily present. With **obsessive-compulsive disorder** , a person is obsessed with unwanted, unpleasant thoughts and/or compulsively engages in repetitive behaviors or mental acts, perhaps as a way of coping with the obsessions. Post-traumatic stress disorder is a similar disorder, although classified as a trauma- and stressor-related disorder.

Link to Learning

Learn more about each of the psychological disorders through the National Institute of Mental Health.

Or for an interesting application of the various mental disorders, take a look at this YouTube playlist showing disorders as they are characterized in popular media. These case studies were developed by students in Dr. Caleb Lack's psychology class.

Post-traumatic stress disorder is a disorder in which the experience of a traumatic or profoundly stressful event, such as combat, sexual assault, or natural disaster, produces a constellation of symptoms that must last for one month or more. These symptoms include intrusive and distressing memories of the event, flashbacks, avoidance of stimuli or situations that are connected to the event, persistently negative emotional states, feeling detached from others, irritability, proneness toward outbursts, and a tendency to be easily startled.

In another module, we discuss dissociative disorders, somatic symptom, and related disorders. The main characteristics of **dissociative disorders** are that people become dissociated from their sense of self, resulting in memory and identity disturbances.

Dissociative disorders listed in the DSM-5 include dissociative amnesia, depersonalization/derealization disorder, and dissociative identity disorder. A person with dissociative amnesia is unable to recall important personal information, often after a stressful or traumatic experience. Depersonalization/derealization disorder is characterized by recurring episodes of depersonalization (i.e., detachment from or unfamiliarity with the self) and/or derealization (i.e., detachment from or unfamiliarity with the world). A person with dissociative identity disorder exhibits two or more well-defined and distinct personalities or identities, as well as memory gaps for the time during which another identity was present. Dissociative identity disorder has generated controversy, mainly because some believe its symptoms can be faked by patients if presenting its symptoms somehow benefits the patient in avoiding negative consequences or taking responsibility for one's actions. The diagnostic rates of this disorder have increased dramatically following its portrayal in popular culture. However, many people legitimately suffer over the course of a lifetime with this disorder.

Somatic symptom disorders were previously known as "somataform disorders." These include somatic symptom disorder, illness anxiety disorder, conversion disorder, and fictitious disorder. You will read about the various symptoms, epidemiology, how individuals present with these problems, and a brief overview of possible causes. These disorders relate to a person experiencing physical ailments that are not fully explained by a medical condition.

Mood disorders are discussed in another module. A mood disorder involving unusually intense and sustained sadness, melancholia, or despair is known as major depressive disorder. Milder but still prolonged depression can be diagnosed as dysthymia. Bipolar disorder is characterized by mood states that vacillate between sadness and euphoria; a diagnosis of bipolar disorder requires experiencing at least one manic episode, which is defined as a period of extreme euphoria, irritability, and increased activity. Mood disorders appear to have a genetic component, with genetic factors playing a more prominent role in bipolar disorder than in depression. Both biological and psychological factors are important in the development of depression. People who suffer from mental health problems, especially mood disorders, are at heightened risk for suicide.

Next, we discuss feeding and eating disorders. Disordered eating can have significant health consequences, and **eating disorders** are a major health concern. Anorexia nervosa is an eating disorder characterized by the maintenance of a bodyweight well below average through starvation and/or excessive exercise. Individuals suffering from anorexia nervosa often have a distorted body image, referenced in literature as a type of body dysmorphia, meaning that they view themselves as overweight even though they are not. People suffering from bulimia nervosa engage in binge eating behavior that is followed by an attempt to compensate for a large amount of consumed food. Avoidant/restrictive food intake disorder is an eating or feeding disturbance associated with an apparent lack of interest in eating or food. Pica is a disorder characterized by an appetite for substances that are largely non-nutritive, such as ice, soap, hair, paper, metal, soil, stones, glass, or chalk.

Many people experience disturbances in their sleep at some point in their lives. **Sleep-wake disorders** involve problems with the quality, timing, and amount of sleep, which result in daytime distress and impairment in functioning. Sleep-wake disorders often occur along with medical conditions or other mental health conditions, such as depression, anxiety, or cognitive disorders.

Substance–related disorders are discussed in a separate module. They result when a craving for, the development of, a tolerance to, and difficulties in controlling the use of a particular substance or a combination of different substances, as well as withdrawal syndromes when a person ceases to use the substance(s). Other addictive disorders also include gambling disorder and other behavioral addictions.

Next, we cover the topics of **gender dysphoria, paraphilic disorders,** and **sexual dysfunctions**. Today more than ever before, mental health professionals are seeing patients seeking treatment in response to dissatisfaction with their sexual functioning. Such dissatisfaction most commonly stems from a sexual dysfunction, but may also be the result of a sexual deviation. Sexual dysfunction disorders include sexual desire disorders, arousal disorders, orgasm disorders, and pain disorders. From a clinical perspective, there has been some effort to define sexual deviation under the umbrella of the sexual paraphilias. Dissatisfaction can also stem from gender dysphoria, or the distress a person feels due to a mismatch between their gender identity and their biological sex.

The DSM-5 classifies **psychotic disorders** that involve psychosis, or a break in reality, like in schizophrenia, delusional disorder, brief psychotic disorder, schizophreniform disorder, schizoaffective disorder, substance/medication-induced psychotic disorder, and psychotic disorder due to another medical condition. The most common psychotic disorder in this domain is schizophrenia, which is a severe disorder characterized by delusions and hallucinations, often causing a breakdown in one's ability to function in life.

The DSM-5 recognizes 10 **personality disorders**, organized into three clusters. The disorders in Cluster A include those characterized by a personality style that is odd and eccentric. These include paranoid, schizoid, and schizotypal personality disorders. Cluster B includes personality disorders characterized chiefly by a personality style that is impulsive, dramatic, highly emotional, and erratic (antisocial, histrionic, narcissistic, and borderline) and those in Cluster C are characterized by a nervous and fearful personality style (avoidant, dependent, and obsessive-compulsive).

Neurodevelopmental disorders are covered in another module, along with **elimination disorders** and disruptive, impulse-control, and conduct disorders. The neurodevelopmental disorders are a group of disorders that are typically diagnosed during childhood and are characterized by developmental deficits in personal, social, academic, and intellectual realms; these disorders include attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder. The major features of autism spectrum disorder include deficits in social interaction and communication and repetitive movements or interests. ADHD is characterized by a pervasive pattern of inattention and/or hyperactive and impulsive behavior that interferes with normal functioning. Genetic and neurobiological factors contribute to the development of ADHD, which can persist well into adulthood and is often associated with poor, long-term outcomes.

Disruptive, **impulse–control, and conduct disorders** refer to a group of disorders that include oppositional defiant disorder, conduct disorder, intermittent explosive disorder, kleptomania, and pyromania. These disorders can cause people to behave angrily or aggressively toward people or property.

Finally, we learn about **neurocognitive disorders**—disorders that describe decreased mental function due to a medical disease other than a psychiatric illness. It is often used synonymously (but incorrectly) with dementia. Although **Alzheimer's disease** accounts for the majority of cases of neurocognitive disorders, several other conditions can similarly affect memory, thinking, and reasoning, and the motor system. In addition to Alzheimer's, these conditions include frontotemporal degeneration, Huntington's disease, Lewy body disease, traumatic brain injury (TBI), Parkinson's disease, prion disease, and dementia/neurocognitive issues due to HIV infection.

Glossary

comorbidity: co-occurrence of two disorders in the same individual

diagnosis: determination of which disorder a set of symptoms represents

***Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5):** authoritative index of mental disorders and the criteria for their diagnosis; published by the American Psychiatric Association (APA)

externalizing problems: problems related with disruptive behavior that cause conflicts in relationships with others

internalizing problems: problems that involve emotional alterations of anxiety disorders and depression

***International Classification of Diseases* (*ICD*):** authoritative index of mental and physical diseases, including infectious diseases, and the criteria for their diagnosis published by the World Health Organization (WHO)

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1.8: International Classification of Diseases

Learning Objectives

- Explain how the *International Classification of Diseases* is used to classify mental disorders

International Classification of Diseases

A second classification system, the *International Classification of Diseases* (*ICD*), is also widely recognized. Published by the World Health Organization (WHO), the *ICD* was developed in Europe shortly after World War II and, like the *DSM*, has been revised several times. The *ICD* is much larger and more comprehensive than the *DSM* because it deals with all manners of healthcare and is not specific only to mental health. The categories of psychological disorders in both the *DSM* and *ICD* are similar, as are the criteria for specific disorders; however, some differences exist. Although the *ICD* is used for clinical purposes, this tool is also used to examine the general health of populations and to monitor the prevalence of diseases and other health problems internationally (WHO, 2013). The ***ICD-11,*** in development for many years but officially adopted in January 2022, is the eleventh revision and replaced the *ICD-10* as the global standard for coding health information and causes of death. The *ICD-11* has not yet been officially implemented in the United States, so until at least 2025, clinicians still use the *ICD-10-CM*.



Figure 1.8.1: The *ICD* is used globally while the *DSM* is rarely used outside of the United States.

Overview

The following mental disorders have been newly added to the *ICD-11*, but were already included in the American *ICD-10-CM* adaption: binge eating disorder, Bipolar II disorder, body dysmorphic disorder, excoriation disorder, frotteuristic disorder, hoarding disorder, and intermittent explosive disorder.

The following mental disorders have been newly added to the *ICD-11*, and are not in the *ICD-10-CM*: avoidant/restrictive food intake disorder, body integrity dysphoria, catatonia, complex post-traumatic stress disorder, gaming disorder (generally defined as the problematic, compulsive use of video games that results in significant impairment to an individual's ability to function in various life domains over a prolonged period of time), olfactory reference disorder (a psychiatric condition in which there is a persistent false belief and preoccupation with the idea of emitting abnormal body odors which the patient thinks are foul and offensive to other individuals), and prolonged grief disorder.

Other notable changes include the following:

- Distinct personality disorders have been collapsed into a single personality disorder diagnosis, using a dimensional (as opposed to categorical) model. Personality disorders are classified as mild, moderate, or severe.
- All subtypes of schizophrenia (e.g., paranoid, hebephrenic, catatonic) have been removed. Instead, a dimensional model is used with the category symptomatic manifestations of primary psychotic disorders, which allows the coding for positive symptoms, negative symptoms, depressive symptoms, manic symptoms, psychomotor symptoms, and cognitive symptoms.
- Persistent mood disorders, including cyclothymia and dysthymia, have been deleted.
- The *ICD-10* differentiated between phobic anxiety disorders, such as agoraphobia, and other anxiety disorders, such as generalized anxiety disorder. The *ICD-11* merges both groups together as anxiety or fear-related disorders.
- All pervasive developmental disorders are merged into one category, autism spectrum disorder, except for Rett syndrome, which is moved to the developmental anomalies chapter.
- Hyperkinetic disorder is renamed attention deficit hyperactivity disorder, and a distinction in subtypes is made between predominantly inattentive, predominantly hyperactive-impulsive, and combined. Hyperkinetic conduct disorder has been removed.

- Acute stress reaction has been moved out of the mental disorder chapter, and placed in the chapter "Factors influencing health status or contact with health services." Thus, in the *ICD-11*, acute stress reaction is no longer considered a mental disorder.

The *ICD-11* features five new chapters. Those most relevant to mental disorders are the new chapters on "Sleep-Wake Disorders" and "Conditions Related to Sexual Health."

ICD chapters

Below is a list of all chapters of the *ICD-11*. You can **view the *ICD-11* here** or learn more about it in this ***ICD-11* reference guide**.

1. Certain Infectious or Parasitic Diseases
2. Neoplasms
3. Diseases of the Blood or Blood-Forming Organs
4. Diseases of the Immune System
5. Endocrine, Nutritional or Metabolic Diseases
6. Mental, Behavioral, or Neurodevelopmental Disorders
7. Sleep-Wake Disorders
8. Diseases of the Nervous System
9. Diseases of the Visual System
10. Diseases of the Ear or Mastoid Process
11. Diseases of the Circulatory System
12. Diseases of the Respiratory System
13. Diseases of the Digestive System
14. Diseases of the Skin
15. Diseases of the Musculoskeletal System or Connective Tissue
16. Diseases of the Genitourinary System
17. Conditions Related to Sexual Health
18. Pregnancy, Childbirth, or the Puerperium
19. Certain Conditions Originating in the Perinatal Period
20. Developmental Anomalies
21. Symptoms, Signs, or Clinical Findings Not Elsewhere Classified
22. Injury, Poisoning or Certain Other Consequences of External Causes
23. External Causes of Morbidity or Mortality
24. Factors Influencing Health Status or Contact with Health Services
25. Codes for Special Purposes
26. Supplementary Chapter Traditional Medicine Conditions—Module I
27. Supplementary Section for Functioning Assessment
28. Extension Codes

Case Study

A study that compared the use of the two classification systems found that worldwide the *ICD* is more frequently used for clinical diagnosis, whereas the *DSM* is more valued for research (Mezzich, 2002). Most research findings concerning the etiology and treatment of psychological disorders are based on criteria set forth in the *DSM* (Oltmanns & Castonguay, 2013). The *DSM* also includes more explicit disorder criteria, along with an extensive and helpful explanatory text (Regier et al., 2012). The *DSM* is the classification system of choice among U.S. mental health professionals, and the modules in this course are based on the *DSM* paradigm.

Compassionate View of Psychological Disorders



Figure **1.8.2**: It's important to be considerate of those dealing with psychological disorders.

As these disorders are outlined, please bear two things in mind. First, remember that psychological disorders represent *extremes* of inner experience and behavior. If, while reading about these disorders, you feel that these descriptions begin to personally characterize you, do not worry—this moment of enlightenment probably means nothing more than you are normal. Each of us experiences episodes of sadness, anxiety, and preoccupation with certain thoughts—times when we do not quite feel ourselves. These episodes should not be considered problematic unless the accompanying thoughts and behaviors become extreme and have a disruptive effect on one's life.

Second, understand that people with psychological disorders are far more than just embodiments of their disorders. We do not use terms such as schizophrenics, depressives, or phobics because they are labels that objectify people who suffer from these conditions, thus promoting biased and disparaging assumptions about them. It is important to remember that a psychological disorder is not what a person *is*; it is something that a person *has*—through no fault of his or her own. As is the case with cancer or diabetes, those with psychological disorders suffer debilitating, often painful conditions that are not of their own choosing. These individuals deserve to be viewed and treated with compassion, understanding, and dignity.

Watch It

Watch this *CrashCourse Psychology* video to better understand the history of diagnosing psychological disorders and how they are classified.



You can view the transcript for "Psychological Disorders: Crash Course Psychology #28" here (opens in new window).

Glossary

International Classification of Diseases (*ICD*): authoritative index of mental and physical diseases, including infectious diseases, and the criteria for their diagnosis; published by the World Health Organization (WHO)

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1.9: Classification and Labeling

Learning Objectives

- Discuss arguments and objections surrounding the *DSM* classification system
- Describe problems associated with classification and labeling

Advantages and Disadvantages of the DSM-5 Classification System

With any system of classification like *DSM-5*, there will always be strengths and weaknesses. One of the major strengths of the *DSM* system is the wide acceptance and use of the system. Mental health professionals in the United States routinely utilize diagnostic systems in their work if for no other reason than to allow their clients to receive treatment in hospitals and reimbursement from health care providers. Each category of disorder has a numeric code taken from the *ICD* coding system, used for health service (including insurance) administrative purposes.

The limitations of the *DSM* system are reflected in the terminology related to diagnosis itself. Since the DSM-3, the goal was to improve the uniformity and validity of psychiatric diagnosis in the wake of a number of critiques, including the famous **Rosenhan experiment** (see box below). There was also felt a need to standardize diagnostic practices within the United States and with other countries, after research showed that psychiatric diagnoses differed between Europe and the United States.[1]

A closer look: The Rosenhan Experiment

The Rosenhan experiment was carried out by David Rosenhan, a Stanford University professor, and published by the journal *Science* in 1973 under the title "On Being Sane in Insane Places." It was an experiment conducted to determine the reliability and validity of psychiatric diagnosis. The experimenters feigned hallucinations to enter psychiatric hospitals, and acted normally afterwards. They were diagnosed with psychiatric disorders and were given antipsychotic drugs.

The study was c onsidered an important and influential criticism of psychiatric diagnosis. It has been argued that the experiment was fabricated; nonetheless, the study concluded, "it is clear that we cannot distinguish the sane from the insane in psychiatric hospitals," and it also illustrated the dangers of dehumanization and labeling in psychiatric institutions. It suggested that the use of community mental health facilities that concentrated on specific problems and behaviors rather than psychiatric labels might be a solution, and recommended education to make psychiatric workers more aware of the social psychology of their facilities.

Here is a seven-minute video summing up the study.

Reliability and Validity

The revisions and refinements in the *DSM* classification system have been largely driven by the need to improve reliability and validity. **Reliability** measures how consistent a diagnosis is and how reliably the categories can be judged. **Validity** looks at how accurate the diagnosis is and how valid the categories are in the sense of discriminating among disorders that have distinctive etiologies and possibly require different treatments.

Reliability Vs. Validity

To be useful, any diagnostic system must demonstrate **reliability** and **validity**. The revisions of the *DSM* from the third edition forward have been mainly concerned with **diagnostic reliability**—the degree to which different diagnosticians agree on a diagnosis. If clinicians and researchers frequently disagree about the diagnosis of a patient, then research into the causes and effective treatments of those disorders cannot advance. To be considered reliable, or consistent, different evaluators using the system should arrive at the same diagnoses when they evaluate the same people. For example, a diagnosis of major depressive disorder, a common mental illness, had a poor reliability kappa statistic of 0.28, indicating that clinicians frequently disagreed on diagnosing this disorder in the same patients. The most reliable diagnosis was major neurocognitive disorder, with a kappa of 0.78.[2]

Critics assert, for example, that many *DSM-5* revisions or additions lack empirical support; **inter-raterreliability,** or the degree of agreement among raters, is low for many disorders; several sections contain poorly written, confusing, or contradictory information; and the psychiatric drug industry unduly influenced the manual's content (many *DSM-5* workgroup participants had ties to pharmaceutical companies).footnote]Welch, Steven; Klassen, Cherisse; Borisova, Oxana; Clothier, Holly (2013). "The *DSM-5* controversies: How should psychologists respond?". Canadian Psychology. 54(3): 166–175. doi:10.1037/a0033841[/footnote]

Diagnostic validity concerns whether the diagnosis measures what it claims to measure. In psychiatry, there is a particular issue with assessing the validity of the diagnostic categories themselves. In this context,

- **content validity** may refer to symptoms and diagnostic criteria.
- **concurrent validity** looks at whether the markers that indicate a disorder actually correlate with the disorder, and if these markers are true over time and across multiple people and measures. It may also look at the appropriate alignment of a treatment response to a disorder.
- **predictive validity** may refer mainly to diagnostic stability over time and the relationship between the diagnosis and the ability to predict people's behavior in certain situations.
- **discriminant validity** may involve delimitation from other disorders.

Robins and Guze proposed in 1970 what were to become influential formal criteria for establishing the validity of psychiatric diagnoses. The five phases of diagnostic validation used by Robins and Guze were (1) clinical characteristics of the syndrome and of the patients who develop it (including core symptoms, demographic characteristics, and precipitating factors); (2) exclusionary criteria differentiating the syndrome from other known disorders; (3) family studies; (4) laboratory data (radiological, chemical, pathologic, and psychological evidence); and (5) follow-up studies (for diagnostic stability, course, and treatment response).[3] Once the characteristics of disorder, the exclusionary criteria, family, lab, and follow-up studies are completed, there should be enough information to deem the *DSM* content as a valid description of the mental disorder.



Figure **1.9.1**: Radiology laboratory studies like MRIs are included in the criteria for validity.

These were incorporated into the Feighner Criteria and Research Diagnostic Criteria that have since formed the basis of the *DSM* and *ICD* classification systems. [4]

Other critics also believe the *DSM* needs to become more sensitive to the importance of cultural and ethnic factors in diagnostic assessment. They encourage us to consider and understand that the symptoms or problem behaviors included as diagnostic criteria in the *DSM* were largely determined by a consensus of mostly U.S.-trained psychiatrists, psychologists, and social workers. Imagine that the American Psychiatric Association had asked Asian-trained or Latin American–trained professionals to develop their diagnostic manual? Might there might have been different diagnostic criteria or even different diagnostic categories? [5]

Overall, the bulk of limitations of the *DSM* system listed so far are related to the diagnostic system in general. It is worthwhile to be alert to the criticisms of the *DSM* system, particularly since they serve as a reminder that mental health professionals aim to help the individual and not the disorder. Furthermore, the *DSM* system has been useful to insurance companies who adopt its use to establish coverage for certain clinical disorders, and has been helpful in allowing researchers and clinicians to have a common language with which to discuss clients.

Differences between Categorical and Dimensional Understanding of Behavior

Both the *DSM* and *ICD* systems represent a **categorical approach** to the description of mental disorders. As the name implies, a categorical approach attempts to categorize mental disorders into distinct diagnoses. The categorical approach is based on the idea that a person either meets criteria for a specific disorder or they do not. Traditional categorical models of classification, which are based on the presence or absence of symptoms, do not take into account levels of expression of a characteristic or the presence of

any underlying **dimension**. This conceptualization allows a mental health professional to make a dichotomous decision (i.e., yes, the person meets criteria or no, the person does not meet criteria for that disorder). A *dimension* refers to a continuum on which an individual can have various levels of a characteristic, in contrast to the dichotomous categorical approach in which an individual does or does not possess a characteristic.

The *DSM-4* organized diagnoses using five separate axes (clinical disorders, personality disorders, general medical disorders, and then sections on psychosocial and environmental factors, and the global assessment of functioning (which measured the severity of a disorder). An **axis** is defined as a category of information regarding one dimension of an individual's functioning. In the *DSM-IV-TR* system, an individual was diagnosed on five different domains, or "axes." The *DSM-5* got rid of the axis system, as there was no scientific justification for grouping things by axis. Originally, the **multiaxial system** was intended to allow professionals to characterize clients in a multidimensional way (medical, psychological, developmental, and environmental/social status), but rationale for removal of the multi-axial system was based on unclear boundaries between medical and psychiatric diagnoses, inconsistent use of Axis IV by clinicians and researchers (psychosocial and environmental problems), and poor psychometric and clinical validity of Axis V (Global Assessment of Functioning).[6]

The most important change in *DSM-5* was the inclusion of dimensions in diagnoses; for example, how severely ill is a patient with schizophrenia or depression? The dimensional approach focuses on varying levels of different behaviors that a person exhibits, rather than whether or not a person meets criteria for a particular disorder. The **dimensional approach** is also included in Section III ("Emerging Measures and Models"). This section includes assessment measures and diagnoses not considered well-established enough to be part of the main system. For example, an "Alternative DSM-5 Model for Personality Disorders" is described.[7] The decision to retain the old *DSM-4* categorical model for personality disorders in *DSM-5* was controversial (currently the ten personality disorders are grouped into three general categories), and efforts continue to persuade the American Psychiatric Association to replace it with the dimensional model in *DSM 5.1*. [8]

Since the categorical model is widely used in clinical practice and has a significant body of research supporting it, its common usage is compelling to laypeople when they are judging the credibility of professional opinion. Therefore, the dimensional approach is often further criticized for being difficult to interpret and less accessible. It is, however, widely used in some professional settings as the established approach, for example by forensic psychologists. [9]

Regardless of whether you use a categorical or dimensional approach to understand a person's functioning, it is crucial to remember that behavior can change over time.

Problems Associated with Classification and Labeling

Finally, but probably most importantly, any diagnostic system such as the DSM system allows for individuals to be labeled for behavior that may or may not be an important part of their character. **Labeling** occurs when information about a person's diagnostic classification is communicated in a negative manner that leads to stigma for the individual with a mental disorder.

The "Mentally Ill"

The social construction of deviant behavior plays an important role in the labeling process that occurs in society. This process involves not only the labeling of criminally deviant behavior, which is behavior that does not fit socially constructed norms, but also labeling that which reflects stereotyped or stigmatized behavior of the "mentally ill." The **labeling theory** was first applied to the term "mentally ill" in 1966 when Thomas J. Scheff published *Being Mentally Ill*. Scheff challenged common perceptions of mental disorders by claiming that mental disorder is manifested solely as a result of societal influence. Scheff argued that society views certain behaviors and actions as deviant and, in order to come to terms with and understand these actions, often places the label of mental disorder on those who exhibit them. Certain expectations are then placed on these individuals and, over time, they unconsciously change their behavior to fulfill them. Criteria for different mental illnesses are not consistently fulfilled by those who are diagnosed with them because all of these people suffer from the same disorder, they are simply fulfilled because the *"mentally ill"* believe they are supposed to act a certain way so, over time, come to do so.[10]

Scheff's theory had many critics, most notably Walter Gove who consistently argued against Scheff with an almost opposite theory; he believed that society has no influence at all on mental disorder. Instead, any societal perceptions of the *"mentally ill"* come about as a direct result of these people's behaviors. Most sociologists' views of labeling and mental disorder have fallen somewhere between the extremes of Gove and Scheff. On the other hand, it is almost impossible to deny, given both common sense and

research findings, that society's negative perceptions of "crazy" people has had some effect on them. It seems that, realistically, labeling can accentuate and prolong the issues termed "mental illness", but it is rarely the full cause.[11]

Labeling theory posits that self-identity and the behavior of individuals may be determined or influenced by the terms used to describe or classify them. It is associated with the concepts of **self-fulfilling prophecy** and **stereotyping**. However, the label of "mentally ill" may help a person seek help, for example, psychotherapy or medication. Labels, while they can be stigmatizing, can also lead those who bear them down the road to proper treatment and (hopefully) recovery. If one believes that "being mentally ill" is more than just believing one should fulfill a set of diagnostic criteria, then one would probably also agree that there are some who are labeled "mentally ill" who need help. It has been claimed that this could not happen if there were no way to categorize (and therefore label) them, although there are actually plenty of approaches to these phenomena that don't use categorical classifications and diagnostic terms, such as spectrum or continuum models. Here, people vary along different dimensions, and everyone falls at different points on each dimension.

The issue at stake is that DSM-5 may lead to the increasingly widespread "medicalization" of psychology. It is suggested that – also due to its impact via the social media – DSM-5 is likely to turn into a true "social representation" (Moscovici et al., 2001) with the power to strongly influence clinical practice, pushing it in the direction of the large-scale prescription of drugs.[12] Allen Frances, Chair of the DSM-IV Task Force, came to be a remarkably prolific and vocal critic of the proposed changes that came out in the DSM-5. In a BMJ editorial, he described the 'grave' consequences of 'false positive epidemics' of disorders that would be constituted through inappropriate usage of new diagnostic entities; in so doing, DSM-5 would 'expand the territory of mental disorder and thin the ranks of the normal'. In other words, it would help to further 'medicalize' society.[13]

Glossary

axis: a category of information regarding one dimension of an individual's functioning

categorical approach: attempts to categorize mental disorders into distinct diagnoses

diagnostic reliability: the degree to which different diagnosticians agree on a diagnosis; consistency and reproducibility of a given result

diagnostic validity: concerns whether the diagnosis measures what it claims to measure

dimension: refers to a continuum on which an individual can have various levels of a characteristic

inter-raterreliability: the degree of agreement among raters

labeling: occurs when information about a person's diagnostic classification is communicated in a negative manner that leads to stigma for the individual with a mental disorder.

multiaxial system: was intended to allow professionals to characterize clients in a multidimensional way (medical, psychological, developmental, and environmental/social status)

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1.10: Mental Disorders and Stigma

Learning Objectives

- Describe types of stigma associated with mental disorders and ways to challenge these stereotypes



Figure **1.10.1**: Though awareness of mental health issues continues to increase, there is still a stigma associated with many mental disorders that should be addressed, both at an individual and societal level.

Public-opinion studies have confirmed the widespread existence of negative stereotypes about individuals with mental health problems: they are considered dangerous, unpredictable, and difficult to talk to. These negative stereotypes can have detrimental consequences on the users of mental health services through processes of **public stigma**, **self-stigma**, and **stigma expectations** with regard to such outcomes as self-efficacy, depressive symptoms, self-esteem, life satisfaction, client satisfaction, work, income, and social relationships, in addition to treatment continuation and medication adherence. A **stereotype,** in general, refers to an expectation that people might have about every person of a particular group. A stereotype is distinct from stigma. **Public stigma** refers to a set of negative attitudes and beliefs that motivate individuals to fear, reject, avoid, and discriminate against people with mental illness (Corrigan and Penn 1999).[1]**Self-stigma** is thought to be particularly damaging, and is said to occur when individuals internalize stigmatizing social attitudes and come to believe the negative societal conceptions and stereotypes associated with their condition (Corrigan and Watson, 2002; Watson et al., 2007; Corrigan and Rao, 2012). Whether stigma is only anticipated or directly experienced, self-stigmatizing beliefs can lead to withdrawal from social support, rejection of help, avoidance of treatment, treatment withdrawal, and limited prospects for recovery (Link and Cullen, 1990; Link, 2001; Corrigan, 2004; Vogel et al., 2007).[2]

With regard to culture and mental health stigma, **stigma expectations**—"the extent to which individuals believe that 'most people' (the community at large) will devalue and discriminate against a mental patient"—do not differ between individuals with and without mental health problems or history of treatment. They interpret this as an indication that such expectations constitute a component of culture. In addition, stigma expectations lead individuals with mental health problems to use adaptive strategies (e.g., secrecy or withdrawal). They thus do not necessarily believe in these negative stereotypes; they only know (and fear) that the stereotypes will be applied to them once they have been "labeled."

Applying this logic, cultural stereotypes might hinder the utilization of mental health services due to fear of discrimination and devaluation. In this vein, a fear of disclosure can be viewed as the core reason why users of mental health services keep their psychiatric status secret. The preference for nondisclosure; the anticipation of social judgement, rejection, and discrimination; and the anticipation of disrespect from professionals are but a few of the many reasons why individuals refrain from seeking help, even if they do not personally adhere to such stigma beliefs. Stigma thus constitutes a major barrier to the utilization of mental health services. [3]

Stigma of Mental Illness

The word **stigma** referred originally to a mark or brand on Greek slaves, clearly separating them from free men. It is a label that causes us to regard certain people as different, defective, and set apart from mainstream members of society.

In his 1963 book,[4], Goffman defines stigma as "a trait which is deeply discrediting." He clearly says that stigmatization is an interactive social process, but some interpret his work as indicating that the blame lies with the person carrying the stigma. More

recent authors have used the term stigma in a wider sense—for example, to refer to the reaction of other people, or even to include the attitude and behavior of both the victim and the perpetrator.

It is important for researchers to define which concept of stigma they are working with. For some, stigma takes on the two forms as described below:

- **Enacted stigma** — (external stigma, discrimination) refers to the experience of unfair treatment by others.
- **Felt stigma** — (internal stigma or self-stigmatization) refers to the shame and expectation of discrimination that prevents people from talking about their experiences and stops them from seeking help.

Felt stigma can be as damaging as enacted stigma since it leads to withdrawal and restriction of social support. Many psychiatric disorders can be hidden once the person has recovered. This allows people to pass as normal so long as no one finds out. In his classic treatise, Goffman (1963)[5] delineates between people who are *discredited*—whose stigma is clearly known or visible—and people who are *discreditable*—whose stigma is unknown and can be concealable. Goffman describes people with a **discreditable condition**—e.g., bipolar disorder in remission—as having to decide how much to tell and to whom. By careful information management, they can minimize social rejection and enacted stigma. Individuals who live with a **discrediting condition**—such as schizophrenia with tardive dyskinesia—will have difficulties with information management and are at higher risk of experiencing enacted stigma. [6][7]

> *Does the stigmatized individual assume his differentness is known about already or is evident on the spot, or does he assume it is neither known about by those present nor immediately perceivable by them? In the first case one deals with the plight of the discredited, in the second with that of the discreditable. This is an important difference.*
>
> — Goffman (1963, p. 4)[8]

Watch It

This short clip highlights the importance of removing the stigma surrounding mental health conditions.



You can view the transcript for "#StigmaFree: Mayim Bialik" here (opens in new window).

#Stigma Free

Words can hurt. Many derogatory words and phrases are used in relation to mental illness. However, these words maintain the stereotyped image and not the reality about mental illness. "Mentally ill people are nuts, crazy, wacko." How often have we heard comments like these or seen these types of portrayals in movies, television shows, or books? We may even be guilty of making comments like them ourselves. Is there any truth behind these portrayals, or is that negative view based on our ignorance and fear? Try not to use these words, and encourage others not to use them. It is more appropriate to refer to "a person who has a mental illness" when speaking about someone. Each of those preconceptions about people who have a mental illness is based on false information. Very few people who have a mental illness are dangerous to society. Most can hold jobs, attend school, and live independently. A person who has a mental illness cannot simply decide to get over it any more than someone who has a different

chronic disease such as diabetes, asthma, or heart disease can. A mental illness, like those other diseases, is caused by a physical problem in the body.

In fact, not all brain diseases are categorized as mental illnesses. Disorders such as epilepsy, Parkinson's disease, and multiple sclerosis are brain disorders, but they are considered neurological diseases rather than mental illnesses. Interestingly, the lines between mental illnesses and these other brain or neurological disorders is somewhat blurring. As scientists continue to investigate the brains of people who have mental illnesses, they are learning that mental illness is associated with changes in the brain's structure, chemistry, and function, and that mental illness does indeed have a biological basis.[9]

Stigma and Illness

Stigma has been defined as an attribute that is deeply discrediting. This stigmatized trait sets the bearer apart from the rest of society, bringing with it feelings of shame and isolation. Often, when a person with a stigmatized trait is unable to perform an action because of the condition, other people view the person as the problem rather than viewing the condition as the problem. More recent definitions of stigma focus on the results of stigma—the prejudice, avoidance, rejection, and discrimination directed at people believed to have an illness, disorder, or other trait perceived to be undesirable. Stigma causes needless suffering, potentially causing a person to deny symptoms, delay treatment and refrain from daily activities. As mentioned earlier, Stigma can exclude people from access to housing, employment, insurance, and appropriate medical care. Thus, stigma can interfere with prevention efforts, and examining and combating stigma is a public health priority.

The Substance Abuse and Mental Health Services Administration (SAMHSA) and the CDC have examined public attitudes toward mental illness in two surveys. In the 2006 HealthStyles survey, only one-quarter of young adults between the ages of 18 and 24 believed that a person with mental illness can eventually recover (HealthStyles survey). In 2007, adults in 37 states and territories were surveyed about their attitudes toward mental illness, using the 2007 Behavioral Risk Factor Surveillance System Mental Illness and Stigma module. This study found that

- 78% of adults with mental health symptoms and 89% of adults without such symptoms agreed that treatment can help persons with mental illness lead normal lives.
- 57% of adults without mental health symptoms believed that people are caring and sympathetic to persons with mental illness.
- Only 25% of adults with mental health symptoms believed that people are caring and sympathetic to persons with mental illness.

These findings highlight both the need to educate the public about how to support persons with mental illness and the need to reduce barriers for those seeking or receiving treatment for mental illness.

Challenging Stereotypes about Mental Illness

Recovery from mental illness is a complex process. As with all serious illness, the well-being of recovering individuals is affected by the attitudes that surround them. Despite increasing sensitivity about most disabilities, mental illness all too often remains a target for ridicule and misrepresentation in advertising, entertainment, and the mainstream media.

Most of what we know as individuals comes not from personal experience, but from the stories that surround us from birth. In the past, it was families, religious institutions, schools, and respected members of the community who instilled cultural attitudes. But "Today, this is done by the mass media," says George Gerbner, founder of the Cultural Environment Movement, and a researcher whose career includes 30 years of monitoring the cultural impact of television on society. Television is, in Gerbner's words, "the wholesale distributor of the stigma of mental illness." Gerbner's research has shown that characters portrayed on television as having mental illnesses have four times the violence rate and six times the victimization rate of other characters. Gerbner notes that "Violence and retribution are shown as inherent in the illness itself and thus inescapable. No other group in the dramatic world of television suffers and is shown to deserve such a dire fate."

The portrayal of mental illness in the movies is similarly distorted. In the late 1980s, Steven E. Hyler of Columbia University and his colleagues identified six categories of psychiatric characters in films: homicidal maniac, narcissistic parasite, seductress, enlightened member of society, rebellious free spirit, and zoo specimen. Hyler concluded that these predominantly negative stereotypes had a damaging effect on the viewing public and on the patients themselves, their family members, and policy makers. More recently, Otto F. Wahl of George Mason University, an authority on public images of mental illness, found that in the decade

from 1985 to 1995, Hollywood released more than 150 films with characters who have mental illnesses, the majority of them killers and villains. There can be no doubt that Hollywood stereotypes are a large part of what people know, or think they know, about people with psychiatric vulnerabilities. Newspaper reports about mental illness are often more accurate than the characters one sees in TV entertainment and movies. Still, people with psychiatric histories generally are reported negatively. In 1991, researchers Russell E. Shain and Julie Phillips found that 86% of all print stories dealing with former mental patients focused on violent crime. Media stereotypes of persons with mental illness as villains, failures, buffoons—together with the misuse of terms like "schizophrenia" and "psychotic" in negative contexts—have far-reaching consequences. On the most deeply personal level, biased stereotypes damage the sense of self-worth of millions of persons diagnosed with serious psychiatric illnesses. On the social and economic levels, negative stereotyping may result in large-scale discrimination against an entire class of people in the areas of housing, employment, health insurance, and medical treatment.

Increasingly, the media are doing better work; at times, their efforts are excellent. Diana Ross's moving and realistic portrayal of schizophrenia in *Out of Darkness*, an ABC television drama, was praised by mental health activists. "Good" characters with mental illness are appearing from time to time in prime-time television entertainment. Feature stories about the achievements of individuals diagnosed with mental illness—such as a New York Times business section feature about John Forbes Nash, Jr., the winner of a Nobel Prize for economics—also help shatter stereotypes. In 1959, Nash began showing clear signs of mental illness, and spent several years at psychiatric hospitals being treated for paranoid schizophrenia. After 1970, his condition slowly improved, allowing him to return to academic work by the mid-1980s. His struggles with his illness and his recovery became the basis for Sylvia Nasar's biography, *A Beautiful Mind*, as well as a film of the same name starring Russell Crowe as Nash. And increasingly, people with first-hand experience of mental illness are writing books, appearing on television news and talk shows, producing documentaries and radio programs, and contributing articles to the print media. The disparity between mental illness as it is perceived by much of the public and mental illness as it is lived and experienced is a gulf to be bridged.

Link to Learning

There are countless examples of mental disorders in movies and TV shows, but only sometimes do they get it right. This video clip from Mojo highlights some of the more accurate portrayals of mental disorders in media.

Glossary

discreditable condition: a condition that is unknown and can be concealable

discrediting condition: a condition that is clearly known or visible

enacted stigma: refers to the experience of unfair treatment by others

felt stigma: refers to the shame and expectation of discrimination that prevents people from talking about their experiences and stops them seeking help

public stigma: refers to a set of negative attitudes and beliefs that motivate individuals to fear, reject, avoid, and discriminate against people with mental illness

self-stigma: occurs when individuals internalize stigmatizing social attitudes and come to believe the negative societal conceptions and stereotypes associated with their condition

stereotype: an expectation that people might have about every person of a particular group

stigma: a label that causes us to regard certain people as different, defective, and set apart from mainstream members of society

stigma expectations: the extent to which individuals believe that "most people" (the community at large) will devalue and discriminate against someone with a mental disorder

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1.11: Introduction to Evaluating and Assessing Mental Disorders

What you'll learn to do: describe and discuss various psychological assessment techniques



In this section, we discuss the "clinical method," or an approach to understanding and treating mental disorders that begins with a particular individual's personal history and is designed around that individual's psychological needs. Taking a personal history along with a diagnostic interview allow the mental health professional to fully establish a clinical diagnosis. You will see, as we discuss cases in future modules, that a comprehensive assessment can prove valuable in helping to understand the nature of an individual's symptoms and potential directions for treatment.

Psychological assessment usually involves a comprehensive assessment of the individual. Psychological assessment can be seen as a formal evaluation of an individual's cognitive, personality, and psychosocial functioning. The process typically involves checking the integration of information from multiple sources, such as tests of normal and abnormal personality, tests of ability or intelligence, and tests of interests or attitudes as well as information from personal interviews. In this section, we'll learn more about diagnostic tools and assessment tools used to gather information about an individual.

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1.12: The Diagnostic Process

Learning Objectives

- Explain the steps and people involved in the diagnostic process, including the case formulation

The word **diagnosis** commonly refers to the identification of the nature and cause of an illness. When a mental health professional diagnoses a client or patient, the first steps can be summarized as gathering information, narrowing down the options, and formulating a diagnostic impression.

The diagnostic process involves gathering relevant information from a person through a detailed interview that includes finding out the person's main concerns, their symptoms, and their life history. This information includes the results from psychological tests or questionnaires and may include information obtained from the person's family and/or from previous treatment records. Mental health professionals use this first phase of working with clients before proceeding with the treatment itself. More specifically, before a diagnosis is given, they determine whether the person's symptoms match the *DSM*'s criteria for a particular mental disorder, and if there is a significant level of disturbance in the person's cognitive, emotional, or behavioral functioning.

Clients, Consumers, and Patients

Why all the different terms? **Patient** and **client** are terms that carry different connotations, often depending on the method of treatment, or treatment site. For many professionals, a *patient* is any recipient of health care services performed by healthcare professionals, and *client* reflects that people in treatment collaborate with those who treat them, notably, the **clinician**—a professional who works directly with patients or clients and may diagnose, treat, and otherwise care for them. A term adopted by many users of psychiatric services is *consumer*." This term was chosen to eliminate the "patient" label and restore the person to an active role as a user or consumer of services.[1] The terms *clients* and *consumers* imply a much more active role on the part of the individual and therefore imply that healthcare professionals are not the only ones who have expertise in healthy functioning.

Diagnostic Procedures

The main goal of the information-gathering model is to accurately diagnose, plan treatments, and evaluate treatment effectiveness. The key to diagnosis is gaining as clear a description as possible of a client's or patient's symptoms. Mental health professionals listen to what the client or patient reports, and they observe quite a number of things, such as behavior and emotional expression. Routine diagnostic practice in mental health services typically involves an interview known as a **mental status examination**, where evaluations are made of appearance and behavior, self-reported symptoms, mental health history, and current life circumstances.

We will discuss a variety of assessment tools used by mental health professionals to determine the extent to which these symptoms correspond with the diagnostic criteria of a given disorder. This approach to diagnosis is more systematic, and allows one to determine the exact nature of a client's symptoms, the length of time the client has experienced these symptoms, and any associated symptoms.

Elements of a Diagnosis

By listening to clients as they describe the experience of their symptoms, and following up with assessment tools (not always, but often), the mental health professional begins to formulate the **principal diagnosis**—or the reason for the visit. This means determining the single diagnosis that is most relevant to the person's chief complaint or need for treatment; this diagnosis will be the main focus of clinical attention or treatment.

After determining the primary reason the individual is seeking professional help, a **differential diagnosis** is commonly used. Two different diagnoses can be attached to a patient who is exhibiting symptoms that could fit into either diagnosis. For example, a patient who has been diagnosed with bipolar disorder may also be given a differential diagnosis of borderline personality disorder, given the similarity in the symptoms of both conditions. Where multiple alternatives are possible, this method is akin to the process of elimination or ruling out of alternative diagnoses, a crucial step in the diagnostic process.

Many patients have additional diagnoses. **Comorbidity** refers to the presence of more than one diagnosis occurring in an individual at the same time. In the context of mental health, comorbidity often refers to disorders that are often coexistent with each other, such as substance use disorder and depression (the most common comorbidities involve the concurrence of substance use disorders with other mental disorders). Multiple diagnoses are usually presented in a hierarchy descending from the condition of most significance to that of the least concern.

The Case Formulation

Once the mental health professional makes a formal diagnosis, he or she then analyzes the factors that may have influenced the patient or client's current psychological state. A **clinical formulation**, also known as case formulation or case conceptualization, is that analysis, or a theoretically based explanation of the information obtained from a clinical assessment. It offers a hypothesis about the cause and nature of the presenting problems (e.g., background history, presenting concerns, and manifestation and progression of behavioral signs and symptoms over time), and is considered an adjunct or alternative approach to the more categorical approach of psychiatric diagnosis. These formulations are used to communicate a hypothesis and provide a framework for developing the most suitable treatment approach, rather than labels or diagnostic codes. It gives a rich description of the client's personal history and pieces together how the disorder evolved. It is most commonly used by clinical psychologists and psychiatrists and is deemed to be a core component of these professions. Mental health nurses and social workers may also use formulations. As mental health professionals, we need to consider both the purpose of our communication and the audience for that communication. As professionals, we must also consider the potential misuse of documents by others, which will influence the way that we present our case formulation.

Types of Formulation

Different psychological schools or models utilize clinical formulations, including cognitive-behavioral therapy (CBT) and related therapies: systemic therapy, psychodynamic therapy, and applied behavior analysis. The structure and content of a clinical formulation is determined by the psychological model (which we will examine more closely in another module). Most systems of formulation contain the following broad categories of information:

- symptoms and problems
- precipitating stressors or events
- predisposing life events or stressors
- an explanatory mechanism that links the preceding categories together
- a description of the precipitants and maintaining influences of the person's problems

Many psychologists use an **integrative psychotherapy approach to formulation,** or the integration of elements from different schools of psychotherapy in the treatment of a client. This is to take advantage of the benefits of resources from each model the psychologist is trained in, according to the patient's needs. The case formulation may pull from several psychological models and formations, such as those mentioned below:

- A cognitive-behavioral model of formulation described by Jacqueline Persons has seven components: problem list, core beliefs, precipitants and activating situations, origins, working hypothesis, treatment plan, and predicted obstacles to treatment.
- A psychodynamic formulation would consist of a summarizing statement, description of nondynamic factors, description of core psychodynamics using a specific model (such as ego psychology, object relations, or self psychology), and prognostic assessment that identifies the potential areas of resistance in therapy.
- Behavioral case formulations used in applied behavior analysis and behavior therapy are built on a rank list of problem behaviors. Such functional analysis is also used in acceptance and commitment therapy. Functional analysis looks at *setting events* (ecological variables, history effects, and motivating operations); *antecedents*; *behavior chains*; *the problem behavior*; and the *consequences*, short- and long-term, for the behavior

Watch It

Watch this video to see all the components that go into a case formulation for a client. The video specifically mentions one type of therapy, acceptance and commitment therapy (ACT), but the concepts apply generally to all types of psychotherapy.



You can view the transcript for "ACT Case Conceptualization: Assessing the 6 Core Processes" here (opens in new window).

Cultural Formulation

A **cultural formulation** is the systematic review of a person's cultural background and the role of culture in the manifestation of symptoms and dysfunction. It is argued that a cultural perspective can help psychiatrists become aware of the hidden assumptions and limitations of current psychiatric theory and practice and can identify new approaches appropriate for treating the increasingly diverse populations seen in psychiatric services around the world. The *DSM-5* includes a **Cultural Formulation Interview** that aims to help clinicians contextualize diagnostic assessment. A related approach to cultural assessment involves cultural consultation that works with interpreters and cultural brokers to develop a cultural formulation and treatment plan that can assist clinicians.

Cross-cultural psychiatry (also known as ethnopsychiatry, transcultural psychiatry, or cultural psychiatry) is a branch of psychiatry concerned with the cultural context of mental disorders and the challenges of addressing ethnic diversity in psychiatric services. It looks at whether psychiatric classifications of disorders are appropriate to different cultures or ethnic groups. It often argues that psychiatric illnesses represent social constructs as well as genuine medical conditions, and as such have social uses peculiar to the social groups in which they are created and legitimized. It studies psychiatric classifications in different cultures, whether informal (e.g., category terms used in different languages) or formal (for example the World Health Organization's *ICD*, the American Psychiatric Association's *DSM*, or the Chinese Society of Psychiatry's *CCMD*). The field has increasingly had to address the process of globalization. It is said every city has a different culture and that the urban environment, and how people adapt or struggle to adapt to it, can play a crucial role in the onset or worsening of mental illness.

Reflecting advances in medical anthropology, the *DSM-5* replaced the term **culture-bound syndrome** (or folk illness—a combination of psychiatric and somatic symptoms that are considered to be a recognizable disease only within a specific society or culture) with a set of terms covering cultural concepts of distress: cultural syndromes (which may not be bound to a specific culture but circulate across cultures), cultural idioms of distress (local modes of expressing suffering that may not be syndromes), causal explanations (that attribute symptoms or suffering to specific causal factors rooted in local ontologies), and folk diagnostic categories (which may be part of ethnomedical systems and healing practices).

Dig Deeper

One argument to support the concept of culture-bound syndromes comes from research on *dhat syndrome*, as it has been specified as a culture-bound syndrome specific to the culture of the Indian subcontinent. The *DSM-4* listed it as a folk diagnostic term used in India to refer to severe anxiety and hypochondriacal concerns associated with the discharge of semen, whitish discoloration of the urine, and feelings of weakness and exhaustion. Similar to *jiryan* (India), *sukra prameha* (Sri Lanka), and *shen-k'uei* (China), *dhat syndrome* in the *DSM-5* is listed as a term that was coined in South Asia little more than half a century ago to account for common clinical presentations of young male patients who attributed their various symptoms to semen loss. Despite the name, it is not a discrete syndrome but rather a cultural explanation of distress for patients who refer to diverse symptoms, such as anxiety, fatigue, weakness, weight loss, impotence, other multiple somatic complaints, and depressive mood. The cardinal feature is anxiety and distress about the loss of *dhat* in the absence of any identifiable physiological dysfunction.

> Research in health care settings has yielded diverse estimates of the syndrome's prevalence (e.g., 64% of men attending psychiatric clinics in India for sexual complaints; 30% of men attending general clinics in Pakistan). Related conditions in *DSM-5* are major depressive disorder, persistent depressive disorder (dysthymia), generalized anxiety disorder, somatic symptom disorder, illness anxiety disorder, erectile disorder, early (premature) ejaculation, and other specified or unspecified sexual dysfunction. [2]

Planning the Treatment

Following an initial assessment, the client or patient and therapist come to an agreement called the **treatment plan**. This is a type of contract that specifies the goals of treatment; treatment procedures; and a regular schedule for the time, place, and duration of their treatment sessions. Sometimes this treatment contract is written down explicitly, but more often it is discussed between the individual seeking therapy and therapist.[3] Services are based in psychiatric hospitals or in the community. Determining the treatment site (most often private therapist's outpatient clinic or office) is an important part of planning treatment. Community mental health centers (CMHCs) provide for community-based care as an alternative to institutionalization. At these centers, patients can be treated while working and living at home.

The importance of the **therapeuticrelationship**, also known as therapeutic alliance, between client and therapist is often regarded as crucial to psychotherapy. **Psychotherapy** focuses on the use of psychological methods, particularly when based on regular personal interaction with adults, to help a person change behavior and overcome problems in desired ways. Psychotherapists may be mental health professionals such as psychiatrists, psychologists, mental health nurses, clinical social workers, marriage and family therapists, or professional counselors. Psychotherapy aims to improve an individual's well-being and mental health; to resolve or mitigate troublesome behaviors, beliefs, compulsions, thoughts, or emotions; and to improve relationships and social skills. There is also a range of psychotherapies designed for children and adolescents, which typically involve play. Certain psychotherapies are considered evidence-based for treating diagnosed mental disorders. Others have been criticized as pseudoscience. There are over a thousand different psychotherapy techniques, some being minor variations, while others are based on very different conceptions of psychology, ethics (how to behave professionally), or techniques. Most involve one-to-one sessions between the client and therapist, but some are conducted with groups, including families.

In practice, clinical and counseling psychologists work with individuals, couples, families, or groups in a variety of settings, including private practices, hospitals, mental health organizations, schools, businesses, and non-profit agencies. Other than providing psychological treatment (psychotherapy), **clinical and counseling psychologists** can offer a range of professional services, including administering and interpreting psychological assessment and testing, and providing expert testimony (forensics).

Although clinical and counseling psychologists and psychiatrists share the same fundamental aim—the alleviation of mental distress—their training, outlook, and methodologies are often different. Perhaps the most significant difference is that **psychiatrists** are licensed physicians, and, as such, psychiatrists are apt to use the medical model to assess mental health problems and to also employ psychotropic medications as a method of addressing mental health problems. Psychologists generally do not prescribe medication, although in some jurisdictions they do have prescription privileges. In five U.S. states (New Mexico, Louisiana, Illinois, Iowa, and Idaho), psychologists with post-doctoral clinical psychopharmacology training have been granted prescriptive authority for mental health disorders. Clinical and counseling psychologists receive extensive training in psychological test administration, scoring, interpretation, and reporting, while psychiatrists are not trained in psychological testing.

Goals of Treatment

Therapy includes immediate, short-term, and long-term goal setting. Goals are an essential part of the treatment plan as they help a client work towards cognitive and behavioral changes. For example, a therapist may use the SMART goals approach by setting goals that are specific, measurable, achievable, realistic, and time-limited.

- **S**pecific—The goal is to be as clear as possible, such as setting a goal to reduce social anxiety levels so that a client can eat lunch with coworkers by the end of the month.

- **M**easurable—The goal should be something you can measure, such as tallying up the number of social interactions a client has, or reducing feelings of anxiety from a 9/10 level to a 4/10 level.
- **A**chievable—The goals should be feasible and within reach. If a person has severe social anxiety, having them host a party or go out every night for a week would be difficult short-term goals that are likely unachievable.
- **R**ealistic and Resourced—Is the goal realistic and possible given the time and resources available to you?
- **T**ime-limited—The goal should have a specific time frame when it will be reached.[4]

Link to Learning

This APA article, "Understanding Psychotherapy and How It Works" provides a good introductory overview of what you can expect during psychotherapy.

Evidence-Based Practice and Ethical Duties

Professional competence—the ability to accurately assess problems, diagnose psychological disorders, recommend an appropriate course of treatment, and successfully carry out that treatment—varies depending on the degree to which the clinician keeps up to date with the latest research and effectively evaluates the evidence. The APA requires that clinicians be trained in **evidence-based practice (EBP)** to be equipped to appraise the range of evidence regarding the efficacy of different forms of psychotherapy, to recognize the strengths and limitations of clinical intuition, and to understand the importance of patient preferences and values as well as the relevance of the socio-cultural context in treating clients.

Why does evidence-based practice (EBP) matter for the ethical practice of psychological treatments? Evidence carries ethical imperatives. Both the decision about what is considered to be beneficial in psychotherapy, and the current paucity of research regarding the potential negative effects of psychological treatments, carry ethical implications. It is argued that the failure to pay attention to psychotherapy research effectively risks undermining key requisites included in professional codes of practice for clinical psychology, psychiatry, social work, and allied fields. First, evidence-based practice (EBP) bears repercussions for the clinician's duty of professional competence, or the responsibility to acquire and apply accurate knowledge. Second, EBP is relevant to the duty to respect patient autonomy—namely, the patient's right to make informed decisions concerning his or her treatment plans.

Glossary

client: reflects that people in treatment collaborate with those who treat them

clinician: health care professional who works directly with patients and may diagnose, treat, and otherwise care for patients.

clinical and counseling psychologists: mental health professional with training in behavioral science who provide direct service to clients

clinical formulation/case formulation: analyzes the factors that may have influenced the client's current psychological state

confidentiality: therapist cannot disclose confidential communications to any third party, unless mandated or permitted by law

couples therapy: when two people in an intimate relationship, such as husband and wife, attend counseling together to resolve conflict

cross-cultural psychiatry: a branch of psychiatry concerned with the cultural context of mental disorders and the challenges of addressing ethnic diversity in psychiatric services

culture-bound syndrome: a combination of psychiatric and somatic symptoms that are considered to be a recognizable disease only within a specific society or culture

cultural formulation: a systematic review of a person's cultural background and the role of culture in the manifestation of symptoms and dysfunction

cultural formulation interview: aims to help clinicians contextualize diagnostic assessment

diagnosis: refers to the identification of the nature and cause of an illness or the identification of the nature and cause of something

family therapy: special form of group therapy consisting of one or more families

group therapy: treatment modality in which five to 10 people with the same issue or concern meet together with a trained clinician

individual therapy: treatment modality in which the client and clinician meet one-on-one

intake: therapist's first meeting with the client in which the therapist gathers specific information to address the client's immediate needs

integrative psychotherapy approach to formulation: the integration of elements from different schools of psychotherapy in the treatment of a client

mental status examination (**MSE**): a structured way of observing and describing a patient's psychological functioning at a given point in time under the domains of appearance, attitude, behavior, mood, and affect speech, thought process, thought content, perception, cognition, insight, and judgment

patient: any recipient of health care services performed by healthcare professionals

psychiatrists: licensed physicians (MD) with specialized training in diagnosing and treating people with psychological disorders

psychologist: health care professional offering psychological services

strategic family therapy: when a therapist guides the therapy sessions and develops treatment plans for each family member for specific problems that can be addressed in a short amount of time

structural family therapy: therapist examines and discusses with the family the boundaries and structure of the family: who makes the rules, who sleeps in the bed with whom, how decisions are made, and what are the boundaries within the family

therapeutic relationship: the relationship between a health care professional and a client (or patient)

treatment plan: collaborative goal setting or outline of how therapy takes place

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1.13: Psychological Assessment

Learning Objectives

- Describe the types and purposes of psychological assessment

Psychological Tests

Psychological tests are written, visual, or verbal evaluations administered to assess the cognitive and emotional functioning of clients or patients. These tests can include questionnaires and interviews, which are also designed to measure unobserved constructs. As we mentioned earlier, useful psychological test/scale must be both valid (i.e., there is evidence to support the idea that the test or scale measures what it is purported to measure and "how well it does so") and reliable (i.e., internally consistent or give consistent results over time, across raters, etc.). Therefore, a test should meet the criterion of standardization.

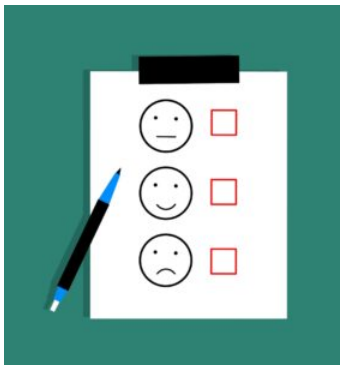


Figure **1.13.1**: A critical part of the treatment process involves utilizing assessments to gather information that can provide direction for how to move forward with a client.

One of the main advantages of standardized testing is that the results can be empirically documented; therefore, the test scores can be shown to have a relative degree of validity and reliability**,** as well as results that are generalizable and replicable. A **standardized test** is a test that is administered and scored in a consistent, or "standard," manner. Standardized tests are designed in such a way that the questions, conditions for administering, scoring procedures, and interpretations are consistent and are administered and scored in a predetermined, standard manner.

Psychological assessment is most often used in the *psychiatric*, *medical*, *legal*, educational, or *psychological clinic* settings. This type of clinical assessment can be thought of as a broad range of measurement techniques, all which involve having people provide scorable information about their psychological functioning. The types of assessments and the purposes for them differ among these settings.

In the psychiatric setting, the common needs for assessment are to determine risks, whether a person should be admitted or discharged, the location the patients should be held, and what therapy the patient should be receiving. Within this setting, psychologists need to be aware of the legal responsibilities that they can legally do in each situation.

Within a medical setting, psychological assessments are used to find a possible underlying psychological disorder, emotional factors that may be associated with medical complaints, an assessment for neuropsychological deficit, a psychological treatment for chronic pain, and the treatment of chemical dependency. There has been greater importance placed on the patient's neuropsychological status as neuropsychologists are becoming more concerned with the functioning of the brain.

Psychological assessment also has a role in the legal setting. Psychologists might be asked to assess the reliability of a witness, the quality of the testimony a witness gives, the competency of an accused person, or determine what might have happened during a crime. They also may help support a plea of insanity or to discount a plea. Judges may use the psychologist's report to change the sentence of a convicted person, and parole officers work with psychologists to create a program for the rehabilitation of a parolee. Problematic areas for psychologists include predicting how dangerous a person will be. There is currently no accurate measure for this prediction; however, there is often a need for this prediction to prevent dangerous people from returning to society.

Psychologists may also be called on to assess a variety of things within an education setting. They may be asked to assess the strengths and weaknesses of children who are having difficulty in the school systems (via intelligence testing), assess behavioral difficulties, assess a child's responsiveness to an intervention, or help create an educational plan for a child. The assessment of children also allows for the psychologists to determine if the child will be willing to use the resources that may be provided. Due to rigorous efforts in developing norms, and developing reliability and validity measures, certain measures such as the Wechsler intelligence scales for both adults and children are seen as the strongest psychological instruments around.

In a psychological clinic setting, psychological assessment can be used to determine characteristics of the client that can be useful for developing a treatment plan. Within this setting, psychologists often are working with clients who may have medical or legal problems or sometimes with students who were referred by their school psychologist.

Some psychological assessments have been validated for use when administered via computer or the internet. However, caution must be applied to these test results as it is possible to fake in electronically mediated assessment. Many electronic assessments do not truly measure what is claimed, such as the Meyers-Briggs personality test. Although one of the most well-known personality assessments, it has been found both invalid and unreliable by many psychological researches and should be used with caution.[1][2]

Types of Psychological Assessment

Psychological measures generally fall within one of several categories, including the following:

- **Intelligence & achievement tests**—These tests are designed to measure certain specific kinds of cognitive functioning (often referred to as IQ) in comparison to a norming group. These tests, such as the WISC-IV and the WAIS, attempt to measure such traits as general knowledge, verbal skill, memory, attention span, logical reasoning, and visual/spatial perception. Several tests have been shown to predict accurately certain kinds of performance, especially scholastic.
- **Personality tests**—Tests of personality aim to describe patterns of behavior, thoughts, and feelings. They generally fall within two categories: objective and projective. Objective measures, such as the *MMPI*, are based on restricted answers—such as yes/no, true/false, or a rating scale—which allow for the computation of scores that can be compared to a normative group. Projective tests, such as the *Rorschach* inkblot test, allow for open-ended answers, often based on ambiguous stimuli. Other commonly used personality assessment measures include the *PAI* and the *NEO-PI-R*.
- **Neuropsychological tests**—Neuropsychological tests consist of specifically designed tasks used to measure psychological functions known to be linked to a particular brain structure or pathway. They are typically used to assess impairment after an injury or illness known to affect neurocognitive functioning, or when used in research, to contrast neuropsychological abilities across experimental groups.
- **Diagnostic Measurement Tools**—Clinical psychologists are able to diagnose psychological disorders and related disorders found in the *DSM-5* and *ICD-10*. Many assessment tests have been developed to complement the clinicians clinical observation and other assessment activities. Some of these include the *SCID-5*, the most widely used.
- **Clinical observation**—Clinical psychologists are also trained to gather data by observing behavior. The clinical interview is a vital part of the assessment, even when using other formalized tools, which can employ either a structured or unstructured format. Such assessment looks at certain areas, such as general appearance and behavior, mood and affects, perception, comprehension, orientation, insight, memory, and content of the communication. One psychiatric example of a formal interview is the **mental status examination**, which is often used in psychiatry as a screening tool for treatment or further testing.

Link to Learning

This video clip demonstrates how a clinician conducts a mental state examination with a client, Glen, who is struggling with alcohol use and other symptoms following the death of his father (note that there is some strong language in the clip). As you watch, consider the techniques used by the clinician as she gathers information about Glen.

Glossary

clinical interview: a face-to-face encounter between a mental health professional and a patient in which the former observes the latter and gathers data about the person's behavior, attitudes, current situation, personality, and life history

mental status examination (MSE): a medical process where a clinician working in the field of mental health systematically examines a patient's mind and the way they look, think, feel, and behave

psychological assessment: a broad range of measurement techniques, all which involve having people provide scorable information about their psychological functioning

psychological tests: written, visual, or verbal evaluations administered to assess the cognitive and emotional functioning of clients or patients

standardization: a psychometric criterion that clearly specifies a test's instructions for administration and scoring

standardized test: a test that is administered and scored in a consistent, or "standard," manner

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1.14: Clinical Assessments and the Mental Status Examination

Learning Objectives

- Describe methods for clinical interviews and the mental status examination

The Importance of Assessment

Across clinical and research domains, mental health assessment and diagnosis are carried out using interviews and questionnaires that determine the presence, severity, frequency, and duration of a broad range of psychiatric symptoms. The question content of these assessment tools is often based on classification systems that we have previously discussed, notably the *DSM-5* and/or *ICD*, where pre-defined patterns of symptom criteria have been grouped together and designated as specific mental health disorders. Their design ranges from more open-ended, clinician-led interviews typically used to make a formal psychiatric diagnosis (e.g., The Structured Clinical Interview for *DSM* Disorders (SCID), to more quantitatively designed auxiliary questionnaires (e.g., The Patient Health Questionnaire (PHQ-9) that provide multidimensional assessments of symptom experience and severity to support diagnosis and treatment evaluation in clinical practice. These interviews are used to investigate underlying etiologies and treatment effectiveness in clinical trials and academic research studies.[1]

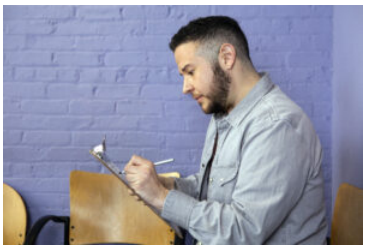


Figure **1.14.1**: Patients may be asked various questions about their mental health to help reach a diagnosis.

When an individual begins therapy, collateral information is also collected about personal, occupational, or medical history, such as from records or from interviews with parents, spouses, teachers, or previous therapists or physicians. Many psychologists do some level of assessment when providing services to clients or patients, and may use for example, simple checklists to assess some traits or symptoms, but psychological assessment is a more complex, detailed, in-depth process. Typical types of focus for psychological assessment are to provide a diagnosis; to assess a particular area of functioning or disability, often for school settings; to help select a type of treatment or to assess treatment outcomes; to help courts decide issues such as child custody or competency to stand trial; to help assess job applicants or employees; or to provide career development counseling or training.

A top-perspective of this landscape of mental health assessment reveals a huge range of interviews and questionnaires available for use. This diversity of choice means there is no shortage of options when searching for assessment tools for clinical use or to suit the needs of a clinical research study. However, this diversity can also make it a real challenge to decide which questionnaire(s) or interview(s) to select for clinical diagnosis or evaluation. For example, there have been more than 280 different questionnaires developed over the last century to assess symptoms of depression that differ in terms of which iteration of the *DSM* they align to; the degree to which they consider co-morbid symptoms; whether they are computer-based or paper-based; and whether they are self-rated, parent-rated or clinician-led. Knowing which questionnaire to choose to obtain a suitable assessment of an individual's mental health is therefore not always a straightforward exercise for even the most experienced researcher or clinician.

The Clinical Interview

The clinical interview is the most widely used means of assessment. A **clinical interview** is a face-to-face encounter between a mental health professional and a patient in which the professional observes the patient and gathers data about the person's behavior, attitudes, current situation, personality, and life history. The interview may be unstructured, in which open-ended questions are asked; structured, in which a specific set of questions according to an interview schedule are asked; or semi-structured, in which there is a pre-set list of questions but clinicians are able to follow up on specific issues that catch their attention.

Unstructured Interview

An **unstructured interview** or non-directive interview is an interview in which questions are not pre-arranged. These non-directive interviews are considered to be the opposite of a structured interview that offers a set amount of standardized questions. They tend to be more informal and free flowing than a structured interview, much like an everyday conversation. The chief feature of the unstructured interview is the idea of using probing questions aimed at determining the client's reason for being in treatment, symptoms, health status, family background, and life history that are designed to be as open as possible. Open-ended questions that have no prepared response choices enable and empower the client to shift the direction of the interview and to bring in unanticipated information. It can require a skillful clinician to bring a talkative respondent back on topic, or to help a client end an awkward silence. However, these open-ended questions give the ability for the respondent to reply about a topic that neither the interviewee nor the interviewer may have thought about before. Some evidence shows that using open-ended questions in interviews results in greater reporting of sensitive subjects, such as domestic violence, or socially disapproved behavior than when closed-ended questions on a self-reporting questionnaire are used. A typical clinical interview will cover the following:

- age and sex
- rearson for referral
- education and work history
- current social situation
- physical and mental health history
- drug/alcohol use and current medication
- family history
- behavioral observations

Structured Interview

A **structured interview** (also known as a standardized interview) can provide a diagnosis or classify the client's symptoms into a *DSM-5* disorder. The aim of this approach is to ensure that each interview is presented with exactly the same questions in the same order. This aim ensures that answers can be reliably aggregated. The **Structured Clinical Interview for DSM-5 Disorders (SCID-5)** is one of the most widely used clinical interviews. It is a diagnostic exam and covers the diagnoses most commonly seen in clinical settings.

Both of these methods (unstructured and structured) have their pros and cons. A highly unstructured interview and informal observations provide key findings about the patient that are both efficient and effective. A potential issue with an unstructured, informal approach is that the clinician may overlook certain areas of functioning or not notice them at all. Or they might focus too much on presenting complaints. The highly structured interview, although very precise, can cause the clinician to make the mistake of focusing a specific answer to a specific question without considering the response in terms of a broader scope or life context. They may fail to recognize how the patient's answers all fit together.

As mentioned above, a **semi-structured** interview is another option. In this situation, the clinician follows a general outline of questions designed to gather essential information, but is free to ask the questions in any particular order and to branch off into other directions to follow up on relevant information.

There are many ways that the issues associated with the interview process can be mitigated. The benefits to more formal standardized evaluation types such as batteries and tests are many. First, they measure a large number of characteristics simultaneously. These include personality, cognitive, or neuropsychological characteristics. Second, these tests provide empirically quantified information. The obvious benefit to this is that we can more precisely measure patient characteristics as compared to any kind of structured or unstructured interview. Third, all these tests have a standardized way of being scored and being administered. Each patient is presented with a standardized stimulus that serves as a benchmark that can be used to determine their characteristics. These types of tests eliminate any possibility of bias and produce results that could be harmful to the patient and cause legal and ethical issues. Fourth, tests are *normed*. This means that patients can be assessed not only based on their comparison to a "normal" individual ,but how they compare to the rest of their peers who may have the same psychological issues that they face. Normed tests allow the clinician to make a more individualized assessment of the patient. Fifth, standardized tests that we commonly use today are both valid and reliable. We know what specific scores mean, how reliable they are, and how the results will affect the patient.

Most clinicians agree that a balanced battery of tests is the most effective way of helping patients. Clinicians should not become victims of blind adherence to any one particular method. A balanced battery of tests allows there to be a mix of formal testing processes that allow the clinician to start making their assessment while conducting more informal, unstructured interviews with the same patient may help the clinician to make more individualized evaluations and help piece together what could potentially be a very complex, unique-to-the-individual kind of issue or problem.

Mental Status Examination (MSE)

Mental status examination, or MSE, is a medical process where a clinician working in the field of mental health (usually a psychotherapist, social worker, psychiatrist, psychiatric nurse, or psychologist) systematically examines a patient's mind and the way they look, think, feel, and behave. It is an important part of the clinical assessment process. It is a structured way of observing and describing a patient's psychological functioning at a given point in time under the domains of appearance, attitude, behavior, and mood and affect speech, thought process, thought content, perception, cognition, insight, and judgment. There are some minor variations in the subdivision of the MSE and the sequence and names of MSE domains.

The purpose of the MSE is to obtain a comprehensive, cross-sectional description of the patient's mental state, which, when combined with the biographical and historical information of the psychiatric history, allows the clinician to make an accurate diagnosis and formulation, which are required for coherent treatment planning. The data are collected through a combination of direct and indirect means: unstructured observation while obtaining the biographical and social information, focused questions about current symptoms, and formalized psychological tests.

The MSE is a core skill of qualified (mental) health personnel. It is a key part of the initial psychiatric assessment in an outpatient or psychiatric hospital setting. It is a systematic collection of data based on observation of the patient's behavior while the patient is in the clinician's view during the interview. The purpose is to obtain evidence of symptoms and signs of mental disorders, including danger to self and others, that are present at the time of the interview. Further, information on the patient's insight, judgment, and capacity for abstract reasoning is used to inform decisions about treatment strategy and the choice of an appropriate treatment setting. It is carried out in the manner of an informal inquiry, using a combination of open and closed questions, supplemented by structured tests to assess cognition. The MSE can also be considered part of the comprehensive physical examination performed by physicians and nurses, although it may be performed in a cursory and abbreviated way in non–mental-health settings. Information is usually recorded as free-form text using the standard headings, but brief MSE checklists are available for use in emergency situations, for example by paramedics or emergency department staff. The information obtained in the MSE is used, together with the biographical and social information of the psychiatric history, to generate a diagnosis, a psychiatric formulation, and a treatment plan.

The MSE accesses behavioral and cognitive functioning. Behavioral components include appearance and general behavior, level of consciousness and attentiveness, motor and speech activity, mood and affect, thought and perception, attitude and insight, and the reaction evoked by the examiner. Cognitive components include alertness, language, memory, constructional ability, and abstract reasoning. There are many versions of MSE that differ around the world, but there is a broad commonality and goal in constructing an overall picture of a patient's mental health.

Sample Mental Status Examination

Appearance: 25-year-old African American female, appears stated age, wearing paper hospital scrubs that have been cut to reveal abdomen with vertical abdominal scar visible, and multiple tattoos of various names visible on forearms bilaterally

Behavior: not in acute distress, difficult to redirect for interviewing, inappropriately laughing and smiling

Motor Activity: minimal psychomotor agitation present; regular gait; regular posturing; no tics, tremors, or EPS present

Speech: hyperverbal, fluent, pressured rate, regular rhythm, regular volume, happy tone

Mood: "fantastic"

Affect: elated, inappropriate, congruent

Thought Process: flight of ideas

Thought Content: denies suicidal ideations, denies homicidal ideations; grandiose delusions elicited of being "an angel on a mission"

Perceptions: endorses auditory hallucinations of God commanding her to go to California; denies visual hallucinations.; does not appear to be actively responding to internal stimuli

Cognition:

Sensorium/orientation: alert and oriented to person, place, and date

Attention/concentration: poor; unable to spell WORLD forward and backward

Memory: able to recall 3/3 objects immediately and after one minute; recent memory—intact to breakfast this morning; long-term memory—intact to what high school she attended

Abstract reasoning: intact with ability to identify a bird and tree as both living

Insight: poor

Judgment: poor

Watch It

This video gives examples of the types of questions asked and information gathered during a mental status examination.

Dig Deeper: Cultural Considerations

The outcome of the MSE is a comprehensive description of how the client looks, thinks, feels, and behaves. There are potential problems when the MSE is applied in a cross-cultural context, as when the clinician and patient are from different cultural backgrounds. For example, the patient's culture might have different norms for appearance, behavior, and display of emotions. Culturally normative spiritual and religious beliefs need to be distinguished from delusions and hallucinations—these may seem similar to one who does not understand that they have different roots. Cognitive assessment must also take the patient's language and educational background into account. Clinician's racial bias is another potential confounder.[2] [3]

Link to Learning

This video clip demonstrates how a clinician conducts a mental state examination with a client, Glen, who is struggling with alcohol use and other symptoms following the death of his father (note that there is some strong language in the clip). As you watch, consider the techniques used by the clinician as she gathers information about Glen.

Glossary

clinical interview: a face-to-face encounter between a mental health professional and a patient in which the former observes the latter and gathers data about the person's behavior, attitudes, current situation, personality, and life history

mental status examination (MSE): a medical process where a clinician working in the field of mental health systematically examines a patient's mind and the way they look, think, feel and behave

mini–mental state examination (MMSE): a brief neuropsychological screening test for dementia

semi-structured interview: a pre-set list of questions but clinicians are able to follow up on specific issues that catch their attention

Structured Clinical Interview for DSM-5 Disorders (SCID-5): a diagnostic exam and covers the diagnoses most commonly seen in clinical settings.

structured interview (also known as a standardized interview): approach to ensure that each interview is presented with exactly the same questions in the same order

unstructured interview: non-directive interview, or an interview in which questions are not prearranged

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1.15: Intelligence Testing

Learning Objectives

- Explain the purposes and types of intelligence testing (including the Wechsler and Stanford-Binet intelligence tests and the Kaufman Assessment Battery for Children)

Intelligence is among the oldest and longest studied topics in all of psychology. The development of assessments to measure this concept is at the core of the development of psychological science itself.



Figure **1.15.1**: Starting from early primary school years, researchers and clinicians assess intelligence through standardized test batteries (i.e., IQ tests), as these tests are intended to be suitable for a wide age range.

When might an IQ test be used? What do we learn from the results, and how might people use this information? While there are certainly many benefits to intelligence testing, it is important to also note the limitations and controversies surrounding these tests. For example, IQ tests have sometimes been used as arguments in support of insidious purposes, such as the eugenics movement (Severson, 2011). The infamous Supreme Court Case, *Buck vs. Bell*, legalized the forced sterilization of some people deemed "feeble-minded" through this type of testing, resulting in about 65,000 sterilizations (*Buck vs. Bell*, 274 U.S. 200; Ko, 2016). Today, only professionals trained in psychology can administer IQ tests, and the purchase of most tests requires an advanced degree in psychology. Other professionals in the field, such as social workers and psychiatrists, cannot administer IQ tests.

In psychology, human intelligence is commonly assessed by IQ scores that are determined by IQ tests. However, there are critics of IQ tests who, while they do not dispute the stability of IQ test scores or the fact that they predict certain forms of achievement rather effectively, do on the other hand argue that to base a concept of intelligence on IQ test scores alone is to ignore many important aspects of mental ability.

For instance, individual differences in general intelligence are one of the strongest predictors of occupational attainment, social mobility, and job performance. People with higher general intelligence in childhood or early adulthood also tend to have better overall physical health, and have a longer life expectancy. Further, children with higher childhood IQ have a lower risk of developing dementia and of being diagnosed with schizophrenia spectrum disorder, major depression, or any anxiety disorder in adulthood. Understanding how differential IQ levels become associated with these life outcomes is crucial not only from a scientific point of view, but also for developing public policy and determining effective interventions.

Why Measure Intelligence?

The value of IQ testing is most evident in educational or clinical settings. Children who seem to be experiencing learning difficulties or severe behavioral problems can be tested to ascertain whether the child's difficulties can be partly attributed to an IQ score that is significantly different from the mean for his or her age group. Without IQ testing—or another measure of intelligence—children and adults needing extra support might not be identified effectively. While IQ tests have sometimes been used as arguments in support of insidious purposes, such as the eugenics movement (Severson, 2011), the following case study demonstrates the usefulness and benefits of IQ testing.

Why Test IQ?

Candace, a 14-year-old girl experiencing problems at school in Connecticut, was referred for a court-ordered psychological evaluation. She was in regular education classes in ninth grade and was failing every subject. Candace had never been a stellar student but had always been passed to the next grade. Frequently, she would curse at any of her teachers who called on her in

class. She also got into fights with other students and occasionally shoplifted. When she arrived for the evaluation, Candace immediately said that she hated everything about school, including the teachers, the rest of the staff, the building, and the homework. Her parents stated that they felt their daughter was picked on because she was of a different race than the teachers and most of the other students. When asked why she cursed at her teachers, Candace replied, “They only call on me when I don’t know the answer. I don’t want to say, ‘I don’t know’ all the time and look like an idiot in front of my friends. The teachers embarrass me.” She was given a battery of tests, including an IQ test. Her score on the IQ test was 68.

What does Candace’s score say about her ability to excel or even succeed in regular education classes without assistance? Why were her difficulties never noticed or addressed?

Types of IQ Tests and Tasks

There are a wide variety of IQ tests that use slightly different tasks and measures to calculate an overall IQ score. The most commonly used test series is the Wechsler Adult Intelligence Scale (WAIS) and its counterpart, the Wechsler Intelligence Scale for Children (WISC). Other commonly used tests include the original and updated version of Stanford-Binet, and the Kaufman Assessment Battery for Children. We will discuss all of these.

Stanford-Binet Intelligence Test

The person best known for formally pioneering the measurement of intellectual ability is Alfred Binet. Binet was particularly interested in the development of intelligence, a fascination that led him to observe children carefully in the classroom setting. Along with his colleague Theodore Simon, Binet created a test of children’s intellectual capacity. Simon and Binet created individual test items that should be answerable by children of given ages. For instance, a child who is three should be able to point to her mouth and eyes, a child who is nine should be able to name the months of the year in order, and a twelve-year-old ought to be able to name sixty words in three minutes. Their assessment became the first IQ test.

IQ or *intelligence quotient* is a name given to the score of the Binet-Simon test. The score is derived by dividing a child’s mental age (the score from the test) by their chronological age to create an overall quotient. These days, the phrase *IQ* does not apply specifically to the Binet-Simon test and is used to generally denote intelligence or a score on any intelligence test. In the early 1900s, the Binet-Simon test was adapted by a Stanford professor named Lewis Terman to create what is, perhaps, the most famous intelligence test in the world, the Stanford-Binet (Terman, 1916). The major advantage of this new test was that it was **standardized.** Based on a large sample of children, Terman was able to plot the scores in a normal distribution, shaped like a bell curve (see Fig. 1). To understand a normal distribution, think about the height of people. Most people are average in height, with relatively fewer being tall or short, and fewer still being extremely tall or extremely short. Terman (1916) laid out intelligence scores in exactly the same way, allowing for easy and reliable categorizations and comparisons between individuals.

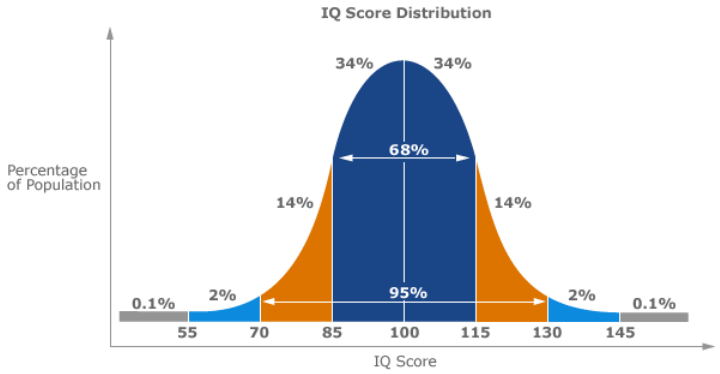


Figure **1.15.2**: The bell curve showing the normal distribution of IQ, with 100 being the average score.

Weschler Intelligence Scales

A look at another modern intelligence test—the Wechsler Adult Intelligence Scale (WAIS)—can provide clues to a definition of intelligence itself. Motivated by several criticisms of the Stanford-Binet test, psychologist David Wechsler sought to create a superior measure of intelligence. He was critical of the way that the Stanford-Binet relied so heavily on verbal ability and was also suspicious of using a single score to capture all intelligence. To address these issues, Wechsler created a test that tapped a wide

range of intellectual abilities. The the Wechsler Adult Intelligence Scale (WAIS) assesses people's ability to remember, compute, understand language, reason well, and process information quickly (Wechsler, 1955). David Wechsler's approach to testing intellectual ability was based on the fundamental idea that there are, in essence, many aspects to intelligence. The Stanford-Binet test reflected mostly verbal abilities, while the Wechsler test also reflected nonverbal abilities. The Stanford-Binet has also been revised several times and is now similar to the Wechsler in several aspects, but the Wechsler continues to be the most popular test in the United States.

The current version of the test, the WAIS-IV, which was released in 2008, comprises 10 core subtests and five supplemental subtests, with the 10 core subtests yielding scaled scores that sum to derive the Full Scale IQ. With the WAIS-IV, the verbal/performance IQ scores from previous versions were removed and replaced by the index scores. The General Ability Index (GAI) was included, which consists of the similarities, vocabulary, and information subtests from the verbal comprehension index and the block design, matrix reasoning and visual puzzles subtests from the perceptual reasoning index. The General Ability Index (GAI) is clinically useful because it can be used as a measure of cognitive abilities that are less vulnerable to impairments of processing speed and working memory.

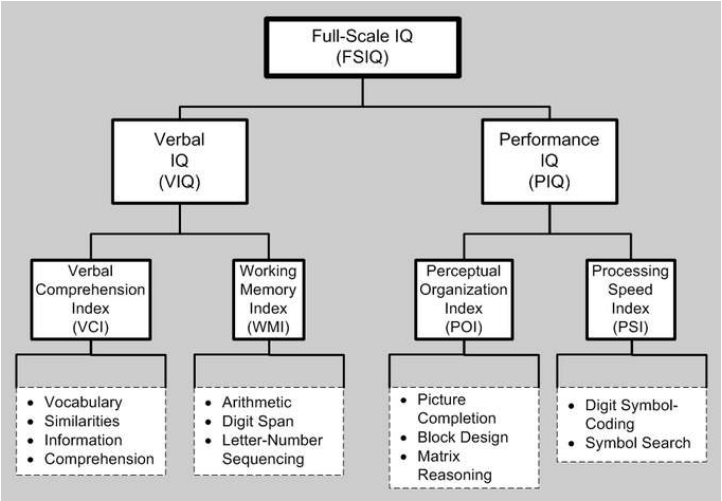


Figure **1.15.3**: The WAIS uses a variety of components to determine a person's IQ score, including verbal, memory, perceptual, and processing skills.

Index Scores and Scales

There are four index scores representing major components of intelligence:

- Verbal Comprehension Index (VCI)
- Perceptual Reasoning Index (PRI)
- Working Memory Index (WMI)
- Processing Speed Index (PSI)

Two broad scores, which can be used to summarize general intellectual ability, can also be derived:

- Full Scale IQ (FSIQ), based on the total combined performance of the VCI, PRI, WMI, and PSI
- General Ability Index (GAI), based only on the six subtests that the VCI and PRI comprise.

Tasks grouped by index

Index	Task	Core?	Description	Proposed abilities measured
Task	Similarities	✓	Describe how two words or concepts are similar.	Abstract verbal reasoning; semantic knowledge
Task	Vocabulary	✓	Name objects in pictures or define words presented to them.	Semantic knowledge; verbal comprehension and expression
Verbal Comprehension **Task**	Information	✓	General knowledge questions	Degree of general information acquired from culture
Task	Comprehension		Questions about social situations or common concepts	Ability to express abstract social conventions, rules, and expressions
Task	Block Design	✓	Put together red-and-white blocks in a pattern according to a displayed model; this is timed, and some of the more difficult puzzles award bonuses for speed	Visual spatial processing and problem solving; visual motor construction
Task	Matrix Reasoning	✓	View an array of pictures with one missing square, and select the picture that fits the array from five options	Nonverbal abstract problem solving, inductive reasoning
Perceptual Reasoning **Task**	Visual Puzzles	✓	View a puzzle in a stimulus book and choose from among pieces of which three could construct the puzzle	Visual spatial reasoning
Task	Picture Completion		Select the missing part of a picture	Ability to quickly perceive visual details
Task	Figure Weights		View a stimulus book that pictures shapes on a scale (or scales) with one empty side and select the choice that keeps the scale balanced	Quantitative reasoning
Working Memory **Task**	Digit Span	✓	Listen to sequences of numbers orally and to repeat them as heard, in reverse order, and in ascending order	Working memory, attention, encoding, auditory processing

	Task	Arithmetic	✓	Orally administered arithmetic word problems; timed	Quantitative reasoning, concentration, mental manipulation
	Task	Letter-Number Sequencing		Recall a series of numbers in increasing order and letters in alphabetical order	Working memory, attention, mental control
Processing Speed	**Task**	Symbol Search	✓	View rows of symbols and target symbols, and mark whether or not the target symbols appear in each row	Processing speed
	Task	Coding	✓	Transcribe a digit-symbol code using a key. The task is time-limited	Processing speed, associative memory, graphomotor speed
	Task	Cancellation		Scan arrangements of shapes and mark specific target shapes within a limited amount of time	Processing speed

The Wechsler Intelligence Scale for Children (WISC)

The **Wechsler Intelligence Scale for Children** (**WISC**) is an individually administered intelligence test for children between the ages of 6 and 16. The Fifth Edition is the most recent version. The WISC-V generates a Full Scale IQ that represents a child's general intellectual ability. It also provides five primary index scores: Verbal Comprehension Index, Visual Spatial Index, Fluid Reasoning Index, Working Memory Index, and Processing Speed Index. Five ancillary composite scores can be derived from various combinations of primary or primary and secondary subtests.



Figure 1.15.4: The (WISC), developed by David Wechsler, is an individually administered intelligence test for children between the ages of six and 16. The Fifth Edition (WISC-V; Wechsler, 2014) is the most recent version.

Five complementary subtests yield three complementary composite scores to measure related cognitive abilities relevant to assessment and identification of specific *learning disabilities*, particularly *dyslexia* and *dyscalculia*. Variation in testing procedures and goals can reduce time of assessment to 15–20 minutes for the assessment of a single primary index, or increase testing time to three or more hours for a complete assessment, including all primary, ancillary, and complementary indices.

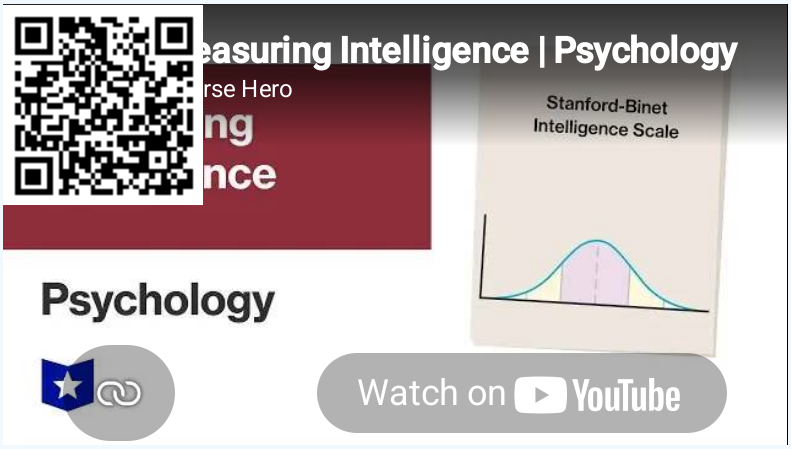
Kaufman Assessment Battery for Children

The **Kaufman Assessment Battery for Children** (**KABC**) is a clinical instrument (psychological diagnostic test) for assessing cognitive development. Its construction incorporates several recent developments in both psychological theory and statistical methodology. The test was developed by Alan S. Kaufman and Nadeen L. Kaufman in 1983 and revised in 2004. The Kaufman Assessment Battery for Children **(**KABC) also gives special attention to certain emerging testing needs, such as use with handicapped groups, application to problems of learning disabilities, and appropriateness for cultural and linguistic minorities. The authors rightly caution, however, that success in meeting these special needs must be judged through practical use over time. The

KABC-II helps to identify an individual's strengths and weaknesses in cognitive ability and mental processing. The information provided by the KABC-II can facilitate clinical and educational planning, treatment planning, and placement decisions.

Watch It

This video explains some of the intelligence tests as well as some of the complications in measuring intelligence.



You can view the transcript for "Measuring Intelligence | Psychology" here (opens in new window).

Glossary

IQ: intelligence quotient, or name given to the score of the Binet-Simon test; the score is derived by dividing a child's mental age (the score from the test) by their chronological age to create an overall quotient

Kaufman Assessment Battery for Children (KABC): a clinical instrument (psychological diagnostic test) for assessing cognitive development

Stanford–Binet Intelligence Scales: an individually administered intelligence test that was revised from the original Binet–Simon Scale by Lewis Terman, a psychologist at Stanford University

Wechsler Adult Intelligence Scale (WAIS): an IQ test designed to measure intelligence and cognitive ability in adults and older adolescents

Wechsler Intelligence Scale for Children (WISC): an individually administered intelligence test for children between the ages of six and 16

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1.16: Personality Testing

Learning Objectives

- Describe personality testing, including self-report inventories and projective personality tests

Personality tests are techniques designed to measure one's personality. They are used to diagnose psychological problems as well as to screen candidates for college and employment. There are two types of personality tests: self-report inventories and projective tests. The **Minnesota Multiphasic Personality Inventory (MMPI)** is one of the most common self-report inventories. It asks a series of true/false questions that are designed to provide a clinical profile of an individual. Projective tests use ambiguous images or other ambiguous stimuli to assess an individual's unconscious fears, desires, and challenges. The Rorschach Inkblot Test, the TAT, the RISB, and the C-TCB are all forms of projective tests.

Self-Report Inventories

Self-report inventories are a kind of objective test used to assess personality. They are standardized questions with fixed response categories that the test-taker completes independently. They typically use multiple-choice items or numbered scales, which represent a range from one (strongly disagree) to five (strongly agree). They often are called *Likert scales* after their developer, Rensis Likert (1932). Self-report inventories are generally easy to administer and cost-effective. There is also an increased likelihood of test-takers being inclined to answer in ways that are intentionally or unintentionally more socially desirable, exaggerated, biased, or misleading.

	Strongly Disagree	Somewhat Disagree	No Opinion	Somewhat Agree	Strongly Agree
I am easygoing.	O	O	O	O	O
I have high standards.	O	O	O	O	O
I enjoy time alone.	O	O	O	O	O
I work well with others.	O	O	O	O	O
I dislike confrontation.	O	O	O	O	O
I prefer crowds over intimacy.	O	O	O	O	O

Figure **1.16.1**: If you've ever taken a survey, you are probably familiar with Likert-type scale questions. Most personality inventories employ these types of response scales.

One of the most widely used personality inventories is the Minnesota Multiphasic Personality Inventory (MMPI), first published in 1943, with 504 true/false questions, and updated to the MMPI-II in 1989, with 567 questions. The original Minnesota Multiphasic Personality Inventory (MMPI) was based on a small, limited sample, comprised mostly of Minnesota farmers and psychiatric patients; the revised inventory was based on a more representative, national sample to allow for better standardization. The MMPI-II takes one to two hours to complete. Responses are scored to produce a clinical profile composed of 10 scales: hypochondriasis, depression, hysteria, psychopathic deviance (social deviance), masculinity versus femininity, paranoia, psychasthenia (obsessive/compulsive qualities), schizophrenia, hypomania, and social introversion. Considering that some of the characteristics of the MMPI may include tendencies of mental illness, such as depression, hysteria, social deviance, paranoia, obsessive/compulsive behaviors, schizophrenia, and some risk for alcohol abuse, the inventory test may help clinicians in assessment and diagnosis. In 2008, the test was again revised, using more advanced methods, to the MMPI-II-RF. Despite the new test's advantages, the MMPI-II is more established and is still more widely used. Typically, the tests are administered by computer. Although the MMPI was originally developed to assist in the clinical diagnosis of psychological disorders, it is now also used for occupational screening, such as in law enforcement, and in college, career, and marital counseling (Ben-Porath & Tellegen, 2008).

	True	False
1. I like gardening magazines.	O	O
2. I am unhappy with my sex life.	O	O
3. I feel like no one understands me.	O	O
4. I think I would enjoy the work of a teacher.	O	O
5. I am not easily awakened by noise.	O	O

Figure **1.16.2**: These true/false questions resemble the kinds of questions you would find on the MMPI.

Big Five Personality Traits

In psychological trait theory, the Big Five personality traits, also know as the OCEAN model, is a suggested taxonomy, or grouping, for personality traits, developed from the 1980s onwards. When factor analysis (a statistical technique) is applied to personality survey data, it reveals semantic associations: some words used to describe aspects of personality are often applied to the same person. For example, someone described as conscientious is more likely to be described as “always prepared” rather than “messy.” These associations suggest five broad dimensions used in common language to describe the human personality and psyche.

The theory identifies these five factors:

- openness to experience (inventive/curious vs. consistent/cautious)
- conscientiousness (efficient/organized vs. extravagant/careless)
- extraversion (outgoing/energetic vs. solitary/reserved)
- agreeableness (friendly/compassionate vs. challenging/callous)
- neuroticism (sensitive/nervous vs. resilient/confident)

As related to mental illness, the Big Five personality traits focus on the trait of neuroticism. Neuroticism is the tendency to experience negative emotions, such as anger, anxiety, or depression. It is sometimes called emotional instability, or is reversed and referred to as emotional stability. According to Hans Eysenck’s (1967) theory of personality, neuroticism is interlinked with low tolerance for stress or aversive stimuli. Neuroticism is a classic temperament trait that has been studied in temperament research for decades, before it was adapted by the FFM. Those who score high in neuroticism are emotionally reactive and vulnerable to stress. They are more likely to interpret ordinary situations as threatening. They can perceive minor frustrations as hopelessly difficult. They also tend to be flippant in the way they express emotions. Their negative emotional reactions tend to persist for unusually long periods of time, which means they are often in a bad mood. For instance, neuroticism is connected to a pessimistic approach toward work, to the certainty that work impedes personal relationships, and to higher levels of anxiety from the pressures at work. Furthermore, those who score high on neuroticism may display more skin-conductance reactivity than those who score low on neuroticism. These problems in emotional regulation can diminish the ability of a person scoring high on neuroticism to think clearly, make decisions, and cope effectively with stress. Lacking contentment in one’s life achievements can correlate with high neuroticism scores and increase one’s likelihood of falling into clinical depression. Moreover, individuals high in neuroticism tend to experience more negative life events, but neuroticism also changes in response to positive and negative life experiences. Also, individuals with higher levels of neuroticism tend to have worse psychological well-being.

At the other end of the scale, individuals who score low in neuroticism are less easily upset and are less emotionally reactive. They tend to be calm, emotionally stable, and free from persistent negative feelings. Freedom from negative feelings does not mean that low-scorers experience a lot of positive feelings, however.

Neuroticism is similar but not identical to being neurotic in the Freudian sense (i.e., neurosis.) Some psychologists prefer to call neuroticism by the term emotional instability to differentiate it from the term neurotic in a career test.

Neuroticism

Below are sample self-assessment statements designed to measure neuroticism.

- I get irritated easily.
- I get stressed out easily.
- I get upset easily.
- I have frequent mood swings.
- I worry about things.
- I am much more anxious than most people.
- I am relaxed most of the time. (*reversed*)
- I seldom feel blue. (*reversed*)

Hans Eysenck's Theory of Personality

The two personality dimensions extraversion and neuroticism were described in his 1947 book *Dimensions of Personality*. It is common practice in personality psychology to refer to the dimension *extraversion* as *E* and to the dimension *neuroticism* as *N*. The combination of extraversion and neuroticism may help explain maladaptive behavior and tie into early Greek theories.

E and N provided a two-dimensional space to describe individual differences in behavior. Eysenck noted how these two dimensions were similar to the four personality types first proposed by the Greek physician Galen.

- High N and high E = Choleric type
- High N and low E = Melancholic type
- Low N and high E = Sanguine type
- Low N and low E = Phlegmatic type

The third dimension, psychoticism, was added to the model in the late 1970s, based upon collaborations between Eysenck and his wife, Sybil B. G. Eysenck.

Psychoticism may be divided into narrower traits such as impulsivity and sensation-seeking; these may, in turn, be further subdivided into even more specific traits—for example, impulsivity may be divided into narrow impulsivity (unthinking responsivity), risk-taking, non-planning, and liveliness. Sensation seeking has also been analyzed into a number of separate facets. Psychoticism ties into mental illness as well as neuroticism to aid in understanding specific characteristics of diagnosis. For example, impulsivity could be consistent with Bipolar I and II diagnosis, as well as borderline personality disorder.

Projective Tests

Another method for assessment of personality is **projective testing,** sometimes called performance-based testing. This kind of test relies on one of the defense mechanisms proposed by Freud—projection—as a way to assess unconscious processes. During this type of testing, a series of ambiguous cards is shown to the person being tested, who then is encouraged to project his feelings, impulses, and desires onto the cards—by telling a story, interpreting an image, or completing a sentence. Some examples of projective tests are the Rorschach Inkblot Test, the Thematic Apperception Test (TAT), the Contemporized-Themes Concerning Blacks test, the TEMAS (Tell-Me-A-Story), and the Rotter Incomplete Sentence Blank (RISB). If an evaluator scores the Rorschach using the Exner scoring system, the test is considered a valid and reliable measure. However, the validity of the other projective tests is questionable, and the results are often not usable for court cases (Goldstein, n.d.).

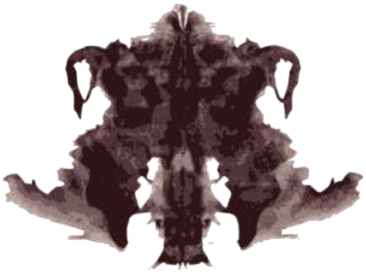


Figure **1.16.3**: An example of the images shown to those taking the Rorschach Inkblot Test.

The **Rorschach Inkblot Test** was developed in 1921 by a Swiss psychologist named Hermann Rorschach (pronounced “ROAR-shock”). It is a series of symmetrical inkblot cards that are presented to a client by a psychologist. Upon presentation of each card, the psychologist asks the client, “What might this be?” What the test-taker sees reveals unconscious feelings and struggles (Piotrowski, 1987; Weiner, 2003). The Rorschach has been standardized using the Exner system and is effective in measuring depression, psychosis, and anxiety.



Figure 1.16.4: This image from the (TAT) can be used in counseling settings.

A second projective test is the **Thematic Apperception Test (TAT)**, created in the 1930s by Henry Murray, an American psychologist, and a psychoanalyst named Christiana Morgan. A person taking the Thematic Apperception Test (TAT) is shown eight to 12 ambiguous pictures and is asked to tell a story about each picture. The stories give insight into their social world, revealing hopes, fears, interests, and goals. The storytelling format helps to lower a person’s resistance divulging unconscious personal details (Cramer, 2004). The Thematic Apperception Test (TAT) has been used in clinical settings to evaluate psychological disorders; more recently, it has been used in counseling settings to help clients gain a better understanding of themselves and achieve personal growth. Standardization of test administration is virtually nonexistent among clinicians, and the test tends to be modest to low on validity and reliability (Aronow, Weiss, & Rezinkoff, 2001; Lilienfeld, Wood, & Garb, 2000).

A third projective test is the **Rotter Incomplete Sentence Blank (RISB)** developed by Julian Rotter in 1950. There are three forms of this test for use with different age groups: the school form, the college form, and the adult form. The tests include 40 incomplete sentences that people are asked to complete as quickly as possible. The average time for completing the test is approximately 20 minutes, as responses are only one or two words in length. This test is similar to a word association test, and like other types of projective tests, it is presumed that responses will reveal desires, fears, and struggles. The Rotter Incomplete Sentence Blank (RISB) is used in screening college students for adjustment problems and in career counseling (Holaday, Smith, & Sherry, 2010; Rotter & Rafferty 1950).

1. I feel...	
2. I regret...	
3. At home...	
4. My mother...	
5. My greatest worry...	

Figure 1.16.5: These incomplete sentences resemble the types of questions on the RISB. How would you complete these sentences?

For many decades, these traditional projective tests have been used in cross-cultural personality assessments. However, it was found that test bias limited their usefulness (Hoy-Watkins & Jenkins-Moore, 2008). It is difficult to assess the personalities and lifestyles of members of widely divergent ethnic/cultural groups using personality instruments based on data from a single culture or race (Hoy-Watkins & Jenkins-Moore, 2008). To address this need, Robert Williams developed the first culturally specific projective test designed to reflect the everyday life experiences of African Americans (Hoy-Watkins & Jenkins-Moore, 2008). The updated version of the instrument is the **Contemporized-Themes Concerning Blacks Test (C-TCB)** (Williams, 1972). The Contemporized-Themes Concerning Blacks Test (C-TCB) contains 20 color images that show scenes of African-American lifestyles. When the C-TCB was compared with the TAT for African Americans, it was found that use of the Contemporized-

Themes Concerning Blacks Test (C-TCB) led to increased story length, higher degrees of positive feelings, and stronger identification with the C-TCB (Hoy, 1997; Hoy-Watkins & Jenkins-Moore, 2008).

The **TEMAS Multicultural Thematic Apperception Test (TEMAS)** is another tool designed to be culturally relevant to minority groups, especially Hispanic youths. TEMAS—standing for "Tell Me a Story" but also a play on the Spanish word *temas* (themes)—uses images and storytelling cues that relate to minority culture (Constantino, 1982).

Watch It

Watch this CrashCourse video to better understand how personality is measured:



You can view the transcript for "Measuring Personality: Crash Course Psychology #22" here (opens in new window).

Glossary

Contemporized-Themes Concerning Blacks Test (C-TCB): projective test designed to be culturally relevant to African Americans, using images that relate to African-American culture

Minnesota Multiphasic Personality Inventory (MMPI): personality test comprising a series of true/false questions in order to establish a clinical profile of an individual

projective test: personality assessment in which a person responds to ambiguous stimuli, revealing hidden feelings, impulses, and desires

Rorschach Inkblot Test: projective test that employs a series of symmetrical inkblot cards that are presented to a client by a psychologist in an effort to reveal the person's unconscious desires, fears, and struggles

Rotter Incomplete Sentence Blank (RISB): projective test that is similar to a word association test in which a person completes sentences in order to reveal their unconscious desires, fears, and struggles

self-report inventory: standardized questions with fixed response categories that the test-taker completes independently.

TEMAS Multicultural Thematic Apperception Test: projective test designed to be culturally relevant to minority groups, especially Hispanic youths, using images and storytelling that relate to minority culture

Thematic Apperception Test (TAT): projective test in which people are presented with ambiguous images, and they then make up stories to go with the images in an effort to uncover their unconscious desires, fears, and struggles

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1.17: Cognitive and Behavioral Assessments

Learning Objectives

- Describe how cognitive and behavioral assessments are used to inform psychotherapy

Cognitive Assessment



Figure **1.17.1**: Cognitive assessments are most commonly used as the first type of test to determine if there is a possible neurocognitive deficit, such as a mild neurocognitive disorder (formerly called dementia) or Alzheimer's disease.

Cognitive assessments are useful to test for cognitive or neurological impairments—deficiencies in knowledge, thought process, or judgment. Psychiatrists often perform cognitive testing during the mental status exam. However, when cognitive impairment is suspected, the cognitive assessment can obtain a more detailed analysis by surveying the neuropsychological domains. This detailed investigation of cognition can diagnose major cognitive impairment (i.e., dementia) and mild cognitive impairment, evaluate traumatic brain injuries, help determine decision-making capacity, and survey intellectual dysfunction.

There are many established tools used to conduct cognitive assessments. Each is carefully constructed to evaluate neuropsychological domains such as memory, language, executive function, abstract reasoning, attention, and visuospatial skills. Available assessment tools range from those designed to evaluate a single neuropsychological domain, to mental status screens that survey multiple neuropsychological domains, to the most extensive test—a complete neuropsychological exam that assesses each neuropsychological domain. When a cognitive deficit is hypothesized, it is common to use other assessment measures as well, such as neuroimaging like CT scans or fMRI.

Most clinicians will use an established mental status screening tool such as the Mini-Mental Status Exam (MMSE) or Montreal Cognitive Assessment (MoCA) to determine if cognitive impairment is present. Mental status screens are short, efficient, and well-researched modalities designed to evaluate multiple cognitive domains. A cognitive assessment, along with a good history, physical exam, and appropriate labs and imaging, can establish a diagnosis or decide if further evaluation is necessary.

If a screening test is inconclusive or more information is required, a complete neuropsychological evaluation is an option. A full neuropsychological evaluation would ideally identify the patient's specific deficits, differentiate between neurological and psychological etiologies, differentiate between Alzheimer's dementia and other dementias, localize the deficits, and help formulate a personalized management plan. The exam is non-invasive and involves a battery of assessments performed by a trained professional. This comprehensive evaluation can take up to a full day to complete. While a full neuropsychological evaluation is the most detailed assessment, it is unnecessary for all patients who have a diagnosis or suspicion of cognitive impairment. However, it can serve as a helpful resource if there are questions or concerns about diagnosis or care.

How to Use

When performing a cognitive assessment, the clinician must take a good patient history and perform a physical exam; this ensures that the patients receive a thorough evaluation while strengthening the caregiver-patient relationship. If the assumption is that the patient has cognitive impairment before considering other diagnoses, the patient may feel that the clinician has dismissed them due to their age, level of education, or other reasons. A thorough examination can also help identify any behavior or personality disorders potentially contributing to the patient's chief complaints, as mild cognitive impairments or dementia often coexist with behavioral and personality disturbances. Cognitively impaired patients cannot express themselves fully, so it is very beneficial to have someone who has a close relationship with the patient present to help establish baseline levels of functioning.

Before deciding upon a particular testing modality, one should compare all the available tests to find the test that is best suited for both the administrator and the patient. One should be mindful that some institutions may have a preferred testing modality.

A variety of cognitive assessment screens exist, and each has instructions, templates (if applicable), and often its own website. Below is a shortlist of some of the more popular screening tools used and their relative strengths and weaknesses.

Mini-Mental State Exam (MMSE)

The Mini-Mental Status Exam (MMSE) usually takes less than ten minutes to administer, is easy to use, and has been researched thoroughly since 1975. However, what was once the gold standard in cognitive assessments, the MMSE is now used less frequently due to copyright laws and additional costs.

Montreal Cognitive Assessment (MoCA)

The Montreal Cognitive Assessment (MoCA) is another popular screening tool that takes approximately ten minutes to complete. It evaluates visuospatial skills, attention, language, abstract reasoning, delayed recall, executive function, and orientation. The MoCA covers more domains than the MMSE and, as a consequence, has greater sensitivity and specificity. The associated website includes specific adaptations for different populations, many different languages, printable versions of the test, and training opportunities.

Mini-Cog

The Mini-Cog is one of the faster cognitive assessment screens used. It consists of two parts: a three-item recall and a clock-drawing test. The delayed three-item recall tests memory while the clock drawing test evaluates cognitive function, language, executive function, and visuospatial skills. The Mini-Cog website also gives detailed instructions for administrators.

Saint Louis University Mental Status Exam (SLUMS)

Initially developed for the veteran population, Saint Louis University Mental Status Exam (SLUMS) is another tool with an online, printable form for testing. Their website has an instructional outline for administrators, training opportunities, and a wide range of language options from which to choose.

Other modalities include but are not limited to the Blessed Orientation-Memory-Concentration Test, Kokmen Short Test of Mental Status, Memory Impairment Screen, Ottawa 3DY, Brief Alzheimer's Screen, Caregiver-completed AD8, and many other dementia-screening scales.

The results of these assessments require review in the context of each patient. Each administrator should remember that a screening test is not a substitute for a diagnostic workup. Lastly, it bears mentioning that no current data support the use of cognitive assessments in asymptomatic patients.

Neuropsychological Domains

Cognitive assessments evaluate for cognitive impairment by assessing the neuropsychological domains. A brief explanation of the frequently tested domains follows.

Language

The language domain involves naming, reading, writing, and repeating words. Some practitioners will evaluate the language by noting the patient's communication skills throughout the interview. There are many ways to test for language. Two neurocognitive tests include the Boston Naming Test and the Controlled Oral Word Association. It should be noted that there is a part of the language domain that can become mildly impaired with normal aging. Expressive aphasia, which is the inability to find words, can become impaired with normal aging.

Executive Function

This assessment encompasses organizing, planning, working memory, mental flexibility, list-making, and executing tasks. An example of executive function impairment might be a patient who cannot follow recipes or cook as well as they used to. Often, executive function is tested by naming as many categorical items as possible; for example, name as many animals as possible in one minute. Other neuropsychological tests include the Trail Making Tests A and B and the Wisconsin Sorting Test.

Abstract Reasoning

Abstract reasoning refers to analyzing information, detecting patterns and relationships, or solving problems on an intangible and theoretical level. An example of abstract reasoning skills would be the ability to identify patterns and/or relationships between things that do not appear to be similar. Another example would be the ability to solve problems without the knowledge that it would normally take to solve them. Abstract reasoning is often tested by having the patient describe similes, analogies, proverbs, or sayings. For example, the relationship between an airplane and a bicycle is that they are both modes of transportation. Some neuropsychological abstract reasoning tests include the Shipley-2 Abstract Test, Gorham's Proverbs Test, Conceptual Level Analogy Test, and Verbal Concept Attainment Test.

Memory

Memory is the mechanism that takes information and then encodes, stores, and retrieves it for later use. Different kinds of memory make this domain very complicated. Memory divides into short-term and long-term memory. Short-term memory is capable of taking small pieces of information and utilizing it for a brief period. Long-term memory subdivides into procedural and declarative, which further divides into episodic and semantic. Procedural memory is the storing of information used to perform or complete tasks that are done often, like driving a car. Declarative memory is the storing and recall of facts and events such as a family member's birthday. Episodic memory is contextual information, storing or remembering things from a specific experience. An example of episodic memory is the patient remembering what he or she did for his or her last birthday. Semantic memory is more general knowledge or factual based memory and would include learned subjects such as math.

Because memory is so complex, it is essential to recognize and document what exactly is under evaluation during this part of the assessment. Memory impairment can be easy to pinpoint from the patient's history, but it can also masquerade as other things, such as having trouble learning new information. It is also worth noting that normal aging can slightly impair memory. A normal aging patient's activities of daily living will remain intact.

Attention/Concentration

Testing for attention and concentration often take place together. They are frequently tested by spelling words backward and/or serially subtracting numbers from a large starting point, such as the MoCA, where the examiner asks the patient to subtract seven from 100 in five increments. Some clinicians observe the patient and assess their level of attention throughout the interview. An example of a neuropsychological test that acknowledges attention and concentration is the Connors Continuous Performance Test.

Visuospatial Skills

This concept is a person's ability to conceptualize and manipulate two- and three-dimensional objects. Testing often consists of copying figures, block designs, or clock drawings. This skill set may be difficult to assess while taking a history, but could present as a patient suddenly having difficulty with parallel parking a car or getting into small accidents. In neuropsychology, an example of a test used for these skills is the Rey-Osterrieth Complex Figure Copy Test.

Behavioral Assessment

Behavioral assessment involves the identification and measurement of particular behaviors and the variables affecting their occurrence. In educational, clinical, and organizational settings, accurate assessment is key to developing effective programs of behavior modification or behavior therapy.

Direct assessment involves observing and recording situational factors surrounding a problem behavior (e.g., antecedent and consequent events). So, for example, a member of an IEP team may observe disruptive behavior in the setting that it is likely to occur, and record the data. If a child is disruptive or acting out in school, a psychologist may want to sit in the classroom to observe their behavior or have a recording of their classroom behavior. With this direct observation, they can identify and record specific behaviors and use techniques to code specific things (such as the number of times the child gets up from their seat).

The observer may use a matrix or scatter plot to chart the relationship between specific instructional variables and student responses. These techniques also will be useful in identifying possible environmental factors (e.g., seating arrangements), activities (e.g., independent work), or temporal factors (e.g., mornings) that may influence the behavior. These tools can be developed specifically to address the type of variable in question, and can be customized to analyze specific behaviors and situations (e.g., increments of five minutes, 30 minutes, one hour, or even a few days). Regardless of the tool, observations that occur consistently

across time and situations, and that reflect both quantitative and qualitative measures of the behavior in question, are recommended.

Self-monitoring is also used as a behavioral assessment tool. For example, a person suffering from PTSD may be asked to monitor and record the frequency and circumstances of their panic attacks or feelings of anxiety.

Behavioral rating scales can also be used as a way to document behavior. These are surveys either filled out by the individual or another observer, such as a parent or caretaker. For example, the **Child Behavior Checklist** (CBCL) is a widely used caregiver report form identifying problem behavior in children. It is a component in the Achenbach System of Empirically Based Assessment developed by Thomas M. Achenbach. The school-age version of the Child Behavior Checklist (CBCL) (CBCL/6-18) instructs a respondent who knows the child well (usually a parent or other close caregiver) to report on the child's problems. Alternative measures are available for teachers (the Teacher's Report Form) and the child (the Youth Self Report, for youths age 11 to 18 years). The school-age checklist contains 118 problem behavior questions.

The main scoring for the Child Behavior Checklist (CBCL) is based on statistical groupings of sets of behaviors that typically occur together. The original scale used principal components analysis to group the items, and more recent research has used confirmatory factor analysis to test the structure. Similar questions are grouped into a number of *syndrome scale scores*, and their scores are summed to produce a raw score for that syndrome. The eight empirically-based syndrome scales are listed below:

1. Aggressive Behavior
2. Anxious/Depressed
3. Attention Problems
4. Rule-Breaking Behavior
5. Somatic Complaints
6. Social Problems
7. Thought Problems
8. Withdrawn/Depressed

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1.18: Putting It Together- Psychological Disorders

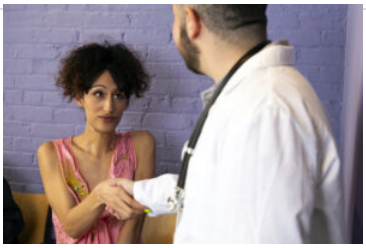


Figure **1.18.1**: Don't be afraid to ask for help if you think you need it. Psychologists, counselors, and doctors are trained to assist you.

This chapter covered a lot of ground and gave you a foundation for understanding psychological disorders. In this module, you learned about how mental disorders are defined and classified using the *DSM-5*, and how psychologists, psychiatrists and other mental health professionals identify mental illnesses. You also learned about mental health treatment in the past compared to treatment today, and how mental health professionals use diagnostic tools to assess mental disorders.

One tendency while reading through this material is for students to self-diagnose, as it is easy to identify with various descriptions of mental disorders. Be wary of self-diagnosis as you may see symptoms that aren't there or miss symptoms or signs of something more serious. If you do think you may be suffering from a mental illness (defined as significant disturbances in thoughts, feelings, and behaviors causing impairment in one's life), then please seek for help from a counselor, doctor, or other trained professional. Hopefully, your reading from this module has also helped you to better understand mental illness and be less inclined to judge or stigmatize those who are suffering from or managing an illness.

Watch It

Watch this video from architect and graphic designer Federico Babina to see his interpretation of various mental disorders listed in the *DSM-5* that we will examine in this course. How might you change or alter his depictions? What would you add to this list? Keep this artistic representation in mind as you work through the course content and consider how *you* might visually or artistically represent the experience of a mental disorder.



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CHAPTER OVERVIEW

2: Research and Ethics in Abnormal Psychology

A scientific approach is crucial for studying mental disorders. This section covers research methodologies, ethical considerations, and legal implications in clinical practice. Students learn about experimental and correlational research, epidemiological studies, and ethical standards that govern psychological research and treatment.

2.1: Why It Matters- Psychological Research
2.2: Introduction to Research
2.3: The Scientific Process
2.4: Descriptive Research and Case Studies
2.5: Descriptive and Epidemiological Research
2.6: Correlational Research
2.7: Experiments
2.8: Understanding Research
2.9: Introduction to Ethics in Abnormal Psychology
2.10: Ethics in Research
2.11: Standards and Competencies in Psychology
2.12: Ethical Standards in Clinical Psychology
2.13: Legal and Forensic Issues in Abnormal Psychology
2.14: Putting It Together- Psychological Research

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2.1: Why It Matters- Psychological Research

Why learn about research and ethics in abnormal psychology?

How do you know what you know? And how do you know that what you believe to be true is really true? Many of us might just say that we know what we know because of what we have experienced in life. People are naturally inquisitive, so they often come up with questions about things they see or hear, and they often develop ideas or hypotheses about why things are the way they are. Sometimes we just have a gut feeling about certain things based on phenomena we have observed. However, if we are to be objective, one way of finding out if we are accurate in our beliefs is to conduct research. Consider the types of headlines that you might read in the popular media:

- Diet of fish 'can prevent' teen violence[1]
- Social isolation may have a negative effect on intellectual abilities[2]

Each of these statements is biased in one way or another. Both contain a direct claim that the writer wishes the audience to accept; however, there is some problem with design, methodology, or analysis making it possible to account for the findings by considering other variables or even a rival hypothesis. It is possible the research findings were reported incorrectly. Statements like the ones above should only be made if there were empirical research studies to back up the results or findings. Results from the studies need to be conveyed in an objective manner. Furthermore, only a specific type of experimental research design should be used before making these statements.

This issue is important in abnormal psychology. Is the will to live a consequence or predictor of depression? [3] We may think we know the answer based on our life experiences or based on our clinical practice, but w hat we think we know about mental health and where mental disorder comes from should be verified and clarified in rigorous research studies.

In their research, psychologists use case studies, naturalistic observation, and surveys to describe behavior. They use correlation to assess the relationship between variables and use experimentation to uncover cause-effect relationships. Researchers use statistics to describe their data, assess relationships between variables, and determine whether differences are significant.

Watch It

Watch the CrashCourse video to review the main types of research studies that you learn about in this module, then read through the following examples and see if you can come up with your own design for each type of study.



You can view the transcript for "Psychological Research: Crash Course Psychology #2" here (opens in new window).

1. Hinsliff, G. (2003, September 14). Diet of fish 'can prevent' teen violence. *The Observer*. Retrieved from www. theguardian.com/politics/2003/sep/14/science.health. ↵
2. Social isolation may have a negative effect on intellectual abilities. (2007, October 30). *Medical News Today*. Retrieved from http://www.medicalnewstoday.com/ releases/87087.php. ↵

3. Carmel S, Tovel H, Raveis VH, O'Rourke N. Is a Decline in Will to Live a Consequence or Predictor of Depression in Late Life? J Am Geriatr Soc. 2018 Jul;66(7):1290–1295. doi: 10.1111/jgs.15394. Epub 2018 Apr 20. PMID: 29676436. ↵

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2.2: Introduction to Research

What you'll learn to do: examine how descriptive, correlational, and experimental research is used to study abnormal behavior

As you learned in the previous module, the scientific approach led to major advances in understanding abnormal behavior and treating mental disorders. The essence of the scientific method is objectivity. It expresses the idea that the claims, methods, and results of science are not, or should not be, influenced by particular perspectives, value commitments, community bias, or personal interests, to name a few relevant factors. In addition, researchers must always be open to alternative explanations that could account for their findings. Many researchers have a personal interest in what they are studying and they become involved in the pursuit of knowledge in areas that relate to experiences in their own lives, particularly in the field of abnormal psychology. Clinical psychologists may wonder whether a particular kind of experience led to an individual's symptoms, whether a certain treatment will be effective to treat the symptoms of a disorder, or they may speculate about the role of genetic predispositions. In either case, when conducting research, however, they do not let their personal biases get in the way of collecting the data or interpreting the findings. The ideal approach to answering these questions involves a progression through a set of steps in which psychological researchers propose a hypothesis, conduct a study, and collect and analyze the data.



In this section, we will take a closer look at how to examine research and the main types of studies used: descriptive, experimental, and correlational. Descriptive, or qualitative, methods include the case study, naturalistic observation, surveys, epidemiological research, archival research, longitudinal research, and cross-sectional research.

When scientists passively observe and measure phenomena, it is called correlational research. Here, psychologists do not intervene and change behavior as they do in experiments. In correlational research, they identify patterns of relationships, but usually cannot infer what causes what. Importantly, with correlational research, you can examine only two variables at a time, no more and no less.

Experiments are conducted in order to determine cause-and-effect relationships. In ideal experimental design, the only difference between the experimental and control groups is whether participants are exposed to the experimental manipulation. Each group goes through all phases of the experiment, but each group will experience a different level of the independent variable: the experimental group is exposed to the experimental manipulation and the control group is not exposed to the experimental manipulation. The researcher then measures the changes that are produced in the dependent variable in each group. Once data is collected from both groups, it is analyzed statistically to determine if there are meaningful differences between the groups. Eventually, results are communicated through publication in scientific journals.

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2.3: The Scientific Process

Learning Objectives

- Explain how scientific research and the scientific method are used to address questions about behavior

The goal of all scientists is to better understand the world around them. Psychologists focus their attention on understanding behavior, as well as the cognitive (mental) and physiological (body) processes that underlie behavior. In contrast to other methods that people use to understand the behavior of others, such as intuition and personal experience, the hallmark of scientific research is that there is evidence to support a claim. Scientific knowledge is **empirical**: It is grounded in objective, tangible evidence that can be observed time and time again, regardless of who is observing.

While behavior is observable, the mind is not. If someone is crying, we can see the behavior. However, the reason for the behavior is more difficult to determine. Is the person crying due to being sad, in pain, or happy? Or is he or she suffering from depression? Sometimes we can learn the reason for someone's behavior by simply asking a question like, "Why are you crying?" However, there are situations in which an individual is either uncomfortable or unwilling to answer the question honestly, or is incapable of answering. For example, a depression rating scale may be used to assign a score to a person's behavior where that score can determine whether that person should be evaluated more thoroughly for a depressive disorder. In some circumstances, the psychologist may have to be creative in finding ways to better understand behavior. This module explores how scientific knowledge is generated, and how important that knowledge is in forming decisions in our personal lives and in the public domain.

Process of Scientific Research

Scientific knowledge is advanced through a process known as the scientific method. Basically, ideas (in the form of theories and hypotheses) are tested against the real world (in the form of empirical observations), and those empirical observations lead to more ideas that are tested against the real world, and so on.

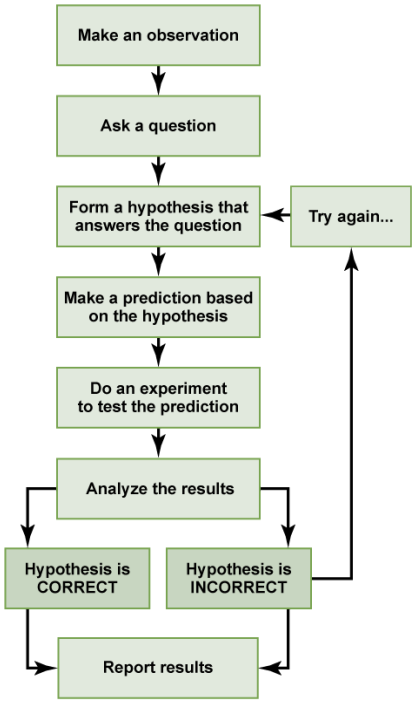


Figure **2.3.1**: The scientific method is a process for gathering data and processing information. It provides well-defined steps to standardize how scientific knowledge is gathered through a logical, rational, problem-solving method.

The basic steps in the scientific method are outlined below:

- Observe a natural phenomenon and define a question about it.
- Make a hypothesis, or potential solution to the question.

- Test the hypothesis.
- If the hypothesis is true, find more evidence or find counter-evidence.
- If the hypothesis is false, create a new hypothesis or try again.
- Draw conclusions and repeat—the scientific method is never-ending and no result is ever considered perfect.

In order to ask an important question that may improve our understanding of the world, a researcher must first observe natural phenomena. By making observations, a researcher can define a useful question. After finding a question to answer, the researcher can then make a prediction (a hypothesis) about what he or she thinks the answer will be. The hypothesis is usually a statement about the relationship between two or more variables. After making a hypothesis, the researcher will then design an experiment to test his or her hypothesis and evaluate the data gathered. This data will either support or refute the hypothesis. Based on the conclusions drawn from the data, the researcher will then find more evidence to support the hypothesis, look for counter-evidence to further strengthen the hypothesis, revise the hypothesis and create a new experiment, or continue to incorporate the information gathered to answer the research question.

Basic Principles of the Scientific Method

Two key concepts in the scientific approach are theory and hypothesis. A **theory** is a well-developed set of ideas that proposes an explanation for observed phenomena that can be used to make predictions about future observations. A **hypothesis** is a testable prediction that is arrived at logically from a theory. It is often worded as an if-then statement (e.g., if I study all night, then I will get a passing grade on the test). The hypothesis is extremely important because it bridges the gap between the realm of ideas and the real world. As specific hypotheses are tested, theories are modified and refined to reflect and incorporate the result of these tests.

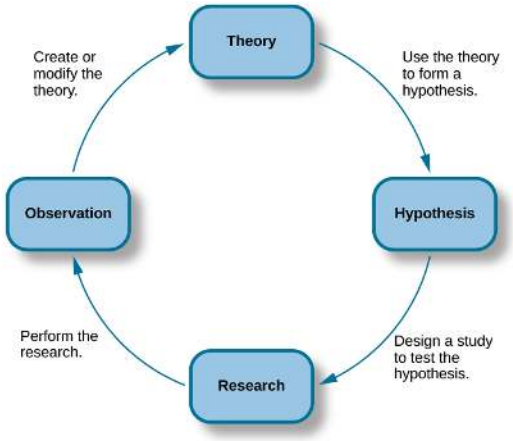


Figure **2.3.2**: The scientific method of research includes proposing hypotheses, conducting research, and creating or modifying theories based on results.

Other key components in following the scientific method include verifiability, predictability, falsifiability, and fairness. **Verifiability** means that an experiment must be replicable by another researcher. To achieve verifiability, researchers must make sure to document their methods and clearly explain how their experiment is structured and why it produces certain results.

Predictability in a scientific theory implies that the theory should enable us to make predictions about future events. The precision of these predictions is a measure of the strength of the theory.

Falsifiability refers to whether a hypothesis can be disproved. For a hypothesis to be falsifiable, it must be logically possible to make an observation or do a physical experiment that would show that there is no support for the hypothesis. Even when a hypothesis cannot be shown to be false, that does not necessarily mean it is not valid. Future testing may disprove the hypothesis. This does not mean that a hypothesis *has* to be shown to be false, just that it can be tested.

To determine whether a hypothesis is supported or not supported, psychological researchers must conduct hypothesis testing using statistics. Hypothesis testing is a type of statistics that determines the probability of a hypothesis being true or false. If hypothesis testing reveals that results were statistically significant, this means that there was support for the hypothesis and that the researchers

can be reasonably confident that their result was not due to random chance. If the results are not statistically significant, this means that the researchers' hypothesis was not supported.

Fairness implies that all data must be considered when evaluating a hypothesis. A researcher cannot pick and choose what data to keep and what to discard or focus specifically on data that support or do not support a particular hypothesis. All data must be accounted for, even if they invalidate the hypothesis.

Link to Learning

Want to participate in a study? Visit this Psychological Research on the Net website and click on a link that sounds interesting to you in order to participate in online research.

Why the Scientific Method Is Important for Psychology

The use of the scientific method is one of the main features that separates modern psychology from earlier philosophical inquiries about the mind. Compared to chemistry, physics, and other natural sciences, psychology has long been considered one of the social sciences because of the subjective nature of the things it seeks to study. Behavioral and social science research helps predict, prevent, and manage mental illness—in individuals and in whole populations. This research also helps people change their behaviors, understand, and adhere to treatments. Psychologists and other scientists use the *scientific method* to advance the description, explanation, prediction, and control of abnormal behavior.

Many of the constructs that psychologists are interested in—such as depression and anxiety—are subjective and cannot be directly measured. Major mental disorders like depression and anxiety affect about one in four American adults in a given year. Research into the causes and effective treatments are essential. In the course of treating a patient, a psychologist will take records of problems, insights, and techniques that were important in the patient's treatment, and they often rely on behavioral observations and self-reported data, which are considered by some to be illegitimate or lacking in methodological rigor. Applying the scientific method to psychology therefore helps to standardize the approach to investigating complex human behaviors, thus allowing psychologists to create evidence-based strategies that solve problems and improve lives.

The scientific method allows psychological data to be to collected and used to interpret in order to better understand and solve some of society's most challenging problems. Research data can be replicated and confirmed in many instances, under different circumstances, and by a variety of researchers. Through replication of experiments, new generations of psychologists can reduce errors and broaden the applicability of theories. It also allows theories to be tested and validated instead of simply being conjectures that could never be verified or falsified. Following the scientific method allows psychologists to gain a stronger understanding of how the human mind works.

Glossary

empirical: grounded in objective, tangible evidence that can be observed time and time again, regardless of who is observing

fairness: implies that all data must be considered when evaluating a hypothesis

falsifiable: able to be disproven by experimental results

hypothesis: (plural: hypotheses) tentative and testable statement about the relationship between two or more variables

predictability: implies that a theory should enable us to make predictions about future events

theory: well-developed set of ideas that proposes an explanation for observed phenomena

verifiability: an experiment must be replicable by another researcher

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2.4: Descriptive Research and Case Studies

Learning Objectives

- Explain the importance and uses of descriptive research, especially case studies, in studying abnormal behavior

Types of Research Methods

There are many research methods available to psychologists in their efforts to understand, describe, and explain behavior and the cognitive and biological processes that underlie it. Some methods rely on observational techniques. Other approaches involve interactions between the researcher and the individuals who are being studied—ranging from a series of simple questions; to extensive, in-depth interviews; to well-controlled experiments.

The three main categories of psychological research are descriptive, correlational, and experimental research. Research studies that do not test specific relationships between variables are called **descriptive, or qualitative, studies**. These studies are used to describe general or specific behaviors and attributes that are observed and measured. In the early stages of research, it might be difficult to form a hypothesis, especially when there is not any existing literature in the area. In these situations designing an experiment would be premature, as the question of interest is not yet clearly defined as a hypothesis. Often a researcher will begin with a non-experimental approach, such as a descriptive study, to gather more information about the topic before designing an experiment or correlational study to address a specific hypothesis. Descriptive research is distinct from **correlational research**, in which psychologists formally test whether a relationship exists between two or more variables. **Experimental research** goes a step further beyond descriptive and correlational research and randomly assigns people to different conditions, using hypothesis testing to make inferences about how these conditions affect behavior. It aims to determine if one variable directly impacts and causes another. Correlational and experimental research both typically use hypothesis testing, whereas descriptive research does not.

Each of these research methods has unique strengths and weaknesses, and each method may only be appropriate for certain types of research questions. For example, studies that rely primarily on observation produce incredible amounts of information, but the ability to apply this information to the larger population is somewhat limited because of small sample sizes. Survey research, on the other hand, allows researchers to easily collect data from relatively large samples. While surveys allow results to be generalized to the larger population more easily, the information that can be collected on any given survey is somewhat limited and subject to problems associated with any type of self-reported data. Some researchers conduct archival research by using existing records. While existing records can be a fairly inexpensive way to collect data that can provide insight into a number of research questions, researchers using this approach have no control on how or what kind of data was collected.

Correlational research can find a relationship between two variables, but the only way a researcher can claim that the relationship between the variables is cause and effect is to perform an experiment. In experimental research, which will be discussed later, there is a tremendous amount of control over variables of interest. While performing an experiment is a powerful approach, experiments are often conducted in very artificial settings, which calls into question the validity of experimental findings with regard to how they would apply in real-world settings. In addition, many of the questions that psychologists would like to answer cannot be pursued through experimental research because of ethical concerns.

The three main types of descriptive studies are case studies, naturalistic observation, and surveys.

Clinical or Case Studies

Psychologists can use a detailed description of one person or a small group based on careful observation. **Case studies** are intensive studies of individuals and have commonly been seen as a fruitful way to come up with hypotheses and generate theories. Case studies add descriptive richness. Case studies are also useful for formulating concepts, which are an important aspect of theory construction. Through fine-grained knowledge and description, case studies can fully specify the causal mechanisms in a way that may be harder in a large study.

Sigmund Freud developed many theories from case studies (Anna O., Little Hans, Wolf Man, Dora, etc.). For example, he conducted a case study of a man, nicknamed “Rat Man,” in which he claimed that this patient had been cured by psychoanalysis.

The nickname derives from the fact that among the patient's many compulsions, he had an obsession with nightmarish fantasies about rats.

Today, more commonly, case studies reflect an up-close, in-depth, and detailed examination of an individual's course of treatment. Case studies typically include a complete history of the subject's background and response to treatment. From the particular client's experience in therapy, the therapist's goal is to provide information that may help other therapists who treat similar clients.

Case studies are generally a single-case design, but can also be a multiple-case design, where replication instead of sampling is the criterion for inclusion. Like other research methodologies within psychology, the case study must produce valid and reliable results in order to be useful for the development of future research. Distinct advantages and disadvantages are associated with the case study in psychology.

A commonly described limit of case studies is that they do not lend themselves to **generalizability**. The other issue is that the case study is subject to the bias of the researcher in terms of how the case is written, and that cases are chosen because they are consistent with the researcher's preconceived notions, resulting in biased research. Another common problem in case study research is that of reconciling conflicting interpretations of the same case history.

Despite these limitations, there are advantages to using case studies. One major advantage of the case study in psychology is the potential for the development of novel hypotheses of the cause of abnormal behavior for later testing. Second, the case study can provide detailed descriptions of specific and rare cases and help us study unusual conditions that occur too infrequently to study with large sample sizes. The major disadvantage is that case studies cannot be used to determine causation, as is the case in experimental research, where the factors or variables hypothesized to play a causal role are manipulated or controlled by the researcher.

Single-Case Experimental Designs

The lack of control available in the traditional case study research strategy led researchers to develop more sophisticated methods, such as single-subject research, which provides the statistical framework for making inferences from quantitative case-study data.



Figure **2.4.1**: Antipsychotics are the treatment of choice in managing schizophrenia and other psychotic disorders. Several major trials have been conducted examining the clinical difference between typical antipsychotics and atypical antipsychotics and how the selection may affect the quality of life.

The **single-case experimental design** (sometimes called *single-participant research designs*), is particularly useful for studies of treatment effectiveness. In single-case experimental designs**,** the same research participant serves as the subject in both the experimental and control conditions. One of the most common forms of the single-case experimental design is the **A-B-A-B design,** or reversal design**,** reflecting the alternation between conditions, or phases A and B. The AB design is a two-part or phase design composed of a baseline ("A" phase) with no changes, and a treatment or intervention ("B") phase. If there is a change, then the treatment may be said to have had an effect. However, it is subject to many possible competing hypotheses, making strong conclusions difficult. The A-B-A-B design, or reversal design, is a variant on the AB design. It introduces ways to control for the competing hypotheses and allows for stronger conclusions. The reversal design (ABAB) is the most powerful of the single-subject research designs because it shows a strong reversal from baseline ("A") to treatment ("B") and back again. In an ABAB design, researchers observe behaviors in the "A" phase, institute treatment in the "B" phase, and then repeat the process. If the variable returns to baseline measure without treatment and then resumes its effects when reapplied, the researcher can have greater confidence in the efficacy of that treatment. However, many interventions cannot be reversed for ethical reasons (e.g., involving self-injurious behavior like smoking). It may be unethical to end an experiment on a baseline measure if the treatment is self-sustaining and highly beneficial and/or related to health. Control condition participants may also deserve the benefits of research once all data has been collected. It is a researcher's ethical duty to maximize benefits and to ensure that all participants have access to those benefits when possible.

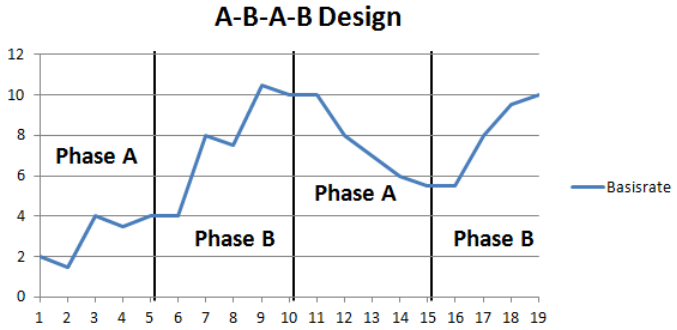


Figure **2.4.2**: The investigator looks for evidence that the change in the observed behavior occurred coincident with treatment. If the problem behavior declines whenever treatment is introduced (during the first and second treatment phases) but returns (is "reversed") to baseline levels during the reversal phase, the experimenter can be reasonably confident the treatment had the intended effect.

Link to Learning: Famous Case Studies

Some well-known case studies that related to abnormal psychology include the following:

- Harlow—Phineas Gage
- Breuer & Freud (1895)—Anna O.
- Cleckley's case studies: on psychopathy (The Mask of Sanity) (1941) and multiple personality disorder (The Three Faces of Eve) (1957)
- Freud and Little Hans
- Freud and the Rat Man
- John Money and the John/Joan case
- Genie (feral child)
- Piaget's studies
- Rosenthal's book on the murder of Kitty Genovese
- Washoe (sign language)
- Patient H.M.

Naturalistic Observation

If you want to understand how behavior occurs, one of the best ways to gain information is to simply observe the behavior in its natural context. However, people might change their behavior in unexpected ways if they know they are being observed. How do researchers obtain accurate information when people tend to hide their natural behavior? As an example, imagine that your professor asks everyone in your class to raise their hand if they always wash their hands after using the restroom. Chances are that almost everyone in the classroom will raise their hand, but do you think hand washing after every trip to the restroom is really that universal?

This is very similar to the phenomenon mentioned earlier in this module: many individuals do not feel comfortable answering a question honestly. But if we are committed to finding out the facts about handwashing, we have other options available to us.

Suppose we send a researcher to a school playground to observe how aggressive or socially anxious children interact with peers. Will our observer blend into the playground environment by wearing a white lab coat, sitting with a clipboard, and staring at the swings? We want our researcher to be inconspicuous and unobtrusively positioned—perhaps pretending to be a school monitor while secretly recording the relevant information. This type of observational study is called **naturalistic observation**: observing behavior in its natural setting. To better understand peer exclusion, Suzanne Fanger collaborated with colleagues at the University of Texas to observe the behavior of preschool children on a playground. How did the observers remain inconspicuous over the duration of the study? They equipped a few of the children with wireless microphones (which the children quickly forgot about) and observed while taking notes from a distance. Also, the children in that particular preschool (a "laboratory preschool") were accustomed to having observers on the playground (Fanger, Frankel, & Hazen, 2012).



Figure **2.4.3**: In naturalistic observation, psychologists take their research into the streets, homes, restaurants, schools, and other settings where behavior can be directly observed.

It is critical that the observer be as unobtrusive and as inconspicuous as possible: when people know they are being watched, they are less likely to behave naturally. For example, psychologists have spent weeks observing the behavior of homeless people on the streets, in train stations, and bus terminals. They try to ensure that their naturalistic observations are unobtrusive, so as to minimize interference with the behavior they observe. Nevertheless, the presence of the observer may distort the behavior that is observed, and this must be taken into consideration (Figure 1).

The greatest benefit of naturalistic observation is the validity, or accuracy, of information collected unobtrusively in a natural setting. Having individuals behave as they normally would in a given situation means that we have a higher degree of ecological validity, or realism, than we might achieve with other research approaches. Therefore, our ability to **generalize** the findings of the research to real-world situations is enhanced. If done correctly, we need not worry about people modifying their behavior simply because they are being observed. Sometimes, people may assume that reality programs give us a glimpse into authentic human behavior. However, the principle of inconspicuous observation is violated as reality stars are followed by camera crews and are interviewed on camera for personal confessionals. Given that environment, we must doubt how natural and realistic their behaviors are.

The major downside of naturalistic observation is that they are often difficult to set up and control. Although something as simple as observation may seem like it would be a part of all research methods, participant observation is a distinct methodology that involves the researcher embedding themselves into a group in order to study its dynamics. For example, Festinger, Riecken, and Shacter (1956) were very interested in the psychology of a particular cult. However, this cult was very secretive and wouldn't grant interviews to outside members. So, in order to study these people, Festinger and his colleagues pretended to be cult members, allowing them access to the behavior and psychology of the cult. Despite this example, it should be noted that the people being observed in a participant observation study usually know that the researcher is there to study them.[1]

Another potential problem in observational research is **observer bias**. Generally, people who act as observers are closely involved in the research project and may unconsciously skew their observations to fit their research goals or expectations. To protect against this type of bias, researchers should have clear criteria established for the types of behaviors recorded and how those behaviors should be classified. In addition, researchers often compare observations of the same event by multiple observers, in order to test **inter-rater reliability**: a measure of reliability that assesses the consistency of observations by different observers.

Surveys

Often, psychologists develop surveys as a means of gathering data. **Surveys** are lists of questions to be answered by research participants, and can be delivered as paper-and-pencil questionnaires, administered electronically, or conducted verbally (Figure 3). Generally, the survey itself can be completed in a short time, and the ease of administering a survey makes it easy to collect data from a large number of people.

Surveys allow researchers to gather data from larger samples than may be afforded by other research methods**.** A **sample** is a subset of individuals selected from a **population**, which is the overall group of individuals that the researchers are interested in. Researchers study the sample and seek to generalize their findings to the population.

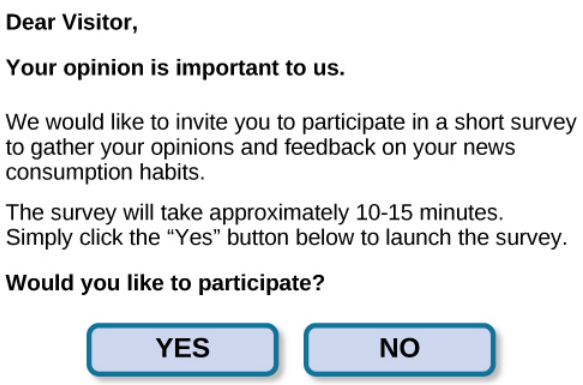


Figure **2.4.4**: Surveys can be administered in a number of ways, including electronically administered research, like the survey shown here. (credit: Robert Nyman)

There is both strength and weakness in surveys when compared to case studies. By using surveys, we can collect information from a larger sample of people. A larger sample is better able to reflect the actual diversity of the population, thus allowing better generalizability. Therefore, if our sample is sufficiently large and diverse, we can assume that the data we collect from the survey can be generalized to the larger population with more certainty than the information collected through a case study. However, given the greater number of people involved, we are not able to collect the same depth of information on each person that would be collected in a case study.

Another potential weakness of surveys is something we touched on earlier in this module: people do not always give accurate responses. They may lie, misremember, or answer questions in a way that they think makes them look good. For example, people may report drinking less alcohol than is actually the case.

Any number of research questions can be answered through the use of surveys. One real-world example is the research conducted by Jenkins, Ruppel, Kizer, Yehl, and Griffin (2012) about the backlash against the U.S. Arab-American community following the terrorist attacks of September 11, 2001. Jenkins and colleagues wanted to determine to what extent these negative attitudes toward Arab-Americans still existed nearly a decade after the attacks occurred. In one study, 140 research participants filled out a survey with 10 questions, including questions asking directly about the participant's overt prejudicial attitudes toward people of various ethnicities. The survey also asked indirect questions about how likely the participant would be to interact with a person of a given ethnicity in a variety of settings (such as, "How likely do you think it is that you would introduce yourself to a person of Arab-American descent?"). The results of the research suggested that participants were unwilling to report prejudicial attitudes toward any ethnic group. However, there were significant differences between their pattern of responses to questions about social interaction with Arab-Americans compared to other ethnic groups: they indicated less willingness for social interaction with Arab-Americans compared to the other ethnic groups. This suggested that the participants harbored subtle forms of prejudice against Arab-Americans, despite their assertions that this was not the case (Jenkins et al., 2012).

Glossary

A-B-A-B design: an experimental design in which the a person is given treatment, or experimental condition (B), to compare against the baseline (A), and this repeats in order to determine effectiveness

clinical or case study: observational research study focusing on one or a few people

correlational research: tests whether a relationship exists between two or more variables

descriptive research: research studies that do not test specific relationships between variables; they are used to describe general or specific behaviors and attributes that are observed and measured

experimental research: tests a hypothesis to determine cause-and-effect relationships

generalizability: inferring that the results for a sample apply to the larger population

inter-rater reliability: measure of agreement among observers on how they record and classify a particular event

naturalistic observation: observation of behavior in its natural setting

observer bias: when observations may be skewed to align with observer expectations

population: overall group of individuals that the researchers are interested in

sample: subset of individuals selected from the larger population

single-case experimental design: when the same research participant serves as the subject in both the experimental and control conditions

survey: list of questions to be answered by research participants—given as paper-and-pencil questionnaires, administered electronically, or conducted verbally—allowing researchers to collect data from a large number of people

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2.5: Descriptive and Epidemiological Research

Learning Objectives

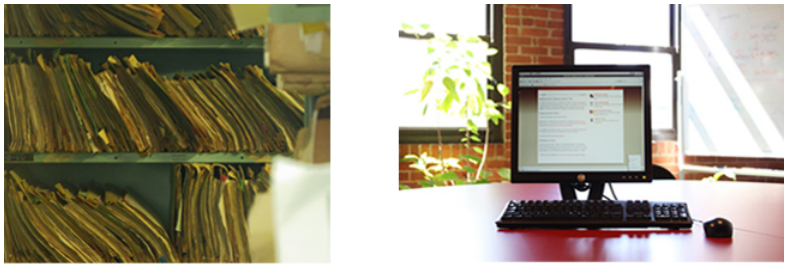
- Describe how archival, longitudinal, cross-sectional, and epidemiological research are valuable to abnormal psychology
- Differentiate between prevalence and incidence

Other types of descriptive research include archival research, longitudinal and cross-sectional studies, and epidemiological studies.

Archival Research

Some researchers gain access to large amounts of data without interacting with a single research participant. Instead, they use existing records to answer various research questions. This type of research approach is known as archival research. **Archival research** relies on looking at past records or data sets to look for interesting patterns or relationships.

Archival research is generally more complex and time-consuming than research that involves the summary, collation and/or synthesis of existing research, and it is frequently also undertaken in other disciplines within the social sciences, including psychology. For example, a psychologist may seek out and extract evidence from original mental institution records from the 1900s in order to determine the prevalence of depressive symptoms in patients at the time. There has been much speculation about modern environments causing an epidemic of depression.[1] Archival research could provide important information about the increasing prevalence (Figure **2.5.1**).



(a) (b)

Figure **2.5.1**: A researcher doing archival research examines records, whether archived (a) physically or (b) electronically. (credit "paper files": modification of work by "Newtown graffiti"/Flickr; "computer": modification of work by INPIVIC Family/Flickr)

In comparing archival research to other research methods, there are several important advantages and disadvantages.

Advantages of Archival Research

- Archival research minimizes the response biases of subjects because the researcher is not present while the data is recorded.
- Archival data is very plentiful and has already been collected. This makes it easier and often less costly than alternative research methods.
- Archival research is effective in helping to confirm that the results and theories derived from experiments reflect the "real world" and do not just exist in artificial or simplistic laboratory settings.
- This approach can help researchers create new ideas for hypotheses and experiments.

Limitations of Archival Research

- Not all archives endure, and those that do not may not have been randomly lost. When the survival of records is selective, there may be bias in the remaining archival data.
- People make mistakes in entering data in archives or there may be biases when data is recorded. For example, suicides may be recorded as accidental deaths to help maintain the privacy of the victims' families.
- Sometimes definitions change so that even though long-term records use the same label, what is being included may change over time. For example, the definition of family may change from comprising a mother, a father, and children to same-sex couples or single-parent families.
- Perhaps most importantly, we cannot conclude causal relations from archival research. After all, the researcher did not control and manipulate the variables that may have played a role. Because the data were not assembled to answer a particular research

question, it might not be exactly the data the researcher requires.

Longitudinal and Cross-Sectional Research

Sometimes we want to see how people change over time. When we test the same group of individuals repeatedly over an extended period of time, we are conducting longitudinal research. **Longitudinal** research is a research design in which data-gathering is administered repeatedly over an extended period of time. For example, we may survey a group of individuals about their dietary habits at age 20; retest them a decade later at age 30; and then again at ages 40, 50, and 60 to possibly find correlations between diet and early-onset dementia. In this case, the longitudinal design is used to uncover risk factors of certain diseases, like Alzheimer's. Longitudinal studies are also used in social-personality and clinical psychology to study rapid fluctuations in behaviors, thoughts, and emotions from moment to moment or day to day.

Another approach is **cross-sectional** research. In cross-sectional research, a researcher compares multiple segments of the population at the same time. Using the dietary habits example above, the researcher might directly compare different groups of people by age. Instead of observing a group of people for 20 years to see how their dietary habits changed from decade to decade, the researcher would study a group of 20-year-old individuals and compare them to a group of 30-year-old individuals and then to a group of 40-year-old individuals, and so on. While cross-sectional research requires a shorter-term investment, it is also limited by differences that exist between the different generations (or cohorts) that have nothing to do with age per se, but rather reflect the social and cultural experiences of different generations of individuals that make them different from one another.

Longitudinal research is a powerful approach because the same individuals are involved in the research project over time, which means that the researchers need to be less concerned with differences among cohorts affecting the results of their study. Often longitudinal studies are employed when researching various mental disorders in an effort to understand particular risk factors. Such studies often involve tens of thousands of individuals who are followed for several decades. Given the enormous number of people involved in these studies, researchers can feel confident that their findings can be generalized to the larger population.

To illustrate this concept, consider the following research tracked by **Alzheimer's Disease Neuroimaging Initiative** (ADNI). Alzheimer's Disease Neuroimaging Initiative (ADNI) is a multisite study that aims to improve clinical trials for the prevention and treatment of Alzheimer's disease (AD). Researchers at 63 sites in the U.S. and Canada track the progression of Alzheimer's disease (AD) in the human brain with neuroimaging, biochemical, and genetic biological markers. This knowledge helps to detect the earliest signs of Alzheimer's disease (AD) and to track the disease, to find better clinical trials for the prevention and treatment of AD, and to make all data and samples available for sharing with clinical trial designers and scientists worldwide. Alzheimer's Disease Neuroimaging Initiative (ADNI) enrolls participants between the ages of 55 and 90 who are recruited at 57 sites in the U.S. and Canada. One group has dementia due to AD, another group has mild memory problems known as mild cognitive impairment (MCI), and the final control group consists of healthy elderly participants. Studies using ADNI cross-sectional and longitudinal MRI, PET, genetics, cognitive, biological fluid, and autopsy data have reported that AD pathology is already present in people with no outward sign of memory loss. These cognitively normal people may already have subtle brain atrophy.

Cohortstudies are one type of longitudinal study that samples a **cohort** (a group of people who share a defining characteristic, typically who experienced a common event in a selected period, such as birth or graduation) and perform cross-section observations at intervals through time. The Minnesota Center for Twin and Family Research (or MCTFR) is a series of behavioral genetic longitudinal studies of families with twin or adoptive offspring conducted by researchers at the University of Minnesota. It seeks to identify and characterize the genetic and environmental influences on the development of psychological traits. The primary cohorts of participants include the Minnesota Twin Family Study, Sibling Interaction and Behavior Study, Minnesota Twin Registry, and a variety of other cohorts of participants.

In twin studies, researchers identify individuals with a specific disorder who are members of an MZ or a DZ twin pair and then study the other twin in the pairs. The Minnesota Twin Family Study is a **twin study** established in June 1989 with 1,300 same-gendered twin pairs ages 11 or 17, with an additional cohort of 500 such pairs recruited around 2004. Twins were born between 1972 and 2000. All twins born in Minnesota at that time were eligible to participate using birth registry data. Both identical and fraternal twins share certain aspects of their environment. This allows researchers to estimate the relative impact of environmental and genetic influences on **phenotypes,** an individual's observable or expressed traits**.** The focus of the Minnesota Twin Family Study (MTFS) is on behavioral phenotypes, such as academic outcomes, cognitive abilities, personality, and interests; family and social relationships; mental and physical health; physiological measurements.

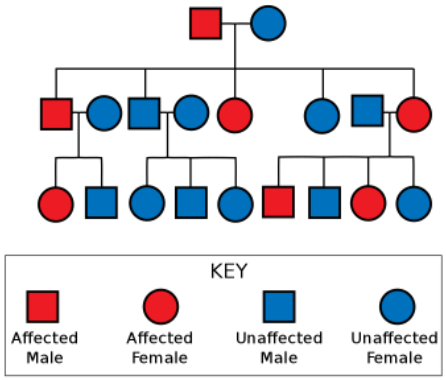


Figure **2.5.2**: Pedigree chart showing an inheritance pattern consistent with autosomal dominant transmission. Behavioral geneticists have used pedigree studies to investigate the genetic and environmental basis of behavior.

Clearly, this type of research is important and potentially very informative. While the name *behavioral genetics* connotes a focus on genetic influences, the field broadly investigates genetic and environmental influences, using research designs that allow removal of the confounding of genes and environment. The field has seen renewed prominence with research on inheritance of behavior and mental illness in (typically using twin and family studies) (Figure 2).

As with any research strategy, longitudinal research is not without limitations. For one, these studies require an incredible time investment by the researcher and research participants. Given that some longitudinal studies take years, if not decades, to complete, the results will not be known for a considerable period of time. In addition to the time demands, these studies also require a substantial financial investment. Many researchers are unable to commit the resources necessary to see a longitudinal project through to the end.

Research participants must also be willing to continue their participation for an extended period of time, and this commitment can be problematic. People move, get married and take new names, get ill, and eventually die. Even without significant life changes, some people may simply choose to discontinue their participation in the project. As a result, the **attrition** rates, or reduction in the number of research participants due to dropouts, in longitudinal studies are quite high and increase over the course of a project. For this reason, researchers using this approach typically recruit many participants fully expecting that a substantial number will drop out before the end. As the study progresses, they continually check whether the sample still represents the larger population and make adjustments as necessary.

Epidemiological Studies

The **epidemiological method** examines rates of occurrence of abnormal behavior in the population as a whole and in various subgroups classified according to factors such as race, ethnicity, gender, or social class. One type of epidemiological study is the survey method, which is used to discover rates of occurrence of various disorders. Often, occurrence of a single disease entity is set as an event. The events can be characterized by **incidence** rates (the number of new cases occurring during a specific period of time) and **prevalence** rates (the overall number of cases of a disorder existing in the population during a given period of time). Prevalence rates, then, include both new and continuing cases.

Full epidemiological studies are expensive and laborious undertakings. Before any study is started, a case must be made for the importance of the research. Epidemiological studies only point to potential causal factors in psychological disorders, as they lack the power of experiments. Epidemiological (and other observational) studies typically highlight *associations* between exposures and outcomes, rather than causation. Moreover, many research questions are impossible to study in experimental settings, due to concerns around ethics and study validity. By finding that disorders cluster in certain groups for example, researchers can identify distinguishing characteristics that place these groups or regions at higher risk. In the process of an epidemiological study, researchers identify predisposing factors that increase the likelihood of getting a disorder, such as genetic history, age, and gender, enabling/disabling factors such as exercise and diet, precipitation factors, and reinforcing factors such as excessive environmental stresses. They also look for patterns and similarities in the cases that may identify major risk factors for developing the disorder.



Figure 2.5.3: The United States Substance Abuse and Mental Health Services Administration (SAMHSA) collects and reports on the epidemiology of mental disorders.

Yet such epidemiological studies cannot control for selection factors; that is, because epidemiological studies can rarely be conducted in a laboratory, the results are often polluted by uncontrollable variations in the cases. This pollution often makes the results difficult to interpret. Therefore, they must be tested further in experimental studies.

The Substance Abuse and Mental Health Services Administration of the U.S. government (SAMHSA) is charged with improving the quality and availability of treatment and rehabilitative services in order to reduce illness, death, disability, and the cost to society resulting from substance abuse and mental illnesses. Surveys are conducted yearly to establish the frequency of use of illegal substances within the population. The World Health Survey was implemented by WHO in 2002–2004 in partnership with 70 countries to generate information on the health of adult populations, including psychological disorders and health systems. The total sample size in these cross-sectional studies includes over 300,000 individuals. These surveys provide valuable epidemiological data. We will rely upon and report about some of this epidemiological data when we learn about specific mental disorders.

Glossary

adoption studies: studies that compare the trait and behavior similarity between an adoptee and his or her biological versus adoptive relatives

archival research: method of research using past records or data sets to answer various research questions or to search for interesting patterns or relationships

attrition: reduction in number of research participants as some drop out of the study over time

cohort: a group of people who share a defining characteristic, typically who experienced a common event in a selected period, such as birth or graduation

cohortstudies: one type of longitudinal study which sample a cohort and perform cross-section observations at intervals through time

cross-sectional research: compares multiple segments of a population at a single time

incidence: the number of new cases occurring during a specific period of time

longitudinal research: studies in which the same group of individuals is surveyed or measured repeatedly over an extended period of time

phenotypes: an individual's observable or expressed traits

prevalence: the overall number of cases of a disorder existing in the population during a given period of time

twin study: study in which researchers identify individuals with a specific disorder who are members of an MZ or a DZ twin pair and then study the other twin in the pairs

1. Hidaka B. H. (2012). Depression as a disease of modernity: explanations for increasing prevalence. *Journal of affective disorders*, 140(3), 205–214. https://doi.org/10.1016/j.jad.2011.12.036↵

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2.6: Correlational Research

Learning Objectives

- Explain correlational research, including what a correlation coefficient tells us about the relationship between variables

Correlational Research

One of the primary methods used to study abnormal behavior is the correlational method. **Correlation** means that there is a relationship between two or more variables (such between the variables of negative thinking and depressive symptoms), but this relationship does not necessarily imply cause and effect. When two variables are correlated, it simply means that as one variable changes, so does the other. We can measure correlation by calculating a statistic known as a correlation coefficient. A **correlation coefficient** is a number from negative one to positive one that indicates the strength and direction of the relationship between variables. The association between two variables can be summarized statistically using the correlation coefficient (abbreviated as *r*).

The number portion of the correlation coefficient indicates the strength of the relationship. The closer the number is to one (be it negative or positive), the more strongly related the variables are, and the more predictable changes in one variable will be as the other variable changes. The closer the number is to zero, the weaker the relationship, and the less predictable the relationships between the variables becomes. For instance, a correlation coefficient of 0.9 indicates a far stronger relationship than a correlation coefficient of 0.3. If the variables are not related to one another at all, the correlation coefficient is zero. The example above about negative thinking and depressive symptoms is an example of two variables that we might expect to have a relationship to each other. When higher values in one variable (negative thinking) are associated with higher values in the other variable (depressive symptoms), there is a *positive correlation* between the variables.

The sign—positive or negative—of the correlation coefficient indicates the direction of the relationship. Positive correlations carry positive signs; negative correlations carry negative signs. A **positive correlation**means that the variables move in the same direction. Put another way, it means that as one variable increases so does the other, and conversely, when one variable decreases so does the other. A **negative correlation**means that the variables move in opposite directions. If two variables are negatively correlated, a decrease in one variable is associated with an increase in the other and vice versa.

Other examples of positive correlations are the relationship between depression and disturbance in normal sleep patterns. One might expect then that scores on a measure of depression would be positively correlated with scores on a measure of sleep disturbances.

One might expect a negative correlation to exist between between depression and self-esteem. The more depressed people are, the lower their scores are on the Rosenberg self-esteem scale (RSES), a self-esteem measure widely used in social-science research. Keep in mind that a negative correlation is not the same as no correlation. For example, we would probably find no correlation between depression and someone's height.

In correlational research, scientists passively observe and measure phenomena. Here, we do not intervene and change behavior, as we do in experiments. In correlational research, we identify patterns of relationships, but we usually cannot infer what causes what. Importantly, with correlational research, you can examine only two variables at a time, no more and no less.

As mentioned earlier, correlations have predictive value. So, what if you wanted to test whether spending on others is related to happiness, but you don't have $20 to give to each participant? You could use a correlational design—which is exactly what Professor Dunn did, too. She asked people how much of their income they spent on others or donated to charity, and later she asked them how happy they were. Do you think these two variables were related? Yes, they were! The more money people reported spending on others, the happier they were.

More Details about the Correlation

To find out how well two variables correspond, we can plot the relationship between the two scores on what is known as a scatterplot (Figure 1). In the scatterplot, each dot represents a data point. (In this case it's individuals, but it could be some other unit.) Importantly, each dot provides us with two pieces of information—in this case, information about how good the person rated

the past month (*x*-axis) and how happy the person felt in the past month (*y*-axis). Which variable is plotted on which axis does not matter.

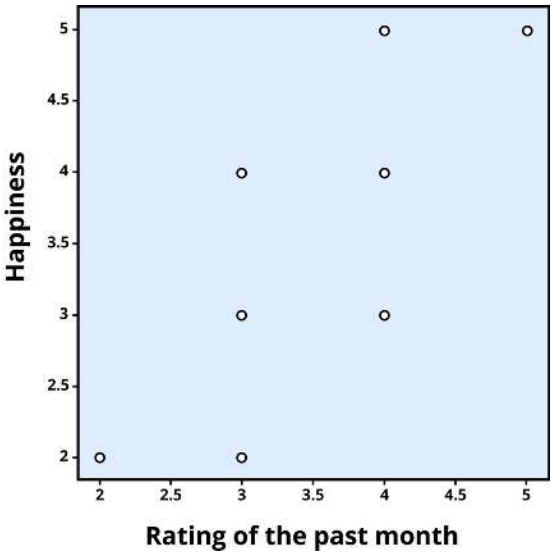


Figure **2.6.1**: Scatterplot of the association between happiness and ratings of the past month, a positive correlation (*r* = .81). Each dot represents an individual.

For the example above, the direction of the association is positive. This means that people who perceived the past month as being good reported feeling more happy, whereas people who perceived the month as being bad reported feeling less happy.

In a scatterplot, the dots form a pattern that extends from the bottom left to the upper right (just as they do in Figure 1). The *r* value for a positive correlation is indicated by a positive number (although, the positive sign is usually omitted). Here, the *r* value is 0.81.

Figure 2 shows a negative correlation, the association between the average height of males in a country (*y*-axis) and the pathogen prevalence, or commonness of disease, of that country (*x*-axis). In this scatterplot, each dot represents a country. Notice how the dots extend from the top left to the bottom right. What does this mean in real-world terms? It means that people are shorter in parts of the world where there is more disease. The *r* value for a negative correlation is indicated by a negative number—that is, it has a minus (−) sign in front of it. Here, it is −0.83.

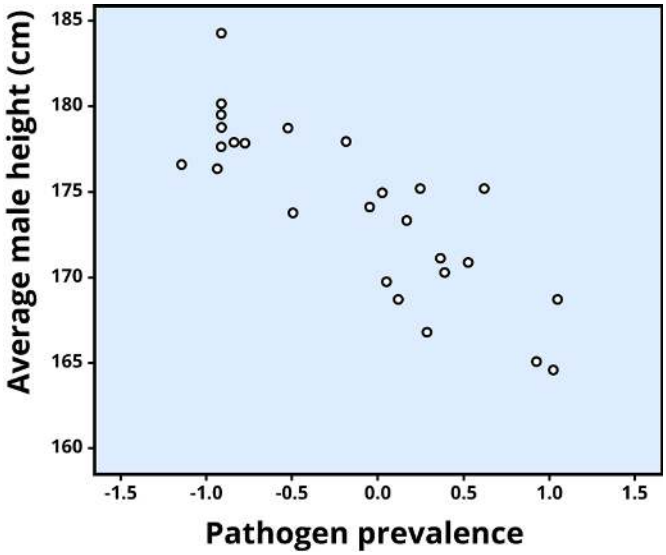


Figure **2.6.2**: Scatterplot showing the association between average male height and pathogen prevalence, a negative correlation (*r* = –0.83). Each dot represents a country (Chiao, 2009).

The strength of a correlation has to do with how well the two variables align. Recall that in Professor Dunn's correlational study, spending on others positively correlated with happiness: the more money people reported spending on others, the happier they

reported to be. At this point, you may be thinking to yourself, “I know a very generous person who gave away lots of money to other people but is miserable!” Or maybe you know of a very stingy person who is happy as can be. Yes, there might be exceptions. If an association has many exceptions, it is considered a weak correlation. If an association has few or no exceptions, it is considered a strong correlation. A strong correlation is one in which the two variables always, or almost always, go together. In the example of happiness and how good the month has been, the association is strong. The stronger a correlation is, the tighter the dots in the scatterplot will be arranged along a sloped line.[1]

Problems with correlation

If generosity and happiness are positively correlated, should we conclude that being generous causes happiness? Similarly, if height and pathogen prevalence are negatively correlated, should we conclude that disease causes shortness? From a correlation alone, we can’t be certain. For example, in the first case it may be that happiness causes generosity, or that generosity causes happiness. Or, a third variable might cause both happiness *and* generosity, creating the illusion of a direct link between the two. For example, wealth could be the third variable that causes both greater happiness and greater generosity. This is why correlation does not mean causation—an often repeated phrase among psychologists.[2]

Correlation Does Not Indicate Causation

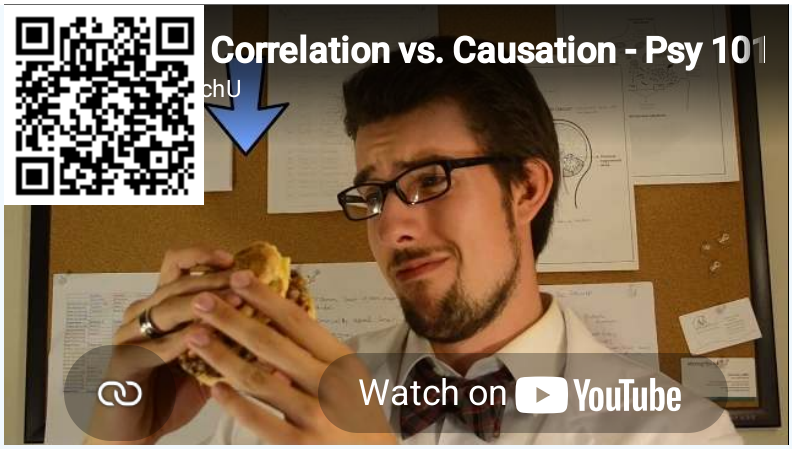
Correlational research is useful because it allows us to discover the strength and direction of relationships that exist between two variables. However, correlation is limited because establishing the existence of a relationship tells us little about **cause and effect**. While variables are sometimes correlated because one does cause the other, it could also be that some other factor, a **confounding variable**, is actually causing the systematic movement in our variables of interest. In the depression and negative thinking example mentioned earlier, stress is a confounding variable that could account for the relationship between the two variables.

Even when we cannot point to clear confounding variables, we should not assume that a correlation between two variables implies that one variable causes changes in another. This can be frustrating when a cause-and-effect relationship seems clear and intuitive. Think back to our example about the relationship between depression and disturbance in normal sleep patterns. It seems reasonable to assume that sleep disturbance might cause a higher score on a measure of depression, just as a high degree of depression might cause more disturbed sleep patterns, but if we were limited to **correlational research**, we would be overstepping our bounds by making this assumption. Both depression and sleep disturbance could be due to an underlying physiological disorder or any to other third variable that you have not measured.

Unfortunately, people mistakenly make claims of causation as a function of correlations all the time. While correlational research is invaluable in identifying relationships among variables, a major limitation is the inability to establish causality. The correlational method does not involve manipulation of the variables of interest. In the previous example, the experimenter does not manipulate people’s depressive symptoms or sleep patterns. Psychologists want to make statements about cause and effect, but the only way to do that is to conduct an experiment to answer a research question. The next section describes how investigators use experimental methods in which the experimenter manipulates one or more variables of interest and observes their effects on other variables or outcomes under controlled conditions.

Watch It

In this video, we discuss one of the best methods psychologists have for predicting behaviors: correlation. But does that mean that a behavior is absolutely going to happen? Let’s find out!



You can view the transcript for "#5 Correlation vs. Causation – Psy 101" here (opens in new window).

Glossary

cause-and-effect relationship: changes in one variable cause the changes in the other variable; can be determined only through an experimental research design

confirmation bias: tendency to ignore evidence that disproves ideas or beliefs

confounding variable: unanticipated outside factor that affects both variables of interest,\; often gives the false impression that changes in one variable causes changes in the other variable, when, in actuality, the outside factor causes changes in both variables

correlation: the relationship between two or more variables; when two variables are correlated, one variable changes as the other does

correlation coefficient: number from -1 to +1, indicating the strength and direction of the relationship between variables, and usually represented by *r*

negative correlation: two variables change in different directions, with one becoming larger as the other becomes smaller; a negative correlation is not the same thing as no correlation

positive correlation: two variables change in the same direction, both becoming either larger or smaller

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1. Scollon, C. N. (2020). Research designs. In R. Biswas-Diener & E. Diener (Eds), Noba textbook series: Psychology. Champaign, IL: DEF publishers. Retrieved from http://noba.to/acxb2thy↵
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2.7: Experiments

Learning Objectives

- Describe the experimental process and its importance to abnormal psychology

Causality: Conducting Experiments and Using the Data

As you have learned, the only way to establish that there is a cause-and-effect relationship between two variables is to conduct a scientific experiment. **Experiment** has a different meaning in the scientific context than in everyday life. In everyday conversation, we often use it to describe trying something for the first time, such as experimenting with a new hair style or a new food. However, in the scientific context, an experiment has precise requirements for design and implementation.

Experimental Hypothesis

In order to conduct an experiment, a researcher must have a specific **hypothesis** to be tested. As you have learned, hypotheses can be formulated either through direct observation of the real world or after careful review of previous research. For example, if you think that children should not be allowed to watch violent programming on television because doing so would cause them to behave more violently, then you have basically formulated a hypothesis—namely, that watching violent television programs causes children to behave more violently. How might you have arrived at this particular hypothesis? You may have younger relatives who watch cartoons featuring characters using martial arts to save the world from evildoers with an impressive array of punching, kicking, and defensive postures. You notice that after watching these programs for a while, your young relatives mimic the fighting behavior of the characters portrayed in the cartoon (Figure 1).



Figure **2.7.1**: Seeing behavior like this right after a child watches violent television programming might lead you to hypothesize that viewing violent television programming leads to an increase in the display of violent behaviors (credit: Emran Kassim).

These sorts of personal observations are what often lead us to formulate a specific hypothesis, but we cannot use limited personal observations and anecdotal evidence to rigorously test our hypothesis. Instead, to find out if real-world data supports our hypothesis, we have to conduct an experiment.

Designing an Experiment

The most basic experimental design involves two groups: the experimental group and the control group. The two groups are designed to be the same except for one difference— experimental manipulation. The **experimental group** gets the experimental manipulation—that is, the treatment or variable being tested (in this case, violent TV images)—and the **control group** does not. Since experimental manipulation is the only difference between the experimental and control groups, we can be sure that any differences between the two are due to experimental manipulation rather than chance.

In our example of how violent television programming might affect violent behavior in children, we have the experimental group view violent television programming for a specified time and then measure their violent behavior. We measure the violent behavior in our control group after they watch nonviolent television programming for the same amount of time. It is important for the control group to be treated similarly to the experimental group, with the exception that the control group does not receive the experimental

manipulation. Therefore, we have the control group watch non-violent television programming for the same amount of time as the experimental group.

We also need to precisely define, or operationalize, what is considered violent and nonviolent. An **operational definition** is a description of how we will measure our variables, and it is important in allowing others understand exactly how and what a researcher measures in a particular experiment. In operationalizing violent behavior, we might choose to count only physical acts like kicking or punching as instances of this behavior, or we also may choose to include angry verbal exchanges. Whatever we determine, it is important that we operationalize violent behavior in such a way that anyone who hears about our study for the first time knows exactly what we mean by violence. This aids peoples' ability to interpret our data as well as their capacity to repeat our experiment should they choose to do so.

Once we have operationalized what is considered violent television programming and what is considered violent behavior from our experiment participants, we need to establish how we will run our experiment. In this case, we might have participants watch a 30-minute television program (either violent or nonviolent, depending on their group membership) before sending them out to a playground for an hour where their behavior is observed and the number and type of violent acts is recorded.

Ideally, the people who observe and record the children's behavior are unaware of who was assigned to the experimental or control group, in order to control experimenter bias. **Experimenter bias** refers to the possibility that a researcher's expectations might skew the results of the study. Remember, conducting an experiment requires a lot of planning, and the people involved in the research project have a vested interest in supporting their hypotheses. If the observers knew which child was in which group, it might influence how much attention they paid to each child's behavior as well as how they interpreted that behavior. By being blind to which child is in which group, we protect against those biases. This situation is a **single-blind study**, meaning that one of the groups (participants) are unaware as to which group they are in (experiment or control group) while the researcher who developed the experiment knows which participants are in each group.



Figure 2.7.2: Providing the control group with a placebo treatment protects against bias caused by expectancy. (credit: Elaine and Arthur Shapiro)

In a **double-blind study**, both the researchers and the participants are blind to group assignments. Why would a researcher want to run a study where no one knows who is in which group? Because by doing so, we can control for both experimenter and participant expectations. If you are familiar with the phrase placebo effect, you already have some idea as to why this is an important consideration. The **placebo effect** occurs when people's expectations or beliefs influence or determine their experience in a given situation. In other words, simply expecting something to happen can actually make it happen.

The placebo effect is commonly described in terms of testing the effectiveness of a new medication. In a placebo condition, the placebo has inert ingredients.

Imagine that you work in a pharmaceutical company, and you think you have a new drug that is effective in treating depression. To demonstrate that your medication is effective, you run an experiment with two groups: The experimental group receives the medication, and the control group does not. But you don't want participants to know whether they received the drug or not.

Why is that? Imagine that you are a participant in this study, and you have just taken a pill that you think will improve your mood. Because you expect the pill to have an effect, you might feel better simply because you took the pill and not because of any drug actually contained in the pill—this is the placebo effect.

To make sure that any effects on mood are due to the drug and not due to expectations, the control group receives a placebo (in this case a sugar pill). Now everyone gets a pill, and once again neither the researcher nor the experimental participants know who got

the drug and who got the sugar pill. Any differences in mood between the experimental and control groups can now be attributed to the drug itself rather than to experimenter bias or participant expectations (Figure 2).

When it comes to abnormal psychology, it is common practice in studies evaluating therapy effectiveness to have a placebo condition. In a **placebo condition**, participants receive a treatment similar to the experimental treatment, but lacking the key feature of the treatment of interest. If the study is evaluating effectiveness of therapy, psychologists must design the placebo in a way that is very similar, but not the same as the actual therapy. Ideally, we would want the placebo participants to receive treatments in the same modality, or form in which the clinician offers psychotherapy, as the experimental group participants, including frequency and duration.

Independent and Dependent Variables

In a research experiment, we strive to study whether changes in one thing cause changes in another. To achieve this, we must pay attention to two important variables, or things that can be changed, in any experimental study: the independent variable and the dependent variable. An **independent variable** is manipulated or controlled by the experimenter. In a well-designed experimental study, the independent variable is the only important difference between the experimental and control groups. A **dependent variable** is what the researcher measures to see how much effect the independent variable had. In our example, the dependent variable is the number of violent acts displayed by the experimental participants. Examples of independent variables in experimental research in abnormal psychology include different types of drug treatments for psychological treatments, treatment factors such as brief versus long-term treatment or inpatient versus outpatient treatment, and experimental manipulations such as consuming alcoholic versus non-alcoholic beverages. Examples of dependent variables include behavioral variables such as measures of adjustment, activity levels, eating behavior, and smoking behavior; physiological variables such as measures of physiological responses such as heart rate, blood pressure, and brain wave activity; and self-report variables such as measures of anxiety, mood, or marital or life satisfaction.

We expect that the dependent variable will change as a function of the independent variable. In other words, the dependent variable *depends* on the independent variable. A good way to think about the relationship between the independent and dependent variables is with this question: what effect does the independent variable have on the dependent variable?

Issues to Consider

In research on the causes of abnormal behavior, it may be difficult to design a true experimental study. Many of the variables that psychologists are interested in are precisely ones that the researcher cannot control. One very important question is, can the variable be considered truly “independent”? Take the example of depression that we illustrated in our discussion about correctional research. Depression in those examples can never be an independent variable because the researcher cannot manipulate it. Another example is that we cannot randomly assign people to groups based on their gender. *Quasi-experiments* have independent variables that already exist such as age, gender, and eye color. Experimenters are also limited by ethical constraints. For instance, you would not be able to conduct an experiment designed to determine if experiencing abuse as a child leads to pedophilic disorder among adults. To conduct such an experiment, you would need to randomly assign some experimental participants to a group that receives abuse, and that experiment would be unethical.

A study that investigates differences among groups from a target population without random assignment are known as **quasi-experimental**. That is to say that this type of study specifically lacks the element of random assignment to treatment or control groups. **Random assignment** is critical for sound experimental design and means that all participants have an equal chance of being assigned to either group. However, in a quasi-experimental design, assignment to a given treatment condition is based on something other than random assignment. Depending on the type of quasi-experimental design, the researcher might have control over the assignment to the treatment condition but use some criteria other than random assignment (e.g., a cutoff score) to determine which participants receive the treatment, or the researcher may have no control over the treatment condition assignment and the criteria used for assignment may be unknown.

On their own, quasi-experimental designs do not allow one to make definitive causal inferences; however, they provide necessary and valuable information that cannot be obtained by experimental methods alone.

Most “true” experimental studies in abnormal psychology test not the causes of abnormal behavior, but the effectiveness of particular treatments where it is possible to design randomly assigned control and experimental groups. Researchers use traditional,

experimental design or randomized, controlled trials to evaluate a treatment's effectiveness by comparing the groups on dependent variables such as the effectiveness of a new treatment, as discussed earlier in the placebo condition. There may be more than one experimental group, depending on the nature of the study.

Glossary

cause-and-effect relationship: changes in one variable cause the changes in the other variable; can be determined only through an experimental research design

confirmation bias: tendency to ignore evidence that disproves ideas or beliefs

control group: serves as a basis for comparison and controls for chance factors that might influence the results of the study—by holding such factors constant across groups so that the experimental manipulation is the only difference between groups

correlation: relationship between two or more variables; when two variables are correlated, one variable changes as the other does

dependent variable: variable that the researcher measures to see how much effect the independent variable had

double-blind study: experiment in which both the researchers and the participants are blind to group assignments

experimental group: group designed to answer the research question; experimental manipulation is the only difference between the experimental and control groups, so any differences between the two are due to experimental manipulation rather than chance

experimenter bias: researcher expectations skew the results of the study

independent variable: variable that is influenced or controlled by the experimenter; in a sound experimental study, the independent variable is the only important difference between the experimental and control group

operational definition: description of what actions and operations will be used to measure the dependent variables and manipulate the independent variables

placebo condition: participants receive a treatment similar to the experimental treatment, but lacking the key feature of the treatment of interest

placebo effect: people's expectations or beliefs influencing or determining their experience in a given situation

quasi-experimental: a study that investigates differences among groups from a target population without random assignment

random assignment: method of experimental group assignment in which all participants have an equal chance of being assigned to either group

single-blind study: experiment in which the researcher knows which participants are in the experimental group and which are in the control group

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2.8: Understanding Research

Learning Objectives

- Explain how reliability and validity are important in understanding research findings

Interpreting Experimental Findings

Once data is collected from both the experimental and the control groups, a **statistical analysis** is conducted to find out if there are meaningful differences between the two groups. A statistical analysis determines how likely any difference found is due to chance (and thus not meaningful). In psychology, group differences are considered meaningful, or significant, if the odds that these differences occurred by chance alone are 5% or less. Stated another way, if we repeated this experiment 100 times, we would expect to find the same results at least 95 times out of 100.

The greatest strength of experiments is the ability to assert that any significant differences in the findings are caused by the independent variable. This occurs because random selection, random assignment, and a design that limits the effects of both experimenter bias and participant expectancy should create groups that are similar in composition and treatment. Therefore, any difference between the groups is attributable to the independent variable, and now we can finally make a causal statement. If we find that watching a violent television program results in more violent behavior than watching a nonviolent program, we can safely say that watching violent television programs causes an increase in the display of violent behavior.

Reporting Research

When psychologists complete a research project, they generally want to share their findings with other scientists. The American Psychological Association (APA) publishes a manual detailing how to write a paper for submission to scientific journals. Unlike an article that might be published in a magazine like *Psychology Today*, which targets a general audience with an interest in psychology, scientific journals generally publish peer-reviewed journal articles aimed at an audience of professionals and scholars who are actively involved in research themselves.

Link to Learning

The Online Writing Lab (OWL) at Purdue University can walk you through the APA writing guidelines.



Figure **2.8.1**: Journal articles that have been peer-reviewed are considered to be more credible and reliable.

A peer-reviewed journal article is read by several other scientists (generally anonymously) with expertise in the subject matter. These peer reviewers provide feedback—to both the author and the journal editor—regarding the quality of the draft. Peer reviewers look for a strong rationale for the research being described, a clear description of how the research was conducted, and evidence that the research was conducted in an ethical manner. They also look for flaws in the study's design, methods, and statistical analyses. They check that the conclusions drawn by the authors seem reasonable given the observations made during the research. Peer reviewers also comment on how valuable the research is in advancing the discipline's knowledge. This commentary helps prevent unnecessary duplication of research findings in the scientific literature and, to some extent, ensures that each research article provides new information. Ultimately, the journal editor will compile all the peer reviewer feedback and determine whether the article will be published in its current state (a rare occurrence), published with revisions, or not accepted for publication.

Peer review provides some degree of quality control for psychological research. Poorly conceived or executed studies can be weeded out, and even well-designed research can be improved by the revisions suggested. Peer review also ensures that the

research is described clearly enough to allow other scientists to **replicate** it, meaning they can repeat the experiment using different samples to determine **reliability**. Replicability is an important feature of science. It means that a study should produce the same results if repeated exactly, either by the same researcher or by another. Sometimes replications involve additional measures that expand on the original finding. In any case, each replication serves to provide more evidence to support the original research findings. Successful replications of published research make scientists more apt to adopt those findings, while repeated failures tend to cast doubt on the legitimacy of the original article and lead scientists to look elsewhere. For example, it would be a major advancement in the medical field if a published study indicated that taking a new drug helped individuals achieve a healthy weight without changing their diet. But if other scientists could not replicate the results, the original study's claims would be questioned.

✔ Dig Deeper: The Vaccine-Autism Myth and the Retraction of Published Studies



Figure **2.8.2**: Some people still think vaccinations cause autism. (credit: modification of work by UNICEF Sverige)

Some scientists have claimed that routine childhood vaccines cause some children to develop autism, and, in fact, several peer-reviewed publications published research making these claims. Since the initial reports, large-scale epidemiological research has suggested that vaccinations are not responsible for causing autism and that it is much safer to have your child vaccinated than not. Furthermore, several of the original studies making this claim have since been retracted.

A published piece of work can be rescinded when data is called into question because of falsification, fabrication, or serious research design problems. Once rescinded, the scientific community is informed that there are serious problems with the original publication. Retractions can be initiated by the researcher who led the study, by research collaborators, by the institution that employed the researcher, or by the editorial board of the journal in which the article was originally published. In the vaccine-autism case, the retraction was made because of a significant conflict of interest in which the leading researcher had a financial interest in establishing a link between childhood vaccines and autism (Offit, 2008). Unfortunately, the initial studies received so much media attention that many parents around the world became hesitant to have their children vaccinated (Figure 2). For more information about how the vaccine/autism story unfolded, as well as the repercussions of this story, take a look at Paul Offit's book, *Autism's False Prophets: Bad Science, Risky Medicine, and the Search for a Cure*.

Reliability and Validity

Reliability and **validity** are two important considerations that must be made with any type of data collection. Reliability refers to the ability to consistently produce a given result. In the context of psychological research, this would mean that any instruments or tools used to collect data do so in consistent, reproducible ways. Unfortunately, being consistent in measurement does not necessarily mean that you have measured something correctly. This is where validity comes into play. Validity refers to the extent to which a given instrument or tool accurately measures what it's supposed to measure. While any valid measure is by necessity reliable, the reverse is not necessarily true. Researchers strive to use instruments that are both highly reliable and valid. In short, reliability is about the consistency of a measure, and validity is about the accuracy of a measure.

? The 'Real-World Approach' and Its Problems

A popular goal in psychological science is to understand human cognition and behavior in the "real world." In contrast, researchers have typically conducted their research in experimental research settings, a.k.a. the "psychologist's laboratory." Critics have often questioned whether psychology's laboratory experiments permit generalizable results. This is known as the "real world or the lab" dilemma[1].

In many studies and research designs, there may be a trade-off between **internal validity** (the extent to which a piece of evidence supports a claim about cause and effect, within the context of a particular study)and **external validity** (the extent to

which the results of a study can be generalized to and across other situations, people, stimuli, and times). Attempts to increase internal validity may also limit the generalizability of the findings, and vice versa. This situation has led many researchers call for "ecologically valid" experiments. By that, they mean that experimental procedures should resemble "real-world" conditions. They criticize the lack of **ecological validity** in many laboratory-based studies with a focus on artificially controlled and constricted environments.

Some researchers think external validity and ecological validity are closely related in the sense that causal inferences based on ecologically valid research designs often allow for higher degrees of generalizability than those obtained in an artificially produced lab environment. However, this again relates to the distinction between generalizing to some population (closely related to concerns about ecological validity) and generalizing across subpopulations that differ on some background factor. Some findings produced in ecologically valid research settings may hardly be generalizable, and some findings produced in highly controlled settings may claim near-universal external validity. Thus, external and ecological validity are independent—a study may possess external validity but not ecological validity, and vice versa.

Glossary

ecological validity: in the behavioral sciences, refers to the judgment of whether a given study's variables and conclusions are sufficiently relevant to its population (e.g., the "real-world" context)

external validity: the extent to which the results of a study can be generalized to and across other situations, people, stimuli, and times

internal validity: the extent to which a piece of evidence supports a claim about cause and effect within the context of a particular study

reliability: consistency and replicability of a given result

replicate: repeating an experiment using different samples to determine the research's reliability

statistical analysis: determines how likely any difference between experimental groups is due to chance

validity: accuracy of a given result in measuring what it is designed to measure

1. Holleman, G. A., Hooge, I., Kemner, C., & Hessels, R. S. (2020). The 'Real-World Approach' and Its Problems: A Critique of the Term Ecological Validity. Frontiers in psychology, 11, 721. https://doi.org/10.3389/fpsyg.2020.00721↵

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2.9: Introduction to Ethics in Abnormal Psychology

What you'll learn to do: describe ethical standards and concerns in abnormal psychology



In this section, we will learn more about what researchers can and cannot do in regards to studying abnormal behavior. We will also examine how forensic issues, such as pleading insanity, relate to our understanding of mental illness.

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2.10: Ethics in Research

Learning Objectives

- Explain how research involving humans and animals is regulated

Today, scientists agree that good research is ethical in nature and is guided by a basic respect for human dignity and safety. However, as you will read in the Tuskegee Syphilis Study, this expectation has not always been the case. Modern researchers must demonstrate that the research they perform is ethically sound. This section presents how ethical considerations affect the design and implementation of research conducted today.

Research Involving Human Participants

Any experiment involving the participation of human subjects is governed by extensive, strict guidelines designed to ensure that the experiment does not result in harm. Any research institution that receives federal support for research involving human participants must have access to an **institutional review board (IRB)**. The institutional review board (IRB) is a committee of individuals often made up of members of the institution's administration, scientists, and community members (Figure 1). The purpose of the institutional review board (IRB) is to review proposals for research that involves human participants. The IRB reviews these proposals with the principles mentioned above in mind, and generally, approval from the IRB is required in order for the experiment to proceed.



Figure 2.10.1: An institution's IRB meets regularly to review experimental proposals that involve human participants. (credit: modification of work by Lowndes Area Knowledge Exchange (LAKE)/Flickr)

An institution's IRB requires several components in any experiment it approves. For one, each participant must sign an informed consent form before they can participate in the experiment. An **informed consent form** provides a written description of what participants can expect during the experiment, including potential risks and implications of the research. It also lets participants know that their involvement is completely voluntary and can be discontinued without penalty at any time. Furthermore, the informed consent guarantees that any data collected in the experiment will remain completely confidential. In cases where research participants are under the age of 18, the parents or legal guardians are required to sign the informed consent form.

While the informed consent form should be as honest as possible in describing exactly what participants will be doing, sometimes deception is necessary to prevent participants' knowledge of the exact research question from affecting the results of the study. **Deception** involves purposely misleading experiment participants in order to maintain the integrity of the experiment, but not to the point where the deception could be considered harmful. For example, if we are interested in how our opinion of someone is affected by their attire, we might use deception in describing the experiment to prevent that knowledge from affecting participants' responses. In cases where deception is involved, participants must receive a full **debriefing** upon conclusion of the study—complete, honest information about the purpose of the experiment, how the data collected will be used, the reasons why deception was necessary, and information about how to obtain additional information about the study.

Dig Deeper: Ethics and Informed Consent

In October 1998, 36-year-old Joseph Santana, a patient at the state-run Bronx Psychiatric Center, died during a trial of antipsychotic drugs. His cries for help were ignored as he was considered delusional. The next month, another ethical concern was raised after the University of Maryland, the University of California at Los Angeles, and the National Institute of Mental

Health in Maryland violated federal regulations during drug trials because they did not get the proper consent from patients—some were taken off of medication for schizophrenia that had been improving their condition.[1]

As a result of the investigation, the institutions changed their practices. The Office of Protection from Research Risk is designed to protect research subjects. Informed consent is permission granted in the knowledge of the possible consequences, typically that which is given by a patient to a doctor for treatment with full knowledge of the possible risks and benefits.

You be the judge: Can a patient suffering from a severe mental disorder give informed consent? How were the participants in these drug trials harmed as a function of the research?

Below are links to FDA regulations governing human subject protection and the conduct of clinical trials:

- Protection of Human Subjects (Informed Consent) (21 CFR Part 50)
- Financial Disclosure by Clinical Investigators (21 CFR Part 54)
- Institutional Review Boards (21 CFR Part 56)
- Good Laboratory Practice for Nonclinical Laboratory Studies (21 CFR Part 58)

Read more about a controversial subject: Are Psychiatrists Betraying Their Patients?

Read more about incapacity to give informed consent owing to mental disorder.

Research Involving Animal Subjects

Many psychologists conduct research involving animal subjects. Often, these researchers use rodents (Figure **2.10.2**) or birds as the subjects of their experiments—the APA estimates that 90% of all animal research in psychology uses these species (American Psychological Association, n.d.). Because many basic processes in animals are sufficiently similar to those in humans, these animals are acceptable substitutes for research that would be considered unethical in human participants.



Figure **2.10.2**: Rats, like the one shown here, often serve as the subjects of animal research. The laboratory rat has contributed enormously to neuroscience research over the years. It has also been a popular animal model for Alzheimer's disease.

This does not mean that animal researchers are immune to ethical concerns. Indeed, the humane and ethical treatment of animal research subjects is a critical aspect of this type of research. Researchers must design their experiments to minimize any pain or distress experienced by animals serving as research subjects.

Whereas IRBs review research proposals that involve human participants, animal experimental proposals are reviewed by an **Institutional Animal Care and Use Committee (IACUC)**. An Institutional Animal Care and Use Committee (IACUC) consists of institutional administrators, scientists, veterinarians, and community members. This committee is charged with ensuring that all experimental proposals require the humane treatment of animal research subjects. It also conducts semi-annual inspections of all animal facilities to ensure that the research protocols are being followed. No animal research project can proceed without the committee's approval.

Glossary

debriefing: when an experiment involved deception, participants are told complete and truthful information about the experiment at its conclusion

deception: purposely misleading experiment participants in order to maintain the integrity of the experiment

informed consent: process of informing a research participant about what to expect during an experiment, any risks involved, and the implications of the research, and then obtaining the person's consent to participate

Institutional Animal Care and Use Committee (IACUC): committee of administrators, scientists, veterinarians, and community members that reviews proposals for research involving animal participants

Institutional Review Board (IRB): committee of administrators, scientists, and community members that reviews proposals for research involving human participants

1. Mosher, Loren L. (1999) *Psychology Today*. https://www.psychologytoday.com/us/articles/199909/are-psychiatrists-betraying-their-patients↵

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2.11: Standards and Competencies in Psychology

Learning Objectives

- Explain the main ethical principles as presented in the APA's Ethical Principles of Psychologists and Code of Conduct

Competencies

Competencies have been defined as "a measurable pattern of knowledge, skill, abilities, behaviors, and other characteristics that an individual needs to perform work roles or occupational functions successfully" (Rodriguez et al., 2002, p. 310). Thus, competencies specify what individuals need to do and the behaviors they should undertake for certain activities, tasks, or roles to perform their professional responsibilities effectively (Schippmann et al., 2000). Competencies are often combinations of the knowledge, skills, and abilities necessary to perform a given role (Campion et al., 2011). In practice, various competencies often overlap and cannot be acquired or attained easily in a linear fashion.

Prahalad and Hamel (1990) first introduced the concept of a **core competency** as a concept in management theory and defined it as "a harmonized combination of multiple resources and skills that distinguish a firm in the marketplace."[1] A multitude of authors have subsequently proposed various combinations of core competencies for psychology (e.g., Rodolfa et al., 2005; Fouad et al., 2009); however, until recently, no unified set of core competencies existed.

Competencies
Psychological knowledge and skills underpinning the core competencies
KN: possess the necessary knowledge
SK: possesses the necessary skills
Professional behavior competencies
PE: practices ethically
AP: acts professionally
ER: relates appropriately to clients and others
WD: works with diversity and demonstrates cultural competence
EP: operates as an evidence-based practitioner
SR: reflects on work
Professional activities competencies
SG: sets relevant goals
PA: conducts psychological assessments and evaluations
PI: conducts psychological interventions
CO: communicates effectively and appropriately

Figure 2.11.1: Competencies identified by the International Association of Applied Psychology (IAAP) and International Union of Psychological Science (IUPsyS) (2016)

The Ethics Code

The first version of a code of ethics was published by the APA in 1953. The need for such a document came after psychologists were taking on more professional and public roles post-World War II. A committee was developed and reviewed situations submitted by psychologists in the field who felt they had encountered ethical dilemmas. Since its origin, there have been nine revisions with the most recent published in 2002 and amended in 2010.

Despite the development and use of a complete ethical code, there have still been ethical violations and controversies. For instance, although the APA takes an explicit stance against conversion therapy, this treatment remains controversial amongst many psychologists and religious groups and is still being practiced by some.

? Conversion Therapy and Controversy

Gay or straight: are people "born that way"? The consensus in the medical community is, "yes, people are born that way." People can't change who they are, and they shouldn't even try.

Conversion therapy is the pseudoscientific practice of trying to change an individual's sexual orientation from homosexual or bisexual to heterosexual using psychological, physical, or spiritual interventions. The term **reparative therapy** has been used as a synonym for conversion therapy. There is no reliable evidence that sexual orientation can be changed, and medical institutions warn that conversion therapy practices are ineffective and potentially harmful.

Some fundamentalist religious groups have used religious justification for the therapy. However, the International Rehabilitation Council for Torture Victims describes conversion therapy as a form of torture, and now various jurisdictions around the world have passed laws against conversion therapy.

The American Psychiatric Association (APA) opposes psychiatric treatment "based upon the assumption that homosexuality *per se* is a mental disorder or based upon the *a priori* assumption that a patient should change his/her sexual homosexual orientation" and describes attempts to change a person's sexual orientation by practitioners as unethical.

There is evidence that LGBT persons resort to psychotherapy at higher rates than the non-LGBT population (Bieschke et al., 2000; King et al., 2007); hence, they may be exposed to higher risk for harmful or ineffective therapies, not only as a vulnerable group, but also as frequent users. There is an identified need for clinicians to be able to work effectively with minority clients, namely LGBT people. The APA's guidelines for psychotherapy with lesbian, gay, and bisexual client (American Psychological Association, 2000, 2012) serve as a main reference. These ethical guidelines highlight, among several issues, the need for clinicians to recognize that their own attitudes and knowledge about the experiences of sexual minorities are relevant to the therapeutic process with these clients and that, therefore, mental health care providers must look for appropriate literature, training, and supervision.

There is also some disagreement within the field about the ethical implications of using a treatment that may be less effective than another known treatment, although some psychologists argue that all therapy treatments are equally effective (see: the Dodo bird verdict). The APA was also implicated in helping the Central Intelligence Agency to continue "enhanced interrogation techniques" of detainees under the Bush administration. This presented an obvious violation of the organization's code of ethics and has been addressed by the APA in the form of reports, responses to media outlets, amendments to policies, and rejections of the allegations.

Ethical Principles of Psychologists and Code of Conduct

The APA's Ethical Principles of Psychologists and Code of Conduct (often referred to as the Ethics Code) "provide(s) guidance for psychologists and standards of professional conduct that can be applied by the APA and by other bodies that choose to adopt them."

Areas covered include but are not limited to the clinical, counseling, and school practice of psychology; research; teaching; supervision of trainees; public service; policy development; social intervention; development of assessment instruments; conducting assessments; educational counseling; organizational consulting; forensic activities; program design and evaluation; and administration. The Ethics Code applies to these activities across a variety of contexts, such as in-person, postal, telephone, internet, and other electronic transmissions. These activities shall be distinguished from the purely private conduct of psychologists, which is not within the purview of the Ethics Code.

- Click here to view the American Psychological Association's Ethical Principles of Psychologists and Code of Conduct

General Principles

There are five general principles that serve as the ideals to which psychologists should aspire within the profession. The principles represent ethical goals but do not explicitly inform or instruct adherence to the goals; instead, the principles aim to influence and to guide professional behavior with respect to the psychologist, research subjects, students, and the individuals who seek psychological services.

Principle A: Beneficence and Non-maleficence

The beneficence and non-maleficence principle of the APA general principles guides psychologists to perform work that is beneficial to others yet does not hurt anyone in the process of carrying out that work. Psychologists are to remain aware of their professional influence and the potential consequences therein on individuals and groups who seek counsel with the psychologist, especially with respect to preventing misuse or abuse, while additionally maintaining awareness of how the psychologist's own physical and mental health may influence their work. Among professional interactions and research, psychologists ought to respect and protect the rights and welfare of patients and participants.

Principle B: Fidelity and Responsibility

The fidelity and responsibility principle of the APA general principles inspires psychologists to cultivate a professional and scientific environment built upon trust, accountability, and ethical considerations. Psychologists are bound to the community by way of their profession and must conduct themselves in a responsible and ethical manner while also maintaining a similar check on colleagues. Furthermore, psychologists are expected to altruistically devote some of their time to the community.

Principle C: Integrity

The integrity principle of the APA general principles aims to encourage psychologists to engage in honest, transparent practices within all aspects of the field of psychology. That is, psychologists should not engage in behavior that could be misconstrued as dishonest, exploitative, or otherwise malicious. When deception is appropriately used (most likely during psychological research), psychologists have a responsibility to mitigate the effects of its use on the overall field.

Principle D: Justice

The justice principle of the APA general principles states that people are entitled to the advances made within the field of psychology and to the services offered by professionals within the field. Furthermore, psychologists should prevent unjust practices by remaining aware of their biases, level of competence, and area and limits of expertise.

Principle E: Respect for People's Rights and Dignity

The APA general principle concerning respect for people's rights and dignity recognizes individuals' rights to privacy and confidentiality. Psychologists are to respect the individuals' rights while also acknowledging the worth of the individual by taking judicious precautions and engaging in positive, professional interactions, avoiding the influence of any personal bias towards the individual or group. This entails awareness of the vulnerabilities experienced by any particular population of people and necessitates understanding of and respect for diversity, including, but not limited to, factors concerning gender, race, religion, disability, and socioeconomic status.

Glossary

competencies: a measurable pattern of knowledge, skill, abilities, behaviors, and other characteristics that an individual needs to perform work roles or occupational functions successfully

1. Prahalad, C.K. and Hamel, G. (1990) "The core competence of the corporation Archived 2014-07-14 at the Wayback Machine", Harvard Business Review (v. 68, no. 3) pp. 79–91. ↵

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- A Competency Model of Psychology Practice: Articulating Complex Skills and Practices. **Authored by**: Kathryn Maree von Treuer1 and Nick Reynolds. **Located at**: https://www.frontiersin.org/articles/10.3389/feduc.2017.00054/full. **License**: *CC BY: Attribution*
- Conversion therapy. **Provided by**: Wikipedia. **Located at**: https://en.Wikipedia.org/wiki/Conversion_therapy. **License**: *CC BY-SA: Attribution-ShareAlike*
- Sexual orientation and gender identity: review of concepts, controversies and their relation to psychopathology classification systems.. **Authored by**: Carla Moleiro and Nuno Pinto. **Located at**: https://www.frontiersin.org/articles/10.3389/fpsyg.2015.01511/full. **Project**: Frontiers in Psychology. **License**: *CC BY: Attribution*

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2.12: Ethical Standards in Clinical Psychology

Learning Objectives

- Describe ethical standards and expectations for psychologists including competence, informed consent, confidentiality, relationships with clients and others, and record keeping

Ethical Standards

In addition to the five ethical principles previously mentioned, the Ethics Code also describes ethical standards related to situations many psychologists may encounter in their professional roles. There are 10 standards contained within the Ethics Code, which we briefly summarize below—those related to resolving ethical issues, competence, human relations, privacy and confidentiality, advertising and public statements, record keeping and fees, education and training, research and publication, assessment, and therapy. The ethical standards are enforced for the benefit of the psychologists, clients, students, and other individuals that work with psychologists. Any psychologist that is a member of the APA is expected to adhere to the ethical standards. Any violation of an ethical standard may result in sanctions ranging from termination of APA membership to loss of licensure.

Resolving Ethical Issues

This section of the code explains how psychologists should resolve ethical conflicts between professional organizations, how to cooperate with professional ethics committees, and how to report ethical violations.

Competence

This ethical standard establishes the fact that psychologists must work within the boundaries of their own competence, based on their training, experience, consultation, and supervision. The different types of mental health–related problems and potential treatments are highly varied. It is impossible for psychologists to be competent in all areas, and in fact, unethical to attempt to portray themselves as such. If a psychologist feels that he/she has not been trained to ensure competence in a specific area to treat a client, he or she should make an appropriate **referral**, which is the act of directing a patient to a therapist, physician, agency, or institution for evaluation, consultation, or treatment. This ethical standard also provides psychologists with guidelines on providing services in emergency situations.

Watch It

In addition to maintaining general competence in the field, psychologists also need to demonstrate cultural competence, showing respect and understanding when working with people from varied cultural backgrounds.

In this video, Lilian Comas-Díaz discusses why this is so important as she talks about her book “Multicultural Care: A Clinician’s Guide to Cultural Competence.”



You can view the transcript for "Interview with APA author Lillian Comas-Díaz" here (opens in new window).

Human Relations

The human relations section of the Ethics Code provides criteria for psychologists on how to approach situations related to the process of working with people in a helping field; how to identify and avoid unfair discrimination, sexual harassment, and other types of verbal and nonverbal harassment. These types of behavior have strong adverse influences on mental health. As such, psychologists must be particularly vigilant in identifying and avoiding these kinds of behavior.

Some treatments have been shown to cause harm, and as such, should be avoided. Situations where a clinician has more than one relationship with the client, beyond just being a client, can be difficult to navigate and should be avoided as multiple relationships can also lead to conflicts of interest. A psychologist refrains from entering into a multiple relationship if the multiple relationship could reasonably be expected to impair the psychologist's objectivity, competence, or effectiveness in performing his or her functions as a psychologist, or otherwise risks exploitation or harm to the person with whom the professional relationship exists. The APA Ethics Code definition states that a **multiple relationship** arises when a psychologist is in a professional role with an individual, and (1) at the same time is in another role with the same person, (2) at the same time is in a relationship with a person closely associated with or related to the person with whom the psychologist has the professional relationship, or (3) promises to enter into another relationship in the future with the person or a person closely associated with or related to the person.[1]

This section also provides guidance for cooperating with other professionals, which is often a situation faced in multidisciplinary treatment teams. Guidance is provided for providing and obtaining informed consent for treatment. Another section outlines how to provide psychological services to or through organizations. And lastly, guidance is provided for how to navigate situations in which there is an interruption of psychological services for various reasons.

? Therapist-client Relationships

What is it like to open up and reveal your most vulnerable and painful feelings to another person when you are struggling with depression, anxiety, or any mental disorder? The initial step to seeking therapy can be the hardest to take. And once the step is taken, how do clients experience the relationship with their therapist? The therapeutic relationship is considered by many leading clinicians and theorists to be the main vehicle for client change and it is the means by which a therapist and a client hope to engage with each other. Some may argue that the nature and quality of the therapeutic relationship is *the* most significant curative factor in psychotherapy.

In the humanistic approach, Carl Rogers identified a number of necessary and sufficient conditions that are required for therapeutic change to take place. These include the three core conditions: *congruence* (a state in which a person's ideal self and actual experience are consistent), *unconditional positive regard*, and *empathy*.

In psychoanalysis, the therapeutic relationship, described by Freud (1912) as a friendly affectionate feeling, has been theorized to consist of three parts: the working alliance, transference/countertransference, and the real relationship. The **working alliance** may be defined as the relationship between the client and counselor, aimed at improving outcomes for the patient. **Transference** can be described as the client's distorted perceptions of the therapist, caused when a client redirects feelings they have about someone else, like a significant other or parent, toward the therapist. **Countertransference** is defined as redirection of a therapist's feelings toward a client—or, more generally, as a therapist's emotional entanglement with a client. All of these feelings can have a great effect on the therapeutic relationship. In contrast to a social relationship, the focus of the therapeutic relationship is on the client's needs and goals. Goals are what the client hopes to gain from therapy, based on their presenting concerns. The bond forms from trust and confidence that what the therapist and client agree need to be done to reach the client's goals will bring the client closer to their goals.

Client characteristics like social attitudes toward psychological disorders and attachment style have been found to be related to client use of counseling as well as expectations and outcome. Stigma against mental illness can keep people from acknowledging problems and seeking help. In terms of attachment style, clients with avoidance styles have been found to perceive greater risks and fewer benefits to counseling, and are less likely to seek professional help, than securely attached clients. Those with anxious attachment styles perceive greater benefits as well as risks to counseling. Educating clients about expectations of counseling can improve client satisfaction, treatment duration, and outcomes, and is an efficient and cost-effective intervention.

Psychologists are also interested in how culture relates to help-seeking, counseling process, and outcome. Recent research suggests that clients who are Black are at risk for experiencing racial micro-aggression from counselors who are White.[2] Efficacy for working with clients who are lesbians, gay, or bisexual might be related to therapist demographics, gender, sexual identity development, sexual orientation, and professional experience. Clients who have multiple oppressed identities might be especially at risk for experiencing unhelpful situations with counselors, so counselors might need help with gaining expertise for working with clients who are transgender, lesbian, gay, bisexual, or transgender people of color, and other oppressed populations. The Guidelines for Psychological Practice with Lesbian, Gay and Bisexual Clients provide assistance to psychologists in areas such as religion and spirituality, the differentiation of gender identity and sexual orientation, socioeconomic and workplace issues, and the use and dissemination of research on LGBT issues.

With so many potential forces driving an individual away from seeking therapy, the initial step can indeed be hard to take. Thus, the therapeutic relationship requires the client to be willing to work with the therapist in a partnership, break old patterns, and take the opportunity to talk, think, relate, and grow.

Privacy and Confidentiality

The privacy and confidentiality section is written to help provide psychologists with guidelines for maintaining appropriate confidentiality and respecting the privacy of the clients and patients under their care. Specific guidelines are provided for maintaining confidentiality for the psychologist's patients as well as discussing the limits of confidentiality with them. In certain situations where the safety of the patient or others is at risk, confidentiality must be broken as law enforcement needs to be notified. Sections on disclosures and consultations provide guidance on how and when psychologists should disclose information and how to ethically consult with other professionals while maintaining appropriate levels of confidentiality.

? HIPPA

Most of us feel that our health information is private and should be protected. That is why there is a federal law that sets rules for health care providers and health insurance companies about who can look at and receive our health information. This law, called the Health Insurance Portability and Accountability Act of 1996 (HIPAA), gives you rights over your health information, including the right to get a copy of your information, make sure it is correct, and know who has seen it.

By law, your health information can be used and shared for specific reasons not directly related to your care, like making sure doctors give good care, making sure nursing homes are clean and safe, reporting when the flu is in your area, or reporting as required by state or federal law. In many of these cases, you can find out who has seen your health information. You can do the following:

- Learn how your health information is used and shared by your doctor or health insurer. Generally, your health information cannot be used for purposes not directly related to your care without your permission. For example, your doctor cannot give it to your employer, or share it for things like marketing and advertising, without your written authorization. You probably received a notice telling you how your health information may be used on your first visit to a new health care provider or when you got new health insurance, but you can ask for another copy anytime.
- Let your providers or health insurance companies know if there is information you do not want to share. You can ask that your health information not be shared with certain people, groups, or companies. If you go to a clinic, for example, you can ask the doctor not to share your medical records with other doctors or nurses at the clinic. You can ask for other kinds of restrictions, but they do not always have to agree to do what you ask, particularly if it could affect your care. Finally, you can also ask your health care provider or pharmacy not to tell your health insurance company about care you receive or drugs you take, if you pay for the care or drugs in full and the provider or pharmacy does not need to get paid by your insurance company.
- Ask to be reached somewhere other than home. You can make reasonable requests to be contacted at different places or in a different way. For example, you can ask to have a nurse call you at your office instead of your home or to send mail to you in an envelope instead of on a postcard.

Advertising and Other Public Statements

The advertising and other public statements section is designed to guide psychologists through the process of advertising their practice and making other types of public statements.

Record Keeping and Fees

The record-keeping and fees standard is developed to guide psychologists in maintaining records of professional and scientific work in confidentiality. This standard states that maintaining records allow professionals to share information with other professionals if needed, help replicate research findings, and abide by the requirements of the institution and the law.

Education and Training

The education and training standard is designed to help psychologists create high-quality programs that train future psychologists with appropriate knowledge and practice.

Research and Publication

The research and publication standard is developed to highlight research and publication ethics that psychologists are expected to adhere to. This standard emphasizes the necessity of approval by the institute prior to carrying out the research, providing accurate information about the research study, and carrying out the research in accordance with the approval. When obtaining informed consent, details of the study should be presented to the participants, including but not limited to, the objective of the study, the procedures, benefits, and potential risks associated with the study, and the participants' right to decline to participate and withdraw from the study without any penalty. However, for studies that are not expected to cause any harm, such as observing in a naturalistic environment, using anonymous questionnaires, or if permitted by the law, it may not be necessary to obtain informed consent.

Compensation for participation should not be used to persuade an unwilling participant. At the conclusion of the study, each participant should be presented with a summary of the study and the participant should be provided with an opportunity to ask any questions he or she has. Further, if there has been some harm caused to the participant due to participation in the study, necessary steps should be followed to minimize the harm.

This standard also provides guidelines and instructions on animal research. Psychologists are responsible for reporting accurate findings and taking the necessary steps to correct any errors in research and publication. Psychologists are also required to only present original data as their work and share publication credits based on the contributions rather than authority. Psychologists are also expected to be willing to share research data when required for verification and maintain the confidentiality of the participants during the review process.

Assessment

The assessment standard is developed to broadly address guidelines on assessments. This standard states that the psychologist's views should be supported by findings from assessments while reporting the limitations of assessments. Psychologists are responsible for using valid and reliable assessments that are administered in a preferred language by the client. Informed consent is expected to be obtained in accordance to the guidelines on the "Informed Consent" standard, unless the assessment is required by the law, a routine practice, or required to test for the ability to make decisions. Psychologists are required to be appropriately trained in order to administer assessments and are responsible for using current tests.

Therapy

The therapy section is broadly written to guide psychologists through various aspects of providing psychological services. Psychologists are required to obtain **informed consent** from clients prior to treatment by presenting sufficient details about the therapy technique, including how established the treatment is and whether a psychologist in training will be assisting the client. It is necessary to communicate with the client about the potential risks and benefits of therapy, confidentiality and its limits, and the expected length of therapy. When psychologists are required to provide services in a group setting, psychologists are required to identify the role of the psychologist and notify the clients of the limitations of confidentiality. If a psychologist is required to provide services for a client already receiving mental health services from another professional, the psychologist is required to discuss with the client or other identified persons to minimize conflict and harm. Psychologists should not engage in sexual relationships with current clients or with those closely related to clients. Psychologists should not be sexually engaged with past clients within two years of termination, and even after two years of termination, psychologists will have to provide enough details to ensure that this ethical standard is not violated by being involved with a past client. In the event of a termination of employment, necessary steps should be taken in order to ensure client care. Termination of therapy should occur when the client shows significant improvements, does not benefit further from treatment, is being harmed by the treatment, or the clinician is threatened by the client or a person related to the client. Psychologists are expected to prepare clients for termination and provide sources for alternative services.

Glossary

countertransference: redirection of a therapist's feelings toward a client or a therapist's emotional entanglement with a client

informed consent: details provided prior to treatment about the therapy technique, the potential risks and benefits of therapy, confidentiality and its limits, and the expected length of therapy

multiple relationship: occurs when a psychologist is in a professional role with a person and (1) at the same time is in another role with the same person, (2) at the same time is in a relationship with a person closely associated with or related to the person with whom the psychologist has the professional relationship, or (3) promises to enter into another relationship in the future with the person or a person closely associated with or related to the person.

referral: the act of directing a patient to a therapist, physician, agency, or institution for evaluation, consultation, or treatment

transference: a client's distorted perceptions of a therapist

working alliance: the relationship between the client and counselor, aimed at improving outcomes for the client

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2.13: Legal and Forensic Issues in Abnormal Psychology

Learning Objectives

- Explain forensic issues in psychological treatment, such as the insanity defense, competency to stand trial, and understanding the purpose of punishment



Figure 2.13.1: Jared Lee Loughner, a convicted mass murderer, was originally ruled incompetent to stand trial due to a diagnosis of paranoid schizophrenia but was later ruled competent and sentenced to life plus 140 years in federal prison.

Jared Lee Loughner (born September 10, 1988) is a convicted American mass murderer who pled guilty to 19 charges of murder and attempted murder in connection with the January 8, 2011, Tucson shooting. After his arrest, two medical evaluations diagnosed Loughner with paranoid schizophrenia and ruled him incompetent to stand trial. Punishment or treatment? What should we do when someone who is mentally ill but commits a horrendous crime?

In this section, we will discuss the forensic issues in psychological treatment, such as the insanity defense, competency to stand trial, and the purpose of punishment. We will also examine the ethical and legal issues in providing service and the rights of mentally ill patients. What is the legal responsibility or duty of mental health care providers to protect individuals who are being threatened with bodily harm by a patient in their care. Should we honor the right to refuse treatment when someone is psychologically disturbed? Should psychiatrists administer antipsychotics or any other drug deemed necessary to treat patients against their will? Should mentally ill clients with a history of violent behavior be recommended for commitment or involuntary hospitalization? How do we balance the rights of the individual with the rights of society? Does the answer to these questions lie with the criminal justice system or with the mental health system?

Below, we begin by summarizing the major perspectives on ethical and forensic issues in psychological treatment.

Tarasoff v. Regents of the University of California

Tarasoff v. Regents of the University of California, (Cal. 1976), was a case in which the Supreme Court of California held that mental health professionals have a **duty to protect** individuals who are being threatened with bodily harm by a patient. The original 1974 decision mandated warning the threatened individual, but a 1976 rehearing of the case by the California Supreme Court called for a “duty to protect” the intended victim. The professional may discharge the duty in several ways, including notifying police, warning the intended victim, and/or taking other reasonable steps to protect the threatened individual.

History

Prosenjit Poddar was a student from Bengal, India. He entered the University of California, Berkeley, as a graduate student in September 1967 and resided at International House. In the fall of 1968, he attended folk dancing classes at the International House, and it was there that he met Tatiana Tarasoff. They dated, but apparently had different ideas about the relationship. He assumed their relationship was serious. This view was not shared by Tarasoff who, upon learning of his feelings, told him that she was involved with other men and that she was not interested in entering into an intimate relationship with him. This statement gave rise to feelings of resentment in Poddar. He began to stalk her.

After this rebuff, Poddar underwent a severe emotional crisis. He became depressed and neglected his appearance, his studies, and his health. He kept to himself, speaking disjointedly and often weeping. This condition persisted, with steady deterioration, throughout the spring and into the summer of 1969. Poddar had occasional meetings with Tarasoff during this period and tape-recorded their various conversations to try to find out why she did not love him.

During the summer of 1969, Tarasoff went to South America. After her departure, Poddar began to improve and at the suggestion of a friend sought psychological assistance. Prosenjit Poddar was a patient of Dr. Lawrence Moore, a psychologist at UC Berkeley's Cowell Memorial Hospital in 1969. Poddar confided his intent to kill Tarasoff. Dr. Moore requested that the campus police detain Poddar, writing that, in his opinion, Poddar was suffering from paranoid schizophrenia, acute and severe. The psychologist recommended that the defendant be civilly committed as a dangerous person. Poddar was detained but shortly thereafter released, as he appeared rational. Dr. Moore's supervisor, Dr. Harvey Powelson, then ordered that Poddar not be subject to further detention.

In October, after Tarasoff had returned, Poddar stopped seeing his psychologist. Neither Tarasoff nor her parents received any warning of the threat. Poddar then befriended Tarasoff's brother, even moving in with him. Several weeks later, on October 27, 1969, Poddar carried out the plan he had confided to his psychologist, stabbing and killing Tarasoff. Tarasoff's parents then sued Moore and various other employees of the university.

Poddar was subsequently convicted of second-degree murder, but the conviction was later appealed and overturned on the grounds that the jury was inadequately instructed. A second trial was not held, and Poddar was released on the condition that he would return to India.

Opinion of the court

The California Supreme Court found that a mental health professional has a duty not only to a patient, but also to individuals who are specifically being threatened by a patient. This decision has since been adopted by most states in the United States and is widely influential in jurisdictions outside the United States as well.

Subsequent developments

As of 2012, a ***duty to warn or protect*** is mandated and codified in legislative statutes of 23 states, while the duty is not codified in a statute but is present in the common law supported by precedent in 10 states. Eleven states have a permissive duty, and six states are described as having no statutes or case law offering guidance.

In 2018, the Court held that universities should protect students in the *Regents of University of California v. Superior Court of Los Angeles County.*

Commitment of Clients

Starting in the 1960s, there has been a worldwide trend toward moving psychiatric patients from hospital settings to less restricting settings in the community, a shift known as "deinstitutionalization." Because the shift was typically not accompanied by a commensurate development of community-based services, critics say that deinstitutionalization has led to large numbers of people who would once have been inpatients as instead being incarcerated or becoming homeless. In some jurisdictions, laws authorizing court-ordered outpatient treatment have been passed in an effort to compel individuals with chronic, untreated, severe mental illness to take psychiatric medication while living outside the hospital (see Laura's Law, Kendra's Law).

In most jurisdictions, **involuntary commitment** is applied to individuals believed to be experiencing a mental illness that impairs their ability to reason to such an extent that the agents of the law, state, or courts determine that decisions will be made for the individual under a legal framework. This is a proceeding distinct from being found incompetent and limits involuntary treatment to individuals who meet statutory criteria for presenting a danger to self or others. **Involuntary commitment** is a legal process through which an individual who is deemed by a qualified agent to have symptoms of severe mental disorder is detained in a psychiatric hospital (inpatient) where they can be treated involuntarily. This treatment may involve the administration of psychoactive drugs including involuntary administration. In many jurisdictions, people diagnosed with mental health disorders can also be forced to undergo treatment while in the community, this is sometimes referred to as outpatient commitment and shares legal processes with commitment.

A number of civil and human rights activists, anti-psychiatry groups, medical and academic organizations, researchers, and members of the psychiatric survivors movement vigorously oppose involuntary treatment on human rights grounds or on grounds of effectiveness and medical appropriateness, particularly with respect to involuntary administration of mind-altering substances, ECT, and psycho-surgery. Some criticism has been made regarding cost, as well as of conflicts of interest with the pharmaceutical industry. Critics, such as the New York Civil Liberties Union, have denounced the strong racial and socioeconomic biases in forced treatment orders. Supporters of involuntary treatment include organizations such as the National Alliance on Mental Illness (NAMI) and the American Psychiatric Association.

Right to treatment and Refusal of Treatment

In October 1970, Ricky Wyatt was a fifteen-year-old who had always been labeled a "juvenile delinquent." Despite not being indicated with a mental illness, he was a patient at Bryce Hospital (first known as the *Alabama State Hospital for the Insane*) and became the named plaintiff in a landmark class-action lawsuit, *Wyatt v. Stickney*. His aunt, W. C. Rawlins, was one of the employees at Bryce who had been laid off. Together they testified about intolerable conditions and improper treatments designed only to make the patients more manageable. In 1971, the plaintiff class was expanded to include patients at Alabama's two other inpatient mental health facilities. The **right to treatment** emerged as the outcome and resulted in federal minimum standards for the care of people with mental illness or mental retardation who reside in institutional settings.

The standards elaborated in that agreement have served as a model nationwide. Known as the "Wyatt Standards," they are founded on four criteria for evaluation of care:

- humane psychological and physical environment
- qualified and sufficient staff for administration of treatment
- individualized treatment plans
- minimum restriction of patient freedom.

The case of *Wyatt v. Stickney* came to a conclusion after 33 years, through the tenure of nine Alabama governors and fourteen state mental health commissioners, the longest mental health case in national history. The State of Alabama estimates its litigation expenses at over $15 million.

Just as clients have a right to treatment, they also have a right to refuse unwanted treatment. **Informed refusal** is where a person has refused a recommended medical treatment based on an understanding of the facts and the implications of not following the treatment. Informed refusal is linked to the informed consent process, as a patient has a right to consent, but also may choose to refuse.

The individual needs to be in possession of the relevant facts as well as of his or her reasoning faculties, such as not being intellectually disabled or mentally ill and without impairment of judgment at the time of refusing. In cases where an individual is considered unable to give informed refusal, another person (a guardian) may be authorized to give consent on his or her behalf. **Substituted judgment** generally is a decision made by a person on behalf of a person who is incompetent and unable to decide for himself or herself. Substituted judgment with regard to guardianship is the standard that directs the decision making of a guardian.

When refusal of treatment may result in significant damage or death, the interaction needs to be documented to protect the care giver in a potential later litigation against the allegation that the recommendation was either not made or not understood. On occasion, a patient will also refuse to sign the "informed refusal" document, in which case a witness would have to sign that the informed process and the refusal took place. The pregnant patient represents a specific dilemma in the field of informed refusal as her action may result in harm or death to the fetus. Ethicists disagree on how to handle this situation.

Clients also have the right for placement in a **least restrictive alternative** to treatment in an institution. ***O'Connor v. Donaldson***, 422 U.S. 563 (1975), was a landmark decision of the U.S. Supreme Court in mental health law ruling that a state cannot constitutionally confine a non-dangerous individual who is capable of surviving safely in freedom by themselves or with the help of willing and responsible family members or friends. Kenneth Donaldson (confined patient diagnosed with paranoid schizophrenia and civilly committed) had been held for 15 years in Florida State Hospital at Chattahoochee, due to needs of "care, maintenance, and treatment." He filed a lawsuit against the hospital and staff members claiming they had robbed him of his constitutional rights, by confining him against his will. Donaldson won his case since the trial court jury found, upon ample evidence, that the petitioner had violated Donaldson's right to liberty. A finding of mental illness alone cannot justify a State's locking a person up against his

will and keeping him indefinitely in simple custodial confinement. Assuming that that term can be given a reasonably precise content and that the mentally ill can be identified with reasonable accuracy, there is still no constitutional basis for confining such persons involuntarily if they are dangerous to no one and can live safely in freedom.

The Insanity Defense

The **insanity defense** is an affirmative defense by excuse in a criminal case, arguing that the defendant is not responsible for his or her actions due to an episodic or persistent psychiatric disorder at the time of the criminal act. Legal definitions of insanity or mental disorder are varied, and include the **M'NaghtenRule**, the **Durhamrule**, the **ALIrule** (American Legal Institute Model Penal Code rule), and other provisions, often relating to a lack of *mens rea* ("guilty mind").

Being **incapable of distinguishing right from wrong** is one basis for being found to be legally insane as a criminal defense. It originated in the *M'Naghten Rule*, formulated as a reaction to the acquittal in 1843 of Daniel M'Naghten, a Scottish woodturner who assassinated an English civil servant while suffering from paranoid delusions. M'Naghten had mistaken him as the UK Prime Minister, the intended target, and believed he was following the commands of a higher power. The central issue of the *M'Naghten Rules* may be stated as, "did the defendant know what they were doing, or, if so, that it was wrong?"

Legislators added the **irresistible impulse** defense, in which the defendant argues that they should not be held criminally liable for their actions that broke the law, because they could not control those actions, even if they knew them to be wrong. In 1994, Lorena Bobbitt was found not guilty when her defense argued that an *irresistible impulse* led her to cut off her husband's penis with a knife while he was asleep in bed. The husband-wife relationship made world-wide headlines in 1993 and revealed that Lorena has been subjected to years of being raped, beaten, and sodomized by her husband. It is a variant of the M'Naghten Rules that addresses the situation in which defendants knew that what they were going to do was wrong, but had no ability to restrain themselves from doing it.

The **Durham rule** later expanded the insanity defense and established that a jury may determine a defendant is not guilty by reason of insanity because the criminal act was the product of a mental disorder. Examples in which such rules were articulated *State v. Pike* (1869) and *Durham v. United States* (1954). After the 1970s, U.S. jurisdictions have tended to not recognize this argument as it places emphasis on "mental disease or defect" and thus on testimony by psychiatrists and is argued to be somewhat ambiguous. It gave psychiatric and psychological experts too much influence in a decision of insanity and not enough to jurors.

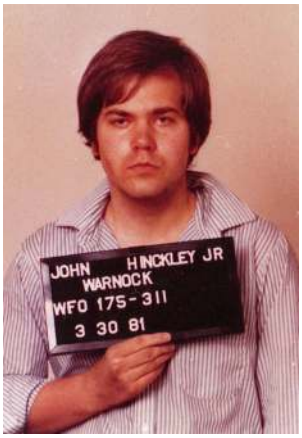


Figure **2.13.2**: On March 30, 1981, United States President Ronald Reagan was shot and wounded by John Hinckley Jr. in Washington, D.C. as he was returning to his limousine after a speaking engagement at the Washington Hilton Hotel. Hinckley believed the attack would impress actress Jodie Foster, with whom he had become obsessed.

The **ALI rule**, or **American Law Institute Model Penal Code rule**, is a recommended rule for instructing juries how to find a defendant in a criminal trial is not guilty by reason of insanity. It broadened the M'Naghten rule. It arose from the case of *United States v. Brawner.* The case overturned the Durham rule (that put substantial dominance on the testimony of experts) gave jurers a specific framework for the insanity defense. The focus is on the acts and mental state of a defendant at the time he committed the acts constituting the crime.

The ALI rule is:

> *"(1) A person is not responsible for criminal conduct if at the time of such conduct as a result of mental disease of defect he lacks substantial capacity either to appreciate the criminality of his conduct or to conform his conduct to the requirements of the law.*
>
> *"(2) As used in this Article, the terms "mental disease or defect" do not include an abnormality manifested only by repeated criminal or otherwise antisocial conduct [Section 4.01]."*[1]

John Hinckley, Jr. is an American who, on March 30, 1981, attempted to assassinate U.S. President Ronald Reagan in Washington, D.C. He wounded Reagan with a bullet from a revolver that ricocheted and hit Reagan in the chest. He also wounded police officer Thomas Delahanty and Secret Service agent Tim McCarthy, and he critically wounded Press Secretary James Brady, who was permanently disabled in the shooting and later died from those injuries. Hinckley was reportedly seeking fame in order to impress actress Jodie Foster, on whom he had an obsessive fixation. He was found not guilty by reason of insanity and remained under institutional psychiatric care until September 10, 2016. Public outcry over the verdict led to the Insanity Defense Reform Act of 1984, which altered the rules for consideration of mental illness of defendants in Federal Criminal Court proceedings in the United States. In January 2015, federal prosecutors announced that they would not charge Hinckley with Brady's death, despite the medical examiner's classification of his death as a homicide. Along with Lynette Fromme and Sara Jane Moore, Hinckley is one of three attempted presidential assassins currently living who are not in prison. Hinckley is portrayed by Steven Flynn in the American television film, *Without Warning: The James Brady Story* (1991).

Competence to Stand Trial

The word **incompetent** is used to describe persons who should not undergo or partake in certain judicial processes, and also for those who lack the mental capacity to make contracts, handle their financial and other personal matters such as consenting to medical treatment, etc. and need a legal guardian to handle their affairs. When it comes to **competence to stand trial** and the law, the right to not be prosecuted while one is incompetent to stand trial has been ruled by the United States Supreme Court to be guaranteed under the due process clause, which acts as a safeguard from arbitrary denial of life, liberty, or property by the government outside the sanction of law. ***Dusky v. United States***, (1960), was a landmark United States Supreme Court case in which the Court affirmed a defendant's right to have a competency evaluation before proceeding to trial.

If the court determines that a defendant's mental condition makes him unable to understand the proceedings, or that he is unable to help in his defense, he is found incompetent. Being determined incompetent is substantially different from undertaking an insanity defense; competence regards the defendant's state of mind at the time of the trial, while insanity regards his state of mind at the time of the crime. A ruling of incompetence may later be reversed. A defendant may recover from a mental illness or disability, and a court may require a defendant to undergo treatment in an effort to render the defendant competent to stand trial. For example, in 1989, Kenneth L. Curtis of Stratford, Connecticut, was found mentally incompetent to stand trial following the murder of his estranged girlfriend. But years later, as he had attended college and received good grades, this ruling was reversed, and he was ordered to stand trial.

Understanding the Purpose of Punishment

Is a mentally ill person convicted of a capital offense able or competent enough to understand the nature and purpose of the death sentence? The case of Alvin Bernard Ford highlights the complexity of the issue. ***Ford v. Wainwright***, 477 U.S. 399 (1986), was a landmark case that upheld the common law rule that t*he insane cannot be executed.* Ford was convicted of murder in 1974 and sentenced to death in Florida. In 1982, while on death row, Ford's mental health diminished to a point resembling paranoid schizophrenia. Ford began referring to himself as Pope John Paul III and reported such accomplishments as thwarting a vast Ku Klux Klan conspiracy to bury dead prisoners inside the prison walls, foiling an attempt by prison guards to torture his female relatives inside the prison, and personally appointing nine new justices to the Florida Supreme Court. A panel of three psychiatrists was eventually called to examine Ford's behavior, and it concluded that while Ford suffered from psychosis and various mental disorders, he was still capable of understanding the nature of the death penalty and the effect that such a penalty would have on him. A death warrant was signed for Ford in 1984. Ford sued the secretary of the Florida Department of Corrections, Louie L. Wainwright, and took his case to the United States Supreme Court, declaring he was not competent to be executed. The court ruled in his favor, then further addressed the procedural issues present in making a determination of insanity for Eighth Amendment concerns (cruel and unusual punishment). The Court, in an opinion by Justice Marshall, reviewed the evolving standards of the

Eighth Amendment to be those consistent with “the progress of a maturing society,” and one not tolerable of acts traditionally branded as “savage and inhumane,” as the execution of the mentally insane. The inmate was transferred to Florida State Hospital for treatment after he was reevaluated and found to be incompetent to be executed. In 1989, a federal district judge ruled that Ford was sane, but defense lawyers appealed that ruling. The appeal was pending when Ford died on February 6, 1991 of natural causes, at 37.

Today, an inmate on death row has a right to be evaluated for competency by a forensic expert to determine if the sentence can be carried out. If the inmate is found incompetent, a forensic professional must provide treatment to aid in his gaining competency so the execution can take place.

Learning Objectives

ALI rule: a recommended rule for instructing juries how to find a defendant in a criminal trial is not guilty by reason of insanity; the focus is on the acts and mental state of a defendant at the time he committed the acts constituting the crime

competence to stand trial: the right to not be prosecuted while one is incompetent to stand trial

Durham rule: established that a jury may determine a defendant is not guilty by reason of insanity because the criminal act was the product of a mental disorder

duty to warn or protect: holds that mental health professionals have a duty to protect individuals who are being threatened with bodily harm by a patient

guardian: a person authorized to give consent on behalf of a person who is unable to decide for himself or herself

incompetent: used to describe persons who should not undergo or partake in certain judicial processes, and also for those who lack mental capacity to make contracts, handle their financial and other personal matters such as consenting to medical treatment, etc., and need a legal guardian to handle their affairs

insanity defense: an affirmative defense by excuse in a criminal case, arguing that the defendant is not responsible for his or her actions due to an episodic or persistent psychiatric disorder at the time of the criminal act

involuntary commitment: civil commitment or involuntary hospitalization—a legal process through which an individual who is deemed by a qualified agent to have symptoms of severe mental disorder is detained in a psychiatric hospital (inpatient) where they can be treated involuntarily

irresistible impulse: defense in which the defendant argues that they should not be held criminally liable for their actions that broke the law because they could not control those actions, even if they knew them to be wrong

least restrictive alternative: law ruling that a state cannot constitutionally confine a non-dangerous individual who is capable of surviving safely in freedom

right to treatment: federal minimum standards for the care of people with mental illness or mental retardation who reside in institutional settings

substituted judgment: a decision made by a person on behalf of a person who is incompetent and unable to decide for himself or herself

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1. Criminal Law—Cases and Materials, 7th ed. 2012, Wolters Kluwer Law & Business; John Kaplan, Robert Weisberg, Guyora Binder, ISBN 978-1-4548-0698-1 ↵

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2.14: Putting It Together- Psychological Research



Figure **2.14.1**: The scientific method.

Psychologists use the scientific method to examine human behavior and mental processes. Some of the methods you learned about include descriptive, experimental, and correlational research designs.

Case Study: a detailed analysis of a particular person, group, business, event, etc. This approach is commonly used to learn more about rare examples with the goal of describing that particular thing.

- Ted Bundy was one of America's most notorious serial killers who murdered at least 30 women and was executed in 1989. Dr. Al Carlisle evaluated Bundy when he was first arrested and conducted a psychological analysis of Bundy's development of his sexual fantasies merging into reality (Ramsland, 2012). Carlisle believes that there was a gradual evolution of three processes that guided his actions: fantasy, dissociation, and compartmentalization (Ramsland, 2012). Read *Imagining Ted Bundy* for more information on this case study.

Naturalistic Observation: a researcher unobtrusively collects information without the participant's awareness.

- Drain and Engelhardt (2013) observed six nonverbal children with autism's evoked and spontaneous communicative acts. Each of the children attended a school for children with autism and were in different classes. They were observed for 30 minutes of each school day. By observing these children without them knowing, Drain and Engelhardt were able to see true communicative acts without any external influences.

Survey: participants are asked to provide information or responses to questions on a survey or structure assessment.

- Educational psychologists can ask students to report their grade point average and what, if anything, they eat for breakfast on an average day. A healthy breakfast has been associated with better academic performance (Digangi's 1999).

Archival research: researchers examine data that has already been collected for other purposes.

- Anderson (1987) tried to find the relationship between uncomfortably hot temperatures and aggressive behavior, which was then looked at with two studies done on violent and nonviolent crime. Based on previous research that had been done by Anderson and Anderson (1984), it was predicted that violent crimes would be more prevalent during the hotter time of year and the years in which it was hotter weather in general. The study confirmed this prediction.

Longitudinal Study: researchers recruit a sample of participants and track them for an extended period of time.

- In a study of a representative sample of 856 children, Eron and his colleagues (1972) found that a boy's exposure to media violence at age eight was significantly related to his aggressive behavior ten years later, after he graduated from high school.

Cross-Sectional Study: researchers gather participants from different groups (commonly different ages) and look for differences between the groups.

- In 1996, Russell surveyed people of varying age groups and found that people in their 20s tend to report being more lonely than people in their 70s.

Correlational Design: two different variables are measured to determine whether there is a relationship between them.

- Thornhill et al. (2003) had people rate how physically attractive they found other people to be. They then had them separately smell t-shirts those people had worn (without knowing which clothes belonged to whom) and rate how good or bad their body odor was. They found that the more attractive someone was, the more pleasant their body order was rated to be.

Experiment: researchers create a controlled environment in which they can carefully manipulate at least one variable to test its effect on another. The key here is that the researchers can cause a change in one variable.

- Clinical psychologists can test a new pharmaceutical treatment for depression by giving some patients the new pill and others an already-tested one to see which is the more effective treatment.

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CHAPTER OVERVIEW

3: Etiology and Treatment of Mental Disorders

Understanding the causes of mental disorders is essential for effective intervention. This book examines biological, psychological, and sociocultural influences on mental health. Various treatment modalities, including psychoanalytic, behavioral, cognitive, humanistic, and biomedical therapies, are analyzed in-depth. The importance of cultural competence in therapy is also highlighted.

3.1: Why It Matters- Etiology and Treatment
3.2: Introduction to Multidimensional Models of Psychopathology
3.3: Multidimensional Approaches to Understanding Mental Illness
3.4: Biological Elements of Psychopathology
3.5: The Brain and Psychopathology
3.6: Biomedical Therapies
3.7: Introduction to Psychodynamic and Humanistic Approaches to Psychopathology
3.8: The Psychoanalytic Model
3.9: Psychoanalytic and Psychodynamic Therapies
3.10: Humanistic and Existential Models
3.11: Introduction to Behavioral and Cognitive Approaches to Psychopathology
3.12: The Behavioral Model
3.13: Behavior Therapy
3.14: The Cognitive Model
3.15: Cognitive-Behavioral Therapy
3.16: Integrative and Emerging Therapies
3.17: Introduction to Modalities and Culture
3.18: Treatment Modalities
3.19: The Sociocultural Perspective and Cultural Competence
3.20: Putting It Together- Etiology, Models, and Therapies

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3.1: Why It Matters- Etiology and Treatment



Figure **3.1.1**: Many forms of therapy have been developed to treat a wide array of problems. These marines who served in Iraq and Afghanistan, together with community mental health volunteers, are part of the Ocean Therapy program at Camp Pendleton, a program in which learning to surf is combined with group discussions. The program helps vets recover, especially vets who suffer from post-traumatic stress disorder (PTSD).

Why does one person in a family develop depression while the other sibling does not? If a person's parent or parents were alcoholic, what is the chance that his or her children will develop the same disorder? These are questions related to *etiology*—the causes of mental disorders. You have already learned about the history of how people and clinicians attempted to explain the development of mental disorders and some of the early treatments that were used as a result of those explanations. In this module, we will expand on what you have learned by exploring the best current scientific understanding of what causes mental disorders, discuss how they arise, and introduce how they can be treated. Understanding these issues is essential to combating the stigma against mental disorders and helping all of us and society find better ways to support those who are struggling with symptoms.

In this module, you will see that treatment approaches to mental illness include biological, psychological, and social interventions, all with the goal of alleviating distress and enhancing the person's ability to cope. Because psychological problems can originate from various sources—biology, genetics, childhood experiences, conditioning, and sociocultural influences—clinicians have developed many different therapeutic techniques and approaches. The Ocean Therapy program shown in Figure 1 uses multiple approaches to support the mental health of veterans in the group.

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3.2: Introduction to Multidimensional Models of Psychopathology

What you'll learn to do: describe how multidimensional models and perspectives aid in understanding the etiology and treatment of mental disorders



As you have learned already, humans have a natural tendency to seek simple answers to the problems we face. In the past, this tendency led to beliefs that mental disorders were caused by divine disfavor, demonic possession, or bodily humors. Unfortunately, this human bias is often wrong, especially when we are trying to understand complex situations. For example, is there a simple answer as to what causes cancer? Pollution? Climate change? Crime? Pandemics? Simple, one-dimensional answers are not able to explain, let alone solve, complex problems because these situations, including mental disorders, are systemic in nature: they are influenced by multiple causes that interact with each other and can change over time.

One example of the tendency to seek simplistic answers is the commonly heard belief that mental disorders are caused by "a chemical imbalance" in the brain. The more we understand about mental disorders, the more we realize that this statement is a fable, an overly simplified, and incorrect understanding of systemic conditions. If we are to make progress in reducing stigma against the mentally ill and improving the effectiveness of treatments, we need to begin by seeking to understand more multidimensional models and explanations of mental disorders and seek to understand how the different elements interact. This section will help you do this, and it will lay the foundation for you to have a better understanding of how psychologists view and treat mental disorders. First, we will take a look at multidimensional models, and then dive deeper into the biological understanding of the etiology and treatment of mental disorders.

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3.3: Multidimensional Approaches to Understanding Mental Illness

Learning Objectives

- Explain why unidimensional explanations of mental disorders are insufficient
- Explain the interaction between biological, psychological, and social factors in the development of psychopathology
- Describe how the diathesis-stress model and gene-environment correlation models explain the etiology of mental disorders

Problems with Unidimensional Models in Understanding Mental Illness



Figure **3.3.1**: Just as a car is a system of parts working together, our brains and bodies are complex systems with a multitude of parts and pieces. Mental disorders do not have any single, simple biological or psychological explanations but are systemic outcomes, influenced by multiple factors, including biological, psychological, social, and cultural forces that regularly interact.

A **system** is a set of elements that interact or are interdependent on one another, but forms a coherent whole and interacts with its environment and/or other systems. For example, the car or bus you use for transportation is a mechanical system. It has multiple parts that interact such as the fuel system, the electrical system, the engine, the drive train, the wheels, etc. All these parts work together in a unified way to create movement and speed. If any part of the system fails, such as the engine, the electrical system, or the wheels, the system experiences dysfunction and may no longer work well. In this example, the car is a system and its purpose is transportation or movement. Cars or buses interact with street systems, highway systems, traffic patterns and so, forth as well as gas stations and more, which represent other, larger systems of which it is a part. Similarly, our own bodies are a biological system made up of interdependent parts.

Our brains, heart, circulatory system, respiration, digestion, etc., are all interdependent parts that work together. If one part of the system becomes damaged or disabled such as by high blood pressure, diabetes, or a broken leg, it affects the entire system. Our body/biological systems together make life, and of course, we interact with our environment and many other social systems around us such as the medical system, families, schools, churches, and more.

Multidimensional Approaches to Mental Disorders



Figure **3.3.2**: Multidimensional approaches to explaining behavior contend that mental illness is not simply caused by “chemicals in the brain.”

Understanding how systems work, and how interdependent the parts are, helps us to realize why simplistic, unidimensional explanations of mental disorders such as that they are caused by a “chemical imbalance”—a reductionistic or overly simplified biological explanation—or alternatively, that they are caused by a negative relationship with one’s mother—an overly simplified psychological and social explanation—are inadequate to fully understand the disorder and its causes. Unidimensional explanations limit both our ability to understand how a mental disorder develops and may limit our ability to find effective treatments. If we are

to make progress in reducing stigma against the mentally ill and improving the effectiveness of treatments, we need to begin by seeking to understand more **multidimensional models** and consider how multiple systems interact in contributing to mental disorders.

To be fair, there are a few specific conditions that mimic mental disorders that can be attributed to a single, unitary cause. An example of this is untreated syphilis, which can spread throughout the body, creating damage in multiple organ systems including the brain and lead to psychosis and dementia, possibly even death. However, these types of conditions today are rare, and would not be classified as mental disorders because they are thought of more as medical conditions with biological treatments. In contrast, mental disorders such as attention deficit hyperactivity disorder, major depression, anorexia nervosa, or schizophrenia do not have any single, simple biological or psychological explanation; they are systemic outcomes, influenced by multiple factors, including biological (genetics, hormones, neurotransmitters, and other systems), psychological (learned behaviors, attitudes, emotional responses, cognitive, or social developmental stage), and social and cultural forces (social support or lack of it, rejection, stress) that frequently interact.

For instance, there are some studies that implicate prenatal exposure to a virus, which has cultural implications as well as biological, in the development of some forms of schizophrenia. **Epigenetics** is a newer field of study shedding light on how social, psychological, and environmental influences can actually change which genes in a person's DNA may activate or may be turned off, having significant implications for the development of disorders. Epigenetic effects are a normal part of human development. Every cell in your body has the same DNA, the same genetic code for an entire human being. But as cells develop in the body, they become specialized, meaning that some genes may be activated in some cells and not in the other cells. A good example are the genes that allow cells to detect light; these are turned on or activated in your eye cells, but are turned off or deactivated in all other cells.[1] Thus, it is not surprising that some environmental events or experiences may activate or deactivate specific genes that may increase or decrease (in the case of protective factors like positive social support) the chance someone may develop a mental disorder. Environmental epigenetic effects do not *change* the DNA itself but influence the expression of our genes. Thus, it is clear that both nature *and* nurture are relevant to all forms of mental disorders, not just one or the other.

This modern, multidimensional approach to understanding mental disorders can help us to avoid labeling persons with mental disorders (or even medical illnesses) as being due to just one cause, thereby reducing stigma. We need to approach each person individually and seek to understand their own biological, psychological, and social history to better understand them and their symptoms. You will learn later in this module that most approaches to the treatment of mental disorders arose from unidimensional perspectives on mental disorders; clinicians today (and researchers), even when they may focus on a particular approach or way of treating a mental disorder, will better serve their clients by recognizing that they are approaching only one element of a system and be open to new information and thorough in their assessments.

The next several sections of this module will focus on describing different "models" that explain how mental disorders occur. We begin by exploring multidimensional models and then will explore unidimensional parts or elements of the biopsychosocial system. These elemental models are important because they reflect the history of scientific attempts to understand the etiology of mental illnesses in a coherent way. Newer models built on older ones or were reactions against previous thinking. Although most of them are unidimensional and therefore limited, they each explore different aspects of the whole multidimensional picture and can be useful when we think about what each of them teaches us. These models also lead to the development of different treatment approaches or "orientations."

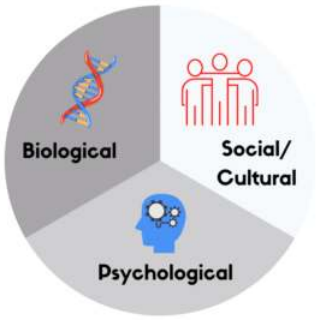


Figure **3.3.3**: The biopsychosocial model emphasizes the interaction between biological, psychological, and social/cultural forces.

The major multidimensional model that is useful in understanding the causes of mental disorders is usually called the **biopsychosocialmodel** (BPS). The biopsychosocial model (BPS) was first proposed by George L. Engel and Jon Romano of the University of Rochester in 1977. As opposed to the medical model, which emphasizes mental illness as disease, this model aimed for a more holistic approach by recognizing that each patient has his or her own thoughts, feelings, and history. In developing the model, Engel framed this model for both medical illnesses and mental problems.

Specifically, Engel revolutionized medical thinking by removing the supposed separation of body and mind. The idea of mind-body dualism goes back at least to the French philosopher René Descartes in the 17th century, but was forgotten during the emergence of the biomedical approach, which largely ignored individual psychological and social development. Engel emphasized that the biomedical approach is flawed because the body alone does not contribute to illness. Instead, the individual's mind (psychological and social factors) plays a significant role in how an illness is caused and how it is treated. Figure **3.3.3** presents a simplified graphical representation of the biopsychosocial model, emphasizing the interaction of all three elements. This model could be used, for example, by clinicians as a way to make sure they are evaluating the medical or genetic conditions a person may have while also exploring the person's psychological experiences and upbringing, and their social and cultural environmental stresses to better understand them. Researchers could use this model as a general way to think about specific research findings, or to explore the interactions between these three major elements of the model.

In the biopsychosocial model (BPS), the *biological element* can include such things as genetic inheritance related to risk of developing a disorder, malfunctions or disruptions of the system of neural transmission between neurons in the brain, potential damage to parts of the brain, infections or diseases that might affect brain or nervous system functioning, and hormonal influences or brain-hormone interactions, among others. As noted above, epigenetic research has found that environmental stressors or events, especially those occurring early in life, may interact with a person's genome (set of all our genes in our DNA), creating expanding biological effects.

The *psychological element* of the BPS includes a wide range of thoughts, feelings, and behaviors including things like: learned behaviors (conditioning or observed behaviors), thought patterns that increase or intensify emotions like sadness or anxiety, events occurring during key developmental stages in life, strengths and weaknesses in ability to cope with different life stressors, sense of confidence or self-efficacy, personality or cognitive development, and more.

The *social/cultural element* of the BPS involves a wide range of external situations that can shape how people respond to events and other people, often by influencing the degree of stress a person experiences in their life. These can include experiences of abuse, being a victim of a crime, experiencing racism and discrimination, poverty, war, the impact of cultural expectations and demands, state of physical health and nutrition, levels and type of stress in the environment (rural living versus city living, for example), extent of social support network (friends, family, and others one can turn to for help and comfort), and more.

? Predisposed for anxiety?

Consider Erin. Erin is born into a family that has strong genetic connections to anxiety. Several of her family members have diagnosable mood and anxiety disorders. Does this mean that Erin will develop a phobia or panic disorder?

Erin likely inherited at least some genes that may make her more likely to react strongly to life events and may shape how her body's stress response works. A lot may also depend on how her parents raise her, as well as the behaviors she sees her family and friends exhibit as she grows up; do they demonstrate effective coping reactions to stress and frightening experiences or do they strongly react to situations and increase symptoms by trying to avoid frightening situations? Do her parents teach her that life is dangerous and unpredictable and that she needs to be on her guard all the time, or do they encourage her to explore, demonstrate how to cope with events that happen, and provide emotional support?

It may also depend a lot on where she lives, whether she experiences severe negative events in life, is discriminated against, or teased and bullied by kids at school. This is only a small set of possible alternatives that could happen in Erin's life, and it is fairly easy to see that whether or not she would develop an anxiety or mood disorder could be influenced by the interaction of these biological, psychological, and social forces. Don't forget that epigenetics would also suggest that it is possible that depending on her life experiences, the way her brain and hormonal systems function might also change as a result, either increasing or decreasing her likelihood of developing a disorder.

In addition, it is important to realize that the particular patterns of how the biopsychosocial model functions also vary by the disorder. Some disorders have a heavier influence due to genetic and other biological factors while other disorders are more heavily influenced by social or psychological elements. If you examine Figure 4(a) for depression, you'll see that biological elements have slightly greater influence on how a person's life will turn out than the other elements, although both the psychological and social/cultural elements still play major roles. In contrast, in Figure 4(b), biological elements have a significantly smaller role in the development of eating disorders while social/cultural and psychological elements have a stronger influence. Even with a condition such as schizophrenia (see Figure 4(c)) which is often incorrectly thought of by many as a "mental disease," the biological elements still contribute *less than* 50% of the influence on whether a person will develop the disorder or not. This statistic is based on multiple studies demonstrating that the **concordance rate** between identical twins is less than half, a measure of the likelihood of an identical twin developing schizophrenia if the co-twin already has it. Said another way, this means that more than half of the time, even with a strongly biologically influenced disorder, an identical twin, a person with identical genes, will not develop schizophrenia. Clearly, social/cultural and psychological elements also play important roles even in this case, and it is possible that epigenetics also has some influence. You will learn more about these issues in later modules.

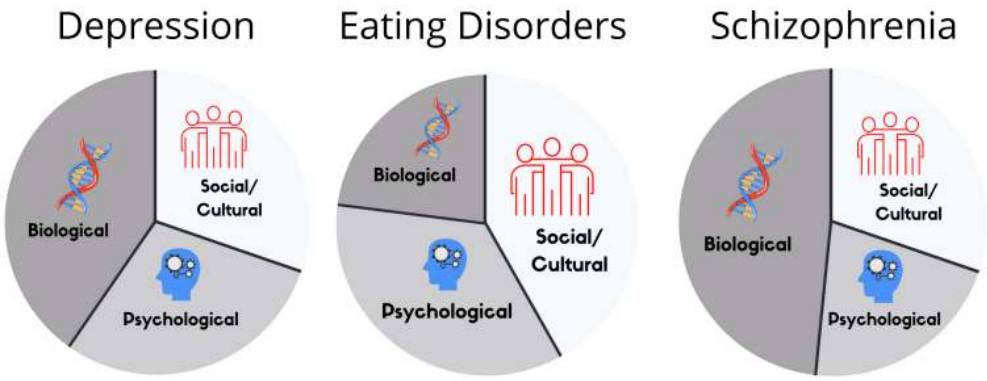
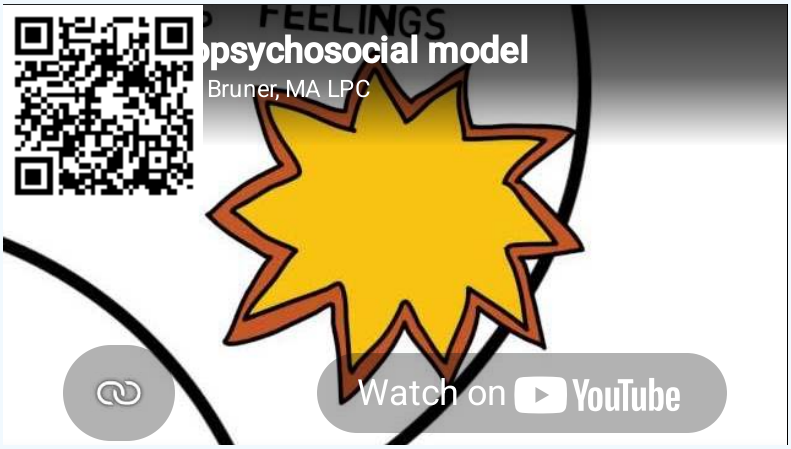


Figure **3.3.4**: Depression has strong biological elements to it, but psychological and social/cultural forces also make significant contributions. (b) Eating disorders are more widely influenced by social and cultural factors. (c) Schizophrenia is the most biologically/genetically influenced set of disorders in this example; however, even with schizophrenia, there is evidence that psychological and social/cultural forces can impact on symptoms and prognosis (future course of the disorder).

Clearly, the biopsychosocial multidimensional model is very useful at helping us understand the elements that contribute to mental disorders in a more complete and useful way, but it does not specifically explain *how* a person like Erin might actually develop a disorder. Fortunately, two BPS-related models have emerged that give us insight into this situation: the diathesis-stress and gene-environment correlation models.

Watch It

This video further explains what some of the biological, psychological, and social factors are that all interact in contributing to our mental wellness.



You can view the transcript for "biopsychosocial model" here (opens in new window).

The Diathesis-Stress Model

The biopsychosocial model gives a broad, general idea of how the three major elements all interact with each other to produce mental disorders. The **diathesis-stress model**, first applied to schizophrenia by Paul Meehl in the 1960s, is more specific and helps us understand why one person might develop a disorder, or why two people from similar backgrounds might develop different disorders. The term *diathesis* comes from the Greek word meaning "predisposition." Professionals sometimes talk about "vulnerability" to a disorder, but these terms are all interchangeable. The key assumption of the diathesis-stress model is that both factors, diathesis (usually genetic or biological) and environmental stress, are necessary in the development of a disorder. It is important to note that a large range of differences exists among individuals' vulnerabilities to the development of a disorder and that the brain and how it functions, contrary to popular belief, continue to change over the lifespan, even at the genetic level, in response to environmental events (Durand & Barlow, 2013).[2]

A given person's diathesis or vulnerability to developing a mental disorder represents the person's pre-existing risk or probability of developing a mental disorder if they are exposed to sufficient stress. For example, a person who grew up in a family with a long history of alcoholism and drug use is much more likely to develop a substance disorder than a person raised in a family with no such history. Diathesis is often thought to refer to genetic, biological, and physiological conditions such as variations in genes or combinations of multiple genes that increase the chance for someone to develop a disorder, but they can also include cognitive, personality-related, and even environmental factors (such as loss of parent early in life). However, remember that there is no simple genetic inheritance that leads to a mental disorder; having a vulnerability or a diathesis for a disorder does not mean the person will actually develop the disorder.

For example, let's assume that the diathesis for a person with a strong family history of alcoholism may include biological effects perhaps like an attraction, stronger than most, for the taste of alcohol, and/or that alcohol has a stronger and more immediate impact on the brain than is true for someone else. However, a lot depends on the interaction with environmental stress. If the person who inherits this diathesis learns alternative ways to cope with stress in their life (rather than drinking) and never, or rarely, drinks alcohol, they may never become an alcoholic. However, if the person follows the example of family members, takes up drinking at a young age, and uses drinking to cope with job loss, relationship problems, and so forth, that person is much more likely to develop alcohol dependence.

Similarly, someone could be exposed to significant life stressors, meaning life events that disrupt a person's stability or functioning in life, and not develop a disorder, if they do not have a diathesis or vulnerability to a disorder or if that diathesis is weak. For example, someone who comes from a family with no history of alcoholism or addiction, may drink and party in college, drink occasionally through life, and may experience divorce, chronic illness, and/or a motor vehicle accident and not develop alcohol dependence. This is because the diathesis-stress model also presumes that for each person there is a **threshold**, a point at which the

person's coping abilities and social, psychological, and biological defenses cannot manage their level of life stress; when the *combination* of a diathesis/vulnerability and life stress exceeds the person's threshold, they will develop a disorder that aligns with their vulnerability.



Figure **3.3.5**: The diathesis-stress model can be compared to a stick that breaks under strain.

Think of picking up a stick on a walk. If you hold the stick in your hands and bend it, it does not immediately break. It may bend and experience strain until it reaches a threshold point at which it will break. Now consider all the factors that may cause that threshold to vary from stick to stick: is the stick green (not dried out), the thickness of the stick, the type of wood the stick comes from, and so on. Each stick will have its own distinctive threshold for how much stress it can take before it breaks.



Figure **3.3.6**: Another metaphor to understand the diathesis-stress model is that of a bucket. Buckets may be of varying sizes and come with different amounts of material already inside, and stressors that cause the water to overflow may trigger a mental disorder in someone who is vulnerable.

Another common metaphor for understanding the diathesis-stress model is the visual image of a bucket filling with water. Genetics influence the size of the bucket we have or the amount of the material that is already inside of the bucket. Life stressors and other events can add water to the bucket, while coping mechanisms and positive experiences can open taps on the bucket to remove some of that stress. We can think of genetic/biological diathesis as the sand that is already inside of the bucket. The larger that vulnerability, the less life stress (the water on top) is necessary to exceed the threshold and trigger the disorder. The reverse is also true; if a person has a very low vulnerability to a particular disorder, the same amount of stress will not exceed the threshold and no disorder will be triggered. Alternatively, the person would have to experience a significant and large amount of stress to exceed the threshold which is possible, but not as likely. This model reinforces the importance of understanding how nature and nurture interact in the etiology of mental disorders.

There are a few other aspects of the diathesis-stress model that are worth considering. Protective factors, such as positive parent-child relationships, positive social networks, effective coping strategies or high self-esteem, can counteract the effects of stressors and prevent or curb the effects of disorder. Many psychological disorders have a window of vulnerability, during which time an individual is more likely to develop the disorder than others. For these reasons, diathesis-stress models are often conceptualized as multi-causal developmental models, which propose that multiple risk factors over the course of development interact with stressors and protective factors contributing to normal development or psychopathology.

The development of the diathesis-stress model was a significant advance from the biopsychosocial model in helping to understand how people would vary in their symptoms and diagnoses even if they had similar genetics or came from similar environments. However, the model was still based mostly in a simple additive idea: the biological diathesis (vulnerability) plus the amount of environmental stress experienced would cause a disorder if it was enough to cross the threshold. A newer model called the gene-environment correlations model or rGE[3], emphasizes, even more, the interactions that can occur between a person's biological elements (genetics, neurotransmitter systems, brain development) and the psychosocial and cultural environment.

Gene-Environment Correlation Models

The rGE (the little *r* is the statistical symbol for *correlation*) model and related studies have greatly expanded since the decoding of the human genome was finished in 2003, creating the possibilities of new research into genes and genetic variants (alternative copies of the same gene such as genes that code for brown eyes versus blue eyes) and how they interact with the environment. However, because this field is rapidly evolving, there are still areas of controversy and new information is emerging regularly.

A review of 315 studies conducted through 2015 found that a large number of them (67–83%) showed significant interactions between genes and the environment. The **gene-environment correlation model (rGE)** is defined as the tendency of individuals to select and/or generate their environment based on genetic features that influence behavior, thoughts, and feelings[4].

In other words, instead of the simple diathesis-stress additive model of a diathesis plus sufficient stress to cross the threshold producing a disorder, the rGE model has demonstrated evidence that a person's genetically influenced personality traits and behaviors may either select for or *create* the stressful life events or experiences that exceed the threshold and trigger the diathesis. The rGE model differs from epigenetics which emphasizes the influence of environmental forces on genetic expression, although that field has also greatly benefited from the mapping of the human genome. Some evidence suggests that cultural forces may also be involved, although this is not yet fully understood. Leighton and colleagues (2017) noted that some gene-environment interactions are robust in western nations like the United States, but have not been replicated in eastern nations. They further concluded that there is an increasing consensus among experts that "most common psychiatric disorders, such as depression and anxiety, are best explained as complex disorders involving dysfunctions in several biological systems in interaction with environmental factors" (p.2).[5].[/footnote]

As an example of how the rGE model works, Jaffee and Price (2007)[6] described a study conducted in 2006 that found that persons with a variant gene that conveyed high risk (diathesis) for alcoholism also showed personality traits, such as less desire to please other people, that increased their risk for a diagnosis of antisocial personality disorder. These personality traits are more likely to lead to disrupted relationships and increased social isolation as well as life events that could generate stress including divorce, never marrying, and potential criminal behavior that could increase a desire to drink, exceeding the threshold and triggering the underlying vulnerability to alcohol dependence.

In the diathesis-stress and gene-environment correlation models, we see the evolution and increased complexity of the biopsychosocial model over time; at the same time, this more complete understanding of the mechanisms involved in how biological forces interact with and are shaped by environmental influences (including psychological and social/cultural elements) holds out promise of greater understanding and new treatments to come. These models also reinforce the inadequacy of simplistic unidimensional explanations of mental disorders, like the "chemical imbalance" explanation. That said, we will now explore different elements of the biopsychosocial model and the treatment approaches they have generated over time.

Glossary

concordance rate: the percentage rate of twins sharing a particular attribute

diathesis-stress model: suggests that people with a predisposition or vulnerability for a disorder (a diathesis) are more likely to develop the disorder when faced with stress; it is one model of psychopathology

epigenetics: the study of how social, psychological, and environmental influences can actually change which genes in a person's DNA may activate or may be turned off—a change in phenotype without a change in genotype

gene-environment correlation model (rGE): is defined as the tendency of individuals to select and generate their environment based on genetic features that influence behavior, thoughts, and feelings

multidimensional models: explanations involving multiple approaches or interacting elements

system: a unified whole made up of parts that interact and are interdependent with each other

1. Shrindhar, N. & Walker, L. (August, 2016). Gene-environment interactions and epigenetics. Retrieved from: https://www.healthandenvironment.org/environmental-health/social-context/gene-environment-interactions↵
2. Durand, V. and Barlow, D., 2013. *Essentials of Abnormal Psychology*. 6th ed. Wadsworth: Cengage Learning. ↵

3. Jaffee, S.R. & Price, T.S. (2007, May). Gene-environment correlations: A review of the evidence and implications for prevention of mental illness. Molecular Psychiatry, 12(5), 432-442. doi:10.1038/sj.mp.4001950. PMC 3703541. ↵
4. Leighton, C., Botto, A., Silva, J.R., Jiménez, P.J., & Luyten, P. (2017). Vulnerability or sensitivity to environment? Methodological issues, trends, and recommendations in Gene-Environment interactions research in human behavior. *Frontiers in Psychiatry*, 8(106), 1–14 ↵
5. [footnote]Leighton, C., Botto, A., Silva, J.R., Jiménez, P.J., & Luyten, P. (2017). Vulnerability or sensitivity to environment? Methodological issues, trends, and recommendations in Gene-Environment interactions research in human behavior. *Frontiers in Psychiatry*, 8(106), 1–14. ↵
6. Jaffee, S.R. & Price, T.S. (2007, May). Gene-environment correlations: A review of the evidence and implications for prevention of mental illness. Molecular Psychiatry, 12(5), 432–442. doi:10.1038/sj.mp.4001950. PMC 3703541. ↵

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3.4: Biological Elements of Psychopathology

Learning Objectives

- Explain polygenic inheritance and genetic heritability in mental disorders
- Explain neural communication and the role of neurotransmitters in neural transmission

Biological Contributions to Mental Disorders

As discussed on the previous page, biological elements such as genetics, brain structures, the endocrine system, and neurotransmitters all play an important role in the development of mental disorders. As explained by the biopsychosocial model, none of these biological factors, on their own can generate a mental disorder, but combined with environmental, psychological, and social or cultural forces, they *interact* to shape how we learn, how our brains function, which genes are expressed or turned off, and the biological foundation of a person's diathesis (or vulnerability) to developing a disorder. Further, because these elements have been heavily studied in recent years, many of the medical or psychiatric treatments that have been developed, including drugs such as Zoloft (sertraline) or Prozac (fluoxetine) and many others, are based on what has been learned regarding these biological systems that contribute to psychopathology. It is important to understand this part of this system in order to better understand human functioning and how medical treatments work.

Genetic Contributions

DNA (deoxyribonucleic acid) is present in the nucleus of every cell of our body, packed into larger structures called chromosomes, and shaped in a double-helix (think of a ladder twisted into a spiral shape; see Figure **3.4.1**). Most human beings have 23 pairs of chromosomes, receiving one set of 23 from our mother and the other set of 23 from our father. The 23rd chromosome, or sex chromosome, differs between males and females. Females have two copies of the X chromosome while males have one X chromosome and one Y chromosome. Our DNA ladder comprises rails made up of sugar and phosphate (blue color in Figure **3.4.1**), and rungs consisting of four base pairs that are linked together (multicolored, horizontal elements in Figure **3.4.1**).



Figure **3.4.1**: DNA, found inside of the nucleus of our cells, contains the genes that influence who we are and how we look and act.

Sections of the DNA strand (the rungs of the ladder) containing many of these base pairs make up what we call **genes**, and they contain the instructions for the construction of proteins. The total of all a person's genes is called their **genome**, and the set of genes that may create a specific outcome, such as hair color, is called the **genotype** (the set of genes that produces something specific). The actual *observable* result of a genotype is called the **phenotype,** and it may differ from what is expected from the genotype due to various factors, including environmental interactions, interactions with other genes, epigenetics, and other factors. The Human Genome Project estimates that human beings have between 20,000 and 25,000 genes. Relevant to mental disorders, another concept is very important to remember. There are some rare medical conditions, such as a form of fatal dementia called Huntington's disease that is caused by a dominant gene. If a person inherits that gene (from either parent), he or she will develop the disorder. However, this is *not* true for any of the known mental disorders discussed in this text. All mental disorders have **polygenic** inheritance; there is no single gene that causes depression, schizophrenia, or anorexia nervosa, for example. Instead, there are multiple (poly) genes that interact to contribute to the development of mental disorders.

Recently, Boyle, Li, and Pritchard, three scientists at Stanford University, proposed an "omnigenic model" hypothesizing that sets of "core genes" may influence most aspects of our lives—from height to the chance of developing schizophrenia—and are each part of large networks of possibly thousands of genes that impact each other. They suggest that core characteristics are not influenced by any singular gene or even group of genes, but by hundreds or thousands of core and periphery genes. As Yong (2017) summarizes, "They're weak individually, but powerful *en* masse."[1]

Another scientist reviewing this omnigenic model, Faraone (2017), gave an example of how this might work in relation to mental disorders: "Let's posit this sequence of events: 1) in utero, the developing [baby's] brain is assaulted by a toxic environment or aberrant core genetic variants that pose a risk to survival. 2) As a reaction to this assault, the regulatory mechanisms governed by peripheral genes make adjustments that decrease the risk of death but increase the risk for psychiatric illness. 3) The initial assault may leave a widespread signature in the brain such as reduced brain volumes (as we see for many psychiatric disorders) and 4) The regulatory responses (#2) up- or down-regulate proteins that directly cause psychiatric symptoms."[2] Thus, it is clear that even a "polygenic" explanation for mental disorders is an overly simplistic statement and as Faraone's (2017) example illustrates, even a biogenic-focused explanation of mental disorders also involves environmental influences.

Twin Studies and Heritability

In the field of behavioral genetics, the Minnesota Study of Twins Reared Apart—a well-known study of the genetic basis for personality—conducted research with twins from 1979 to 1999. In studying 350 pairs of twins, including pairs of identical and fraternal twins reared together and apart, researchers found that identical twins, whether raised together or apart, have very similar personalities (Bouchard, 1994; Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Segal, 2012). These findings suggest the heritability of some personality traits. **Heritability** refers to the proportion of difference among people that is attributed to genetics. In other words, the larger the statistic, the more people will vary on that trait/phenotype due to genetic differences that exist in the population. Some of the traits that the study reported as having more than a 0.50 heritability ratio include leadership, obedience to authority, a sense of well-being, alienation, resistance to stress, and fearfulness. The implication is that some aspects of our personalities are largely influenced by genetics; however, it's important to point out that traits are not determined by a single gene, but by a combination of many genes, as well as by epigenetic factors that control whether the genes are expressed or not.

Heritability measures how much of the variation of a trait can be attributed to genetic factors, as opposed to environmental factors. Thus, the concept of heritability can be expressed in the form of the following question: "What is the proportion of the variation in a given trait within a population that is *not* explained by the environment or random chance?" It is calculated by comparing individual variation among related individuals in a population using various methods and is helpful in seeking to understand the impact of genetics versus environmental influences for a given disorder (think back to the comparison of different versions of the biopsychosocial model depending on the disorder in the last module). A related concept is that of **concordance**, which measures the probability or likelihood that if one person in a pair of people (usually twins) has a specific phenotype, that the other one will also have or will develop that trait.

For example, the graph in Figure **3.4.2** shows concordance estimates for schizophrenia. If you compare two people who are unrelated and one of them has schizophrenia, what is the chance that the other person will develop schizophrenia? In this case, it is very low—around 1%. Other than the overall genome related to being human, these two individuals share very little genetic overlap. However, as you go down the chart, you can see that the more genetic overlap there is between two people, the greater the chance that if one of them has schizophrenia, that the other one will also develop the disorder. So, compared to two non-related individuals whose probability is only 1% or so, if a half-sibling has schizophrenia, then the other person's risk level is about five to six times as high to develop the disorder! In other words, the greater the amount of shared genetic material, the greater the probability that the genes will manifest the phenotype. This suggests a strong impact of genetics on the disorder. Of course, the highest rate of overlap occurs with identical twins who share the same genome. However, even in this highest group, the concordance rate is less than 50%. Thus, there is evidence of strong genetic influences on the development of schizophrenia, but there is also evidence that environmental forces also exert significant influence.

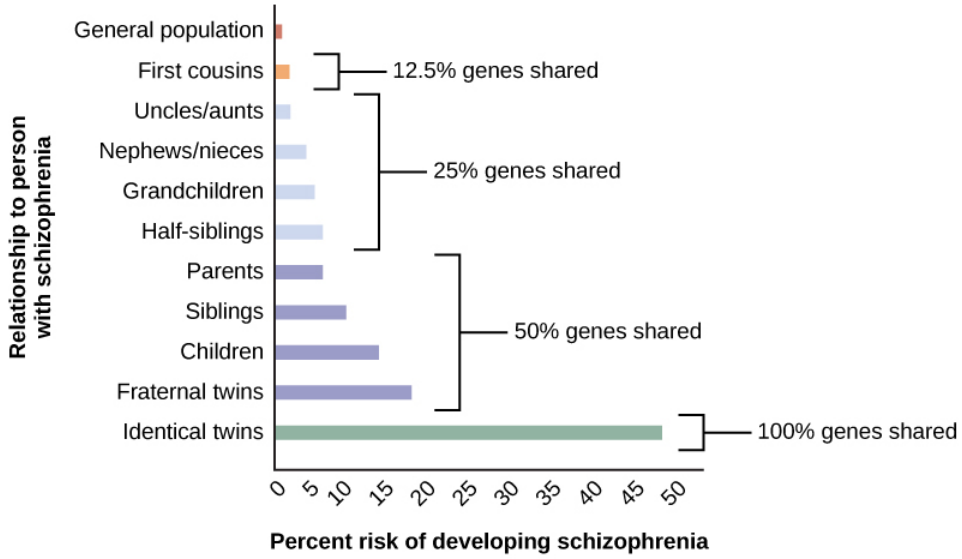


Figure **3.4.2**: A person's risk of developing schizophrenia increases if a relative has schizophrenia. The closer the genetic relationship, the higher the risk due to a higher degree of shared genes.

Findings such as these have led many of today's researchers to search for specific genes and genetic mutations that contribute to mental disorders. Also, sophisticated neural imaging technology in recent decades has revealed how abnormalities in brain structure and function might be directly involved in many disorders, and advances in our understanding of neurotransmitters and hormones have yielded insights into their possible connections. The biological perspective is currently thriving in the study of mental disorders.

Neural Transmission—How Neurons Signal Each Other

Those striving to understand the human mind may study the nervous system. Learning how the body's cells and organs function can help us understand the biological basis of human psychology, which is essential in understanding the biological etiology and treatment of mental disorders. It is especially important to understand how the cells of our brains function; these cells are called neurons.

Neuron Structure

Neurons are the central building blocks of the nervous system, 100 billion strong at birth. Like all cells, neurons consist of several different parts, each serving a specialized function. A neuron's outer surface is made up of a **semipermeable membrane**. This membrane allows smaller molecules and molecules without an electrical charge to pass through it while stopping larger or highly charged molecules. (See Figure **3.4.3**).

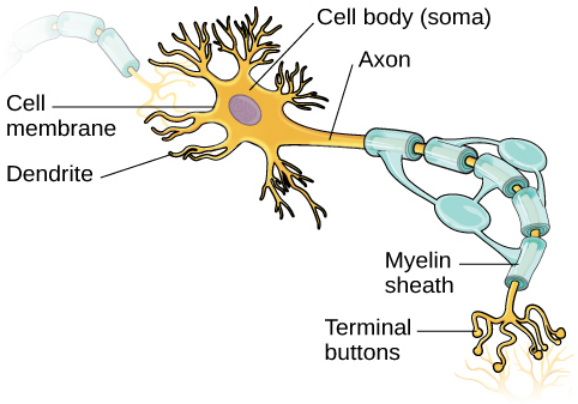


Figure **3.4.3**: This illustration shows a prototypical neuron, which is being myelinated by a glial cell.

The nucleus of the neuron, where all genetic material is stored and proteins are produced including neurotransmitters, is located in the soma, or cell body. The **soma** has branching extensions known as **dendrites**. The neuron is a small information processor, and

dendrites serve as input sites where signals are received from other neurons. These signals are transmitted electrically across the soma and down a major extension from the soma known as the **axon**, which ends at multiple **terminal buttons**. The terminal buttons contain **synaptic vesicles** that house **neurotransmitters**, the chemical messengers of the nervous system.

Axons range in length from a fraction of an inch to several feet. In some axons, glial cells form a fatty substance known as the myelin sheath, which coats the axon and acts as an insulator, increasing the speed at which the signal travels, making neural transmission more efficient. The myelin sheath is crucial for the normal operation of the neurons within the nervous system: the loss of the insulation it provides can be detrimental to normal function. To understand how this works, let's consider an example. PKU, a genetic disorder, causes a reduction in myelin and abnormalities in white matter of cortical and subcortical structures. The disorder is associated with a variety of conditions including severe cognitive deficits, exaggerated reflexes, and seizures (Anderson & Leuzzi, 2010; Huttenlocher, 2000). Another disorder, multiple sclerosis (MS), an autoimmune disorder, involves a large-scale loss of the myelin sheath on axons throughout the nervous system. The resulting interference in the electrical signal prevents the quick transmittal of information by neurons and can lead to a number of symptoms, such as dizziness, fatigue, loss of motor control, and sexual dysfunction. While some treatments may help to modify the course of the disease and manage certain symptoms, there is currently no known cure for multiple sclerosis.

In healthy individuals, the neuronal signal moves rapidly down the axon to the terminal buttons, where synaptic vesicles (shaped like bubbles) release neurotransmitters into the synaptic cleft. The **synaptic cleft** or synapse is a very small space between two neurons and is an important site where communication between neurons occurs. Once neurotransmitters are released into the synaptic cleft, they travel across the small space and bind with corresponding receptors on the dendrite of an adjacent neuron (called the *postsynaptic* neuron because it is *after* the synapse). **Receptors**, proteins on the cell surface where neurotransmitters attach, vary in shape, with different shapes "matching" different neurotransmitters.

Watch It

This video shows the structure and physiology of a neuron.



You can view the transcript for "2-Minute Neuroscience: The Neuron" here (opens in new window).

How does a neurotransmitter "know" which receptor to bind to? The neurotransmitter and the receptor have what is referred to as a lock-and-key relationship—specific neurotransmitters fit specific receptors similar to how a key fits a lock. The neurotransmitter floats in the synapse and binds to any receptor that it fits (see Figure **3.4.4**).

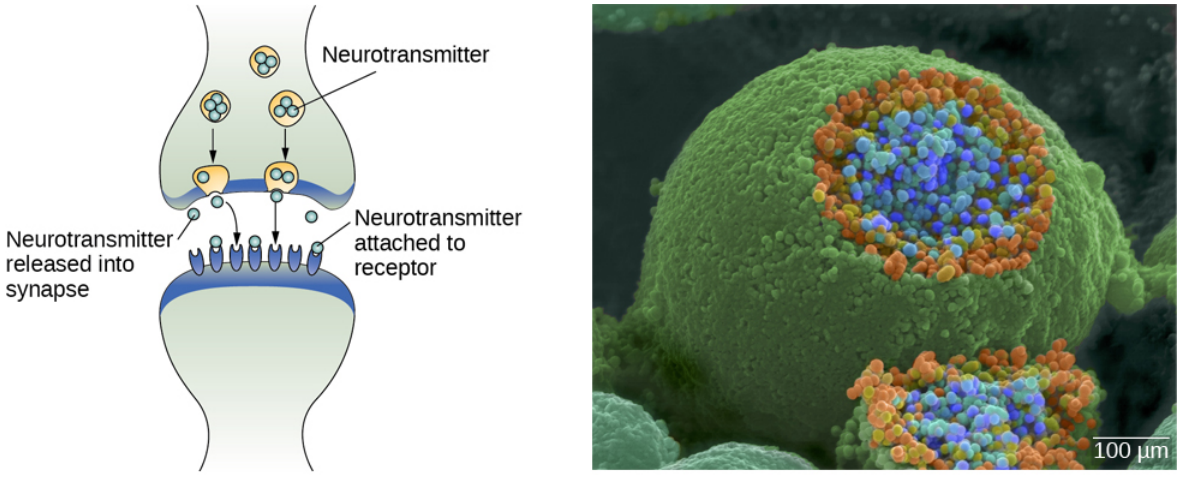


Figure **3.4.4**: (a) The synaptic cleft is the space between the terminal button of one neuron and the dendrite of another neuron. (b) In this pseudo-colored image from a scanning electron microscope, a terminal button (green) has been opened to reveal the synaptic vesicles (orange and blue) inside. Each vesicle contains about 10,000 neurotransmitter molecules. (credit b: modification of work by Tina Carvalho, NIH-NIGMS; scale-bar data from Matt Russell)

Neural Communication

SUGGESTION: As you read through the discussion below, it may help you to get out a piece of paper and summarize with bullets or numbers the process involved in neural transmission. This will help you remember the process and also help you to review the material for quizzes and exams. Alternatively, if you remember visual images better, draw out the process on a picture of a neuron, number the steps along the picture or diagram with a brief description.

To understand the details of neural communication, let's begin at the neuronal membrane. The **neuron** exists in a fluid environment—it is surrounded by extracellular fluid and contains intracellular fluid (i.e., cytoplasm). The neuronal membrane keeps these two fluids separate—a critical role because the electrical signal that passes through the neuron depends on the intracellular and extracellular fluids being electrically different. This difference in charge across the membrane, called the **membrane potential**, provides energy for the signal.

The electrical charge of the fluids is caused by charged molecules (ions) dissolved in the fluids. The semipermeable nature of the neuronal membrane somewhat restricts the movement of these charged molecules, and, as a result, some of the charged particles tend to become more concentrated either inside or outside the cell.

Between signals, the neuron membrane's potential is held in a state of readiness, called the **resting potential**. Like a rubber band stretched out and waiting to spring into action, ions line up on either side of the cell membrane, ready to rush across the membrane when the neuron goes active and the membrane opens its gates (i.e., a sodium-potassium pump that allows movement of ions across the membrane). Ions in high-concentration areas are ready to move to low-concentration areas, and positive ions are ready to move to areas with a negative charge.

In the resting state, sodium (Na^+) is at higher concentrations outside the cell, so it will tend to move into the cell. Potassium (K^+), on the other hand, is more concentrated inside the cell, and will tend to move out of the cell. In addition, the inside of the cell is slightly negatively charged compared to the outside. This provides an additional force on sodium, causing it to move into the cell (see Figure **3.4.5**).

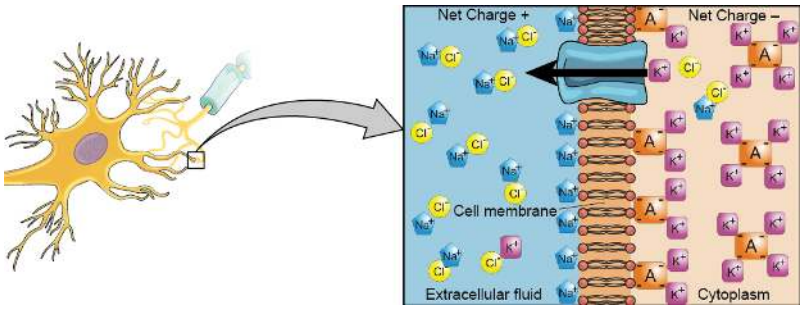


Figure **3.4.5**: At the neuron's resting potential, Na^+ (blue pentagons) is more highly concentrated outside the cell in the extracellular fluid (shown in blue), whereas K^+ (purple squares) is more highly concentrated near the membrane in the cytoplasm or intracellular fluid. Other molecules, such as chloride ions (yellow circles) and negatively charged proteins (brown squares), help contribute to a positive net charge in the extracellular fluid and a negative net charge in the intracellular fluid.

From this resting potential state, the neuron receives a signal and its state changes abruptly (Figure **3.4.6**). When a neuron receives signals at the dendrites—due to neurotransmitters from an adjacent neuron binding to its receptors—small pores, or gates, open on the neuronal membrane, allowing Na^+ ions, propelled by both charge and concentration differences, to move into the cell. With this influx of positive ions, the internal charge of the cell becomes more positive. If that charge reaches a certain level, called the **threshold of excitation**, the neuron becomes active and the action potential begins. This process of when the cell's charge becomes positive, or less negative, is called **depolarization**.

Many additional pores open, causing a massive influx of Na^+ ions and a huge positive spike in the membrane potential, the peak action potential. At the peak of the spike, the sodium gates close and the potassium gates open. As positively charged potassium ions leave, the cell quickly begins repolarization. At first, it **hyperpolarizes**, becoming slightly more negative than the resting potential, and then it levels off, returning to the resting potential.

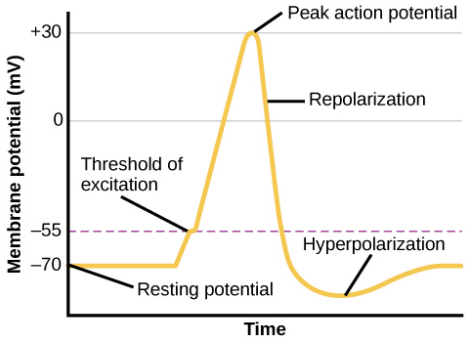


Figure **3.4.6**: During the action potential, the electrical charge across the membrane changes dramatically.

This positive spike constitutes the **action potential**: the electrical signal that typically moves from the cell body down the axon to the axon terminals. The electrical signal moves down the axon like a wave; at each point, some of the sodium ions that enter the cell diffuse to the next section of the axon, raising the charge past the threshold of excitation and triggering a new influx of sodium ions. The action potential moves all the way down the axon, pushing synaptic vesicles loaded with neurotransmitters towards the terminal buttons.

The action potential is an **all-or-none** phenomenon. In simple terms, this means that an incoming signal from another neuron is either sufficient or insufficient to reach the threshold of excitation. There is no in-between, and there is no turning off an action potential once it starts. Think of it like sending an email or a text message. You can think about sending it all you want, but the message is not sent until you hit the send button. Furthermore, once you send the message, there is no stopping it. Presynaptic neurons (those sending the signals via neurotransmitters) can either be excitatory, meaning they are encouraging the postsynaptic neuron to depolarize and "fire" the signal, or they can be inhibitory, attempting to repolarize the neuron and prevent firing of the action potential. Whether the neuron activates the action potential or not depends on the overall sum of these excitatory and inhibitory signals and whether they exceed the neuron's threshold of excitation.

Because it is all or none, the **action potential** is recreated, or propagated, at its full strength at every point along the axon. Much like the lit fuse of a firecracker, it does not fade away as it travels down the axon. It is this all-or-none property that explains the

fact that your brain perceives an injury to a distant body part like your toe as equally painful as one to your nose.

As noted earlier, when the action potential arrives at the terminal button, the synaptic vesicles that had been pushed down the axon by the signal wave release their neurotransmitters into the **synaptic cleft**. The neurotransmitters travel across the synapse and bind to receptors on the dendrites of the adjacent neuron, and the process repeats itself in the new neuron (assuming the signal is sufficiently strong to trigger an action potential). Once the signal is delivered, excess neurotransmitters in the synaptic cleft drift away, are broken down into inactive fragments by enzymes, or are reabsorbed in a process known as reuptake. **Reuptake** involves the neurotransmitter being pumped back into the neuron that released it, in order to clear the synapse (Figure **3.4.7**) in a type of "recycling" that allows the neuron to repackage the neurotransmitters into vesicles for the next signal. Clearing the synapse serves both to provide a clear "on" and "off" state between signals and to regulate the production of neurotransmitter (full synaptic vesicles provide signals that no additional neurotransmitters need to be produced).

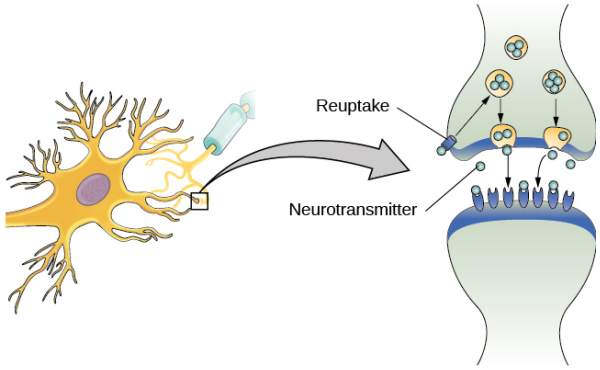


Figure **3.4.7**: Reuptake involves moving a neurotransmitter from the synapse back into the axon terminal from which it was released.

Neuronal communication is often referred to as an electrochemical event. The movement of the action potential down the length of the axon is an electrical event, and movement of the neurotransmitter across the synaptic space represents the chemical portion of the process. Understanding these processes is vital to understanding how modern psychiatry has developed medical treatments for mental disorders. For instance, some common forms of antidepressants like Zoloft, Paxil, or Celexa are classified as Selective Serotonin Reuptake Inhibitors. As this name suggests, their mechanism of action is to blockade reuptake receptors on the presynaptic neuron. This means that serotonin, a neurotransmitter, released into the synapse will not be able to be brought back into the cell and be repackaged; it will remain in the synapse for a longer period of time, creating more opportunities to bind to receptor sites on the postsynaptic neuron.

Other psychiatric drugs work by increasing the production of specific neurotransmitters in the nucleus of the neuron, blockading the receptor sites on the postsynaptic dendrite so the signal is decreased. An older antidepressant called Monoamine Oxidase Inhibitor (MAOI) inhibits the action of the enzymes in the synapse so that the neurotransmitters remain in the synapse longer and thus have a stronger signal. Other non-drug treatments, often used as "second line" interventions when medications are not effective, usually work by influencing how neurons function. One example is Transcranial Magnetic Stimulation (TMS), which involves a noninvasive pulse of magnetic energy to parts of the brain. Remember that neurons' action potentials are triggered by ions that are positive or negative in charge (like the ends of a battery) and thus may be influenced by magnetic fields. Specific medical treatments for mental disorders will be described in the relevant modules for the mental disorders.

Neurotransmitters and Mental Disorders

The exact number of neurotransmitters in the brain is still being determined and not all of them are related to mental disorders. Remember that neurotransmitters can also be defined as excitatory (encouraging the postsynaptic neuron to fire by disrupting the resting potential) or inhibitory (calming down or restoring the resting potential across the membrane of the postsynaptic neuron and preventing an action potential). As research continues in the functioning of the brain and neurons, it is also clear that often neurotransmitter systems interact and mental disorders may be related to the functioning of several neurotransmitter systems together. The mental disorder modules will go into more details about specific neurotransmitter systems, but at this point, our best understanding is that at least five major neurotransmitter systems are involved, in various ways, with mental disorders and may

have other physical effects as well (such as Parkinson's disease as described above). These are the five neurotransmitter systems involved:

- dopamine: related to movement, memory, and as part of the brain's pleasure or reward center
- serotonin: a neuromodulator, meaning that serotonin appears to influence the functioning of other neurotransmitter systems; serotonin is also relevant to pain, sleep, digestion, and mood
- norepinephrine: also related to mood, pain, heart rate, and blood pressure
- GABA: a major inhibitory neurotransmitter, often believed to be related to calming down or relaxation; it has links and connections to alcohol and other substances
- glutamate: an excitatory neurotransmitter, related to learning and memory and other functions

Glossary

action potential: electrical signal that moves down the neuron's axon

agonist: drug that mimics or strengthens the effects of a neurotransmitter

all-or-none: phenomenon that incoming signal from another neuron is either sufficient or insufficient to reach the threshold of excitation

antagonist: drug that blocks or impedes the normal activity of a given neurotransmitter

axon: major extension of the soma

biological perspective: view that psychological disorders like depression and schizophrenia are associated with imbalances in one or more neurotransmitter systems

depolarization: when a cell's charge becomes positive or less negative

dendrite: branch-like extension of the soma that receives incoming signals from other neurons

glial cell: nervous system cell that provides physical and metabolic support to neurons, including neuronal insulation and communication, and nutrient and waste transport

hyperpolarization: when a cell's charge becomes more negative than its resting potential

membrane potential: difference in charge across the neuronal membrane

myelin sheath: fatty substance that insulates axons

neuron: cells in the nervous system that act as interconnected information processors, which are essential for all the tasks of the nervous system

neurotransmitter: chemical messenger of the nervous system

psychotropic medication: drugs that treat psychiatric symptoms by restoring neurotransmitter balance

receptor: protein on the cell surface where neurotransmitters attach

resting potential: the state of readiness of a neuron membrane's potential between signals

reuptake: neurotransmitter is pumped back into the neuron that released it

semipermeable membrane: cell membrane that allows smaller molecules or molecules without an electrical charge to pass through it, while stopping larger or highly charged molecules

soma: cell body

synapse: small gap between two neurons where communication occurs

synaptic vesicle: storage site for neurotransmitters

terminal button: axon terminal containing synaptic vesicles

threshold of excitation: level of charge in the membrane that causes the neuron to become active

1. Yong, E. (2017, June 19). *What If (Almost) Every Gene Affects (Almost) Everything?* https://www.theatlantic.com/science/archive/2017/06/its-like-all-connected-man/530532/. ↵
2. J Psychiatry Brain Sci. 2017; 2(5): S1; https://doi.org/10.20900/jpbs.20170014S1↵

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3.5: The Brain and Psychopathology

Learning Objectives

- Describe the functions of different major areas of the brain and endocrine system and how they are involved in mental disorders

In addition to understanding the basic functioning of neurons, biological approaches to psychopathology also examine the structures of the brain and body that contribute to and are influenced by mental disorders. This large field is known as neuroscience. For example, we know that damage to certain areas of the brain can contribute to specific types of behavioral changes. This section of the module focuses on understanding the interactions between the nervous system and the endocrine (hormonal) system as well as a larger view of the brain and its structures.

The Nervous and Endocrine Systems

What we call the nervous system actually has two main systems: the **central nervous system** (CNS), which consists of the brain and spinal cord, as well as the **peripheral nervous system**, which is further subdivided into the somatic and autonomic nervous systems.

The peripheral nervous system is made up of thick bundles of axons, called nerves, carrying messages back and forth between the CNS and the muscles, organs, and senses in the periphery of the body (i.e., everything outside the CNS). The central nervous system processes all incoming information, sorts it, evaluates risks, decides if information is important or irrelevant, interprets sensory signals such as sound, taste, or touch, activates memory, language comprehension, and more to interact with the world around us and to generate responses that are sent back out from the brain through the spinal cord.

The spinal cord has some immediate reactions to certain stimuli that we call reflexes, but mostly it serves as the conduit or path by which information from the peripheral nervous system is sent to the brain and vice versa. In the peripheral nervous system, the **somaticnervous system** (remember the word *soma* means “body” from our discussion of neurons) transmits sensory and motor signals to and from the central nervous system; it is how we control our “voluntary” muscle movements. For example, when you reach out to pick up your cell phone, the movement of your arm, hand, fingers, and so forth is under voluntary control through the somatic nervous system. In contrast, the **autonomicnervous system** controls the functioning of our organs and glands including involuntary muscle movement, usually things that we are unaware of most of the time like breathing, our heart beating, and the movement of the muscles that pushes our food through the intestines and is part of digestion and elimination.

The autonomic nervous system is also subdivided into the sympathetic and parasympathetic systems (see Figure **3.5.1**). **Sympathetic** activation is related to energy use and maintaining homeostasis such as our body temperature; its major purpose, though, is to enable us to respond to emergencies or threats through fight or flight by mobilizing energy quickly. In contrast, **parasympathetic** activation is associated with normal functioning under relaxed conditions, sometimes referred to as governing “feed and breed” and then “rest and digest.” Although these two systems are often seen as opposites of each other, in reality they often work together, usually in sequence. For example, if you are late for class, the sympathetic NS may boost your ability to run, take in air more quickly, increase your heart rate, and so on to enable you to get to class faster; once you get to your seat, however, you would not want to maintain that level of activation. Your parasympathetic NS (think “parachute” or bringing you back down), then helps to slow your breathing back down, reduce your heart rate and heat levels, and enable you to resume normal functioning. Both systems are also actively involved in sexual activity and many other situations. You will likely hear reference to these systems in the modules to come.

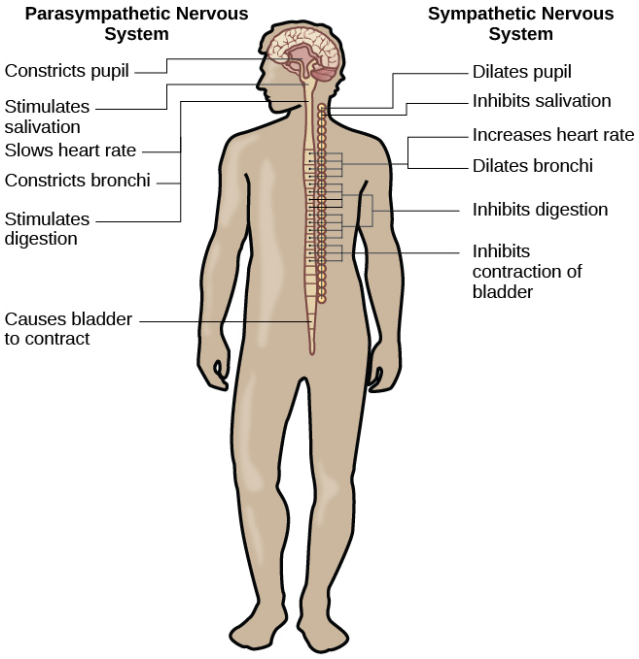


Figure **3.5.1**: The sympathetic and parasympathetic divisions of the autonomic nervous system have the opposite effects on various systems.

Working in conjunction with the nervous system is the **endocrine system**, which consists of a series of glands that produce chemical substances known as **hormones** (Figure **3.5.2**). Like neurotransmitters, hormones are chemical messengers that must bind to a receptor in order to send their signals. In fact, some of the same molecules that constitute neurotransmitters are referred to as hormones when produced by the endocrine system. However, unlike neurotransmitters, which are released in close proximity to cells with their receptors, hormones are secreted into the bloodstream and travel throughout the body, affecting any cells that contain receptors for them. Thus, whereas neurotransmitters' effects are localized, the effects of hormones are widespread. Also, hormones are slower to take effect, and tend to be longer-lasting.

There is a close relationship between the brain and the endocrine system, particularly between the hypothalamus, which has strong connections to the pituitary gland, often called the master gland because it regulates the functioning of other glands. Other important glands related to psychopathology include the adrenal glands, which release epinephrine (also known as adrenaline) and cortisol (one of the body's major stress hormones); the thyroid gland, which is involved in energy metabolism and physical growth; and the reproductive glands (testes in males and ovaries in females), which not only produce eggs and sperm, but also estrogen and testosterone, both of which can impact mood and behavior.

Hormones produced by the endocrine system play a role in relation to many mental disorders. For instance, low levels of thyroid production often mimic major depression, and this is one reason that a blood test is a useful part of early assessment of someone presenting with significant depressive symptoms; the opposite is also true—excessive production of thyroid can mimic mania similar to that of bipolar disorder. The fight-or-flight system, described before in relation to the sympathetic nervous system could not function without activation of the adrenal glands and stress hormones.

The part of the adrenal glands that produces the stress hormone cortisol is heavily involved in what is known as the **hypothalamic-pituitary-adrenocortical axis** or **HPA axis**. An *axis* is a major pathway of neurons that connects to many locations; in this case, the hypothalamic-pituitary-adrenocortical axis (HPA axis) connects the brain and endocrine system and is central in the body's response to stress or threat. Dysfunction of the hypothalamic-pituitary-adrenocortical (HPA) axis is implicated in disorders like depression, anxiety, and post-traumatic stress disorder. There is also evidence that overexposure to stressful events in early development, like chronic abuse or death of a parent, may cause the HPA axis to become overly sensitive to stressful events; sustained high levels of cortisol have also been shown to damage the hypothalamus, creating difficulties in regulating the stress response and leading to increased vulnerability for depression and anxiety later on in life. Again, this illustrates the complex interactions between environmental and biological events and processes.

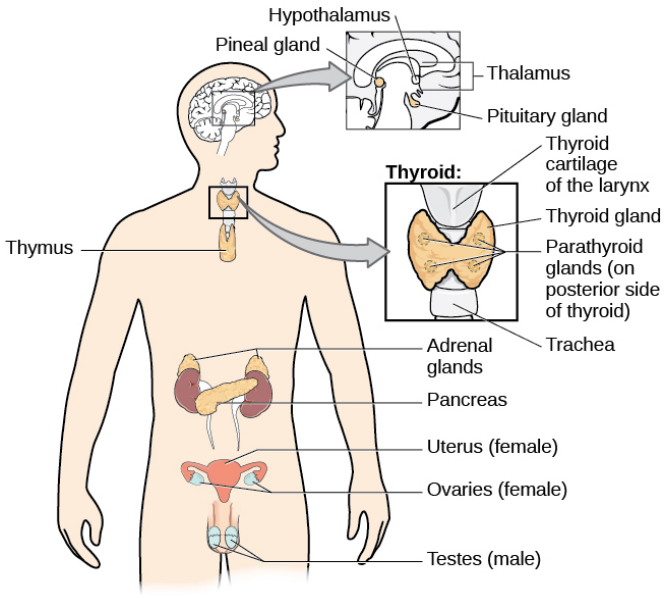


Figure **3.5.2**: The major glands of the endocrine system are shown.

Link to Learning

Learn more about endocrinology, the study of psychology and the endocrine system, from the Noba Psychology article, "Hormones and Behavior."

Brain Structures and Functioning

The two hemispheres of the cerebral cortex are part of the **forebrain** (Figure **3.5.3**), which is the largest part of the brain, accounting for about 80% of the neurons in the central nervous system.[1] Both hemispheres are similar in appearance and operate somewhat independently, communicating through a central structure called the corpus collosum that connects the two. The left hemisphere tends to specialize more in verbal processes while the right hemisphere is more focused on perceiving and manipulating images. The forebrain contains the cerebral cortex and a number of other structures that lie beneath the cortex (called subcortical structures): thalamus, hypothalamus, pituitary gland, and the limbic system (a collection of structures). The cerebral cortex, which is the outer surface of the brain, is associated with higher-level processes such as consciousness, thought, emotion, reasoning, language, and memory.

The **hindbrain** is the lower part of the brainstem and regulates ongoing, mostly automatic systems in the body, including heart rate, breathing, digestion, and other processes we don't often consciously think about. Major structures in the hindbrain include the cerebellum (the purplish flower looking structure in Figure 3), which manages our balance and muscle coordination; the pons (the bulb-shaped structure at the top of the hindbrain), which acts as a connector between the cerebellum and the medulla (located just below the pons) and can transfer messages between the other parts of the brain and the spinal cord; and the medulla, which manages our breathing, heart rate and blood pressure.

Above the hindbrain is the **midbrain** that handles input from our senses and uses this input to help coordinate movement and parts of the reticular activating system, which is involved in managing our sleep/wake cycle and other processes related to tension and arousal. At the top of the brainstem are many of the important structures mentioned above that are involved in different types of mental disorders, including the thalamus and hypothalamus (involved in the HPA axis); the limbic system, which includes the hippocampus and amygdala and is strongly involved in memory and thus our ability to learn; regulation of emotions; and fundamental drives related to eating and drinking, aggression, and sex.

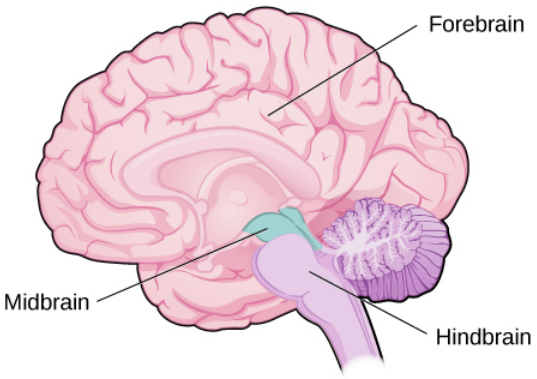


Figure **3.5.3**: The brain and its parts can be divided into three main categories: the forebrain, midbrain, and hindbrain.

Each cerebral hemisphere of the forebrain can be subdivided into four lobes, each associated with different functions.

Brain Lobes

The four lobes of the brain are the frontal, parietal, temporal, and occipital lobes (Figure **3.5.4**). The **frontal lobe** is located in the forward part of the brain, extending back to a fissure known as the central sulcus. The frontal lobe is involved in reasoning, abstract thinking and planning, creativity, motor control, emotion, and language. It contains the **motor cortex**, which is involved in planning and coordinating movement; the **prefrontal cortex**, which is responsible for higher-level cognitive functioning including major inhibitory pathways of neurons that help us regulate our behaviors and reactions; and **Broca's area**, which is essential for language production.

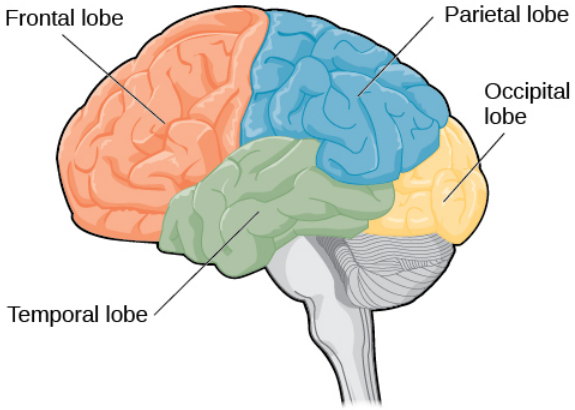


Figure **3.5.4**: The lobes of the brain are shown.

People who suffer damage to Broca's area have great difficulty producing language of any form. For example, Padma was an electrical engineer who was socially active and a caring, involved mother. About twenty years ago, she was in a car accident and suffered damage to her Broca's area. She completely lost the ability to speak and form any kind of meaningful language. There is nothing wrong with her mouth or her vocal cords, but she is unable to produce words. She can follow directions but can't respond verbally, and she can read but no longer write. She can do routine tasks like running to the market to buy milk, but she could not communicate verbally if a situation called for it.

One particularly fascinating area in the frontal lobe is called the "primary motor cortex." This strip running along the side of the brain is in charge of voluntary movements like waving goodbye, wiggling your eyebrows, and kissing. It is an excellent example of the way that the various regions of the brain are highly specialized. Interestingly, each of our various body parts has a unique portion of the primary motor cortex devoted to it. Each individual finger has about as much dedicated brain space as your entire leg. Your lips, in turn, require about as much dedicated brain processing as all of your fingers and your hand combined!

Because the cerebral cortex in general, and the frontal lobe in particular, are associated with such sophisticated functions as planning and being self-aware, they are often thought of as a higher, less primal portion of the brain. Indeed, while other animals

such as rats and kangaroos do have frontal regions in their brains, they do not have the same level of development in the cerebral cortices. The closer an animal is to humans on the evolutionary tree—think chimpanzees and gorillas, the more developed this portion of their brains are.

The brain's **parietal lobe** is located immediately behind the frontal lobe and is involved in processing information from the body's senses. It contains the **somatosensory cortex**, which is essential for processing sensory information from across the body, such as touch, temperature, and pain. The somatosensory cortex is organized topographically, which means that spatial relationships that exist in the body are generally maintained on the surface of the somatosensory cortex. For example, the portion of the cortex that processes sensory information from the hand is adjacent to the portion that processes information from the wrist.

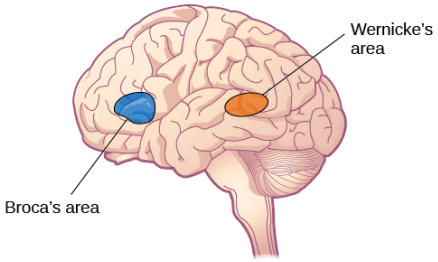


Figure **3.5.5**: Damage to either Broca's area or Wernicke's area can result in language deficits. The types of deficits are very different, however, depending on which area is affected.

The **temporal lobe** is located on the side of the head (*temporal* means "near the temples"), and is associated with hearing, memory, emotion, and some aspects of language. The **auditory cortex**, the main area responsible for processing auditory information, is located within the temporal lobe. **Wernicke's area**, important for speech comprehension, is also located here. Whereas individuals with damage to Broca's area (frontal lobe) have difficulty producing language, those with damage to Wernicke's area can produce sensible language, but they are unable to understand what others are saying.

The **occipital lobe** is located at the very back of the brain, and contains the primary visual cortex, which is responsible for interpreting incoming visual information. The occipital cortex is organized retinotopically, which means there is a close relationship between the position of an object in a person's visual field and the position of that object's representation on the cortex.

Watch It

So how does our understanding of the brain, the nervous system, and the endocrine apply to abnormal psychology? Biological psychologists seek to understand how genetic, biological, and physical components of the body and brain influence mental illness. Watch this video to see examples of the types of research that biological psychologists do, and specifically how research by Dr. Bonnie Nagel from Oregon Health & Science University is examining neurological structures and their connection to alcoholism and substance-abuse disorders.



You can view the transcript for "What is a Biological Psychologist?" here (opens in new window).

Glossary

auditory cortex: strip of cortex in the temporal lobe that is responsible for processing auditory information

autonomic nervous system: controls our internal organs and glands

Broca's area: region in the left hemisphere frontal lobe that is essential for language production

central nervous system (CNS): brain and spinal cord

cerebral cortex: surface of the brain that is associated with our highest mental capabilities

fight-or-flight response: activation of the sympathetic division of the autonomic nervous system, allowing access to energy reserves and heightened sensory capacity so that we might fight off a given threat or run away to safety

forebrain: largest part of the brain; contains the cerebral cortex, the thalamus, and the limbic system, among other structures

frontal lobe: part of the cerebral cortex involved in reasoning, motor control, emotion, and language; contains motor cortex

hindbrain: lower end of the brainstem, mostly regulates autonomic body functions

homeostasis: state of equilibrium—biological conditions, such as body temperature, are maintained at optimal levels

hypothalamic-pituitary-adrenocortical (HPA) axis: pathway connecting the brain and the endocrine system, heavily involved in fight-or-flight reactions to threats

midbrain: top of the brainstem; coordinates sensory input and motor movement and regulates sleep/wake cycles

motor cortex: strip of cortex involved in planning and coordinating movement

occipital lobe: part of the cerebral cortex associated with visual processing; contains the primary visual cortex

parasympathetic nervous system: associated with routine, day-to-day operations of the body

parietal lobe: part of the cerebral cortex involved in processing various sensory and perceptual information; contains the primary somatosensory cortex

peripheral nervous system (PNS): connects the brain and spinal cord to the muscles, organs, and senses in the periphery of the body

prefrontal cortex: area in the frontal lobe responsible for higher-level cognitive functioning

somatic nervous system: relays sensory and motor information to and from the CNS

somatosensory cortex: essential for processing sensory information from across the body, such as touch, temperature, and pain

sulcus (plural: sulci): depressions or grooves in the cerebral cortex

sympathetic nervous system: involved in stress-related activities and functions

temporal lobe: part of cerebral cortex associated with hearing, memory, emotion, and some aspects of language; contains primary auditory cortex

Wernicke's area: important for speech comprehension, located in the temporal lobe

1. Durand, V.M. & Barlow, D.H. (2013). *Essentials of Abnormal Psychology*. Cengage Learning. ↵

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3.6: Biomedical Therapies

Learning Objectives

- Explain different forms of biomedical therapies and brain stimulation treatments

The Modern Advent of Biomedical Treatments

Early attempts to understand mental illness included supernatural theories and related forms of treatment that often included intense physical and emotional interventions such as exorcisms and trephination, as well as some forms of somatogenic theories and treatments. As time passed, still lacking a scientific explanation for mental disorders and their symptoms, society created asylums to isolate individuals suffering from mental disorders, adding to stigma against the mentally ill and creating painful and stressful living conditions, despite the efforts of reformers such as Dorothea Dix. However, near the end of the 19th century, new ideas and theories began to emerge, such as those of Sigmund Freud and his peers, that emphasized more psychogenic explanations for symptoms. The emergence of the fields of psychiatry and psychology began to approach these issues from a more scientific perspective, although they were still mostly unidimensional in approach. The discovery of the syphilis bacteria as a biological agent that could generate symptoms mimicking those of mental disorders as well as early studies and efforts to understand the brain also began to strengthen the appeal of somatogenic (body or biological) treatments as a way to reduce symptoms, which is a strong emphasis that continues today. One of the earliest modern forms of somatogenic or biomedical treatments was the use of lobotomy.

Lobotomy is a form of psychosurgery in which parts of the frontal lobe of the brain are destroyed or their connections to other parts of the brain severed. The goal of lobotomy was usually to calm symptoms in people with serious psychological disorders, such as schizophrenia. Lobotomy was widely used during the twentieth century; indeed, it was so mainstream that António Moniz, a Portuguese neurosurgeon, won a Nobel Prize in physiology or medicine in 1949 for his work on a specific lobotomy procedure. However, lobotomy was always highly controversial, partly because the results were irreversible, and widely criticized as a tool for behavioral control of people who were engaged in behaviors that were not actually clinical symptoms (see NPR's report on Howard Dully). In addition, lobotomy did not actually eliminate the disorder being "treated," and while it did reduce emotional expressions and made patients more "manageable," side effects could include death; seizures; brain damage; and severe impacts on a patient's personality, cognition, and social responsiveness, often referred to as "mental dullness." Women were also given more lobotomies than men, raising significant ethical concerns regarding discrimination. By the 1960s and 1970s, lobotomy fell out of favor in the United States.

Another reason that lobotomy fell out of favor was the development in the 1950s and 1960s of new medications for the treatment of mental disorders; these have now become the most widely used forms of biological treatment. While these medications are often used in combination with psychotherapy, they also are taken by individuals not in therapy. This is known as **biomedical therapy** or treatment. Medications used to treat mental disorders are called psychotropic or psychoactive medications and are prescribed by medical doctors, including psychiatrists. In Louisiana and New Mexico as well as in the military, specially trained psychologists are able to prescribe some types of these medications as well (American Psychological Association, 2014). Specially trained nurse practitioners are also able to prescribe psychotropic drugs under the supervision of a physician.

Neurotransmitters and Psychotropic Drugs

There are several different types of **neurotransmitters** released by different neurons, and we can speak in broad terms about the kinds of functions associated with different neurotransmitters (Table 1). Much of what psychologists know about the functions of neurotransmitters comes from research on the effects of drugs in psychological disorders. **Psychotropic** or psychoactive drugs are any type of drug or chemical that alters the way a person thinks, feels, or behaves; most people have experience with at least some of these compounds, caffeine being the most common example. Some psychoactive drugs are illegal, mostly due to their potential for addiction (e.g., cocaine, heroin, and methamphetamine), but we will focus here on psychoactive drugs used in treatment. Research on the effects of different neurotransmitters, their interactions, and their role in mental disorders is ongoing, and we continue to learn.

Table 1. Major Neurotransmitters and How They Affect Behavior

Neurotransmitter	Involved in and With	Potential Effect on Behavior
Acetylcholine	Muscle action, memory, Alzheimer's disease	Increased arousal, enhanced cognition
Beta-endorphin (sometimes called endogenous or internal opioids)	Pain, pleasure	Decreased anxiety, decreased tension
Dopamine	Mood, sleep, learning, motor functions in Parkinson's, schizophrenia	Increased pleasure, suppressed appetite
Gamma-aminobutyric acid (GABA)	Brain function, sleep, anxiety disorders, epilepsy, major inhibitory neurotransmitter	Decreased anxiety, decreased tension
Glutamate	Memory, learning, major excitatory neurotransmitter	Increased learning, enhanced memory
Norepinephrine	Heart, intestines, alertness, arousal, depression	Increased arousal, suppressed appetite
Serotonin	Mood, sleep, depression, aggression, schizophrenia	Modulates mood, suppressed appetite

Psychotropic drugs can act as agonists or antagonists for a given neurotransmitter system. **Agonists** are chemicals that mimic a neurotransmitter at the receptor site and thus strengthen its effects. An **antagonist** (like an opponent)**,** on the other hand, blocks or impedes the normal activity of a neurotransmitter at the postsynaptic receptor. We know from the biopsychosocial model and epigenetics that there are complex interactions between neurotransmitter systems and also with the environment. For example, there is evidence that obsessive-compulsive disorder (OCD) involves interactions between serotonin systems and dopamine systems, and some studies have shown similar increased levels of serotonin in patients who responded to treatment, whether that treatment involved the use of medications or only psychotherapy with no drug treatment. Although mental disorder symptoms are not caused solely by dysfunctions in a neurotransmitter system, drug treatments have been developed to target specific symptoms and improve functioning.

For example, Parkinson's disease, a progressive nervous system disorder, is associated with low levels of dopamine due to the death of neurons that produce dopamine. Therefore, a common treatment strategy for Parkinson's disease involves using dopamine agonists, which mimic the effects of dopamine by binding to dopamine receptors. In contrast, some symptoms of schizophrenia are associated with overactive dopamine neurotransmission. The antipsychotics used to treat these symptoms are antagonists for dopamine—they block dopamine's effects by binding its receptors without activating them. Thus, they prevent dopamine released by one neuron from signaling information to adjacent neurons. Remember that these issues are complex. For instance, there have been at least *five* different dopamine receptor types in the brain and two more are being investigated; these are further divided into two "families" of dopamine receptors. The effects of dopamine binding to these different postsynaptic receptors may vary depending on which receptor is activated by the neurotransmitter.

In contrast to agonists and antagonists, which both operate by binding to receptor sites, reuptake inhibitors prevent unused neurotransmitters from being transported back to the neuron. This allows neurotransmitters to remain active in the synaptic cleft for longer durations, increasing their effectiveness. Depression, which has been consistently linked with reduced serotonin levels, is commonly treated with selective serotonin reuptake inhibitors (SSRIs). By preventing reuptake, SSRIs strengthen the effect of serotonin, giving it more time to interact with serotonin receptors on the postsynaptic dendrites. Common SSRIs on the market today include Prozac, Paxil, and Zoloft. The drug LSD is structurally very similar to serotonin, and it affects the same neurons and receptors as serotonin.

Psychotropic drugs are not instant solutions for people suffering from psychological disorders. Often, an individual must take a drug for several weeks before seeing improvement, and many psychoactive drugs have significant negative side effects. Furthermore, individuals vary dramatically in how they respond to the drugs, sometimes leading patients to feel that they are being "experimented on." In a way, this perception is true—the doctor is testing which drug and dosages produce the best effect for a given individual. At this moment, we do not have any type of blood test or other way to reveal which dose or which drug would work best for a given

patient. Doctors often base initial doses on national recommendations and types of drugs based on family history. For example, if a patient's older sister was diagnosed with depression and tried several drugs, but she found that Celexa worked better than Paxil, then the psychiatrist might begin the patient's treatment with Celexa. This may work, but it may not. In addition, some people just do not improve with antidepressants at all (Ioannidis, 2008). As we better understand why individuals differ, the easier and more rapidly we will be able to help people in distress.

One area that has received interest recently has to do with an individualized treatment approach. We now know that there are genetic differences in some of the cytochrome P450 enzymes found in our cells and their ability to break down drugs. The general population falls into the following four categories: 1) *ultra-extensive metabolizers* break down certain drugs (like some of the current antidepressants) very, very quickly; 2) *extensive metabolizers* are also able to break down drugs fairly quickly; 3) *intermediate metabolizers* break down drugs more slowly than either of the two above groups; and finally 4) *poor metabolizers* break down drugs much more slowly than all the other groups.

Now consider someone receiving a prescription for an antidepressant—what would the consequences be if they were either an ultra-extensive metabolizer or a poor metabolizer? The ultra-extensive metabolizer would be given antidepressants and told it will probably take four to six weeks to begin working (this is true), but they metabolize the medication so quickly that it will never be effective for them. In contrast, the poor metabolizer given the same daily dose of the same antidepressant may build up such high levels in their blood (because they are not breaking the drug down) that they will have a wide range of side effects and feel really badly—also not a positive outcome. What if instead, prior to prescribing an antidepressant, the doctor could take a blood sample and determine which type of metabolizer a patient actually was? They could then make a much more informed decision about the best dose to prescribe. There are new genetic tests now available to better individualize treatment in just this way. A blood sample can determine (at least for some drugs) which category an individual fits into, but we need data to determine if this test actually is effective for treating depression or other mental illnesses (Zhou, 2009). Currently, this genetic test is expensive and not many health insurance plans cover this screen, but this test may be an important component in the future of psychopharmacology.

Another approach to improve chances for success is for people receiving pharmacotherapy to undergo psychological and/or behavioral therapies as well. Some research suggests that combining drug therapy with other forms of therapy tends to be more effective than any one treatment alone (for one such example, see March et al., 2007).

Common Treatment Approaches

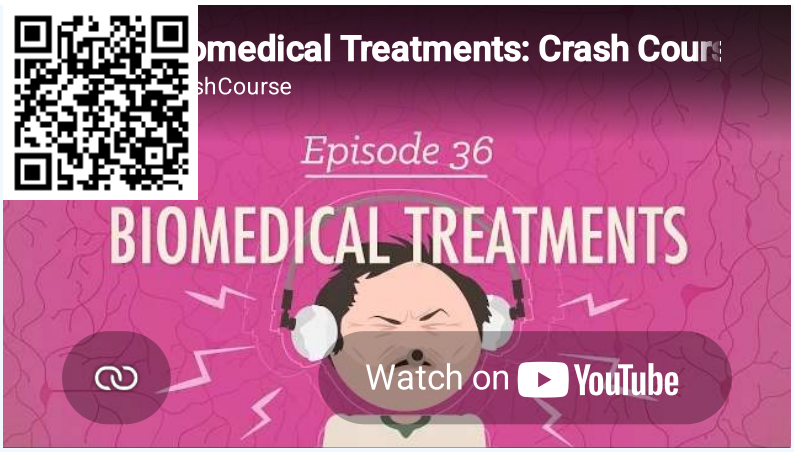
Different types and classes of medications are prescribed for different disorders. For instance, a person with depression might be given an antidepressant, an individual with bipolar disorder might be given a mood stabilizer, and a person diagnosed with schizophrenia might be given an antipsychotic medication. Unlike antibiotics that may target and kill a nasty bacterial infection in the body, psychotropic drugs mostly treat the symptoms of a mental disorder. They can help people feel better so that they can function on a daily basis, but they do not cure the disorder, similar to other medical treatments for high blood pressure or diabetes, for example. Some people may only need to take a psychotropic medication for a short period of time. Others with severe disorders like bipolar disorder or schizophrenia may need to take psychotropic medication for a long time, possibly for the rest of their lives. Table 2 shows the types or classes of medications and how they are used.

Table 2. Commonly Prescribed Psychotropic Medications

Type of Medication	Used to Treat	Example Brand Names of Commonly Prescribed Medications	How They Work	Side Effects
Antipsychotics (developed in the 1950s)	Schizophrenia and other types of severe thought disorders	Haldol, Mellaril, Prolixin, Thorazine	Treat positive psychotic symptoms such as auditory and visual hallucinations, delusions, and paranoia by blocking the neurotransmitter dopamine	Long-term use can lead to tardive dyskinesia, involuntary movements of the arms, legs, tongue, and facial muscles, resulting in Parkinson's-like tremors
Atypical Antipsychotics (developed in the late 1980s)	Schizophrenia and other types of severe thought disorders	Abilify, Risperdal, Clozaril	Treat the negative symptoms of schizophrenia, such as withdrawal and apathy, by targeting both dopamine and serotonin receptors; newer medications may treat both positive and negative symptoms	Can increase the risk of obesity and diabetes as well as elevate cholesterol levels; constipation, dry mouth, blurred vision, drowsiness, and dizziness
Anti-depressants	Depression and increasingly for anxiety	Paxil, Prozac, Zoloft (selective serotonin reuptake inhibitors, [SSRIs]); Tofranil and Elavil (tricyclics)	Alter levels of neurotransmitters such as serotonin and norepinephrine	SSRIs: headache, nausea, weight gain, drowsiness, reduced sex drive Tricyclics: dry mouth, constipation, blurred vision, drowsiness, reduced sex drive, increased risk of suicide
Anti-anxiety agents	Anxiety and agitation that occur in OCD, PTSD, panic disorder, and social phobia	Xanax, Valium, Ativan (Benzodiazepines) Buspar (non-Benzodiazepine)	Depress central nervous system activity	Drowsiness, dizziness, headache, fatigue, lightheadedness
Mood Stabilizers	Bipolar disorder	Lithium, Depakote, Lamictal, Tegretol	Treat episodes of mania as well as depression	Excessive thirst, irregular heartbeat, itching/rash, swelling (face, mouth, and extremities), nausea, loss of appetite
Stimulants	ADHD	Adderall, Ritalin	Improve ability to focus on a task and maintain attention	Decreased appetite, difficulty sleeping, stomachache, headache

Watch It

Watch the selected portion of this CrashCourse video to learn more about research, biomedical therapy, and drug treatments as well as alternative biological treatments for mental disorders.



You can view the transcript for "Biomedical Treatments: Crash Course Psychology #36" here (opens in new window).

Brain Stimulation Treatments



Figure **3.6.1**: The Bergonic Chair was a device used in the World War I era to deliver an early form of ECT.

Brain stimulation therapies can play a role in treating certain mental disorders. These therapies involve activating or inhibiting neuron systems directly using electricity. The electricity can be given directly by electrodes surgically implanted in the brain, or non-invasively through electrodes placed on the scalp. The electricity can also be induced by using magnetic fields applied to the head that penetrate into the brain. While these types of therapies are less frequently used than medication and psychotherapies, they can be helpful for treating certain mental disorders that have not responded to previous treatments with medications and/or psychotherapy or in cases where a rapid response is required such as patients with high suicide risk or catatonia, a form of psychosis in which the patient is unable to move or respond to the outside world and in cases where patients may not be eating or drinking. In other words, they are often considered to be "second line" treatments. In order to reduce the possibility of relapse, many of these treatments may involve follow-up treatment with medications and/or therapy.



Figure **3.6.2**:ECT is often portrayed in the media as scary, but ECT today is administered in a safe and controlled environment and known to be effective in treating severe depression.

Electroconvulsive therapy (ECT; depicted in Figure **3.6.1** and previously called "electroshock therapy," a term that is no longer used) is the best-studied brain stimulation therapy and has the longest history of use. ECT is most often used to treat severe, treatment-resistant depression, but it may also be sometimes useful in other mental disorders, such as bipolar disorder or schizophrenia, especially when used in combination with medications.[1] Two major advantages of ECT over medications are that ECT usually begins to work more quickly, often starting within the first week, and older individuals respond especially quickly. Before ECT is administered, a person is sedated with general anesthesia and given a medication called a muscle relaxant to prevent movement during the procedure, reducing the risk of physical injury. An anesthesiologist monitors breathing, heart rate, and blood pressure during the entire procedure, which is conducted by a trained medical team, including physicians and nurses. At the start of the procedure, electrodes are placed at precise locations on the head. Through the electrodes, an electric current passes through the brain, causing a seizure that lasts generally less than one minute. Because the patient is under anesthesia and has taken a muscle relaxant, it is not painful and the patient cannot

feel the electrical impulses. Five to ten minutes after the procedure ends, the patient awakens. He or she may feel groggy at first as the anesthesia wears off. But after about an hour, the patient usually is alert and can resume normal activities.

A typical course of ECT is administered about three times a week until the patient's depression improves (usually within six to 12 treatments). After that, maintenance ECT treatment is sometimes needed to reduce the chances that symptoms will return. ECT maintenance treatment varies depending on the needs of the individual, and may range from one session per week to one session every few months. As noted above, follow-up treatment may include medications and/or psychotherapy. The most common side effects associated with ECT include headache, upset stomach, muscle aches, and memory loss. Some people may experience more significant memory problems, especially of memories around the time of the treatment. Less commonly, these memory problems may be more severe, but usually, they improve over the days and weeks following the end of an ECT course. Research has found that memory problems seem to be more associated with the traditional type of ECT called bilateral ECT, in which the electrodes are placed on both sides of the head. In unilateral ECT, the electrodes are placed on just one side of the head—typically the right side because it is opposite the brain's learning and memory areas. Unilateral ECT has been found to be less likely to cause memory problems and therefore is preferred by many doctors, patients, and families.



Figure 3.6.3: A woman undergoing transcranial magnetic stimulation.

Repetitive transcranial magnetic stimulation (rTMS) uses a magnet to activate neurons in the brain. First developed in 1985, **repetitive transcranial magnetic stimulation (**rTMS) has been studied as a treatment for depression, psychosis, anxiety, and other disorders. Unlike ECT, in which electrical stimulation is more generalized, repetitive transcranial magnetic stimulation (rTMS) can be targeted to a specific site in the brain. Scientists believe that focusing on a specific site in the brain reduces the chance for the types of side effects associated with ECT. But experts have not yet reached a conclusion as to what site is best. In 2008, rTMS was approved for use by the FDA as a treatment for major depression for patients who do not respond to at least one antidepressant medication in the current episode. It is also used in other countries as a treatment for depression in patients who have not responded to medications and who might otherwise be considered for ECT. The evidence supporting rTMS for depression was mixed until the first large clinical trial, funded by NIMH, was published in 2010. The trial found that 14% achieved remission with rTMS compared to 5% with an inactive (placebo) treatment that mimics real rTMS. After the trial ended, patients could enter a second phase in which everyone, including those who previously received the placebo treatment, was given rTMS. Remission rates during the second phase climbed to nearly 30%.

A typical rTMS session lasts 30 to 60 minutes and does not require anesthesia. During the procedure, an electromagnetic coil is held against the forehead near an area of the brain that is thought to be involved in mood regulation. Then, short electromagnetic pulses are administered through the coil. The magnetic pulses easily pass through the skull and cause small electrical currents that stimulate nerve cells in the targeted brain region. Because this type of pulse generally does not reach further than two inches into the brain, scientists can select which parts of the brain will be affected and which will not be. The magnetic field is about the same strength as that of a magnetic resonance imaging (MRI) scan. Generally, the person feels a slight knocking or tapping on the head as the pulses are administered. There are still disagreements among scientists on the best way to position the magnet on the patient's head or administer the electromagnetic pulses. At this time, it is also unclear if rTMS works best when given as a single treatment or combined with medication and/or psychotherapy. More research is underway to determine the safest and most effective uses of rTMS. Sometimes a person may have discomfort at the site on the head where the magnet is placed. The muscles of the scalp, jaw, or face may contract or tingle during the procedure. Mild headaches or brief light-headedness may result. It is also possible that the procedure could cause a seizure, although documented incidences of this are uncommon. Two large-scale studies on the safety of rTMS found that most side effects, such as headaches or scalp discomfort, were mild or moderate, and no seizures occurred. Because the treatment is relatively new, however, long-term side effects are unknown.

Magnetic seizure therapy (MST) borrows certain aspects from both ECT and rTMS. Like rTMS, magnetic seizure therapy (MST) uses magnetic pulses instead of electricity to stimulate a precise target in the brain. However, unlike rTMS, magnetic seizure therapy

(MST) aims to induce a seizure similar to ECT. The pulses are given at a higher frequency than that used in rTMS, and the patient must be anesthetized and given a muscle relaxant to prevent movement. The goal of MST is to retain the effectiveness of ECT while reducing its cognitive side effects. MST is in the early stages of testing for mental disorders, but initial results are promising. A recent review article that examined the evidence from eight clinical studies found that MST triggered remission from major depression or bipolar disorder in 30–40% of individuals. MST carries the risk of side effects that can be caused by anesthesia exposure and the induction of a seizure. Studies in both animals and humans have found that MST produces fewer memory side effects, shorter seizures, and a shorter recovery time than ECT.

Vagus nerve stimulation (VNS) works through a device, called a pulse generator, implanted under the skin that sends electrical pulses through the left vagus nerve, half of a prominent pair of nerves that run from the brainstem through the neck and down to each side of the chest and abdomen. The vagus nerves carry messages from the brain to the body's major organs (e.g. heart, lungs and intestines) and to areas of the brain that control mood, sleep, and other functions. Vagus nerve stimulation (VNS) was originally developed as a treatment for epilepsy. However, scientists noticed that it also had favorable effects on mood, especially depressive symptoms. Using brain scans, scientists found that the device affected areas of the brain that are involved in mood regulation. The pulses appeared to alter the levels of serotonin, norepinephrine, GABA, and glutamate. In 2005, the U.S. Food and Drug Administration (FDA) approved vagus nerve stimulation (VNS) for use in treating treatment-resistant depression in specific circumstances: the patient is an adult (over 18 years of age), the disorder has persisted over two years or more and is severe or recurrent, and there has been no significant relief after attempting four prior treatments. According to the FDA, it is not intended to be a first-line treatment, even for patients with severe depression. Despite FDA approval, VNS is not frequently used because results of early studies testing its effectiveness for major depression were mixed. But a newer study, which pooled together findings from only controlled clinical trials, found that 32% of depressed people responded to VSN and 14% had a full remission of symptoms after being treated for nearly two years. It is important to note that it may be several months before the patient notices any benefits and not all patients will respond to VNS. Patients undergoing VNS are expected to continue use of other treatments even with the pulse generator in place.

Additionally, there may be complications from VNS such as infection from the implant surgery, or the device may come loose, move around or malfunction, which may require additional surgery to correct. Some patients have no improvement in symptoms and some actually get worse. Other wide effects may include voice changes or hoarseness, cough or sore throat, neck pain, discomfort or tingling where the pulse generator is implanted, breathing problems during exercise (the generator can be temporarily turned off for exercise), and difficulty swallowing. Long-term side effects are unknown at this time.

Deep brain stimulation (DBS) was first developed as a treatment for Parkinson's disease to reduce tremor, stiffness, walking problems, and uncontrollable movements. In deep brain stimulation (DBS), a pair of electrodes is implanted in the brain and controlled by a generator that is implanted in the chest. Stimulation is continuous and its frequency and level are customized to the individual. Deep brain stimulation (DBS) has been studied as a treatment for obsessive-compulsive disorder (OCD) and depression, although its use in treating depression is still considered experimental. A review of all 22 published studies testing DBS for depression found that only three of them were of high quality because they included both a treatment group and a control group. The review found that across the studies, 40–50% of patients showed greater than 50% improvement in symptoms.

Unlike VNS, DBS requires brain surgery, guided by MRI scans of the brain. Once inside the skull, the surgeon threads a slender tube down into the brain to place electrodes on each side of a specific area of the brain. For OCD, the electrodes are placed in an area of the brain (the ventral capsule/ventral striatum) believed to be associated with the disorder. In the case of depression, the first area of the brain targeted by DBS is called Area 25, or the subgenual cingulate cortex. This area has been found to be overactive in depression and other mood disorders. But later research targeted several other areas of the brain affected by depression. The electrodes in the brain are then attached to wires that run from the head down to the chest, where a pair of battery-operated generators are implanted. From here, electrical pulses are continuously delivered over the wires to the electrodes in the brain. Although it is unclear exactly how the device works to reduce OCD or depression, scientists believe that the pulses help to "reset" the area of the brain that is malfunctioning so that it works normally again. Because DBS involves surgery, there is a list of potential side-effects including bleeding in the brain or stroke, infection, disorientation or confusion, mood changes, movement disorders, lightheadedness, and trouble sleeping. Because the procedure is still being studied, other side effects not yet identified may be possible. Long-term benefits and side effects are unknown.

Dig Deeper: Evidence-based Practice

A buzzword in therapy today is evidence-based practice. However, it's not a novel concept but one that has been used in medicine for at least two decades. Evidence-based practice is used to reduce errors in treatment selection by making clinical decisions based

on research (Sackett & Rosenberg, 1995). In any case, evidence-based treatment is on the rise in the field of psychology. So what is it, and why does it matter? In an effort to determine which treatment methodologies are evidence-based, professional organizations such as the American Psychological Association (APA) have recommended that specific psychological treatments be used to treat certain psychological disorders (Chambless & Ollendick, 2001). According to the APA (2005), "Evidence-based practice in psychology (EBPP) is the integration of the best available research with clinical expertise in the context of patient characteristics, culture, and preferences" (p. 1).

The foundational idea behind evidence-based treatment is that best practices are determined by research that has been compiled by comparing various forms of treatment (Charman & Barkham, 2005). These treatments are then operationalized and placed in treatment manuals—trained therapists follow these manuals. The benefits are that evidence-based treatment can reduce variability between therapists to ensure that a specific approach is delivered with integrity (Charman & Barkham, 2005). Therefore, clients have a higher chance of receiving therapeutic interventions that are effective at treating their specific disorder. While EBPP is based on randomized control trials, critics of EBPP reject it, stating that the results of trials cannot be applied to individuals and instead determinations regarding treatment should be based on a therapist's judgment (Mullen & Streiner, 2004).

Glossary

biomedical therapy: treatment that involves medication and/or medical procedures to treat psychological disorders

electroconvulsive therapy (ECT): type of biomedical therapy that involves using an electrical current to induce seizures in a person to help alleviate the effects of severe depression

lobotomy: a form of psychosurgery in which parts of the frontal lobe of the brain are destroyed or their connections to other parts of the brain severed

psychotropic drugs: also referred to as psychoactive drugs; a drug or chemical agent that influences thoughts, emotions, and behavior

1. Sanghani SN, Petrides G, Kellner CH. Electroconvulsive therapy (ECT) in schizophrenia: a review of recent literature. Curr Opin Psychiatry. 2018 May;31(3):213–222. doi: 10.1097/YCO.0000000000000418. PMID: 29528902. ↵

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3.7: Introduction to Psychodynamic and Humanistic Approaches to Psychopathology

What you'll learn to do: describe the key concepts and treatment methods connected to the understanding of mental illness from psychodynamic and humanistic approaches



Figure **3.7.1**: Sigmund Freud's famous couch. Freud is the founder of the psychodynamic theory; while many of his ideas are now considered inaccurate, his approach to treatment and psychotherapy continues to have a profound effect on treatment today.

You have learned about biomedical approaches and treatments for mental illness. Now, we will turn our attention to psychodynamic and humanistic approaches. These perspectives look inward at psychological, rather than biological, causes for mental illness.

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3.8: The Psychoanalytic Model

Learning Objectives

- Describe Freud's concept of the unconscious and explain how the three basic elements of personality interact
- Explain the psychoanalytic concepts of psychosexual development and defense mechanisms

In contrast to the biological aspects of the biopsychosocial model of psychopathology, the next five models described focus mostly on psychological and social/cultural aspects of human beings that contribute to the development of mental disorders. Each of these models was developed in an attempt to investigate and explain aspects of human development, emotions, thoughts, personality, and behaviors that could lead to specific symptoms or disorders. The concepts within these models also suggest treatments or interventions that could reduce suffering and improve a person's ability to adapt to their challenges in life. As we now know, all these models are incomplete, but they each provide different perspectives on psychopathology that can be helpful both in understanding various disorders or problems and that have led to a variety of treatment approaches. The psychodynamic model has a strong emphasis on human development and the effect of psychological processes we may not be consciously aware of.

Sigmund Freud (1856–1939) is probably the most controversial and misunderstood psychological theorist. When reading Freud's theories, it is important to remember that he was a medical doctor, not a psychologist, and his ideas were also related to the times and culture in which he lived. While Freud's focus on biological drives led him to emphasize the impact of sociocultural factors on personality development (due to conflicts between these forces), his followers quickly realized that biology alone could not account for the diversity they encountered as the practice of psychoanalysis spread during the time of the Nazi Holocaust. The antisemitism which was prevalent during this period of time may also have led mainstream psychoanalysts to focus primarily on the universality of the psychological structures of the mind. Furthermore, there was no such thing as a degree in psychology at the time that he received his education, which can help us understand some of the controversy over his theories today. However, Freud was the first to systematically study and theorize the workings of the unconscious mind in the manner that we associate with modern psychology.

In the early years of his career, Freud worked with Josef Breuer, a Viennese physician. During this time, Freud became intrigued by the story of one of Breuer's patients, Bertha Pappenheim, who was referred to by the pseudonym Anna O. (Launer, 2005). Anna O. had been caring for her dying father when she began to experience symptoms such as partial paralysis, headaches, blurred vision, amnesia, and hallucinations (Launer, 2005). In Freud's day, these symptoms were commonly referred to as hysteria. Anna O. turned to Breuer for help. He spent two years (1880–1882) treating Anna O. and discovered that allowing her to talk about her experiences seemed to bring some relief of her symptoms. Anna O. called his treatment the "talking cure" (Launer, 2005). Despite the fact the Freud never met Anna O., her story served as the basis for the 1895 book, *Studies on Hysteria*, which he co-authored with Breuer. Based on Breuer's description of Anna O.'s treatment, Freud concluded that hysteria was the result of sexual abuse in childhood and that these traumatic experiences had been hidden from consciousness. Breuer disagreed with Freud, which soon ended their work together. However, Freud continued to work to refine talk therapy and build his theory on personality.

Levels of Consciousness

To explain the concept of conscious versus unconscious experience, Freud compared the mind to an iceberg (Figure **3.8.1**). He said that only about one-tenth of our mind is **conscious** or self-aware, and the rest of our mind is unconscious. Our **unconscious** refers to mental activity (emotional reactions, motivations) of which we are unaware and are unable to access (Freud, 1923). According to Freud, unacceptable urges and desires are kept in our unconscious through a process called repression. For example, we sometimes say things that we don't intend to say by unintentionally substituting another word for the one we meant. You have probably heard of a Freudian slip, the term used to describe this. Freud suggested that slips of the tongue are actually sexual or aggressive urges, accidentally slipping out of our unconscious. However, speech errors such as this are quite common. Instead of seeing them only as a reflection of unconscious desires, linguists today have found that slips of the tongue tend to occur when we are tired, nervous, or not at our optimal level of cognitive functioning (Motley, 2002).

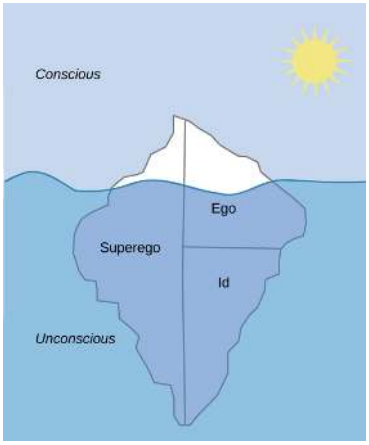


Figure **3.8.1**: Freud believed that we are only aware of a small amount of our mind's activities and that most of it remains hidden from us in our unconscious. The information in our unconscious affects our behavior, although we are unaware of it.

According to Freud, our personality develops from a conflict between two forces: our biological aggressive and pleasure-seeking drives versus our internal (socialized) control over these drives. Our personality is the result of our efforts to balance these two competing forces. Freud suggested that we can understand this by imagining three interacting systems within our minds. He called them the id, ego, and superego (Figure **3.8.2**).

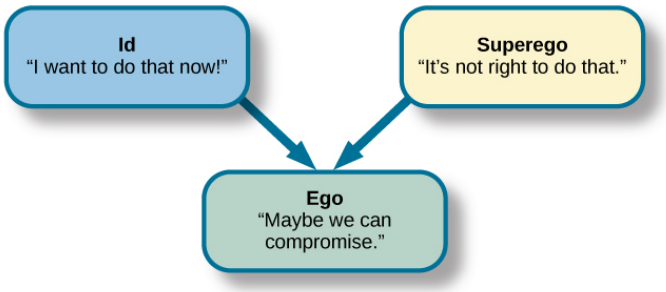


Figure **3.8.2**: The job of the ego, or self, is to balance the aggressive/pleasure-seeking drives of the id with the moral control of the superego.

The unconscious **id** contains our most primitive drives or urges and is present from birth. It directs impulses for hunger, thirst, aggression, and sex. Freud believed that the id operates on what he called the "pleasure principle," in which the id seeks immediate gratification. Through social interactions with parents and others in a child's environment, the ego and superego develop to help control the id. The **superego** develops as a child interacts with others, learning the social rules for right and wrong. The superego acts as our conscience; it is our moral compass that tells us how we should behave. It strives for perfection and judges our behavior, leading to feelings of pride or—when we fall short of the ideal—feelings of guilt. In contrast to the instinctual id and the rule-based superego, the **ego** is the rational part of our personality. It's what Freud considered to be the self, and it is the part of our personality that is seen by others. Its job is to balance the demands of the id and superego in the context of reality; thus, it operates on what Freud called the "reality principle." The ego helps the id satisfy its desires in practical and socially acceptable ways.

The id and superego are in constant conflict because the id wants instant gratification regardless of the consequences, but the superego tells us that we must behave in ways that align with social and cultural expectations and demands. Thus, the ego's job is to find the middle ground. The ego helps satisfy the id's desires in a rational way that will not lead us to feelings of guilt. According to Freud, a person who has a strong ego, which can balance the demands of the id and the superego, has a healthy personality. Freud maintained that imbalances in the system can lead to **neurosis** (a tendency to experience negative emotions), anxiety disorders, or unhealthy behaviors. For example, a person who is dominated by their id might be narcissistic and impulsive. A person with a dominant superego might be controlled by feelings of guilt and deny themselves even socially acceptable pleasures; conversely, if the superego is weak or absent, a person might become a psychopath. An overly dominant superego might be seen in an over-controlled individual whose rational grasp on reality is so strong that they are unaware of their emotional needs, or, in a neurotic person who is overly defensive (overusing ego defense mechanisms).

Stages of Psychosexual Development

Freud believed that personality develops during early childhood: childhood experiences shape our personalities as well as our behavior as adults. He asserted that we develop via a series of stages during childhood. Each of us must pass through these childhood stages, and if we do not have the proper nurturing and parenting during a stage, we will be stuck, or fixated, in that stage, even as adults.

In each **psychosexual stage of development**, the child's pleasure-seeking urges, coming from the id, are focused on a different area of the body, called an erogenous zone. The stages are oral, anal, phallic, latency, and genital (see Table 1).

Freud's psychosexual development theory is quite controversial. To understand the origins of the theory, it is helpful to be familiar with the political, social, and cultural influences of Freud's day in Vienna at the turn of the 20th century. This era combined a climate of sexual repression with limited understanding and education surrounding human sexuality, and strongly defined sexual and social roles for males and females. These cultural forces heavily influenced Freud's perspective. Given that sex was a taboo topic, Freud assumed that negative emotional states (neuroses) stemmed from suppression of unconscious sexual and aggressive urges. For Freud, his own recollections and personal interpretations of patients' experiences and dreams were sufficient proof that psychosexual stages were universal events in early childhood.

Table 1. Freud's Stages of Psychosexual Development

Stage	Age (years)	Erogenous Zone	Major Conflict	Adult Fixation Example
Oral	0–1	Mouth	Weaning off breast or bottle	Smoking, overeating
Anal	1–3	Anus	Toilet training	Neatness, messiness
Phallic	3–6	Genitals	Oedipus/Electra complex	Vanity, overambition
Latency	6–12	None	None	None
Genital	12+	Genitals	None	None

Oral Stage

In the **oral stage** (birth to one year), pleasure is focused on the mouth. Eating and the pleasure derived from sucking (nipples, pacifiers, and thumbs) play a large part in a baby's first year of life. At around one year of age, babies are weaned from the bottle or breast, and this process can create conflict if not handled properly by caregivers. According to Freud, an adult who smokes, drinks, overeats, or bites her nails is fixated in the oral stage of her psychosexual development; she may have been weaned too early or too late, resulting in these fixation tendencies, all of which seek to ease anxiety.

Anal Stage

After passing through the oral stage, children enter what Freud termed the **anal stage** (one to three years). In this stage, children experience pleasure in their bowel and bladder movements, so it makes sense that the conflict in this stage is over toilet training. During this stage of development, children work to master control of themselves. Freud suggested that success at the anal stage depended on how parents handled toilet training. Parents who offer praise and rewards encourage positive results and can help children feel competent. Parents who are harsh in toilet training can cause a child to become so fearful of soiling that they over-control and become fixated at the anal stage, leading to the development of an anal-retentive personality. The anal-retentive personality is stingy and stubborn, has a compulsive need for order and neatness, and might be considered a perfectionist. If parents are too lenient in toilet training, the child may fail to develop sufficient self-control, become fixated at this stage, and develop an anal-expulsive personality. The anal-expulsive personality is messy, careless, disorganized, and prone to emotional outbursts.

Phallic Stage

Freud's third stage of psychosexual development is the **phallic stage** (three to six years), corresponding to the age when children become aware of their bodies and recognize the differences between boys and girls. The erogenous zone in this stage is the genitals. Conflict arises when the child feels a desire for the opposite-sex parent, and jealousy and hatred toward the same-sex parent. For boys, this is called the Oedipus complex, involving a boy's desire for his mother and his urge to replace his father, who is seen as a rival for the mother's attention. At the same time, the boy is afraid his father will punish him for his feelings, so he experiences *castration anxiety*. The Oedipus complex is successfully resolved when the boy begins to identify with his father as an indirect way to have the mother. Failure to resolve the Oedipus complex may result in fixation and development of a personality that might be described as vain and overly ambitious.

Girls experience a comparable conflict in the phallic stage—the Electra complex. The Electra complex, while often attributed to Freud, was actually proposed by Freud's protégé, Carl Jung (Jung & Kerenyi, 1963). A girl desires the attention of her father and wishes to take her mother's place. Jung also said that girls are angry with the mother for not providing them with a penis—hence the term *penis envy*. While Freud initially embraced the Electra complex as a parallel to the Oedipus complex, he later rejected it; yet it remains as a cornerstone of Freudian theory, thanks in part to academics in the field (Freud, 1931/1968; Scott, 2005).

Latency Period

Following the phallic stage of psychosexual development is a period known as the **latency period** (six years to puberty). This period is not considered a stage because sexual feelings are dormant as children focus on other pursuits, such as school, friendships, hobbies, and sports. Children generally engage in activities with peers of the same sex, which serves to consolidate a child's gender-role identity.

Genital Stage

The final stage is the **genital stage** (from puberty on). In this stage, there is a sexual reawakening as the incestuous urges resurface. The young person redirects these urges to other, more socially acceptable partners (who often resemble the other-sex parent). People in this stage have mature sexual

interests, which for Freud meant a strong desire for the opposite sex. Individuals who successfully completed the previous stages, reaching the genital stage with no fixations, are said to be well-balanced, healthy adults.

Defense Mechanisms

Freud believed that feelings of anxiety result from the ego's inability to mediate the conflict between the id and superego. When this happens, Freud believed that the ego seeks to restore balance by reducing anxiety through various protective measures known as **defense mechanisms** (Figure 3.8.3). The ego, usually conscious, resorts to these *unconscious* actions or behaviors to protect itself from becoming overwhelmed. Thus, when we use defense mechanisms, we are unaware that we are using them. Further, they operate in various ways that may distort reality. According to Freud, we all use ego defense mechanisms; they are a normal part of life, but defense mechanisms can also backfire if the underlying conflict is never resolved.

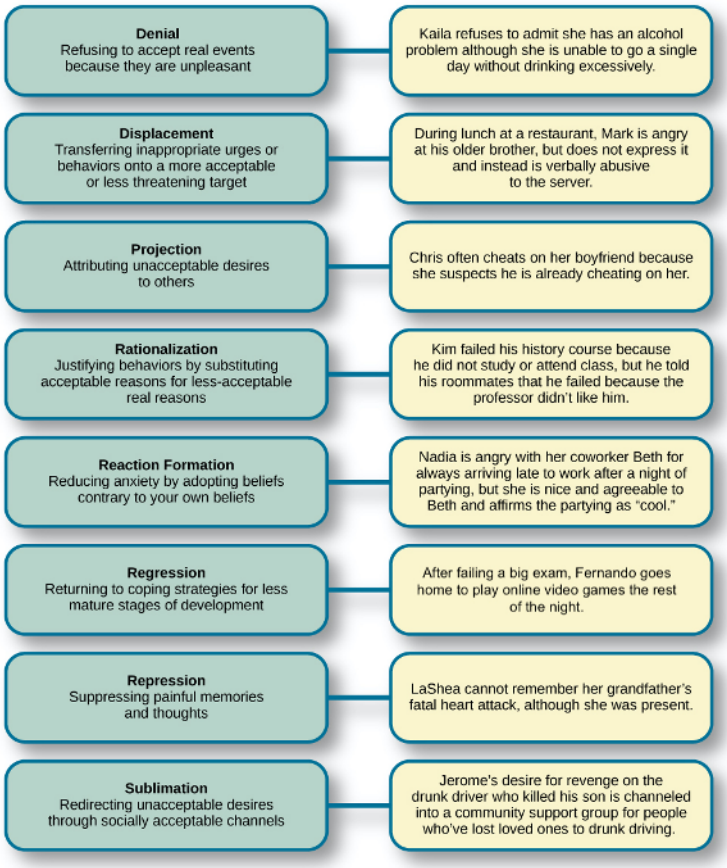


Figure 3.8.3: Defense mechanisms are unconscious, protective behaviors that work to reduce anxiety.

While everyone uses defense mechanisms, Freud believed that overuse of them may be problematic. For example, let's say Aaron Snyder is a high school football player. Aaron's id feels sexually attracted to males. His culturally shaped superego-driven conscious belief is that being gay is immoral and that if he were gay, his family would disown him and he would be ostracized by his peers. Therefore, there is a conflict between his conscious beliefs (being gay is wrong and will result in being ostracized) and his unconscious urges (attraction to males). The idea that he might be gay causes Joe to have feelings of anxiety, possibly overwhelming his ego or ability to balance this internal conflict. How can he decrease his anxiety? Joe may find himself acting very "macho," making gay jokes, and picking on a school peer who is gay. This way, Joe's unconscious impulses are further submerged, and his anxiety is reduced because he "fits in."

There are several different types of defense mechanisms. For instance, in repression, anxiety-causing memories from consciousness are blocked. As an analogy, let's say your car is making a strange noise, but because you do not have the money to get it fixed, you just turn up the radio so that you no longer hear the strange noise. Eventually you forget about it. Similarly, in the human psyche, if a memory is too overwhelming to deal with, it might be **repressed** and thus removed from conscious awareness (Freud, 1920). This repressed memory might cause symptoms in other areas.

Another defense mechanism is **reaction formation**, in which someone expresses feelings, thoughts, and behaviors opposite to their inclinations. In the above example, Aaron made fun of a homosexual peer while himself being attracted to males. In **regression**, an individual acts much younger than their age. For example, a four-year-old child who resents the arrival of a newborn sibling may act like a baby and revert to drinking out of a bottle. In **projection**, a person refuses to acknowledge her own unconscious feelings and instead sees those feelings in someone else. Other defense mechanisms include **rationalization**, **displacement**, and **sublimation**.

Freud's Theories Today

Empirical research assessing psychodynamic concepts has produced mixed results, with some concepts receiving empirical support and others not faring as well. For example, the notion that we express strong sexual feelings from a very early age, as the psychosexual stage model suggests, has not held up to empirical scrutiny. On the other hand, the idea that there are dependent, control-oriented, and competitive personality types—an idea also derived from the psychosexual stage model—does seem useful.

Many ideas from the psychoanalytic perspective have been studied empirically. Luborsky and Barrett (2006) reviewed much of this research; other useful reviews are provided by Bornstein (2005), Gerber (2007), and Huprich (2009). For now, let's look at three psychodynamic hypotheses that have received some empirical support:

- *Unconscious processes influence our behavior as the psychodynamic perspective predicts.* We perceive and process much more information than we realize, and much of our behavior is shaped by feelings and motives of which we are, at best, only partially aware (Bornstein, 2009, 2010). Evidence for the importance of unconscious influences is strong enough that it has become a central element of contemporary cognitive and social psychology (Robinson & Gordon, 2011).
- *We all use ego defenses and they help determine our psychological adjustment and physical health.* People really do differ in the degree that they rely on different ego defenses—so much so that some researchers now study each person's "defense style" (the unique constellation of defenses that we use). It turns out that certain defenses are more adaptive than others: rationalization and sublimation are healthier (psychologically speaking) than repression and reaction formation (Cramer, 2006). Denial is, quite literally, bad for your health because people who use denial tend to ignore symptoms of illness until it's too late (Bond, 2004).
- *Mental representations of self and others do indeed serve as blueprints for later relationships.* Dozens of studies have shown that mental images of our parents, and other significant figures, really do shape our expectations for later friendships and romantic relationships. The idea that you choose a romantic partner who resembles mom or dad is a myth, but it's true that you expect to be treated by others as you were treated by your parents early in life (Silverstein, 2007; Wachtel, 1997).

While many of Freud's ideas have *not* found support in modern research, we cannot discount the contributions that Freud has made to the field of psychology. It was Freud who pointed out that a large part of our mental life is influenced by the experiences of early childhood and takes place outside of our conscious awareness; his theories paved the way for others. More modern versions of some of Freud's ideas are described as **psychodynamic** theories or models while Freud's approach and forms of treatment are described as **psychoanalytic.** Psychodynamic theorists and clinicians that expanded on, updated, or altered some of Freud's basic ideas still maintain a strong focus on unconscious motivations and emotions and how they influence behavior as well as the importance of early development. Some of the founders of later psychodynamic approaches were initially followers of Freud and include Carl Jung, Melanie Klein, John Bowlby, Alfred Adler, and Mary Ainsworth.

Glossary

anal stage: psychosexual stage in which children experience pleasure in their bowel and bladder movements

conscious: mental activity (thoughts, feelings, and memories) that we can access at any time

defense mechanism: unconscious protective behaviors designed to reduce ego anxiety

displacement: ego defense mechanism in which a person transfers inappropriate urges or behaviors toward a more acceptable or less threatening target

ego: aspect of personality that represents the self, or the part of one's personality that is visible to others

genital stage: psychosexual stage in which the focus is on mature sexual interests

id: aspect of personality that consists of our most primitive drives or urges, including impulses for hunger, thirst, and sex

latency period: psychosexual stage in which sexual feelings are dormant

neurosis: tendency to experience negative emotions

oral stage: psychosexual stage in which an infant's pleasure is focused on the mouth

phallic stage: psychosexual stage in which the focus is on the genitals

projection: ego defense mechanism in which a person confronted with anxiety disguises their unacceptable urges or behaviors by attributing them to other people

psychoanalytic model: the personality theories and form of treatment developed by Sigmund Freud

psychodynamic model: modern versions of Freud's original concepts originated by his followers still focusing on unconscious processes and development

psychosexual stages of development: stages of child development in which a child's pleasure-seeking urges are focused on specific areas of the body called erogenous zones

rationalization: ego defense mechanism in which a person confronted with anxiety makes excuses to justify behavior

reaction formation: ego defense mechanism in which a person confronted with anxiety swaps unacceptable urges or behaviors for their opposites

regression: ego defense mechanism in which a person confronted with anxiety returns to a more immature behavioral state

repression: ego defense mechanism in which anxiety-related thoughts and memories are kept in the unconscious

sublimation: ego defense mechanism in which unacceptable urges are channeled into more appropriate activities

superego: aspect of the personality that serves as one's moral compass, or conscience

unconscious: mental activity of which we are unaware and unable to access

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3.9: Psychoanalytic and Psychodynamic Therapies

Learning Objectives

- Describe psychoanalysis and psychodynamic treatment methods

The term *psychotherapy* refers to a type of treatment for mental disorders that is mostly focused on psychogenic models of the etiology of psychopathology such as Freud's psychoanalytic model. It is also often called "talk therapy" to separate it from biomedical approaches that rely more on prescribed drugs or medical interventions, including surgery. Because psychotherapy is more interpersonal and interactive than biomedical interventions, the relationship between the therapist and the client is especially important to success. As noted previously, psychotherapeutic approaches (over 400 of them now) have proliferated significantly as the field of psychology has evolved and as we continue to learn more about learning, memory, emotions, and social aspects of human functioning. Because there are no cures for mental disorders regardless of the intervention approach used, the goals of most types of psychotherapy include reducing the severity of symptoms being experienced, enhancing personal growth, and improving the client's ability to function in life. Therapists also aim to reduce or eliminate client destructive patterns and to encourage new solutions to difficult situations. This goal is reflected in the following poem:

***Autobiography in Five Short Chapters* by Portia Nelson (1993)**

Chapter One

I walk down the street.
There is a deep hole in the sidewalk.
I fall in. I am lost. . . . I am helpless.
It isn't my fault.
It takes forever to find a way out.

Chapter Two

I walk down the same street.
There is a deep hole in the sidewalk.
I pretend I don't see it.
I fall in again.
I can't believe I am in this same place.
But, it isn't my fault.
It still takes a long time to get out.

Chapter Three

I walk down the same street.
There is a deep hole in the sidewalk.
I *see* it is there.
I still fall in . . . it's a habit . . . but,
my eyes are open.
I know where I am.
It is *my* fault.
I get out immediately.

Chapter Four

I walk down the same street.
There is a deep hole in the sidewalk.
I walk around it.

Chapter Five

I walk down another street.

For each psychological model, there are specific interventions and methods that have been developed, founded on the core ideas in the model, to treat the client's symptoms and problems. Starting with the psychoanalytic and psychodynamic model, for the rest of this module we will present the major treatment approaches relevant to each model after describing the model (Table 1). Keep in mind that these major approaches have branched out over time with many specific variations. As noted above, there are now over 400 different types of psychotherapy. The goal here is to help you understand core elements and approaches within each major area.

Table 1. Major Psychotherapy Approaches

Type	Description	Example
Psychoanalysis and Psychodynamic psychotherapy	Talk therapy based on the belief that the unconscious and childhood conflicts impact behavior	Patient talks about his or her past
Play therapy	Psychoanalytical therapy wherein interaction with toys is used instead of talk; used in child therapy	Patient (child) acts out family scenes with dolls
Humanistic therapy	Increase self-awareness and acceptance through focus on conscious thoughts	Patient learns to articulate thoughts that keep him or her from achieving his or her goals
Behavior therapy	Principles of learning applied to change undesirable behaviors	Patient learns to overcome fear of elevators through several stages of relaxation techniques
Cognitive therapy	Awareness of cognitive process helps patients eliminate thought patterns that lead to distress	Patient learns not to overgeneralize failure based on single failure
Cognitive-behavioral therapy	Work to change cognitive distortions and self-defeating behaviors	Patient learns to identify self-defeating behaviors to overcome an eating disorder
Integrative Therapies	Forms of psychotherapy that intentionally integrate different approaches into a new coherent approach	Patient learns self-acceptance and increases personal responsibility for change as they practice cognitive change strategies

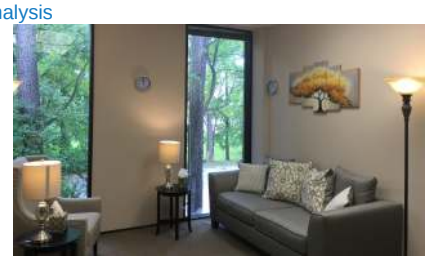


Figure 3.9.1: Unlike the famous couch in Freud's consulting room, where patients were instructed to lie down and face away from Freud, most patients today choose to sit on a couch or chair facing a therapist, as shown in this room.

Psychoanalysis was developed by Sigmund Freud and was the first form of psychotherapy. It was the dominant therapeutic technique in the early 20th century, but it has since waned significantly in popularity. Because Freud believed most of our psychological problems are the result of repressed impulses and trauma experienced in childhood, he developed psychoanalytic methods that he thought would help uncover long-buried feelings and personality conflicts. In a psychoanalyst's office, you might see a patient lying on a couch speaking of dreams or childhood memories, and the therapist using various Freudian methods such as **free association** and dream analysis (Figure 1). In free association, the patient relaxes and then says whatever comes to mind at the moment without attempting to edit or worry how the therapist might react. This enables the therapist to discern unconscious emotions, motives, and drives. Sometimes, free association exercises are applied specifically to childhood recollections. That is, psychoanalysts believe a person's childhood relationships with caregivers often determine the way that person relates to others, and predicts later psychiatric difficulties. However, Freud felt that the ego would at times try to block, or repress, unacceptable urges or painful conflicts during free association. Consequently, a patient would demonstrate resistance to recalling these thoughts or situations. Another approach is **dream analysis**. In Freudian theory, dreams contain not only *manifest* (or literal) content, but also *latent* (or symbolic) content. For example, someone may have a dream that his/her teeth are falling out—the manifest or actual content of the dream. However, dreaming that one's teeth are falling out could be a reflection of the person's unconscious concern about losing his or her physical attractiveness—the latent or metaphorical content of the dream. It is the therapist's job to help discover the latent content underlying the client's manifest content through dream analysis.

Psychoanalysis is a therapy approach that typically takes years. Over the course of time, the patient reveals a great deal about themselves to the therapist. Freud suggested that during this patient-therapist relationship, the patient comes to develop strong feelings for the therapist—maybe positive feelings, maybe negative feelings. Freud called this **transference**: the patient transfers all the positive or negative emotions associated with the patient's other relationships to the psychoanalyst. For example, Crystal is seeing a psychoanalyst. During the years of therapy, she comes to see her therapist as a father figure. She transfers her feelings about her father onto her therapist, perhaps in an effort to gain the love and attention she did not receive from her own father.

Psychoanalysis was once the only type of psychotherapy available, but presently the number of therapists practicing this approach is decreasing around the world. Psychoanalysis is not appropriate for some types of patients, including those with severe psychopathology (including suicidal thinking or intention) or intellectual disability. Further, psychoanalysis is often expensive because treatment usually lasts many years. Still, some patients and therapists find the prolonged and detailed analysis very rewarding. Perhaps the greatest disadvantage of psychoanalysis is the lack of empirical support for its effectiveness. The limited research that has been conducted on psychoanalytic treatments suggests that they do not reliably lead to better mental health outcomes for most people (e.g., Driessen et al., 2010).

Today, Freud's psychoanalytical perspective has been expanded upon by the developments of subsequent theories and methodologies: the **psychodynamic perspective**. This approach to therapy remains centered on the role of people's internal drives and forces, but treatment is less intensive than Freud's original model.

Psychodynamic Approaches to Treatment

Deriving from psychoanalysis, modern psychodynamic therapies share many common elements with psychoanalysis and other psychodynamic treatments, although each approach has its own theories and may not use all the common methods; psychodynamic therapists are usually not formally trained psychoanalysts. These common elements include

- an emphasis on the central importance of internal and unconscious conflicts and their relation to development;
- the identification of defenses mechanisms as behaviors used to avoid unpleasant consequences of conflict;
- a belief that psychopathology develops especially from early childhood experiences;
- a view that internal representations of experiences are organized around interpersonal relations;
- a conviction that life issues and dynamics will re-emerge in the context of the client-therapist relationship as transference and counter-transference;
- the use of free association as a major method for exploration of internal conflicts and problems;
- a focus on interpretations of transference, defense mechanisms, and current symptoms and the working through of these present problems; and
- a trust in insight as critically important for success in therapy.

Although there were many "spin-offs" or descendants of Freud's original psychoanalytic approach, there are at least three major variations: ego psychology, object relations approaches, and self psychology in addition to a more modern form of treatment called *brief psychodynamic therapy*.

Ego psychology resulted from the primary work of Anna Freud (Freud's youngest child), Heinz Hartmann, and David Rapaport. Ego psychology therapists tend to focus primarily on supporting and analyzing the client's ego functioning and ability to adapt to reality. They are mostly concerned with defense mechanisms; ability to adapt to situations; and reality testing, meaning the ability to respond effectively to real-world stresses and demands.

Object relations therapy was primarily expanded by Melanie Klein, Donald Winnicott, Harry Guntrip, and others. In this approach, an *object* is the internal representation in one's mind of a person that one has a relationship with. Typically, in infants, the mother of the child is the first relationship, and the child's internal representation of his or her mother is the first object. Therapy focuses primarily on seeking to explore and understand the client's relationships with others while the person works to maintain a sense of independence as well. Consistent with Freud's emphasis on early childhood experiences, therapists using this approach also assume that the early self and other representations and relationships often play out as the person grows and can affect adult relationships.

Heinz Kohut created self psychology, which is based on the idea that one's sense of self is grounded in a person's personal experiences and the development or lack of self-esteem as a result. Issues of how one differentiates the self from others while still maintaining relationships and establishing boundaries is also central to this approach. These forms of psychodynamic therapies typically do not last as long as psychoanalysis and sessions are not as frequent, although they still tend to be longer treatments than other forms of psychotherapy, mostly because the goals of treatment are to help the person develop more fully or overcome long repressed issues and conflicts. Therapists also often sit facing the client, and there is more direct interpersonal interactions than in psychoanalysis. There are ongoing debates and discussions about the effectiveness of these forms of therapy, but some evidence suggests that they may be effective for some individuals.

Another form of psychodynamic therapy today is called brief psychodynamic therapy. Like other forms of psychodynamic approaches, it still focuses on many of the core ideas and approaches but is much shorter and more focused; use of free association, for example, is not used very much. Many therapists practicing these more intensive forms of psychodynamic approaches are not trying to help the person's personality mature or develop, but instead believe that an intensive focus on one core problem or issue can begin a process of change that is more lasting and does not necessarily require the consistent interventions by the therapist. Initial assessment is very important in this process, and together the therapist and client identify an important issue the client is facing and agree to work on that issue. Because of this assessment, the therapist is more active and involved in discussions and interpretations of behaviors with the client than is typical in longer forms of psychodynamic treatment in order to help the client stay on track and focus on the issue the client decided on. There is some evidence suggesting that these briefer forms of psychodynamic therapy may be more effective overall than other forms of psychodynamic treatment, and aspects of this approach have even been included in more integrative forms of therapy.

Watch It

This video provides an overview of psychodynamic therapy.



You can view the transcript for "What is Psychodynamic Therapy? Video from Harley Therapy" here (opens in new window).



Figure **3.9.2**: This type of play therapy is known as sandplay or sandtray therapy. Children can set up a three-dimensional world using various figures and objects that correspond to their inner state (Kalff, 1991). (credit: Kristina Walter)

Play Therapy

Play therapy (Figure 2) is often used with children since they are not likely to sit on a couch and recall their dreams or engage in traditional talk therapy. It was originally developed to extend the psychodynamic approach to children, who are not as verbal as adults. It is usually used with children at least three years old into adolescence. This technique uses a therapeutic process of play to "help clients prevent or resolve psychosocial difficulties and achieve optimal growth" (O'Connor, 2000, p. 7). The idea is that children play out their hopes, fantasies, and traumas while using dolls, toys, stuffed animals, and sandbox figurines and are able to find release from their emotions (sadness, anger, anxiety) in a productive way.

Play therapy can also be used to help a therapist make a diagnosis. The therapist observes how the child interacts with toys (e.g., dolls, animals, and home settings) in an effort to understand the roots of the child's disturbed behavior. Play therapy can be nondirective or directive. In nondirective play therapy, children are encouraged to work through their problems by playing freely while the therapist observes (LeBlanc & Ritchie, 2001). In directive play therapy, the therapist provides more structure and guidance in the play session by suggesting topics, asking questions, and even playing with the child (Harter, 1977). Although the origins of play therapy were in psychodynamic thought, therapists from humanistic and cognitive-behavioral approaches have adapted play therapy to align with their core concepts as well.

Glossary

biomedical therapy: treatment that involves medication and/or medical procedures to treat psychological disorders

dream analysis: technique in psychoanalysis in which patients recall their dreams and the psychoanalyst interprets them to reveal unconscious desires or struggles

free association: technique in psychoanalysis in which the patient says whatever comes to mind at the moment

psychoanalysis: therapeutic orientation developed by Sigmund Freud that employs free association, dream analysis, and transference to uncover repressed feelings

psychotherapy: (also, psychodynamic psychotherapy) psychological treatment that employs various methods to help someone overcome personal problems or to attain personal growth

transference: process in psychoanalysis in which the patient transfers all the positive or negative emotions associated with the patient's other relationships to the psychoanalyst

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3.10: Humanistic and Existential Models

Learning Objectives

- Describe the key concepts and applications of the humanistic approach to psychopathology

Humanism and Humanistic Approaches

As is common with many of the psychogenic models and the treatment approaches connected to them, the humanistic approach emerged, in part, as a reaction to both psychoanalysis/psychodynamic approaches and the behavioral approach (to be discussed next). Humanists saw behavioral models as mechanical and overly focused on an objective perspective, lacking an understanding and concern for the depth of human experience, consciousness, and personality development. They also saw psychodynamic and analytic approaches as cold, distant, and overly deterministic regarding human development, especially the concept that negative early childhood experiences generated "fixations" or an inability of a person to progress in his or her life. They also believed these approaches were focused primarily on pathology, what is wrong with someone, rather than acknowledging human creativity, growth, and adaptation. Abraham Maslow, one of the major figures in the humanistic movement said, "It is as if Freud supplied us the sick half of psychology and we must now fill it out with the healthy half."[1]

The humanistic perspective within psychology emphasizes the potential for good that is innate to all humans. Two of the most well-known proponents of humanistic psychology are Abraham Maslow and Carl Rogers (O'Hara, n.d.). Abraham Maslow (1908–1970) was an American psychologist who is best known for proposing a hierarchy of human needs in motivating behavior (Figure 1). Maslow asserted that so long as basic needs necessary for survival were met (e.g., food, water, shelter), higher-level needs (e.g., social needs) would begin to motivate behavior. According to Maslow, the highest-level needs relate to self-actualization, a process by which we achieve our full potential.

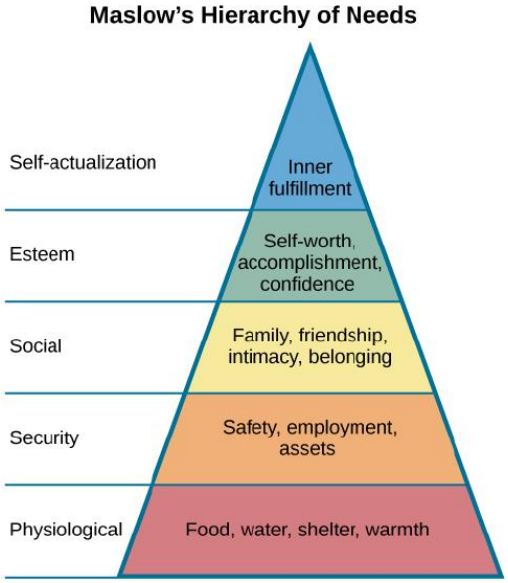


Figure **3.10.1**: Maslow's hierarchy of needs emphasizes that basic needs for food and safety need to be met before higher-level needs can serve as motivators.

Carl Rogers (1902–1987) was also an American psychologist who, like Maslow, emphasized the potential for good that exists within all people. Rogers's focus was to ensure that human developmental processes led to healthier, even more creative, personality functioning. The term *actualizing tendency* was also coined by Rogers, and was a concept that eventually led Abraham Maslow to study self-actualization as one of the needs of humans.

The humanistic psychology perspective is summarized by five core principles or *postulates* of humanistic psychology, first articulated in an article written by James Bugental in 1964 and adapted by Tom Greening, psychologist and long-time editor of the *Journal of Humanistic Psychology*. The five basic principles of humanistic psychology are

1. Human beings, as human, supersede the sum of their parts. They cannot be reduced to components.

2. Human beings have their existence in a uniquely human context, as well as in a cosmic ecology.
3. Human beings are aware and are aware of being aware—i.e., they are conscious. Human consciousness always includes an awareness of oneself in the context of other people.
4. Human beings have the ability to make choices and therefore have responsibility.
5. Human beings are intentional, aim at goals, are aware that they cause future events, and seek meaning, value, and creativity.

Existential Approaches and Treatment

Another contributing element to humanism was **existentialism**, a philosophical approach emphasizing a holistic view of human beings, validating the nature of human emotions, thought, behavior, and choices. Existentialism focuses on the ability, and need, of human beings to define and find their own sense of meaning or purpose in an "absurd" world that does not have any innate meaning to it. In many ways, it echoes the core principles of humanism and the rejection of psychodynamic determinism and the objectivity of behaviorism. Existential approaches differ from humanism in an even stronger emphasis on free will, choice, self-determination, and the search for meaning and purpose.

An important concept in existentialism is angst or anxiety, created mostly by the responsibility that comes with freedom; free will conveys a sense of responsibility for the choices that we make and their future consequences. Angst is also related to the need to accept the inevitability of one's own mortality and future death in order to live a full life. Other important concepts include authenticity, which is a person living the life that they have deliberately chosen to the fullest extent possible, and despair, which differs slightly from the usual use of that term to mean the emotional experience of being frustrated or unable to live the life that a person intended to create. For example, a student who has dreamed for her whole life to become a physician may experience despair if she is not accepted into any medical school. Existentialism is usually tied to the philosophical writings and ideas of Jean-Paul Sartre, although it was shaped by many others, including Søren Kierkegaard, Simone de Beauvoir, Maurice Marleau-Ponty, and Albert Camus. Despite this description of some of the core concepts of existentialism, there was no coherent or single school or discipline of existentialism, but rather many different variations and forms of this philosophical and psychological approach.

This existential philosophical approach has been applied to psychotherapy particularly by Paul Tillich and Irvin Yalom. Tillich claimed that existential psychotherapy focused on dealing with the core issues of living including loneliness, suffering, and the meaningless of life; Yalom believed that universal themes experienced by all people include death, isolation, freedom, and emptiness (related to the concept of despair).[2] Thus, existential therapists do not see anxiety nor despair as a symptom of a disorder, but view it as a normal part of experience when human beings confront these critical aspects of life. With an emphasis on empathy and supporting the client, they may encourage clients to accept the anxiety and use its energy to help them make authentic or genuine choices about their life and to take responsibility for their future experiences rather than just reacting to circumstances. Because human beings exist in relation to others, Yalom, in particular, emphasized the value of group therapy as an effective approach to helping people live a more meaningful and authentic life.[3]

Humanistic or Person-Centered Therapy



Figure 3.10.2: The quality of the relationship between therapist and patient is of great importance in person-centered therapy.

Because humanistic psychology focuses on a holistic view of individuals and believes that humans are inherently good and seek to achieve fulfillment and self-actualization, humanistic psychotherapy is aimed at fostering these goals. In contrast to psychoanalysis, humanistic therapists focus on conscious rather than unconscious thoughts. They also emphasize the patient's present and future, as

opposed to exploring the patient's past. Symptoms are viewed as resulting from inconsistencies between a person's behaviors or actions and their view of themselves and what they want to achieve. Thus, the goal of person-centered therapy (PCT), created by Carl Rogers, is to create conditions under which clients can discover their self-worth, feel comfortable exploring their own identity, and alter their behavior to better reflect this identity.

Unlike a psychoanalytic/psychodynamic approach in which the therapist plays an important role in interpreting what conscious behavior reveals about the unconscious mind, PCT involves the patient taking a lead role in the therapy session. Rogers, in particular, believed that all people have the potential to change and improve, and that the role of therapists is to foster self-understanding in an environment where adaptive change is most likely to occur (Rogers, 1951). Rogers believed that a therapist needed to display three features to maximize the effectiveness of this particular approach: unconditional positive regard, genuineness, and empathy. **Unconditional positive regard** refers to the fact that the therapist accepts their client for who they are, without condition. The therapist never criticizes or expresses negative opinions of the client's actions or remarks, but expresses warmth and empathy. This creates an environment free from approval or disapproval, where patients come to appreciate their value and to behave in ways that are congruent with their own identity. Rogers believed that if these conditions were present, people were more than capable of dealing with and working through their own issues (Thorne & Henley, 2005). In general terms, this type of therapy is often called **nondirective therapy** because the therapist does not give advice or provide interpretations but helps the person to identify conflicts and understand feelings so they can take responsibility for their own direction in life and for their choices. The therapist tends only to ask questions and use active listening to ensure the client feels heard and understood.

One key advantage of person-centered therapy is that it is highly acceptable to patients. In other words, people tend to find the supportive, flexible environment of this approach very rewarding. Because of this, the fundamental aspects of PCT are now widely integrated into most other forms of psychotherapy. For example, most therapists of any orientation find that clients respond well to being treated with nonjudgmental empathy and the use of active listening. These are usually considered the core skills of effective therapy and counseling upon which other specific methods and techniques are built. The main disadvantage to PCT, however, is that findings about its effectiveness are mixed. One possibility for this could be that the treatment is primarily based on *unspecific treatment factors*. That is, rather than using therapeutic techniques that are specific to the patient and the mental problem (i.e., *specific treatment factors*), the therapy focuses on techniques that can be applied to anyone (e.g., establishing a good relationship with the patient) (Cuijpers et al., 2012; Friedli, King, Lloyd, & Horder, 1997). Similar to how "one-size-fits-all" doesn't really fit every person, PCT uses the same practices for everyone, which may work for some people but not others. Further research is necessary to evaluate its utility as a therapeutic approach.

Watch It

Watch this video as Carl Rogers explains his nonjudgmental and accepting approach to person-centered therapy.



You can view the transcript for “Carl Rogers on Person-Centered Therapy Video” here (opens in new window).

Glossary

active listening: a set of skills focused on the effective use of paraphrasing, reflection, and summarizing that aim to accurately capture the client’s thoughts and emotions

existentialism: a philosophical approach emphasizing a holistic view of human beings, validating the nature of human emotions, thought, behavior, and choices

humanistic therapy: therapeutic orientation aimed at helping people become more self-aware and accepting of themselves

mindfulness: a process that tries to cultivate a nonjudgmental, yet attentive, mental state. It is a therapy that focuses on one’s awareness of bodily sensations, thoughts, and the outside environment

nondirective therapy: therapeutic approach in which the therapist does not give advice or provide interpretations, but helps the person identify conflicts and understand feelings

Rogerian (person-centered therapy): non-directive form of humanistic psychotherapy developed by Carl Rogers that emphasizes unconditional positive regard and self-acceptance

unconditional positive regard: fundamental acceptance of a person regardless of what they say or do; term associated with humanistic psychology

1. Maslow, Abraham (1968). *Toward a Psychology of Being*. p. 3. ↵
2. https://www.psychologytoday.com/us/therapy-types/existential-therapy ↵
3. Psychology Today. Existential Therapy. https://www.psychologytoday.com/us/therapy-types/existential-therapy. ↵

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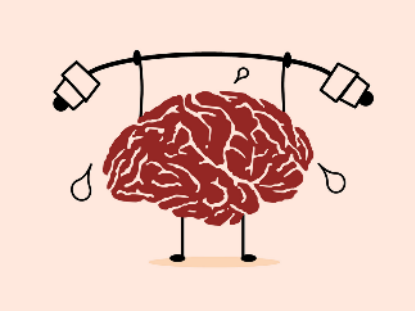
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3.11: Introduction to Behavioral and Cognitive Approaches to Psychopathology

What you'll learn to do: explain the key principles of behaviorism, cognitive psychology, and how cognitive and behavioral methods are used together



In this section, we will look closely at a few other prominent approaches to psychology, including behaviorism with its emphasis on learned behaviors and cognitivism with its emphasis on the power of the mind.

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3.12: The Behavioral Model

Learning Objectives

- Describe the key concepts and applications of the behavioral approach to psychopathology

The Behavioral Perspective

In contrast to the psychodynamic approaches of Freud and the neo-Freudians, which focus on how mental illness connects childhood experiences to inner (unconscious) processes and defense mechanisms, the learning, or behavioral approaches, focus only on observable behavior. The early behaviorists were primarily concerned with the scientific study of human learning and behavior; they rejected Freud's theories and treatments mostly because those relied so heavily on Freud's own theories and "interpretations" of client's behaviors. They emphasize the study of what is observable—actual behaviors by animals and human beings rather than trying to study or evaluate things that could not be seen or tested. John B. Watson wrote an article in which he said, "Psychology as the behaviorist views it is a purely objective experimental branch of natural science. Its theoretical goal is the prediction and control of behavior. Introspection forms no essential part of its methods, nor is the scientific value of its data dependent upon the readiness with which they lend themselves to interpretation."[1]

Behaviorists also do not believe in biological determinism: they do not see personality traits as inborn. Instead, they view personality as significantly shaped by the reinforcements and behavioral consequences that occur in the environment. In other words, people behave in a consistent manner based on prior learning. B. F. Skinner, a strict behaviorist, believed that the environment was solely responsible for all behavior, including the enduring, consistent behavior patterns studied by personality theorists. He proposed that we demonstrate consistent behavior patterns because we have developed certain response tendencies (Skinner, 1953). In other words, we *learn* to behave in particular ways. We increase the behaviors that lead to positive consequences, and we decrease the behaviors that lead to negative consequences.

Skinner disagreed with Freud's idea that personality is fixed in childhood. He argued that personality develops over our entire life, not only in the first few years. Our responses can change as we come across new situations; therefore, we can expect more variability over time in personality than Freud would anticipate. For example, consider a young woman, Greta, a risk-taker. She drives fast and participates in dangerous sports such as hang gliding and kiteboarding. But after she gets married and has children, the system of reinforcements and punishments in her environment changes. Speeding and extreme sports are no longer reinforced, so she no longer engages in those behaviors. In fact, Greta now describes herself as a cautious person.

Over time and through ongoing studies, the field of behaviorism has changed and paved the way toward integrating cognitive elements that also influence learning and shape behavior. Yet, the early principles are still useful and play an important role in treating many types of mental illness. The behavioral model is generally viewed as including three major areas: classical conditioning, operant conditioning, and observational learning/social learning.

Classical Conditioning

Two figures are usually central in reviews of the principles of classical conditioning. The first was Ivan Pavlov (1849–1936), a Nobel-prize winning Russian physiologist, who also studied dogs and discovered the basic principles of **classical conditioning** (Figure **3.12.1**).

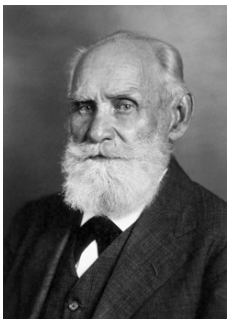


Figure **3.12.1**: Ivan Pavlov's research on the digestive system of dogs unexpectedly led to his discovery of the learning process now known as classical conditioning.

Pavlov's studied the digestive system (Hunt, 2007) in dogs. Over time, Pavlov (1927) noted that the dogs began to salivate not only at the taste of food, but also at the sight of food, at the sight of an empty food bowl, and even at the sound of the laboratory assistants' footsteps. Salivating to food in the mouth is reflexive, so no learning is involved. However, dogs don't naturally salivate at the sight of an empty bowl or the sound of footsteps. To explore this phenomenon, Pavlov designed a series of carefully controlled experiments. He trained the dogs to salivate in response to stimuli that had nothing to do with food, such as the sound of a bell, a light, and a touch on the leg. Through his experiments, Pavlov realized that an organism has two types of responses to its environment: (1) unconditioned (unlearned) responses, or reflexes, and (2) conditioned (learned) responses. *(If you remember that unlearned = unconditioned, and learned = conditioned, it is easier to make sense of the information that follows.)*

In his experiments, the dogs salivated each time meat powder was presented to them. The meat powder in this situation was an **unconditioned stimulus (UCS)**: a stimulus that elicits a reflexive response in an organism. The dogs' salivation was an **unconditioned response (UCR)**: a natural (unlearned) reaction to a given stimulus. Before conditioning, think of the dogs' stimulus and response like this:

Meat powder (UCS) → Salivation (UCR)

In classical conditioning, a **neutral stimulus** is presented immediately before an unconditioned stimulus. Pavlov would sound a tone (like ringing a bell) and then give the dogs the meat powder (Figure 2 below). The tone was the neutral stimulus (NS), which is a stimulus that does not naturally elicit a response. Prior to conditioning, the dogs did not salivate when they just heard the tone because the tone had no association or meaning for the dogs. Quite simply, this pairing means the following:

Tone (NS) + Meat Powder (UCS) → Salivation (UCR)

When Pavlov repeatedly paired the tone with the meat powder, the previously neutral stimulus (the tone) also began to elicit salivation from the dogs. Thus, the neutral stimulus *had changed* and became the **conditioned (learned) stimulus (CS)**, which is a stimulus that elicits a response after repeatedly being paired with an unconditioned stimulus. Eventually, the dogs began to salivate to the tone alone, just as they previously had salivated at the sound of the assistants' footsteps. The behavior caused by the

conditioned stimulus is called the **conditioned response (CR)**. In the case of Pavlov's dogs, they had learned to associate the tone (conditioned stimulus or CS) with being fed, and they began to salivate (conditioned response or CR) in anticipation of food.

Tone (CS) → Salivation (CR)

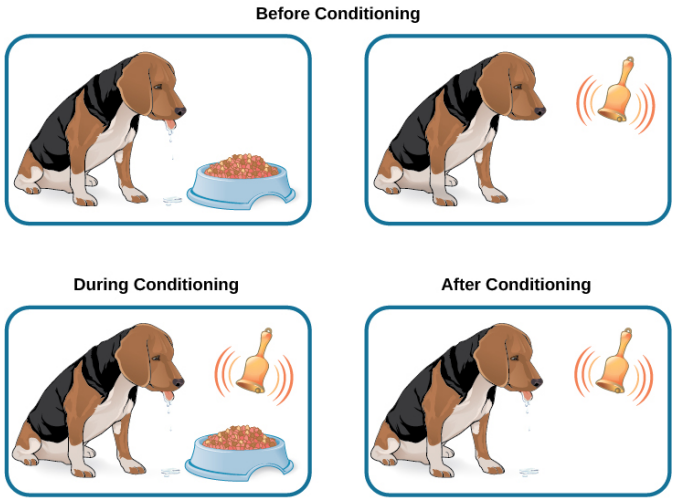


Figure **3.12.2**: Before conditioning, an unconditioned stimulus (food) produces an unconditioned response (salivation), and a neutral stimulus (bell) does not produce a response. During conditioning, the unconditioned stimulus (food) is presented repeatedly just after the presentation of the neutral stimulus (bell). After conditioning, the neutral stimulus alone produces a conditioned response (salivation), thus becoming a conditioned stimulus.

Application to Human Beings

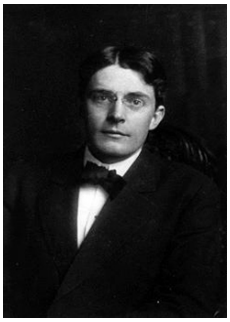


Figure **3.12.2**: John B. Watson used the principles of classical conditioning in the study of human emotion.

The second figure central to classical conditioning was John B. Watson, often regarded as the founder of behaviorism. Watson's ideas were influenced by Pavlov's work. According to Watson, human behavior, just like animal behavior, is primarily the result of conditioned responses. While Pavlov's work with dogs involved the conditioning of reflexes, Watson believed the same principles could be extended to the conditioning of human emotions (Watson, 1919). He first tested this together with his graduate student Rosalie Rayner in a series of studies with a baby called Little Albert. Although this study would be considered unethical today in how it was carried out, Watson and Rayner (1920) demonstrated how fears can be conditioned in humans.

In 1920, Watson was the chair of the psychology department at Johns Hopkins University. There, he came met Little Albert's mother, Arvilla Merritte, who worked at a campus hospital (DeAngelis, 2010). Watson offered her a dollar to allow her son to be the subject of his experiments in classical conditioning. Initially, he was presented with various neutral stimuli, including a rabbit, a dog, a monkey, masks, cotton wool, and a white rat. He was not afraid of any of these things. Then Watson, with the help of Rayner, conditioned Little Albert to associate these stimuli with an emotion: fear.

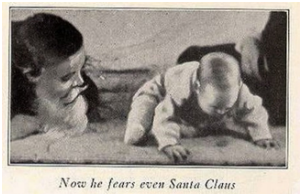


Figure **3.12.3**: Through stimulus generalization, Little Albert came to fear furry things, including Watson in a Santa Claus mask.

For example, Watson handed Little Albert the white rat, and Little Albert enjoyed playing with it (neutral stimulus). Then Watson made a loud sound, by striking a hammer against a metal bar hanging behind Little Albert's head, each time Little Albert touched the rat. Little Albert was frightened by the sound—demonstrating a reflexive fear of sudden loud noises—and began to cry. Watson repeatedly paired the loud sound with the white rat. Soon Little Albert became frightened by the white rat alone. *In this case, what are the UCS, CS, UCR, and CR?(Write out your answers to practice).* Days later, Little Albert demonstrated **stimulus generalization**—he became afraid of other furry things: a rabbit, a furry coat, and even a Santa Claus mask (Figure 3). Watson had succeeded in conditioning a fear response in Little Albert, thus demonstrating that emotions could become conditioned responses. It had been Watson's intention to produce a phobia—a persistent, excessive fear of a specific object or situation—through conditioning alone, thus countering Freud's view that phobias are caused by deep, hidden conflicts in the mind. However, there is no evidence that Little Albert experienced phobias in later years. Little Albert's mother moved away, ending the experiment, and Little Albert himself died a few years later of unrelated causes.

Examples of Classical Conditioning Related to Mental Illness

Classical conditioning can occur in many regular contexts in life from food aversion (getting sick after eating something and then feeling nauseated if you see the same food later on), emotional reactions to people or places, to reactions to chemotherapy (experiencing nausea when seeing a doctor or nurse involved in the treatment even if outside of the hospital or clinic). It also can play a significant role in some forms of mental illnesses. For example, fear conditioning plays a role in creating many anxiety disorders in humans, such as phobias and panic disorders, where people associate cues (such as closed spaces, or a shopping mall) with panic or other emotional trauma (see Mineka & Zinbarg, 2006). Here, rather than a physical response (like dogs drooling), the conditioned stimulus (CS) triggers an emotional reaction. Simple examples may include a child who sees a dog (previously a neutral stimulus) and then gets bitten, increasing the chance of developing a phobia towards dogs (now a CS) or a student, who when younger, gave a presentation in class and was laughed at, contributing to social phobia. Remember that the biopsychosocial model emphasizes multiple interactions in the development of mental illnesses, but these types of learning experiences certainly are relevant and contribute to mental illness.

Classical conditioning can also play a role in drug or alcohol addictions. When a drug is consumed, it can become paired with previously neutral cues that are present at the same time (e.g., rooms, odors, or drug paraphernalia). In this regard, if someone associates a particular smell with the high from the drug, whenever that person smells the same odor later, it may cue behavioral or emotional responses that encourage continued use. But drug cues have an even more interesting property: they can elicit physical or physiological responses that represent the body attempting to compensate for the upcoming effect of the drug (see Siegel, 1989). For example, morphine suppresses pain; however, if someone is used to taking morphine, a cue that signals the "drug is coming soon" can actually make the person more sensitive to pain. Because the person knows a pain suppressant will soon be administered, the body becomes more sensitive, anticipating that "the drug will soon take care of it." Remarkably, such conditioned compensatory responses, in turn, decrease the impact of the drug on the body—because the body has become more sensitive to pain.

This conditioned compensatory response has many implications. For instance, a drug user will be most tolerant to the drug in the presence of cues that have been linked to it (because such cues generate compensatory responses) and so the user increases the dose they are taking to get the same high. As a result, overdose is often not due to an increase in dosage, but to taking the drug in a new place without the familiar cues—which would have otherwise allowed the user to tolerate the drug at the dose being used (see Siegel, Hinson, Krank, & McCully, 1982). Conditioned compensatory responses (which include heightened pain sensitivity and decreased body temperature, among others) might also cause discomfort, thus motivating the drug user to continue usage of the drug to reduce them. This is one of several ways classical conditioning might be a factor in drug addiction and dependence.

Operant Conditioning

The second major area of the behavioral model relevant to mental disorders is operant conditioning. In **operant conditioning**, organisms learn to associate a behavior with its consequence (Table 1). A pleasant consequence makes that behavior more likely to be repeated in the future. For example, Spirit, a dolphin at the National Aquarium in Baltimore, does a flip in the air when her trainer blows a whistle. The consequence is that she gets a fish, making her more likely to flip again when she hears a whistle.

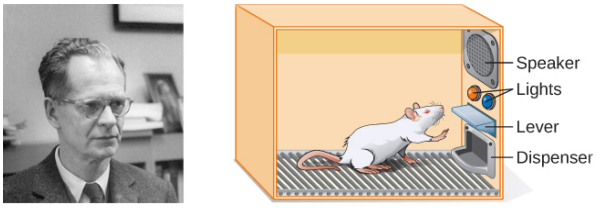
Table 1. Classical and Operant Conditioning Compared

	Classical Conditioning	Operant Conditioning
Conditioning approach	An unconditioned stimulus (such as food) is paired with a neutral stimulus (such as a bell). The neutral stimulus eventually becomes the conditioned stimulus, which brings about the conditioned response (salivation).	The target behavior is followed by reinforcement or punishment to either strengthen or weaken it, so that the learner is more or less likely to exhibit the desired behavior in the future.
Stimulus timing	The stimulus occurs immediately *before* the response.	The stimulus (either reinforcement or punishment) occurs soon *after* the response.

Psychologist B. F. Skinner realized that classical conditioning is limited to existing behaviors that are mostly reflexive; it does not account for new behaviors such as riding a bike. He proposed a theory about how such behaviors are acquired. Skinner believed that behavior is motivated by the *consequences* we receive for the behavior: the reinforcements and punishments. His idea that learning is the result of consequences is based on the **law of effect**, which was first proposed by psychologist Edward Thorndike. According to the law of effect, behaviors engaged in by the organism (instead of an external neutral stimulus) that are followed by consequences that are satisfying to the organism are more likely to be repeated, and behaviors that are followed by unpleasant consequences are less likely to be repeated (Thorndike, 1911). Essentially, if an organism does something that brings about a desired result, the organism is more likely to do it again. If an organism does something that does not bring about a desired result, the organism is less likely to do it again. An example of the law of effect is in employment. One of the reasons (and often the main reason) we show up for work is because we get paid to do so. If we stop getting paid, we will likely stop showing up—even if we love our job.

Working with Thorndike's law of effect as his foundation, Skinner began conducting scientific experiments on animals (mainly rats and pigeons) to determine how organisms learn through operant conditioning (Skinner, 1938). He placed these animals inside an operant conditioning chamber, which has come to be known as a Skinner box (Figure 4). A Skinner box contains a lever (for rats) or disk (for pigeons) that the animal can press or peck for a food reward via the dispenser. Speakers and lights can be associated with certain behaviors. Because the rat has no natural association between pressing a lever and getting food, the rat has to learn this connection. At first, the rat may simply explore its cage, climbing on top of things, burrowing under things, in search of food. Eventually while poking around its cage, the rat accidentally presses the lever, and a food pellet drops in. This voluntary behavior is called an operant behavior because it operates on the environment (i.e., it is an action that the animal itself makes). A consequence that *increases* the frequency of a behavior is called reinforcement or a **reinforcer,** while consequences that *decrease* how often a behavior occurs are referred to as punishment or **punishers**.

Now, once the rat recognizes that it receives a piece of food every time it presses the lever, the behavior of lever-pressing becomes reinforced. That is, the food pellets serve as reinforcers because they strengthen the rat's desire to engage with the environment in this particular manner.



(a) (b)

Figure **3.12.4**: (a) B. F. Skinner developed operant conditioning for the systematic study of how behaviors are strengthened or weakened according to consequences. (b) In a Skinner box, a rat presses a lever in an operant conditioning chamber to receive a food reward. (credit a: modification of work by "Silly rabbit"/Wikimedia Commons)

In an example related to humans, imagine that you are playing a street-racing video game. As you drive through one city course multiple times, you try a number of different streets to get to the finish line. On one of these trials, you discover a shortcut that dramatically improves your overall time, and you feel excited and pleased. You have learned this new path through operant conditioning. That is, by engaging with your environment (operant responses), you performed a sequence of behaviors that that was positively reinforced (i.e., you found the shortest distance to the finish line) and experienced satisfaction and pleasure. And now that you've learned how to drive this course, you will perform that same sequence of driving behaviors (just as the rat presses on the lever) to receive your reward of a faster finish and feeling good.

Another relevant example is striving for a good grade in your class—which could be considered a reward for students (i.e., it produces a positive emotional response). In order to get that reward (similar to the rat learning to press the lever), a student needs to modify their behavior. For example, a student may learn that speaking up in class gets them participation points (a reinforcer), so the student speaks up repeatedly. However, the student also learns that they shouldn't speak up about just anything; talking about topics unrelated to school actually costs points (punishment). Therefore, through the student's freely chosen behaviors, they learn which behaviors are reinforced and which are punished. Keep in mind that when discussing operant conditioning, the terms *reinforcement* and *punishment* are **not** used the same way they are in everyday speech. Also, the use of the terms *positive* and *negative* are also used distinctly, in a mathematical way, with *positive* meaning a reinforcer or punisher being an *added* consequence while *negative* (think of negative numbers in math) is a situation or event that is *subtracted* or removed.

An important distinction of operant conditioning is that it provides a method for studying how consequences influence voluntary behavior. The rat's decision to press the lever is voluntary in the sense that the rat is free to make and repeat that response whenever it wants. Classical conditioning, on the other hand, is just the opposite—depending instead on involuntary behavior (e.g., the dog doesn't choose to drool; it just does). So, whereas the rat must actively participate and perform some kind of behavior to attain its reward, the dog in Pavlov's experiment is a passive participant. One of the lessons of operant conditioning research, then, is that voluntary behavior is strongly influenced by its consequences.

Interactions between Classical and Operant Conditioning

Classical and operant conditioning are usually studied separately. But outside of the laboratory, they frequently occur at the same time. For example, a person who is reinforced for drinking alcohol or eating excessively learns these behaviors in the presence of certain stimuli—a pub, a set of friends, a restaurant, or possibly the couch in front of the TV. These stimuli are also available for association with the reinforcer. In this way, classical and operant conditioning are often intertwined. Generally speaking, any operant behavior that is reinforced or punished usually occurs in the presence of some stimulus or set of stimuli such as the behaviors of others (such as parents), specific environments or situations, stressors, etc. For one thing, the stimulus will come to evoke a system of responses (remember *systems* means interdependent elements) that help the organism prepare for the consequence. For example, a drinker may undergo changes in body temperature when exposed to cues related to drinking alcohol; the eater may salivate and have an increase in insulin secretion in preparing to digest the food. In addition, the stimulus will evoke approach behaviors, moving to continue or complete the behaviors if the outcome is positive, or retreat or avoidance if the outcome is negative/punishing.

Another effect of classical cues is that they motivate ongoing operant behavior (see Balleine, 2005). For example, if a rat has learned via operant conditioning that pressing a lever will give it a drug, in the presence of cues that signal the "drug is coming soon" (like the sound of the lever squeaking), the rat will work harder to press the lever than if those cues weren't present (i.e., there is no squeaking lever sound). Similarly, in the presence of food-associated cues (e.g., smells), a rat (or an overeater) will work harder for food. And finally, even in the presence of punishment cues (like something that signals fear), a rat, a human, or any other organism will work harder to avoid those situations that might lead to trauma. Classical CSs thus have many effects that can contribute to significant patterns of operant behavior.

Extinction: Diminishing Learned Associations

Although the term *extinction* is often associated with dinosaurs or the disappearance of species, it has a specific meaning in behavior theory. After conditioned learning has occurred, the response to the CS can be eliminated if the CS is presented repeatedly without the US. This effect is called **extinction**, and the response is said to become "extinguished." For example, if Pavlov kept ringing the bell but never gave the dog any food afterward, eventually the dog's CR (drooling) would no longer happen when it heard the CS (the bell), because the bell would no longer be a predictor of food. Extinction is important for many reasons. For one thing, it is the basis for many therapies that clinical psychologists use to eliminate maladaptive and unwanted behaviors.

Take the example of a person who has a debilitating fear of spiders: one approach might include systematic exposure to spiders. Whereas, initially the person has a CR (e.g., extreme fear) every time he or she sees the CS (e.g., the spider), after repeatedly being shown pictures of spiders in neutral conditions, pretty soon the CS no longer predicts the CR (i.e., the person doesn't have the fear reaction when seeing spiders, having learned that spiders no longer serve as a cue for that fear). Here, repeated exposure to spiders without an aversive consequence causes extinction.

Psychologists must accept one important fact about extinction, however: it does not necessarily destroy the original learning (see Bouton, 2004). For example, imagine you strongly associate the smell of chalkboards with the agony of middle school detention. Now imagine that, after years of encountering chalkboards, the smell of them no longer recalls the agony of detention (an example of extinction). However, one day, after entering a new building for the first time, you suddenly catch a whiff of a chalkboard and WHAM!, the agony of detention returns. This is called spontaneous recovery: following a lapse in exposure to the CS after extinction has occurred, sometimes re-exposure to the CS (e.g., the smell of chalkboards) can evoke the CR again (e.g., the agony of detention).

Another related phenomenon is the renewal effect: After extinction, if the CS is tested in a new context, such as a different room or location, the CR can also return. In the chalkboard example, the action of entering a new building—where you don't expect to smell chalkboards—suddenly renews the sensations associated with detention. These effects have been interpreted to suggest that extinction inhibits rather than erases the learned behavior, and this inhibition is mainly expressed in the context in which it is learned.

This does not mean that extinction is a bad treatment for behavior disorders. Instead, clinicians can increase its effectiveness by using basic research on learning to help defeat these relapse effects (see Craske et al., 2008). For example, conducting extinction therapies in contexts where patients might be most vulnerable to relapsing (e.g., at work), might be a good strategy for enhancing the therapy's success.

Observational Learning and Social Learning

The third major area of the behavioral model is related to observational learning, which paved the way to the introduction of cognition into behavioral theory, an approach that is now termed social learning theory (Bandura, 1977) because of its emphasis on social interactions and context.[2] **Observational learning** is essentially a cognitive process involving perception and interpretation of behaviors and is important because not all forms of learning are accounted for entirely by classical and operant conditioning. Imagine a child walking up to a group of children playing a game on the playground. The game looks fun, but it is new and unfamiliar. Rather than joining the game immediately, the child opts to sit back and watch the other children play a round or two. Through watching others, the child takes note of the ways in which they behave while

playing the game. This helps the child figure out the rules of the game and even some strategies for doing well at the game. The child may then be able to join in and participate in the game, continuing to learn as he or she does so.

Alfred Bandura's key contribution to learning theory was the idea that much learning is vicarious. Observational learning does not necessarily require reinforcement, but instead hinges on the presence of others referred to as social models. Social models are typically of higher status or authority compared to the observer, examples of which include parents, teachers, and police officers. In the example above, the children who already know how to play the game could be thought of as being authorities—and are therefore social models—even though they are the same age as the observer. By observing how the social models behave, an individual is able to learn how to act in a certain situation. Other examples of observational learning might include a child learning to place her napkin in her lap by watching her parents at the dinner table, or a customer learning where to find the ketchup and mustard after observing other customers at a hot dog stand. Drawing on the behaviorists' ideas about reinforcement, Bandura suggested that whether we *choose* to imitate a model's behavior depends on whether we see the model reinforced or punished, a process he called vicarious reinforcement. Through observational learning, we come to learn what behaviors are acceptable and rewarded in our culture, and we also learn to inhibit deviant or socially unacceptable behaviors by seeing what behaviors are punished.

A related concept that also demonstrates that learning can occur outside of classical or operant conditioning is **latent learning**. In latent learning, an animal or a person observes behavior or gathers information without classical associations or operant rewards or punishers and is capable of demonstrating the behavior later when there is motivation to do so. For example, a young boy may watch his father go out and mow the lawn several times. His parents later buy him a toy lawnmower, and the next time his father goes out to mow, the boy takes his toy mower and follows his father who laughs and smiles at him (reinforcer). The boy had learned the behavioral pattern for mowing the lawn even though he could not use the equipment, was not given formal instruction in what to do, and received no rewards for mowing behavior. However, given an opportunity later with his toy, he successfully imitated the behavior which was now reinforced through play/fun and his father's attention. Some psychologists have suggested this is a significant factor, for instance, in how animals and human beings learn to navigate their physical environment.

Bandura also proposed the concept of **reciprocal determinism**, which is the idea that behavior is controlled or determined by the individual through cognitive processes *and* by the environment through external social stimulus events. We can see the principles of reciprocal determinism at work in observational learning. For example, personal factors determine which behaviors in the environment a person chooses to imitate, and those environmental events in turn are processed cognitively according to other personal factors. One person may experience receiving attention as reinforcing, and that person may be more inclined to imitate behaviors such as boasting when a model has been reinforced for similar behavior. For others, boasting may be viewed negatively, despite the attention that might result—or receiving heightened attention may be perceived as being scrutinized. In either case, the person may be less likely to imitate those behaviors even though the reasons for not doing so would be different. An example of Bandura's reciprocal determinism could occur when a child is acting out in school. The child doesn't like going to school; therefore, they act out in class. This behavior results in teachers and administrators of the school disliking having the child around. When confronted by the situation, the child admits they hate school and other peers don't like him or her. This results in the child acting inappropriately, forcing the administrators who dislike having the child around to create a more restrictive environment for children of this stature. Each behavioral and environmental factor coincides with the child and so forth resulting in a continuous battle on all three levels.

Watch It

Watch this video to review the differences between classical and operant conditioning.



You can view the transcript for "The difference between classical and operant conditioning – Peggy Andover" here (opens in new window).

Glossary

neutral stimulus (NS): a stimulus that does not naturally generate a reaction from the animal or person

unconditioned stimulus (US): a stimulus that naturally generates a reaction from an organism, usually a reflexive reaction

unconditioned response (UCR): a natural and expected reaction to an unlearned stimulus, such as dogs salivating to food or Little Albert crying at a loud noise

conditioned stimulus (CS): a previously neutral stimulus, that through the process of learning in classical conditioning, has now become a learned stimulus that the organism reacts to (a tone, footsteps, or a white rat)

conditioned response (CR): a learned response to the previously neutral stimulus (now a conditioned stimulus) such as crying and trying to avoid the white rat

stimulus generalization: a learned reaction where the organism expands the conditioned stimulus to include related or similar stimuli; the organism has generalized their emotional reaction to multiple stimuli beyond the original CS

law of effect: a principle elaborated by Edward Thorndike stating that behaviors that result in a "satisfying effect" are more likely to occur again in that situation while behaviors that result in a "discomforting effect" are less likely to occur again

operant behavior: a behavior voluntarily emitted by an organism in response to its environment (it operates on the environment)

reinforcement/reinforcer: a consequence of an operant behavior that increases the frequency of its occurrence in the future

punishment/punisher: a consequence of an operant behavior that decreases the frequency of that behavior in the future

extinction: the process by which a previously learned association is weakened to the point that it no longer regularly occurs

spontaneous recovery: following a period of extinction, exposure to the stimulus or environment that was originally associated with the behavior causes the behavior to occur again

observational learning: a process of vicarious learning that occurs through the observation of others and the consequences of their behavior

latentlearning: a form of learning that occurs through observation and experience but is not manifested until later when there is motivation to do so

reciprocal determinism: the principle that a person's behaviors are influenced both by a person's cognitive attitudes and skills as well as by the reactions or consequences of others and the environment

1. Watson, John B. (1913). "Psychology as the Behaviorist Views It". *Psychological Review*. 20 (2): 158–177. doi:10.1037/h0074428 ↵
2. Bandura, A. (1977). Social learning theory. Englewood Cliffs, NJ: Prentice Hall ↵

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3.13: Behavior Therapy

Learning Objectives

- Explain how behavioral principles such as exposure and systematic desensitization and others are used in treating psychological disorders

Behavior Therapy

In psychoanalysis, therapists help their patients look into their past to uncover repressed feelings and resolve internal personality conflicts. In behavior therapy, a therapist employs principles of learning to help clients change undesirable behaviors—rather than digging deeply into one's unconscious. Therapists with this orientation believe that dysfunctional behaviors, like phobias and bedwetting, can be changed by teaching clients new, more constructive behaviors. Behavior therapy employs both classical and operant conditioning techniques to change behavior, but it is important to note that establishing a relationship of trust and empathy with the client (or the parents of the child being treated) is still an important element of successful treatment.

Behavior therapy begins with careful assessment of the person, through a scientifically based process. It is important to determine what the target behavior(s) of treatment will be and to understand how frequently the behavior occurs at the present time. This rate is called the baseline and enables the therapist to evaluate whether the treatment is working by increasing a constructive behavior or decreasing a problematic behavior compared to baseline. It also may involve careful evaluation of the situations in which the behavior occurs in order to understand what cues or stimuli may be associated with the behavior and the context in which the behavior occurs; it may be possible to influence the rate of the behavior by making changes to the environment as well as more direct classical or operant conditioning methods. It is also important to note that for operant conditioning situations, that consequences that may be reinforcing or punishing may be idiosyncratic or unique to the person. While many common consequences are generally perceived as reinforcing (for example money, attention, pleasure) this may not be true for all persons, and behaviors that are often thought of as punishers (such as restriction of privileges or freedom, fines, or other additional costs of the behavior) may not function the same way for all persons, for a variety of reasons.

As an example, a sixteen-year-old girl in a psychiatric inpatient unit suddenly began to increase her frequency of assaults on peers and staff. This behavior resulted in the use of seclusion and sometimes restraints to try and manage her behavior and to provide safety to others. The young woman had a repeated history of sexual abuse that had occurred in her family when she was much younger. This situation was baffling because despite the use of these punishment methods (seclusion, restraints, loss of privileges) aimed at reducing assaultive behavior, her assault rate was increasing. The fact that the target behavior (physical assault on others) was increasing suggested that she was in fact being *reinforced* for that behavior. Careful analysis suggested the possibility that, given her history, she was finding some comfort or possibly pleasure in being touched and handled by young male staff involved in the restraint procedures in addition to the attention given to her by those checking on her to make sure she was physically unharmed. The treatment involved the creation of specific protocols for her in which only female staff intervened and direct attention was minimized (no eye contact, minimal conversation necessary) during these procedures. In addition, a behavior modification program was put in place to encourage prosocial behaviors for which staff would give her more attention, encouragement, and recognition for prosocial behaviors (social reinforcement). This resulted in a successful reduction in assault-related behaviors and gradually improving relationships with staff and peers, although these processes took some time. This example also demonstrates the importance of genuinely understanding these behavior principles rather than relying on common interpretations of behavior or misunderstandings of the meaning and use of reinforcers and punishers. If the target behavior is increasing, it is being reinforced regardless of assumptions people make about the consequences of the behavior.

One type of behavior therapy utilizes classical conditioning techniques. Therapists using these techniques believe that dysfunctional behaviors are conditioned responses. Applying the conditioning principles developed by Ivan Pavlov, these therapists seek to recondition their clients and thus change their behavior. Emmie is eight years old and frequently wets her bed at night. She's been invited to several sleepovers, but she won't go because of her problem. Using a type of conditioning therapy, Emmie begins to sleep on a liquid-sensitive bed pad that is hooked to an alarm. When moisture touches the pad, it sets off the alarm, waking up Emmie. When this process is repeated enough times, Emmie develops an association between urinary relaxation and waking up,

and this stops the bedwetting. Emmie has now gone three weeks without wetting her bed and is looking forward to her first sleepover this coming weekend.

Another commonly used classical conditioning therapeutic technique is **counterconditioning**: a client learns a new response to a stimulus that has previously elicited an undesirable behavior. Two counterconditioning techniques are aversive conditioning and exposure therapy. Aversive conditioning uses an unpleasant stimulus to stop an undesirable behavior. Therapists apply this technique to eliminate addictive behaviors, such as smoking, nail-biting, and drinking. In aversion therapy, clients will typically engage in a specific behavior (such as nail biting) and at the same time are exposed to something unpleasant, such as a mild electric shock a bad taste, or a repulsive odor. After repeated associations between the unpleasant stimulus and the behavior, the client can learn to stop the unwanted behavior.

Aversion therapy has been used effectively for years in the treatment of alcoholism (Davidson, 1974; Elkins, 1991; Streeton & Whelan, 2001). One common way this occurs is through use of a medication called Antabuse. When a person takes Antabuse and then consumes alcohol, they experience uncomfortable side effects including nausea, vomiting, increased heart rate, heart palpitations, severe headache, and shortness of breath. Antabuse is repeatedly paired with alcohol until the client associates alcohol with unpleasant feelings, which decreases the client's desire to consume alcohol. Antabuse creates a conditioned aversion to alcohol because it replaces the original pleasure response with an unpleasant one.

Exposure Therapy

In order to understand the value of exposure therapy, think back to the discussion regarding extinction. Extinction is the gradual disconnection of the relationship between the unconditioned stimuli and the conditioned (learned) stimuli, or in operant conditioning, the disconnection between the operant behavior and a reinforcer (e.g., people would not go to work if they were not being paid). All forms of exposure therapy aim to reduce or eliminate the undesired behavior through these behavioral processes. Exposure therapy is especially useful in treating anxiety disorders and has been widely used in a variety of these disorders.

In exposure therapy, a therapist seeks to treat clients' fears or anxiety by presenting them with the object or situation that causes their anxiety with the idea that due to extinction they will eventually get used to it. This can be done via reality, imagination, or virtual reality. Exposure therapy was first reported in 1924 by Mary Cover Jones, who is considered the mother of behavior therapy. Jones worked with a three-year-old boy named Peter who was afraid of rabbits. Her goal was to replace Peter's fear of rabbits with a conditioned response of relaxation, which is a response that is incompatible with fear. How did she do it? Jones began by placing a caged rabbit on the other side of a room with Peter while he ate his afternoon snack. Over the course of several days, Jones moved the rabbit closer and closer to where Peter was seated with his snack. After two months of being exposed to the rabbit while relaxing with his snack, Peter was able to hold the rabbit and pet it while eating (Jones, 1924; Figure **3.13.1**).

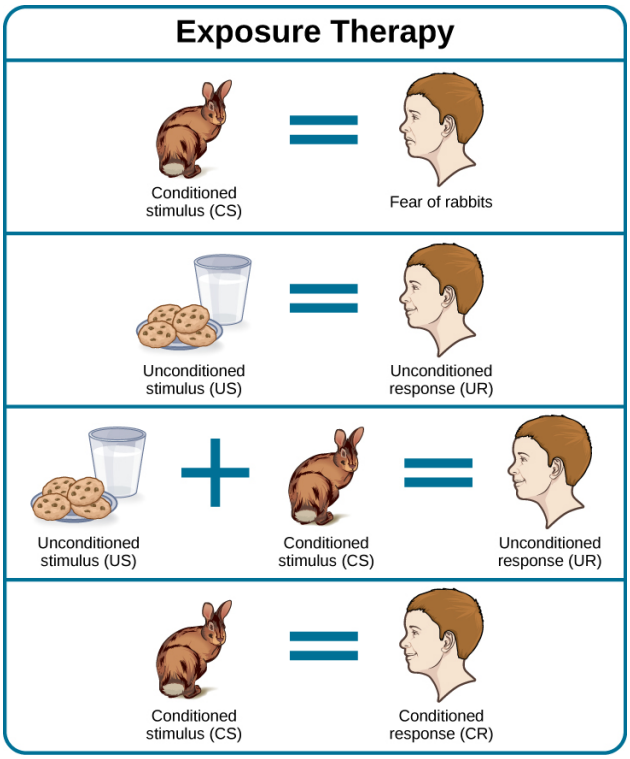


Figure **3.13.1**: Exposure therapy seeks to change the response to a conditioned stimulus (CS). An unconditioned stimulus is presented over and over just after the presentation of the conditioned stimulus. This figure shows conditioning as conducted in Mary Cover Jones' 1924 study.

Thirty years later, Joseph Wolpe (1958) refined Jones's techniques, giving us the behavior therapy technique of exposure therapy that is used today. A popular form of exposure therapy is **systematic desensitization**, wherein a calm and pleasant state is gradually associated with increasing levels of anxiety-inducing stimuli. The idea is that you can't be nervous and relaxed at the same time. Therefore, if you can learn to relax when you are facing environmental stimuli that make you nervous or fearful, you can eventually eliminate your unwanted fear response (Wolpe, 1958; Figure **3.13.2**).



Figure **3.13.2**: This person suffers from arachnophobia (fear of spiders). Through exposure therapy, he is learning how to face his fear in a controlled, therapeutic setting. (credit: "GollyGforce – Living My Worst Nightmare"/Flickr)

How does exposure therapy work? Jayden is terrified of elevators. Nothing bad has ever happened to him on an elevator, but he's so afraid of elevators that he will always take the stairs. That wasn't a problem when Jayden worked on the second floor of an office building, but now he has a new job—on the 29th floor of a skyscraper in downtown Los Angeles. Jayden knows he can't climb 29 flights of stairs in order to get to work each day, so he decided to see a behavior therapist for help. The therapist asks Jayden to first construct a hierarchy of elevator-related situations that elicit fear and anxiety. They range from situations of mild anxiety such as being nervous around the other people in the elevator, to the fear of getting an arm caught in the door, to panic-provoking situations such as getting trapped or the cable snapping. Next, the therapist uses progressive relaxation. She teaches Jayden how to relax each of his muscle groups so that he achieves a drowsy, relaxed, and comfortable state of mind. Once he's in this state, she asks Jayden to imagine a mildly anxiety-provoking situation. Jayden is standing in front of the elevator thinking about pressing the call button.

If this scenario causes Jayden anxiety, he lifts his finger. The therapist would then tell Jayden to forget the scene and return to his relaxed state. She repeats this scenario over and over until Jayden can imagine himself pressing the call button without anxiety. Over time, the therapist and Jayden use progressive relaxation and imagination to proceed through all the situations on Jayden's hierarchy until he becomes desensitized to each one. After this, Jayden and the therapist begin to practice what he only previously envisioned in therapy, gradually going from pressing the button to actually riding an elevator. The goal is that Jayden will soon be able to take the elevator all the way up to the 29th floor of his office without feeling any anxiety.

Sometimes, it's too impractical, expensive, or embarrassing to re-create anxiety-producing situations, so a therapist might employ virtual reality exposure therapy by using a simulation to help conquer fears. Virtual reality exposure therapy has been used effectively to treat numerous anxiety disorders such as the fear of public speaking, claustrophobia (fear of enclosed spaces), aviophobia (fear of flying), and post-traumatic stress disorder (PTSD; a trauma and stressor-related disorder) (Gerardi, Cukor, Difede, Rizzo, & Rothbaum, 2010).

Link to Learning

Virtual reality exposure therapy is being used to treat PTSD in soldiers. Virtual Iraq is a simulation that mimics Middle Eastern cities and desert roads with situations similar to those soldiers experienced while deployed in Iraq. This method of virtual reality exposure therapy has been effective in treating PTSD for combat veterans. Approximately 80% of participants who completed treatment saw clinically significant reduction in their symptoms of PTSD, anxiety, and depression (Rizzo et al., 2010). Watch this Virtual Iraq video that shows soldiers being treated via simulation to learn more.

Operant Conditioning Therapies

Some behavior therapies employ operant conditioning. As noted above, if a behavior is no longer reinforced, it will become extinguished. These principles, defined by Skinner as operant conditioning, can be applied to help people with a wide range of psychological problems. For instance, operant conditioning techniques designed to reinforce desirable behaviors and punish unwanted behaviors are effective behavior modification tools to help children with autism (Lovaas, 1987, 2003; Sallows & Graupner, 2005; Wolf & Risley, 1967). This technique is called **applied behavior analysis** (ABA). In this treatment, a child's behavior is charted and analyzed. The applied behavior analysis (ABA) therapist, along with the caregivers, determines what reinforces the child, what encourages a behavior to continue, and how best to manage a behavior. For example, Nur may become overwhelmed and run out of the room when the classroom is too noisy. Whenever Nur runs out of the classroom, the teacher's aide chases him and places him in a special room where he can relax. Going into the special room and getting the aide's attention are reinforcing for Nur. In order to change Nur's behavior, he must be presented with other options before he becomes overwhelmed, and he cannot receive reinforcement for displaying maladaptive behaviors. Instead, more productive and effective coping strategies need to be identified and then implemented along with specific teaching of the behaviors and reinforcement, including gradual shaping, of the new behaviors which will replace the maladaptive behaviors.

Operant conditioning also plays a significant role in the formation and treatment of anxiety disorders. When a person develops an anxiety disorder, one of the key features is avoidance; the person engages in behaviors in an attempt to avoid confronting the situation or stimulus that creates the anxiety or fear, such as a person afraid of snakes avoiding the houses of friends with pet snakes, movies with snakes, or places where snakes may be found. This leads to a sense of relief or of escaping a danger, and this emotional response is a very powerful form of **negative reinforcement.** Recall that reinforcers *increase* a behavior, so when a person engages in avoidance and feels relieved, they are *more* likely to do so again. The public frequently confuses negative reinforcement with punishment (probably because the common association of the word *negative* means something one doesn't like), but remember that negative reinforcement and punishment are *opposites*. This is precisely why exposure is important. If the anxiety or fear is to be reduced, the avoidant behavior must be reduced through extinction, which comes through exposure.

Aggressive behavior also frequently involves interdependent aspects of positive and negative reinforcement. For example, the bully at school threatens a target peer; the fear reaction by the peer or giving in to the bully's demands are positively reinforcing to the bully. However, the victim's reaction and watching the bully leave without causing them harm also generates feelings of relief or escape and so may negatively reinforce the victim's behavior making it more likely they may give in again to the bully's demands in the future. This is seen in abusive family environments, and unfortunately sometimes in residential or inpatient treatment settings as well as on the playground. Making progress in these areas may also involve punishment for the bully as well as attempts to

eliminate positive reinforcers, but also needs to address the needs of the victim and teaching and reinforcing behaviors that increase or restore their sense of competence and self-esteem.

Operant conditioning methods and procedures are also involved in many other forms of mental disorders ranging from eating disorders, mood disorders like depression, and sexual dysfunctions as well as other disorders.

Another popular operant conditioning intervention is called the token economy. This involves a controlled setting such as an inpatient unit or residential treatment setting where individuals are reinforced for desirable behaviors with tokens, such as a poker chip or points, that can be exchanged for items or privileges. Token economies are often used in psychiatric hospitals to increase patient cooperation and activity levels. Patients are rewarded with tokens when they engage in positive behaviors (e.g., making their beds, brushing their teeth, coming to the cafeteria on time, and socializing with other patients). They can later exchange the tokens for extra TV time, private rooms, visits to the canteen, and so on (Dickerson, Tenhula, & Green-Paden, 2005).

Watch It

This video provides a nice overview of types of behavior therapy.



You can view the transcript for “Behavior Therapy | Psychology” here (opens in new window).

Glossary

counterconditioning: a behavioral treatment method where the client learns a new response to a stimulus that previously resulted in an undesired behavior

exposure therapy: a behavioral treatment method that aims to replace a fearful CS with an incompatible response to create a new conditioned response where the person no longer fears the CS or in operant conditioning, to eliminate negative reinforcement for avoidance behavior

applied behavioral analysis (ABA): a complex form of behavioral therapy involving careful assessment and planning to replace maladaptive behaviors with more prosocial and proactive ones, often by reinforcing behaviors that are incompatible with the previous maladaptive behavior

negative reinforcement: a consequence of an operant behavior that subtracts or reduces the presence of a negative or fearful stimulus and increases avoidant behavior; it is usually associated with the emotions of escape or avoidance

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3.14: The Cognitive Model

Learning Objectives

- Describe the key concepts and applications of the cognitive approach to psychopathology, including rational-emotive-behavioral therapy and cognitive-behavioral therapy

From Behaviorism to the Cognitive Model

Behaviorism's emphasis on objectivity and focus on external behavior had pulled psychologists' attention away from the mind for a prolonged period of time. The early work of the humanistic psychologists redirected attention to the individual human as a whole, and as a conscious and self-aware being. By the 1950s, new disciplinary perspectives in linguistics, neuroscience, and computer science were emerging, and these areas revived interest in the mind as a focus of scientific inquiry. This particular perspective has come to be known as the cognitive revolution (Miller, 2003). By 1967, Ulric Neisser published the first textbook entitled *Cognitive Psychology*, which served as a core text in cognitive psychology courses around the country (Thorne & Henley, 2005).

In the area of clinical work, the impact of the cognitive revolution also became apparent. One of the first therapists to address cognition in psychotherapy was Alfred Adler with his notion of basic mistakes and how they contributed to the creation of unhealthy or useless behavioral and life goals. Adler's work influenced the work of Albert Ellis, who developed the earliest cognitive-based psychotherapy, known today as rational emotive behavior therapy, or REBT. Ellis also credits Abraham Low as a founder of cognitive-behavioral therapy.

Albert Ellis and Rational Emotive Behavior Therapy

Alber Ellis (1957) is known for developing what he called **rational-emotive-behavioral therapy** (REBT; originally called RET—rational emotive therapy). Ellis (1957) used the term *irrational thinking* or irrational thoughts to refer to dysfunctional thoughts that tended to produce significant negative emotions and consequently, maladaptive behaviors. Ellis's approach also had elements of humanism interwoven in the theory, including the concept that all human beings have innate worth and value (so there is no need to worry what others think of us), and that when we allow ourselves to become emotionally upset, we are being unethical to ourselves. Ellis also strongly believed in free will and that engaging in irrational thinking is a type of choice that we make and therefore, can refuse to make as is evident in the title of one of his books written for the public: *How to Stubbornly Refuse to Make Yourself Miserable About Anything—Yes, Anything!* (2019)[1]

The rational-emotive-behavioral therapy (REBT) framework posits that humans have both innate *rational* (meaning self-helping, socially helping, and constructive) and *irrational* (meaning self-defeating, socially defeating, and unhelpful) tendencies and leanings. Rational-emotive-behavioral therapy (REBT) claims that people to a large degree consciously and unconsciously construct emotional difficulties—such as self-blame, self-pity, clinical anger, hurt, guilt, shame, depression and anxiety—and behaviors and behavior tendencies—like procrastination, compulsiveness, avoidance, addiction, and withdrawal—by the means of their irrational and self-defeating thinking, emotional expressions, and behaving. By attaining and ingraining a more rational and self-constructive philosophy of themselves, others, and the world, people often are more likely to behave and experience emotions in more life-serving and adaptive ways. One way that Ellis originally explained this process was using what is known as the A-B-C model.

Imagine that you go to visit a friend, and she opens the door crying. You ask what happened, and she replies that her boyfriend was just cruel to her on the phone. An REBT therapist would help the client analyze the situation this way:

- A = the Activating Event, the situation or stressor that triggered the emotion
- B = the Beliefs or negative thought patterns (irrational thinking)
- C = the emotional Consequence of the Belief

Most people (not REBT therapists) would explain your friend's emotional reaction, and possibly negative behaviors like screaming at him or throwing her phone across the room, by saying that her boyfriend (A) made her sad and thus angry (C). This explanation omits the crucial role of the person's beliefs (B); it assumes that her boyfriend's behaviors are the cause of her emotions and behaviors. However, this explanation robs her of control over her own life and gives her few options for a healthier response.

REBT emphasizes that although the activating event had some influence on her (and one behavioral solution might be to see if such events can be reduced), her beliefs about the situation (B), such as “he hates me” or “no one will ever love me” are the direct causes of her emotional distress and intensify it, creating maladaptive behaviors as well (C).

REBT describes irrational beliefs as having some general characteristics such as being absolute and judgmental—they are not flexible or adaptive and do not deal with the reality of life, which is complex and filled with nuance. REBT would encourage the client to identify patterns in her thinking and then learn to dispute and replace these absolute judgments with beliefs that are more adapted to reality while also being self-accepting such as, “He said mean things, and it really hurt me” or “I don’t like feeling this way; I need to decide if this relationship is healthy.” These more rational beliefs accept the reality of emotional pain from what the boyfriend said, but they do not exaggerate it nor intensify it; they allow her to acknowledge her own emotions, and she retains choice or agency and can evaluate her best options and responses.

Watch It

Watch this video for a description of Albert Ellis’s ABC model of how events or situational triggers (A) can set off dysfunctional thinking (B) with consequences to emotions and behaviors (C).



You can view the transcript for “ABC model of Cognitive Behavioral Therapy” here (opens in new window).

Aaron Beck and Cognitive-Behavioral Therapy

Around the same time that rational emotive therapy, as it was known then, was being developed, Aaron T. Beck was conducting free association sessions in his psychoanalytic practice. During these sessions, Beck noticed that thoughts were not as unconscious as Freud had previously theorized, and that certain types of thinking may be the culprits of emotional distress.

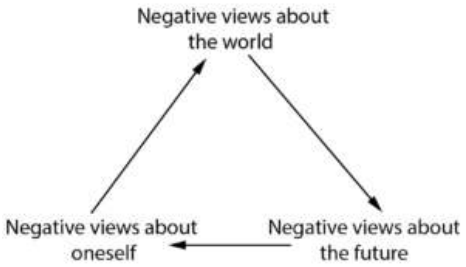


Figure **3.14.1**: Beck’s cognitive triad.

Beck, who began applying analysis of cognitive thought patterns with patients suffering from depression, used the term “automatic thoughts” to refer to the thoughts these patients reported experiencing spontaneously. Beck observed that these thoughts arise from three core belief systems: beliefs about the self, beliefs about the world, and beliefs about the future (Figure 1). Examples of this negative thinking include the following:

- the self—”I’m worthless and ugly,” “I wish I was different,” or “I can’t do anything right”
- the world—”No one values me,” “people ignore me all the time,” or “life is so unfair”

- the future—"Things will never change," "things can only get worse!" or "I'll never feel good again"

Subsequent research has shown that some of these thought patterns, particularly those related to feelings of hopelessness, and especially when coupled with situations or instances involving shame or humiliation, have strong connections to suicidal thinking and behavior. Likewise, other studies have shown connections in college students between pessimistic explanations of one's life and behavior and risk for suicidal behavior in subsequent semesters.

Beck also noted that people with depression will tend to quickly overlook their positive attributes and disqualify their accomplishments as being minor or meaningless. They may also misinterpret the care, goodwill, and concern of others as being based on pity or susceptible to being lost easily if those others knew the "real person" and this misinterpretation fuels further feelings of guilt. The main cognitive distortions according to Beck are summarized below:

- **arbitrary inference**—drawing conclusions from insufficient or no evidence
- **selective abstraction**—drawing conclusions on the basis of just one of many elements of a situation
- **overgeneralization**—making sweeping conclusions based on a single event
- **magnification**—exaggerating the importance of an undesirable event
- **minimization**—underplaying the significance of a positive event
- **personalization**—attributing negative feelings of others to oneself

Both Beck's cognitive therapy and Ellis's REBT share a common foundation that is a core part of all forms of **cognitive-behavioral therapy** (CBT)(Figure 2).

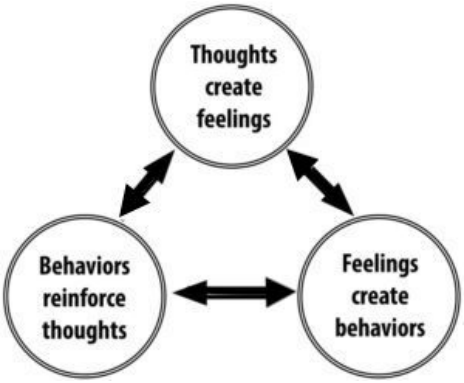


Figure **3.14.2**: Patterns of thoughts, feelings, and behaviors addressed through cognitive-behavioral therapy.

In CBT, there are interdependent and systemic interactions between thoughts, emotions, or feelings and our behaviors. All forms of CBT aim to target dysfunctional thought patterns and also frequently utilize behavioral principles to influence and create more healthy and adaptive behaviors that can then reduce the likelihood of future negative thinking.

Cognitive and Behavioral Integration—The Third Wave

Wilson (2008)[2] described three "waves" of CBT over time as this general form of treatment has evolved. The first wave consisted mostly of the application of behavioral learning principles to psychotherapy, gradually moving towards Bandura's development of social learning theory, and subsequent integration of aspects of cognition into behavioral explanations and interventions. In the second wave, the original versions of both Ellis' REBT and Beck's Cognitive Therapy (CT) were primarily focused just on cognitive aspects of emotional distress and dysfunction with Beck's original work being focused on depression. The underlying foundation of the systemic interactions between thoughts, feelings, and behaviors naturally led to considerations of how cognitive interventions related to and connected with behavioral principles of learning.

In initial studies, cognitive therapy was often contrasted with behavioral treatments to see which treatment was most effective. However, during the 1980s and 1990s, cognitive and behavioral techniques were merged into cognitive behavioral therapy. Pivotal to this merging was the successful development of treatments for panic disorder by David M. Clark in the UK and David H. Barlow in the U.S. Over time, cognitive behavior therapy (CBT) came to be known not only as a therapy, but as an umbrella term for all cognitive-based psychotherapies. These therapies include, but are not limited to, rational emotive behavior therapy (REBT), cognitive therapy, acceptance and commitment therapy, reality therapy/choice theory, cognitive processing therapy, eye movement desensitization and reprocessing (EMDR), and multimodal therapy. All these therapies are a blending of cognitive- and behavior-

based elements. This blending of theoretical and technical foundations from both behavior and cognitive therapies constituted the "third wave" of CBT. In addition, the so-called third wave also includes more integrated forms of therapy that blend CBT methods with other forms of therapeutic interventions. For example, dialectical behavior therapy (DBT), usually used in the treatment of borderline personality disorder, combines CBT principles and practices with techniques for stress tolerance, acceptance, and mindful awareness, all of which are aspects of ancient meditative religious practices, although DBT does not advocate nor describe any religious elements.

In the previous section on behavior therapy, there was an example of systematic desensitization as a form of exposure therapy for phobias. In CBT, the principles of desensitization would be used, but the therapist would also likely spend time with the client before exposure begins to identify irrational thought patterns that contribute to the anxiety such as catastrophizing (similar to Beck's magnification distortion). Catastrophizing thoughts assume that if an event occurs, it will have disastrous, often life-threatening results. A client with a snake phobia, for example, might express the belief that if they see a live snake, they will have a heart attack or that the snake will attack them and seriously injure them. As the exposure process continued, the therapist would seek opportunities for the client to recognize and accept that these beliefs were false or inaccurate (they didn't have a heart attack when the snake was brought into the room; the snake did not viciously attack them but mostly avoided them). This approach combines the learning process that occurs with exposure (extinction) with the disputing and replacement of irrational thoughts with more reality-based adaptive thought patterns. This integration of cognitive and behavioral elements can frequently accelerate change and result in better therapy outcomes than either approach alone.

Watch It

Watch this video for an overview of cognitive-behavioral therapy.



You can view the transcript for "Cognitive Behavioral Therapy (CBT) Simply Explained" here (opens in new window).

Glossary

cognitive-behavioral therapy: type of therapy that focuses on challenging and changing unhelpful cognitive distortions and behaviors, improving emotional regulation, and developing personal coping strategies to target problems; CBT is based on the belief that thought distortions (about your self, others, and the future) and maladaptive behaviors play a role in the development and maintenance of psychological disorders, and that symptoms and associated distress can be reduced by teaching new information-processing skills and coping mechanisms

rational-emotive-behavioral therapy: psychotherapy that posits that people have erroneous beliefs about situations they are involved in, and that these beliefs cause disturbance, but can be disputed with and changed

1. Ellis, A.(2019). How to Stubbornly Refuse to Make Yourself Miserable About Anything - Yes, Anything! (Revised Ed). New York, NY: Citadel Press. ↵

2. Wilson GT (2008). "Behavior therapy." In Corsini RJ, Wedding D (eds.). Current psychotherapies (8th ed.). Belmont, CA: Thomson Brooks/Cole. ↵

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3.15: Cognitive-Behavioral Therapy

Learning Objectives

- Explain how cognitive-behavioral therapy is used to treat mental disorders

Psychotherapy: Cognitive and Cognitive-Behavioral Therapy (CBT)

Cognitive therapy is a form of psychotherapy that focuses on how a person's thoughts lead to feelings of distress. The idea behind cognitive therapy is that how you think determines how you feel and act. Cognitive therapists help their clients change dysfunctional thoughts in order to relieve distress. They help a client see how they misinterpret a situation (cognitive distortion). For example, a client may overgeneralize—because Ray failed one test in his general psychology course, he feels he is stupid and worthless. These thoughts then cause his mood to worsen and may lead to procrastination or distancing himself from the class. Therapists also help clients recognize when they blow things out of proportion. Because Ray failed his psychology test, he has concluded that he's going to fail the entire course and probably flunk out of college altogether. These errors in thinking have contributed to Ray's feelings of distress. His therapist will help him challenge these irrational beliefs, focus on their illogical basis, and correct them with more logical and rational thoughts and beliefs.

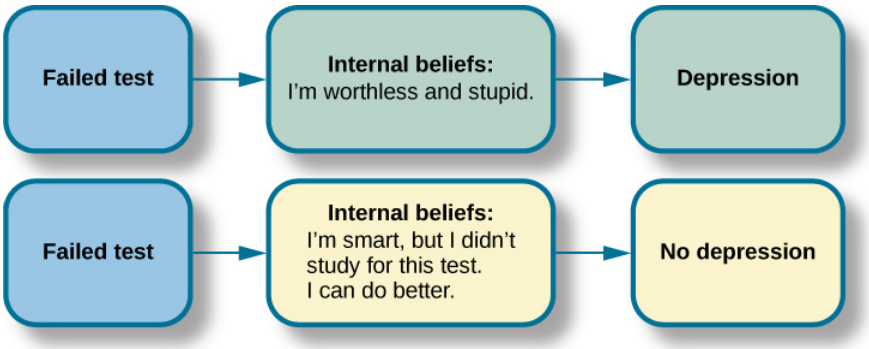


Figure **3.15.1**: Your emotional reactions are the result of your thoughts about the situation rather than the situation itself. For instance, if you consistently interpret events and emotions around the themes of loss and defeat, then you are likely to be depressed. Through therapy, you can learn more logical and adaptive ways to interpret situations.

Link to Learning

View a brief video in which Judith Beck, psychologist and daughter of Aaron Beck, talks about cognitive therapy and conducts a session with a client.



Figure **3.15.2**: In CBT, therapists seek to help clients adopt more adaptive thought patterns and behaviors.

Cognitive-behavioral therapy (CBT) is a very large group of psychotherapeutic approaches that help clients examine how their thoughts affect their behavior. It aims to change cognitive distortions and self-defeating behaviors. For example, if it's your first time meeting new people, you may have the automatic thought, "These people won't like me because I have nothing interesting to share." That thought itself is not what's troublesome; the appraisal (or evaluation) that it might have merit is what's troublesome. The goal of cognitive-behavioral therapy (CBT) is to help people make adaptive, instead of maladaptive, appraisals (e.g., "I do know some interesting things!"). This technique of reappraisal, or cognitive restructuring, is fundamental to cognitive-behavioral therapy (CBT). With cognitive restructuring, it is the therapist's job to help point out when a person has an inaccurate or a

maladaptive thought, so that the patient can either eliminate it or modify it to be more adaptive. In essence, this approach is designed to change the way people think and feel as well as how they act.

Contrary to psychoanalytic and psychodynamic approaches, cognitive therapy and CBT are present-focused; they are aimed at understanding the situations the client is in, their current thoughts and beliefs, and working with them to practice and replace dysfunctional thinking with more healthy thought patterns and behaviors. CBT therapists do have an interest in their client's previous history, mostly as a way to better understand the events that may have led to current patterns of thinking and for understanding how long the client has been experiencing these negative emotions and symptoms. CBT also tends to be shorter in duration than older forms of therapy, often achieving significant benefits to clients in 12–16 sessions or less (depending on the nature of the disorder and severity of symptoms) which makes it equally or more cost-effective than other forms of treatment, including use of long-term medications.

Another distinctive trait of almost all forms of CBT is that sessions are very interactive between the client and the therapist, and treatment involves between-session homework assignments. For example, the therapist may give a patient who compulsively washes her hands for hours a day a worksheet to take home; on this worksheet, the woman is to write down every time she feels the urge to wash her hands, what thoughts she is experiencing at the time, how she deals with the urge, and what behavior she uses to replace that urge. When the patient has her next therapy session, she and the therapist review her homework together as a starting point and an opportunity to help her practice and rehearse new thinking and evaluate her success with behavior change. Given the supportive and interactive nature of CBT, the alliance and relationship between therapist and client is very important.

There are different protocols for delivering cognitive behavioral therapy, with important similarities among them. Use of the term *CBT* may refer to different interventions, including "self-instructions (e.g. distraction, imagery, motivational self-talk), relaxation and/or biofeedback methods, development of adaptive coping strategies (e.g. minimizing negative or self-defeating thoughts), changing maladaptive beliefs about pain, and goal setting"[1]. Treatment is sometimes "manualized" (as in a manual/book), with brief, direct, and time-limited treatments for specific psychological disorders that are technique-driven. CBT is used in both individual and group settings, and the techniques are often adapted for self-help applications. Some clinicians and researchers are more cognitively oriented (e.g., cognitive restructuring), while others are more behaviorally oriented (e.g., live exposure therapy). Interventions such as imaginal exposure therapy combine both approaches.

Persons with medical conditions may also benefit from CBT; evidence supports its use with chronic back pain, fibromyalgia, post spinal cord injuries, insomnia, and pain management in a variety of conditions including breast cancer.

CBT may also be delivered in conjunction with a variety of diverse but related techniques including exposure therapy, stress inoculation, cognitive processing therapy, cognitive therapy, relaxation training, dialectical behavior therapy, and acceptance and commitment therapy. It is therefore a fairly adaptive form of treatment that maintains core principles but is useful and applicable to many specific situations.

One benefit of CBT is that it is an intuitive treatment that makes logical sense to patients. CBT can also be adapted to suit the needs of many different populations. One disadvantage, however, is that CBT does involve significant effort on the patient's part because the patient is an active participant in treatment both in sessions and through between-session homework. The greatest strength of CBT is the abundance of empirical support for its effectiveness. Studies have consistently found CBT to be equally or more effective than other forms of treatment, including medication and other therapies (Butler, Chapman, Forman, & Beck, 2006; Hofmann et al., 2012). For this reason, CBT is considered a first-line treatment for many mental disorders. In depression, for example, CBT has been shown to have the same or better effectiveness than antidepressant medications, but with significantly lower rates of relapse after treatment is completed. The use of CBT in anxiety disorders rather than use of benzodiazepine medications may result in significant benefit without the risk of dependence on the medications and/or subsequent withdrawal and relapse effects.

One client said the following about his cognitive-behavioral therapy:

I have had many painful episodes of depression in my life, and this has had a negative effect on my career and has put considerable strain on my friends and family. The treatments I have received, such as taking antidepressants and psychodynamic counseling, have helped [me] to cope with the symptoms and to get some insights into the roots of my problems. CBT has been by far the most useful approach I have found in tackling these mood problems. It has raised my awareness of how my thoughts impact on my moods. How the way I think about myself, about others and about the world can lead me into depression. It is a practical approach,

which does not dwell so much on childhood experiences, whilst acknowledging that it was then that these patterns were learned. It looks at what is happening now, and gives tools to manage these moods on a daily basis. (Martin, 2007, n.p.)

In total, hundreds of studies have shown the effectiveness of cognitive-behavioral therapy in the treatment of numerous psychological disorders such as depression, PTSD, anxiety disorders, eating disorders, bipolar disorder, and substance abuse (Beck Institute for Cognitive Behavior Therapy, n.d.). For example, CBT has been found to be effective in decreasing levels of hopelessness and suicidal thoughts in previously suicidal teenagers (Alavi, Sharifi, Ghanizadeh, & Dehbozorgi, 2013). Cognitive-behavioral therapy has also been effective in reducing PTSD in specific populations, such as transit workers (Lowinger & Rombom, 2012).

Watch It

Watch this video clip for an overview of CBT.



You can view the transcript for "What is CBT? | Making Sense of Cognitive Behavioural Therapy" here (opens in new window).

Glossary

cognitive-behavioral therapy: form of psychotherapy that aims to change cognitive distortions and self-defeating behaviors

cognitive therapy: form of psychotherapy that focuses on how a person's thoughts lead to feelings of distress, with the aim of helping them change these irrational thoughts

rational emotive therapy (RET): form of cognitive-behavioral therapy

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3.16: Integrative and Emerging Therapies

Learning Objectives

- Describe major conclusions about the common factors that produce effective results with psychotherapy and describe several forms of integrative or eclectic therapies
- Give examples of several relatively new or emerging forms of psychotherapy

Evaluating Various Forms of Psychotherapy and Common Factors

Given that there are so many different types of therapies available today, how does a client decide what type of therapist to approach? One consideration is that some forms of therapy were created by a particular psychologist or psychiatrist and thus may be more restricted by where the psychologist (and others he or she trains) live such as their country or region within a country. For instance, **interpersonal psychotherapy** (IPT), which focuses on coping with or improving relationships, dealing with grief, life transitions such as retirement or divorce, and resolving conflict with others, has been found to be effective in treating depression and other mood disorders. However, most practitioners of interpersonal psychotherapy (IPT) are on the east coast of the United States since this form of therapy began there and many who have been trained in it live in that part of the country. This limited access may change over time as more clinicians are trained and with increased use of online training programs. Another consideration is the effectiveness of the therapy for the mental disorder the client struggles with. Is one technique more effective than another? According to the American Psychological Association, three factors work together to produce successful treatment. The first is the use of evidence-based treatment that is deemed appropriate for the client's particular issues. The second important factor is the clinical expertise of the psychologist or therapist in treating the relevant disorder(s) and how well he or she is trained in the form of therapy. The third factor is the client's characteristics, values, preferences, and culture and if the client feels comfortable and understood. Many people begin psychotherapy feeling like their problem will never be resolved; however, psychotherapy helps people see that there are steps they can take to make their situation better.



Figure **3.16.1**:Therapy comes in many different forms and settings, but one critical factor in its success is the relationship between the therapist and client.

Many studies have explored the effectiveness of psychotherapy. For example, one large-scale study that examined 16 meta-analyses (large-scale summary studies of multiple single study results) of CBT reported that it was equally effective or more effective than other therapies in treating PTSD, generalized anxiety disorder, depression, and social phobia (Butlera, Chapmanb, Formanc, & Becka, 2006). Another study found that CBT was as effective at treating depression (43% success rate) as prescription medication (50% success rate) compared to the placebo rate of 25% (DeRubeis et al., 2005). Another meta-analysis found that psychodynamic therapy was also as effective at treating these types of psychological issues as CBT (Shedler, 2010). However, no studies have consistently found one psychotherapeutic approach more effective than another (Abbass, Kisely, & Kroenke, 2006; Chorpita et al., 2011) across all conditions, nor have they shown any relationship between a client's treatment outcome and the level of the clinician's training or experience (Wampold, 2007).

Advocates of this position emphasize a set of common therapeutic factors that produce beneficial outcomes regardless of the model of therapy used. For example, one critical factor that determines the success of treatment is a person's trusting relationship with the psychologist or therapist (Figure 1). Other key common factors include the following[1]:

- client factors (making connections with already occurring change patterns in the client's life, making therapy relevant to the client's world outside of the therapy session)
- hope and expectancy (enhancing hope and decreasing hopelessness, focusing on realistic interventions, and orienting the client and therapy towards the future)
- novel methods (the therapist provides effective methods that give the client more structure and that offer the client new approaches and new ways to think and behave that address their major concerns)

David Burns (2017), a psychiatrist who popularized and expanded CBT and worked with Aaron Beck, published a recent blog where he points out that when potential bias (or favoritism) from researchers conducting comparative studies is taken into account, most of the different forms of psychotherapy produce approximately similar results. For example, he notes that in studies comparing different treatments for depression, patients who received CBT showed improvement in depression and reduced distortions in their thinking, but so did clients who were treated using other forms of therapy or who received medications. The same was true for other forms of therapy; for example, patients receiving IPT showed improved relationships and decreased conflict with others, but this was also true for other patients who improved even though they had been treated using other interventions. In other words, despite the theory or model of the treatment involved, there were few, if any, specific effects despite the theory's predictions. Burns (2017) concludes that the etiology of mental disorders is highly complex and there is still much we do not know; the different forms of psychotherapy have substantial evidence that they can help with most mental disorders, mostly likely due to common factors.[2]

Eclectic and Integrative Therapies

Most clinicians in graduate school are trained in a specific form of psychotherapy that they naturally use over time. As they help more clients and engage in continuing education in their field, they are often exposed to other approaches and other methods. It is fairly common that many of them, while they continue with the core treatment they were trained in, begin to use techniques and methods from other forms of psychotherapy, perhaps to treat specific symptoms or when one of their usual methods may not work well with a given client. This is often done without necessarily accepting or committing to the theory or model of the technique being used. This is usually referred to as "eclectic" psychotherapy (eclectic meaning taking ideas from a broad range of sources). An eclectic psychotherapist may use one mode of treatment for one patient and a different one for another patient. **Technical eclecticism** is the specific and intentional use of different psychotherapy techniques and methods, seeking to match the client's unique struggles and background with treatments that have been shown to be effective in that context.[3] The difficulty with this approach is gaining sufficient training and experience to be effective at using the different methods.

Prochaska and Norcross (2018) describe another approach called **theoretical integration.** In contrast to technical eclecticism, clinicians using this approach actually blend two or more models of therapy, both theory and techniques, in an attempt to find a more effective approach to helping clients. Among other elements, Prochaska and Norcross (2018) describe eclecticism as "technical . . . choosing from many . . . and applying the parts" while theoretical integration is "theoretical . . . combining many . . . and unifying the parts."[4] Prochaska and Norcross (2018) also describe their own approach called transtheoretical therapy, which is grounded in the transtheoretical model, a theory about how people change over time and adopt new behaviors; their model of therapy promotes the use of change techniques from most major forms of therapy at specific points, depending upon the person's stage of readiness to change.

In one sense, CBT is a broad-scale example of theoretical integration because the underlying model of cognitive therapy includes the relationship between dysfunctional thoughts and behaviors and accepts the role of classical, operant, and observational learning in psychopathology. Here are a couple of other examples:

- Psychodynamic-behavior therapy, created by Paul Wachtel, while still emphasizing the important role of early childhood experiences, argues that these early personality patterns are not fixated, are visible in interpersonal relationships in the present, and can be changed by both helping people recognize these underlying dynamics and modifying how they live in the present using behavioral methods. Prochaska and Norcross (2018) quote Wachtel: "fundamental personality change requires fundamental change in how we handle the events of our daily lives."[5]
- David Burns (2017), mentioned above, created an integrated form of therapy called TEAM CBT. TEAM stands for Testing (regular, ongoing assessment of both client symptoms and the strength of the therapeutic relationship), Empathy (a heavy emphasis on humanistic acceptance of the client and acceptance of client free will and choice), Agenda Setting (accepting and

looking for client resistance to change, addressing it, and building an agenda for change priorities with the client), and Methods (which include a wide range of techniques from CBT, motivational interviewing, Gestalt therapy, interpersonal aspects, relapse prevention and more, selected in collaboration with the client). This therapy is almost a blend of technical eclecticism with theoretical integration built on the foundation of common factors found to be effective across different therapies.[6]

While medical treatments for mental disorders are often seen as incompatible or opposed to psychotherapy, there are various situations where medications and therapy are used simultaneously, often referred to as **combination therapy**; this term is also used to describe situations where physicians or psychiatrists prescribe several different medications in a patient's treatment. In a study of data from 3100 physicians, Cascade, Kalali, and Blier (2007)[7] found that in treating depression, physicians tended to use only one antidepressant in treating depression, but used combination therapy with other drugs if they believed the patient's symptoms were more severe. Psychiatrists were four times more likely to use antidepressant combinations than primary care physicians. These authors also noted that in real-world practice, only one-third of patients with depression achieved remission after sufficient treatment with a single antidepressant and that in other fields of medicine, use of multiple treatment medications is common. A drawback of combined medical treatments for mental disorders is the cost of multiple treatments, but if these treatments prevent hospitalization or suicide attempts, it would be considered cost-effective. Sammons and McGuinness (2015),[8] two prescribing psychologists, note that most patients with mental health problems receive only medications, although they prefer psychotherapy alone or in combination with medications. They also note that there are patients who do not respond to medication alone and also patients who do not respond to psychotherapy alone; for these patients, combined treatment that involves both psychotherapy and medication tends to result in improved outcomes and reduced costs, mostly due to decreased need for healthcare utilization in the future versus those who only achieve partial remission.

For patients with certain disorders, especially those with a greater biological contribution to the etiology such as bipolar disorder or schizophrenia, combination therapy has been a common aspect of practice for some time. The medications are used to address significant symptoms that do not respond well to therapy such as mania or hallucinations or delusions, but psychotherapy is often very helpful in addressing psychosocial needs (such as improving relationships with others, developing self-care or communication skills, etc.), reducing stress to improve treatment outcomes, increasing self-acceptance and the need for treatment, and learning skills to help them prepare for release or living on their own. Most inpatient units and usually residential treatment units serving patients like this often involve them in a psychotherapeutic environment called psychosocial rehabilitation where they learn practical skills in communication, assertiveness, self-care, managing finances, and more, often using forms of CBT.

Mindfulness Approaches

One age-old practice that has seen a resurgence in popularity in recent years is mindfulness. **Mindfulness** is a process that tries to cultivate a nonjudgmental, yet attentive, mental state. It is a therapy that focuses on one's awareness of bodily sensations, thoughts, and the outside environment. Whereas other therapies work to modify or eliminate these sensations and thoughts, mindfulness focuses on non-judgmentally accepting them (Kabat-Zinn, 2003; Baer, 2003). For example, whereas CBT may actively confront and work to change a maladaptive thought, mindfulness therapy works to acknowledge and accept the thought, understanding that the thought is spontaneous and not what the person truly believes. There are two important components of mindfulness: (1) self-regulation of attention and (2) orientation toward the present moment (Bishop et al., 2004). Mindfulness is thought to improve mental health because it draws attention away from past and future stressors, encourages acceptance of troubling thoughts and feelings, and promotes physical relaxation.

Psychologists have adapted the practice of mindfulness as a form of psychotherapy, generally called **mindfulness-based therapy (MBT)**. Several types of MBT have become popular in recent years, including **mindfulness-based stress reduction (MBSR)** (e.g., Kabat-Zinn, 1982) and **mindfulness-based cognitive therapy (MBCT)** (e.g., Segal, Williams, & Teasdale, 2002).

Mindfulness-based stress reduction (MBSR) uses meditation, yoga, and attention to physical experiences to reduce stress. The hope is that reducing a person's overall stress will allow that person to more objectively evaluate his or her thoughts. In mindfulness-based cognitive therapy (MBCT), rather than reducing one's general stress to address a specific problem, attention is focused on one's thoughts and their associated emotions. For example, mindfulness-based cognitive therapy (MBCT) helps prevent relapses in depression by encouraging patients to evaluate their own thoughts objectively and without value judgment (Baer, 2003). Although cognitive behavioral therapy (CBT) may seem similar to this, it focuses on "pushing out" the maladaptive thought, whereas mindfulness-based cognitive therapy focuses on "not getting caught up" in it.



Figure **3.16.2**: Recent improvements in video chat technology along with the proliferation of mobile devices like smartphones and tablets has made online delivery of therapy more commonplace. [Image: Noba, CC BY 2.0, https://goo.gl/BRvSA7]

With growth in research and technology, psychologists have been able to develop new treatment strategies in recent years. Often, these approaches focus on enhancing existing treatments, such as cognitive-behavioral therapies, through the use of technological advances. For example, *internet-* and *mobile-delivered therapies* make psychological treatments more available, through smartphones and online access using specific technologies that encrypt communication and ensure privacy. Clinician-supervised online CBT modules allow patients to access treatment from home on their own schedule—an opportunity particularly important for patients with less geographic or socioeconomic access to traditional treatments. Furthermore, smartphones help extend therapy to patients' daily lives, allowing for symptom tracking, homework reminders, and more frequent therapist contact (Figure **3.16.2**).

Another benefit of technology is cognitive bias modification**.** Here, patients are given exercises, often through the use of video games, aimed at changing their problematic thought processes. For example, researchers might use a mobile app to train alcohol abusers to avoid stimuli related to alcohol. One version of this game flashes four pictures on the screen—three alcohol cues (e.g., a can of beer or the front of a bar) and one health-related image (e.g., someone drinking water). The goal is for the patient to tap the healthy picture as fast as he or she can. Games like these aim to target patients' automatic, subconscious thoughts that may be difficult to direct through conscious effort. That is, by repeatedly tapping the healthy image, the patient learns to "ignore" the alcohol cues, so when those cues are encountered in the environment, they will be less likely to trigger the urge to drink. Approaches like these are promising because of their accessibility; however, they require further research to establish their effectiveness.

Yet another emerging treatment employs **CBT-enhancing pharmaceutical agents**. These are drugs used to improve the effects of therapeutic interventions. Based on research from animal experiments, researchers have found that certain drugs influence the biological processes known to be involved in learning. Thus, if people take these drugs while going through psychotherapy, they are better able to "learn" the techniques for improvement. For example, the antibiotic d-cycloserine improves treatment for anxiety disorders by facilitating the learning processes that occur during exposure therapy. Ongoing research in this exciting area may prove to be quite fruitful.

Watch It

Review each of the types of psychotherapy you've learned about in this module in the following CrashCourse video.



You can view the transcript for “Getting Help – Psychotherapy: Crash Course Psychology #35” here (opens in new window).

Glossary

CBT-enhancing pharmaceutical agents: experimental use of medications used in combination with CBT therapy that enhance the learning processes that occur in CBT therapy

cognitive bias modification: the use of apps or video games to help clients work on and achieve therapeutic outcomes using technology

common factors: a common set of factors thought to underlie all forms of psychotherapy and that may be responsible for most of the beneficial outcomes from therapy

combination therapy: either the use of multiple psychotropic medications to treat a disorder or the simultaneous use of medications together with psychotherapy

Internet and mobile-delivered services: psychotherapy delivered usually in synchronous mode over the phone, tablet, or internet using special technology to ensure privacy and confidentiality

mindfulness: the process of focusing one’s attention and awareness on bodily sensations, thoughts, and awareness of the immediate environment (via sensory input: sight, hearing, touch, etc.) in the present moment without judgment

technical eclecticism therapy: the intentional use of a variety or combination of therapeutic methods or interventions drawn from various therapeutic orientations or models and customized to client symptoms and needs

theoretical integration therapy: the blending and integration of both therapeutic theories/models as well as the methods and approaches used by those theories in a way that is coherent and consistent

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3.17: Introduction to Modalities and Culture

What you'll learn to do: explain different types of treatments modalities used in treating psychopathology and the importance of cultural awareness and competence in working with clients from different backgrounds



Consistent with the biopsychosocial model, there's no single or simple way to treat a mental disorder, but psychotherapy or medicine or a combination of the two is the most common treatment method. However, even the term *psychotherapy* is broader than just one-on-one sessions between a therapist and a client. There are multiple treatment modalities in psychotherapy that are used in different contexts such as group therapy, couples therapy, psychoeducation, and family therapy. Usually, different therapists, using different treatment orientations or models may engage clients in these contexts after modifying their techniques to fit these new environments. Each client is unique, and this means that therapists must also be sensitive and aware of cultural elements that clients bring with them to therapy and that therapists also hold (including biases, cultural privilege, and more) that may influence the therapeutic relationship and the outcome of treatment.

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3.18: Treatment Modalities

Learning Objectives

- Describe the benefits of different types of treatment modalities and how they are used to address specific problems and populations

So far in this module, we have explored the biopsychosocial model of mental disorders, how disorders arise as explained by the diathesis-stress and gene-environment correlation model, and the role of epigenetics. We have also looked at the separate parts of the biopsychosocial model—the biological and psychological/social models of therapy that seek to understand symptoms and how to treat them with a variety of treatment approaches or orientations. In this section, we will be exploring treatment **modalities**. Treatment modalities are not based on any specific theory or model of psychotherapy; instead, they are different environments in which treatment takes place. Couples therapy, for example, can be approached from a range of different therapeutic models like psychodynamic, CBT, interpersonal, or forms of integrated therapy. What is unique is that the environment in which those approaches are used now involves two people, already in a relationship, interacting with the therapist and each other. Each modality is different, may address different populations or needs, and requires specific skills and training on the part of the clinician to increase the chances of success.

Treatment Modalities

There are several modalities, or environments, in which psychotherapeutic treatments take place: individual therapy, group therapy, couples therapy, and family therapy are the most common. In an individual therapy session, a client works one-on-one with a trained therapist. In group therapy, usually five to 10 people meet with a trained group therapist to discuss a common issue (e.g., divorce, grief, eating disorders, substance abuse, or anger management). Couples therapy involves two people in an intimate relationship who are having difficulties and are trying to resolve them. The couple may be dating, partnered, engaged, or married. The therapist helps them resolve their problems as well as implement strategies that will lead to a healthier and happier relationship. Family therapy is a special form of group therapy made up of one or more families. The goal of this approach is to enhance the growth of each individual family member and enhance the functioning of the family as a whole.

Once a person seeks treatment, whether voluntarily or involuntarily, he or she has an **intake** assessment done to assess the person's clinical needs. An intake is the therapist's first meeting with the client. The therapist gathers specific information to address the client's immediate needs and to arrive at a diagnosis, such as the presenting problem, symptoms, the client's support system, and insurance status. Depending on the setting, the intake therapist may also carry out brief aspects of assessment such as those described in Chapter 1. The therapist informs the client about confidentiality, fees, and what to expect in treatment. **Confidentiality** means the therapist cannot disclose confidential communications to any third party without the client's consent unless mandated or permitted by law to do so. During the intake, the therapist and client will work together to discuss treatment goals. Then a treatment plan will be formulated, usually with specific measurable objectives. Also, the therapist and client will discuss how treatment success will be measured and the estimated length of treatment and cost. It is common in mental health centers or in other treatment facilities for one clinician to carry out the initial intake and, depending on the results, to recommend more in-depth assessment, or a specific modality of treatment usually carried out by another clinician. For instance, a client may go through an intake that leads to a preliminary diagnosis and referral to another clinician for group therapy that targets clients with that diagnosis.



(a)



(b)

Figure 3.18.1: Therapy may occur (a) one-on-one between a therapist and client or (b) in a group setting. (credit a: modification of work by Connor Ashleigh, AusAID/Department of Foreign Affairs and Trade)

Individual Therapy

In **individual therapy**, also known as individual psychotherapy or individual counseling, the client and clinician meet one-on-one (usually from 45 minutes to an hour). These meetings typically occur weekly or every other week, and sessions are conducted in a confidential and caring environment (Figure 1). The clinician will work with clients to help them explore their feelings, work through life challenges, identify aspects of themselves and their lives that they wish to change, and set goals to help them work towards these changes. In our previous discussions of treatment approaches, this was the implied environment for treatment. A client might see a clinician for only a few sessions, or the client may attend individual therapy sessions for a year or longer depending on the therapeutic orientation of the therapist. The amount of time spent in therapy also depends on the needs of the client, the nature of the disorder and severity of symptoms, and the client's personal goals. For example, a patient being treated for mild depression by a CBT therapist may be in treatment for eight to 10 total sessions while a client with a personality disorder may be seen by a therapist for months or even more than a year because of the nature of the disorder and the degree of change involved.

Group Therapy



Figure **3.18.2**: In group therapy, usually five to 10 people meet with a trained therapist to discuss a common issue such as divorce, grief, an eating disorder, substance abuse, or anger management. (credit: Cory Zanker)

In **group therapy**, a clinician meets together with several clients with similar problems (Figure **3.18.2**). When children are placed in group therapy, it is particularly important to match clients by age as well as the nature of their problems. One benefit of group therapy is that it can help decrease a client's shame and isolation about a problem while offering needed support, both from the therapist and other members of the group (American Psychological Association, 2014). A nine-year-old sexual abuse victim, for example, may feel very embarrassed and ashamed to talk about his situation. If he is placed in a group with other sexually abused boys, he will realize that he is not alone. A child struggling with poor social skills would likely benefit from a group with a specific curriculum to foster interpersonal skills. The group environment would give the child the opportunity not only to learn along with others, but to practice the skills together in a supportive environment. A woman suffering from post-partum depression could feel less guilty and more supported by being in a group with similar women.

Group therapy also has some specific areas of concern. Members of the group may be afraid to speak in front of other people because sharing secrets and problems with complete strangers can be stressful and overwhelming, especially at first. There may be personality clashes and arguments among group members. There could also be concerns about confidentiality: someone from the group might share what another participant said to people outside of the group. Especially among adults, many of these issues should be specifically addressed and discussed by the therapist at the start of the group. If a client is added soon after a group has already begun, the therapist may meet with them individually to explain the basic aspects of participating in the group, answer questions, and then introduce the client into the group environment and other members.

Another benefit of group therapy is that members can confront each other about their patterns. For those with some types of problems, such as sexual abusers, group therapy is the recommended treatment for this very reason. Group treatment for this population is considered to have several benefits:

Group treatment is more economical than individual, couples, or family therapy. Sexual abusers often feel more comfortable admitting and discussing their offenses in a treatment group where others are modeling openness. Clients often accept feedback about their behavior more willingly from other group members than from therapists. Finally, clients can practice social skills in group treatment settings. (McGrath, Cumming, Burchard, Zeoli, & Ellerby, 2009)

During a group session, the entire group could reflect on an individual's problem or difficulties, and others might disclose what they have done in that situation. When a clinician is facilitating a group, the focus is always on making sure that everyone benefits

and participates in the group and that no one person is the focus of the entire session. Groups can be organized in various ways: some have an overarching theme or purpose, some are time-limited, some have open membership that allows people to come and go, and some are closed. Some groups are structured with planned activities and goals, while others are unstructured: There is no specific plan, and group members themselves decide how the group will spend its time and on what goals it will focus. This can become a complex and emotionally charged process, but it is also an opportunity for personal growth (Page & Berkow, 1994).

Psychoeducation Group Treatment

Groups that have a strong educational component are called **psychoeducation** groups. For example, a group for children who have a parent with cancer might discuss in depth what cancer is, types of treatments are used for cancer, the side effects of treatments such as hair loss, as well as ways to support the family member, and ways to cope with the emotional impact on themselves. For children, group therapy sessions often take place in school. They are led by a school counselor, a school psychologist, or a school social worker. Groups might focus on test anxiety, social isolation, self-esteem, bullying, or school failure (Shechtman, 2002). Whether the group is held in school or in a clinician's office, group therapy has been found to be effective with children facing numerous kinds of challenges (Shechtman, 2002).

Other examples include psychoeducation family therapy with the family members of a person diagnosed with schizophrenia. One of the major goals of such treatment is to allow family members to express their concerns and feel supported, but it is also to receive educational information regarding schizophrenia and to dispute and reduce misinformation, especially because there are many social stereotypes about persons with schizophrenia (such as that they are all violent and dangerous). Psychoeducation in this situation is also intended to educate the family about expressed emotion, meaning emotionally charged interactions with the person with schizophrenia; lowering the level of expressed emotion in the family is important in preventing relapse. Psychoeducational groups often take place as well in inpatient or residential facilities where the goal is to teach patients about life skills such as applying for a job or how to find a dentist, discussions about medications and treatments, or assertiveness training.

Couples Therapy

Couples therapy involves two people in an intimate relationship who are having difficulties and are trying to resolve them (Figure 3). The couple may be dating, partnered, engaged, or married. The primary therapeutic orientation used in couples counseling is cognitive-behavioral therapy (Rathus & Sanderson, 1999), although other orientations also are used depending on the therapist. Couples meet with a therapist to discuss conflicts and/or aspects of their relationship that they want to change. The therapist helps them see how their individual backgrounds, beliefs, and actions are affecting their relationship. Often, a therapist tries to help the couple resolve these problems, as well as implement strategies that will lead to a healthier and happier relationship, such as how to listen, how to argue productively, and how to express feelings. However, sometimes, after working with a therapist, a couple will realize that they are too incompatible and will decide to separate. Some couples seek therapy to work out their problems, while others attend therapy to determine whether staying together is the best solution. Counseling couples in a high-conflict and volatile relationship can be difficult. In fact, psychologists Peter Pearson and Ellyn Bader, who founded the Couples Institute in Palo Alto, California, have compared the experience of the clinician in couples' therapy to be like "piloting a helicopter in a hurricane" (Weil, 2012, para. 7). As with group therapists, being an effective therapist in this modality requires specialized training; couples therapy is not simply conducting individual therapy with two people present. With proper training, it can be a fulfilling, although challenging, occupation.



Figure **3.18.3**: In couples counseling, a therapist helps people work on their relationship. (credit: Cory Zanker)

Family Therapy

Family therapy is a special form of group therapy, consisting of one or more families. Although there are many theoretical orientations in family therapy, one of the most predominant is the systems approach. The family is viewed as an organized system, and each individual within the family is a contributing member who creates and maintains processes within the system that shape behavior (Minuchin, 1985). It may help you to think back to our discussion of systems and how they work at the start of the module. Each member of the family influences and is influenced by the others; they are all interdependent in their relationships. The goal of this approach is to enhance the growth of each family member as well as that of the family as a whole.

Often, dysfunctional patterns of communication that develop between family members can lead to conflict. These patterns will likely be evident in the interactions of family members during sessions and can be addressed directly by the therapist as they are happening. In many cases, one member of the family has problems that detrimentally affect everyone. For example, a mother's depression, a teen daughter's eating disorder, or a father's alcohol dependence could affect all members of the family. The therapist would work with all members of the family to help them cope with the issue, support each other, and to encourage resolution and growth for the individual family member with the problem.

With family therapy, the nuclear family (i.e., parents and children) or the nuclear family plus whoever lives in the household (e.g., grandparent) come into treatment. Family therapists work with the whole family unit to heal the family. In addition to the systems approach, there are other types of family therapy. For example, in structural family therapy, the therapist examines and discusses the boundaries and structures within the family: who makes the rules, who sleeps in the bed with whom, how decisions are made, and what are the boundaries within the family. In some families, the parents do not work together to make rules, or one parent may undermine the other, leading the children to act out. The therapist helps them resolve these issues and learn to communicate more effectively.

In **strategic family therapy**, the goal is to address specific problems within the family that can be dealt with in a relatively short amount of time. Typically, the therapist would guide what happens in the therapy session and design a detailed approach to resolving each member's problem (Madanes, 1991). As with other forms of treatment described in this section of the module, family therapists receive specialized training and supervision beyond that of individual therapy in order to work within this treatment modality.

Other Treatment Environments

In addition to the modalities mentioned above, there are other, broader environments in which psychotherapy takes place:

- **inpatient hospitalization**: treatment occurs within a hospital providing 24-hour care and observation; patients who are sent for inpatient hospitalization have usually been evaluated by a court to represent a risk of harm or danger to themselves or others. A multidisciplinary treatment team usually including a psychiatrist, psychologist, social worker, nursing staff, recreation therapists, and others as needed work together to formulate a plan for careful assessment and treatment of the patient; these treatment plans frequently involve medications, individual and group therapy, possibly psychoeducation, and other treatments including recreation therapy and medical treatment for other problems. These professionals have to communicate effectively with each other and closely coordinate their efforts for the best treatment outcomes. In many states, a person can be confined to an inpatient hospital for a period of observation and assessment lasting 48 or 72 hours with a follow-up report to the court regarding whether there is need for more sustained confinement and extended treatment. Most states also permit a person to voluntarily choose hospitalization for treatment; if the person does not have health insurance, they may be admitted, if space is available, to state-run psychiatric hospitals. Voluntary patients can leave if they choose to do so unless the treatment team believes they could harm themselves or others, in which case the court would have to evaluate the situation and make a decision.
- **partial hospitalization**: usually this type of environment may be chosen if a patient no longer requires 24-hour inpatient care (which is very expensive), but still requires a high degree of coordination among professionals and a variety of both psychiatric and psychotherapeutic care. The patient usually attends treatment only during the day. In addition to the others treatments mentioned above, the patient may receive specialized forms of treatment, such as behavioral training and support to help a patient learn life skills to be able to successfully live on their own and/or skills to be able to secure a job in the near future. At the end of the day, the patient returns home or to a supervised living arrangement where they care for themselves.

- **residential treatment center**: a residential treatment center (RTC), sometimes called a rehab, is a live-in health care facility providing therapy for substance abuse, mental illness, or other behavioral problems. Residential treatment is a more restrictive option than outpatient therapy; however, it is less restrictive than inpatient psychiatric hospitals. For adolescents, most RTCs use a behavior modification paradigm. Others are relationally oriented, focusing on community and interpersonal relationships. Some utilize a community or positive peer-culture model. Generalist programs are usually large (80-plus clients and as many as 250) and level-focused in their treatment approach. That is, in order to manage clients' behavior, they frequently put systems of rewards and punishments in place. Specialist programs are usually smaller (less than 100 clients and as few as 10 or 12). Specialist programs typically are not as focused on behavior modification as generalist programs are. Different RTCs work with different types of problems, and the structure and methods of RTCs varies. Some RTCs are lock-down facilities; that is, the residents are locked inside the premises. In a locked residential treatment facility, clients' movements are restricted. By comparison, an unlocked residential treatment facility allows them to move about the facility with relative freedom, but they are only allowed to leave the facility under specific conditions. Adult RTC programs are similar in many ways, but focus primarily on either adults who have been discharged from inpatient hospitals but who are not yet ready to live on their own, or adults with substance dependence who have struggled to avoid **relapse** and need a more structured environment.
- **outpatient care**: this environment is the "default" for most persons with mental disorders. It can include a range of treatment modalities usually chosen by the client themselves, although some may be court-ordered, especially for persons with lesser criminal offenses or drug charges. The client arranges to meet with an appropriate therapist or psychiatrist for an intake session and together, they decide which modality of treatment would be most effective. This form of treatment is mostly covered through health insurance. Many clients at least initially seek outpatient care from primary care physicians who often begin treatment with medications; some psychiatrists, depending on the diagnosis, may recommend a psychotherapy modality as the primary treatment.
- **forensic psychiatric units**: unlike the other environments described above, forensic psychiatric units represent a blend of criminal justice and mental health treatment environments. Individuals are sent to these units by the court system, and the unit, which is often part of a larger psychiatric inpatient hospital, is frequently stand alone; everyday procedures are managed very much like a jail or prison environment with increased security compared to a typical inpatient treatment unit. The goal and function of this type of facility is also distinctive. Inmates on the unit are defendants charged with criminal offenses and the purpose of the unit is to carry out thorough assessments, most commonly for competency to stand trial or for insanity at the time of the offense and report the results to the court. Some individuals are receiving treatment to restore them to legal competence, meaning that the focus of both psychiatric and psychological care is to enable defendants to be returned to the criminal justice system in order to stand trial. These units may also house some individuals who have been found not guilty by reason of insanity and are ordered for treatment until they are judged safe to return to a regular inpatient treatment or to be released. In addition to medications, inmates may also receive individual therapy to reduce symptoms and improve functioning and often group therapy that frequently involves psychoeducation regarding the court system, to help them understand the charges against them, how the legal system functions, and how to work with an attorney to prepare their defense. An alternate form of this environment is found in prisons with individuals who may have been found guilty but mentally ill (depending on state law); these individuals receive the equivalent of inpatient treatment, but in a prison setting and will continue to serve their sentence even when treatment is successful.

Watch It

Watch this video to learn more about a structural family session. At the five-minute mark, you'll see an example of a therapy session with the family and a counselor.



You can view the transcript for "Family Therapy" here (opens in new window).

Treatment for Addiction

Addiction and substance abuse disorders are difficult to treat because chronic substance use can permanently alter the neural structure in the prefrontal cortex, an area of the brain associated with decision-making and judgment, thus driving a person to continue to use drugs and/or alcohol in spite of negative consequences (Muñoz-Cuevas, Athilingam, Piscopo, & Wilbrecht, 2013). This helps explain why relapse rates tend to be high. About 40–60% of individuals **relapse**, which means they return to abusing drugs and/or alcohol after a period of improvement (National Institute on Drug Abuse [NIDA], 2008).

The goal of substance-related treatment is to help an addicted person stop compulsive drug-seeking behaviors (NIDA, 2012). This means an addicted person will need long-term treatment, similar to a person battling a chronic physical disease such as hypertension or diabetes. Treatment usually includes behavioral therapy and/or medication, depending on the individual (NIDA, 2012). Specialized therapies have also been developed for specific types of substance-related disorders, including alcohol, cocaine, and opioids (McGovern & Carroll, 2003). Substance-related treatment is considered much more cost-effective than incarceration or not treating those with addictions (NIDA, 2012).



Figure 3.18.4: Substance use and abuse costs the United States over $600 billion a year (NIDA, 2012). This addict is using heroin. (credit: "jellymc – urbansnaps"/Flickr)

Specific factors make substance-related treatment much more effective. One factor is the duration of treatment. Generally, the addict needs to be in treatment for at least three months to achieve a positive outcome (Simpson, 1981; Simpson, Joe, & Bracy, 1982; NIDA, 2012). This is due to the psychological, physiological, behavioral, and social aspects of abuse (Simpson, 1981; Simpson et al., 1982; NIDA, 2012).

While individual therapy is used in the treatment of substance-related disorders, group therapy is the most widespread treatment modality (Weiss, Jaffee, de Menil, & Cogley, 2004). The rationale behind using group therapy for addiction treatment is that addicts are much more likely to maintain sobriety in a group format. It has been suggested that this is due to the rewarding and therapeutic benefits of the group, such as support, affiliation, identification, and even confrontation (Center for Substance Abuse Treatment, 2005).

Treatment also usually involves medications to detox the addict safely after an overdose, to prevent seizures and agitation that often occur in detox, to prevent reuse of the drug, and to manage withdrawal symptoms. Getting off drugs often involves the use of drugs—some of which can be just as addictive. Detox can be difficult and dangerous.

Frequently, a person who is addicted to drugs and/or alcohol has **comorbid disorders**, meaning they may have additional diagnoses of other psychological disorders. In cases of comorbidity, the best treatment is thought to address both (or multiple) disorders simultaneously (NIDA, 2012). Behavior therapies are used to treat comorbid conditions, and in many cases, medications are used along with psychotherapy.

Glossary

comorbid disorder: individual who has two or more diagnoses, which often includes a substance abuse diagnosis and another psychiatric diagnosis, such as depression, bipolar disorder, or schizophrenia

confidentiality: therapist cannot disclose confidential communications to any third party, unless mandated or permitted by law

couples therapy: two people in an intimate relationship, such as husband and wife, who are having difficulties and are trying to resolve them with therapy

family therapy: special form of group therapy consisting of one or more families

group therapy: treatment modality in which five to 10 people with the same issue or concern meet together with a trained clinician

individual therapy: treatment modality in which the client and clinician meet one-on-one

intake: therapist's first meeting with the client in which the therapist gathers specific information to address the client's immediate needs

modality: a specific environment in which treatment takes place; it can vary from the number of people involved and the physical setting in which treatment takes place.

psychoeducation: a form of group or family therapy where a large emphasis is placed on educating the participants about symptoms, social skills, and ways to cope with mental disorders

relapse: resumption of drug and/or alcohol use after a period of improvement from substance abuse

strategic family therapy: therapist guides the therapy sessions and develops treatment plans for each family member for specific problems that can be addressed in a short amount of time

structural family therapy: therapist examines and discusses with the family the boundaries and structure of the family: who makes the rules, who sleeps in the bed with whom, how decisions are made, and what are the boundaries within the family

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3.19: The Sociocultural Perspective and Cultural Competence

Learning Objectives

- Describe the impact of culture on key aspects of treatment availability and access and explain the importance of cultural competence to ethical and effective clinical practice

Unlike the other theoretical models described so far that attempt to explain the etiology of mental disorders and lead to a specific form of therapeutic orientation and methods, the sociocultural perspective looks at a person's thoughts, emotions, behaviors, and symptoms in the context of the person's culture and background. Think of it as an additional dimension of assessment and understanding of the person seeking treatment that helps the therapist establish a helpful and understanding relationship with the client. For example, José is an 18-year-old male from a traditional Mexican-American family. José comes to treatment because of depression. During the intake session, he reveals that he is gay and is nervous about telling his family. He also discloses that he is concerned and anxious because his religious background has taught him that homosexuality is morally wrong. How does his religious and cultural background affect him? How might his cultural background affect how his family reacts if José were to tell them he is gay? To be effective in helping José, the therapist has to consider these aspects of his life and identity in order to make useful suggestions or recommendations. To ignore these social and cultural aspects of José's background could lead to recommendations that actually increase his stress and depression.

Multicultural counseling and therapy does not utilize specific methods or techniques; instead, it aims to define treatment goals and use those therapeutic methods consistent with the life experiences and cultural values of clients. It strives to recognize that all client identities include individual, group, and universal dimensions, advocate the use of universal and culture-specific strategies and roles in the healing process and balances the importance of individualism and collectivism in the assessment, diagnosis, and treatment of the client and client systems (Sue, 2001). For example, all persons have aspects of their personality and biology that are unique to them from their genetic makeup, to personal life experiences and learning. Likewise, all persons have group memberships or relationships with culture that shape their identity, attitudes, emotions, and behaviors such as gender, family, age, religion, ethnicity, nationality, economic class, and more. All human beings also share some universal aspects such as basic emotions (happiness, sadness, anxiety, surprise, etc.), desire for acceptance, common life experiences (childhood, puberty, etc.), self-awareness, capacity for language and learning, etc. Some cultures are more individualistic, emphasizing one's own personal perspective and achieving one's own personal goals in life while other cultures are significantly more collectivist, emphasizing conforming behavior and actions that support one's family, group, clan, or tribe.

The sociocultural perspective accepts and integrates the impact of cultural and social norms starting at the beginning of treatment. Therapists who use this perspective work with clients to obtain and integrate information about their cultural patterns into a unique treatment approach based on their particular situation (Stewart, Simmons, & Habibpour, 2012) and acknowledge cultural differences between client and therapist that could influence the therapeutic relationship and treatment. Sociocultural therapy can include individual, group, family, and couples treatment modalities.

As our society becomes increasingly multiethnic and multiracial, mental health professionals must develop **cultural competence** (Figure 3.19.1). This means they must understand; accept; and honestly and openly address issues of gender, race, culture, ethnicity, and other variables in terms of how these shape their own life and attitudes and how they shape the experiences of others. They must also develop strategies to effectively address the needs of various populations for which Eurocentric therapies, which tend to assume individualistic goals are most important (such as achieving one's personal dreams or goals in life) have limited application (Sue, 2004). For example, a counselor whose treatment focuses on individual decision making may be ineffective at helping a Chinese-American client with a collectivist approach to problem solving (Sue, 2004) in which the needs and values of others may be more important than the client's own goals.

Because of these concerns, most professional associations have adopted or incorporated cultural competence into their ethical standards or principles. For example, in 2017, the American Psychological Association adopted 10 basic principles of cultural competence that are "necessary for psychologists working in all domains: practice, research, consultation and education."[1] Among other points, these principles include

- psychologists need to understand that as “cultural beings” they hold personal beliefs and attitudes that shape how they view and interact with others;
- the need to be aware of how social and physical environments impact the lives of research participants, clients, students, and others;
- the need to understand and be aware of historical and ongoing issues of power and privilege, and histories of oppression and seek to address disparities and inequalities as they promote justice, human rights, and access; and
- the promotion of “culturally adaptive” interventions.

It is vital that all therapists understand and acknowledge the impact of social situations such as racial discrimination on mental health. For instance, a recent longitudinal study of almost 700 Black adolescents for a period of 18 years found that perceived racial discrimination predicted increases in anxiety and depression in adulthood among Black males.[2].



Figure **3.19.1**: How do your cultural and religious beliefs affect your attitude toward mental health treatment? (credit “top-left”: modification of work by Staffan Scherz; credit “top-left-middle”: modification of work by Alejandra Quintero Sinisterra; credit “top-right-middle”: modification of work by Pedro Ribeiro Simões; credit “top-right”: modification of work by Agustin Ruiz; credit “bottom-left”: modification of work by Czech Provincial Reconstruction Team; credit “bottom-left-middle”: modification of work by Arian Zwegers; credit “bottom-right-middle”: modification of work by “Wonderlane”/Flickr; credit “bottom-right”: modification of work by Shiraz Chanawala)

Watch It

Watch this short video explains cultural competence and sociocultural treatments.



You can view the transcript for “What is cultural competence and why is it important?” here (opens in new window).

Link to Learning

This link takes you to a published case study of a Muslim-American “hijabi” woman (who follows traditional Muslim dress codes) struggling with an eating disorder and depression. It is a good illustration of the use of culturally competent practices. While the entire article is useful, especially review the initial mental status examination, diagnosis, case summary, course of

treatment, and discussion. This will give you a good idea of how important cultural competence is and some of the major struggles that clients from different backgrounds face.

Understanding Barriers to Treatment

Statistically, ethnic minorities tend to utilize mental health services less frequently than White, middle-class Americans (Alegría et al., 2008; Richman, Kohn-Wood, & Williams, 2007). Why is this so? Perhaps the reason has to do with access and availability of mental health services. Ethnic minorities and individuals of low socioeconomic status (SES) report that barriers to services include lack of insurance, access to transportation, and time required to navigate these elements (Thomas & Snowden, 2002). However, researchers have found that even when income levels and insurance variables are taken into account, ethnic minorities are far less likely to seek out and utilize mental health services. And when access to mental health services is comparable across ethnic and racial groups, differences in service utilization remain (Richman et al., 2007).

In a study involving thousands of women, it was found that the prevalence rate of anorexia was similar across different races, but that bulimia nervosa was more prevalent among Hispanic and African-American women when compared with non-Hispanic Whites (Marques et al., 2011). Although they have similar or higher rates of eating disorders, Hispanic and African American women with these disorders tend to seek and engage in treatment far less than Caucasian women. These findings suggest ethnic disparities in access to care, as well as clinical and referral practices that may prevent Hispanic and African-American women from receiving care, which could include lack of bilingual treatment, stigma, fear of not being understood, family privacy, and lack of education about eating disorders.

Perceptions and attitudes toward mental health services may also contribute to this imbalance. A recent study at King's College, London, found many complex reasons why people do not seek treatment: self-sufficiency and not seeing the need for help, not seeing therapy as effective, concerns about confidentiality, and the many effects of stigma and shame (Clement et al., 2014). And in another study, African Americans exhibiting depression were less willing to seek treatment due to fear of possible psychiatric hospitalization as well as fear of the treatment itself (Sussman, Robins, & Earls, 1987). Instead of mental health treatment, many African Americans prefer to be self-reliant or use spiritual practices (Snowden, 2001; Belgrave & Allison, 2010). For example, it has been found that the Black church plays a significant role as an alternative to mental health services by providing prevention and treatment-type programs designed to enhance the psychological and physical well-being of its members (Blank, Mahmood, Fox, & Guterbock, 2002).



Figure **3.19.2**: Accessible mental health services for those who don't speak English can be difficult to find.

Additionally, people belonging to ethnic groups that already report concerns about prejudice and discrimination are less likely to seek services for a mental illness because they view it as an additional stigma (Gary, 2005; Townes, Cunningham, & Chavez-Korell, 2009; Scott, McCoy, Munson, Snowden, & McMillen, 2011). For example, in one recent study of 462 older Korean Americans (over the age of 60) many participants reported suffering from depressive symptoms. However, 71% indicated they thought depression was a sign of personal weakness, and 14% reported that having a mentally ill family member would bring shame to the family (Jang, Chiriboga, & Okazaki, 2009).

Language differences are a further barrier to treatment. In the previous study on Korean Americans' attitudes toward mental health services, it was found that there were no Korean-speaking mental health professionals where the study was conducted (Orlando and Tampa, Florida) (Jang et al., 2009). Because of the growing number of people from ethnically diverse backgrounds, there is a need for therapists and psychologists to develop knowledge and skills to become culturally competent (Ahmed, Wilson, Henriksen, & Jones, 2011). Those providing therapy must approach the process from the context of the unique culture of each client (Sue & Sue, 2007).

Dig Deeper: Treatment Perceptions

By the time a child is a senior in high school, 20% of his or her classmates—that is one in five—will have experienced a mental health problem (U.S. Department of Health and Human Services, 1999), and 8%—about one in 12—will have attempted suicide (Centers for Disease Control and Prevention, 2014). Of those classmates experiencing mental disorders, only 20% will receive professional help (U.S. Public Health Service, 2000). Why?

Apparently, the public has a negative perception of children and teens with mental health disorders. According to researchers from Indiana University, the University of Virginia, and Columbia University, interviews with over 1,300 U.S. adults show that they believe children with depression are prone to violence and that if a child receives treatment for a psychological disorder, then that child is more likely to be rejected by peers at school.

Bernice Pescosolido, author of the study, asserts that this is a misconception. However, stigmatization of psychological disorders is one of the main reasons why young people do not get the help they need when they are having difficulties. Pescosolido and her colleagues caution that this stigma surrounding mental illness, based on misconceptions rather than facts, can be devastating to the emotional and social well-being of our nation's children.

This warning played out as a national tragedy in the 2012 shootings at Sandy Hook Elementary. In her blog, Suzy DeYoung (2013), co-founder of Sandy Hook Promise (the organization that parents and concerned others set up in the wake of the school massacre) speaks to treatment perceptions and what happens when children do not receive the mental health treatment they desperately need.

> I've become accustomed to the reaction when I tell people where I'm from. Eleven months later, it's as consistent as it was back in January. Just yesterday, inquiring as to the availability of a rental house this holiday season, the gentleman taking my information paused to ask, "Newtown, Connecticut? Isn't that where that . . . that *thing* happened?"
>
> A recent encounter in the Massachusetts Berkshires, however, took me by surprise.
>
> It was in a small, charming art gallery. The proprietor, a woman who looked to be in her 60s, asked where we were from. My response usually depends on my present mood and readiness for the inevitable dialogue. Sometimes it's simply, Connecticut. This time, I replied, "Newtown, Connecticut." The woman's demeanor abruptly shifted from one of amiable graciousness to one of visible agitation.
>
> "Oh my God," she said wide-eyed and open-mouthed. "Did you know her?"
>
> . . . .
>
> "Her?" I inquired
>
> "That woman," she replied with disdain, "that woman that raised that monster."
>
> "That woman's" name was Nancy Lanza. Her son, Adam, killed her with a rifle blast to the head before heading out to kill 20 children and six educators at Sandy Hook Elementary School in Newtown, CT [December 14th, 2012].
>
> When Nelba Marquez Greene, whose beautiful six-year-old daughter, Ana, was killed by Adam Lanza, was recently asked how she felt about "that woman," this was her reply:
>
> "She's a victim herself. And it's time in America that we start looking at mental illness with compassion, and helping people who need it. This was a family that needed help, an individual that needed help and didn't get it. And what better can come of this, of this time in America, than if we can get help to people who really need it?" (pars. 1–7, 10–15)

Fortunately, we are starting to see campaigns related to the de-stigmatization of mental illness and an increase in public education and awareness. Join the effort by encouraging and supporting those around you to seek help if they need it. To learn more, visit the National Alliance on Mental Illness (NAMI) website (http://www.nami.org/). The nation's largest nonprofit mental health advocacy and support organization is NAMI.

Glossary

cultural competence: the therapist's understanding and attention to issues of race, culture, and ethnicity in themselves and in their clients when providing treatment

1. American Psychological Association. *APA adopts new multicultural guidelines*. Monitor on Psychology. https://www.apa.org/monitor/2018/01/multicultural-guidelines. ↵
2. Assari, S., Moazen-Zadeh, E., Caldwell, C.H., & Zimmerman, M.A. (2017). Racial Discrimination during Adolescence Predicts Mental Health Deterioration in Adulthood: Gender Differences among Blacks. *Frontiers in Public Health*. Retrieved from: https://doi.org/10.3389/fpubh.2017.00104↵

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3.20: Putting It Together- Etiology, Models, and Therapies

Putting It Together: Etiology, Models, and Therapies

In this chapter, you learned the following:

- the problems and insufficiency of unidimensional explanations of mental disorders
- the importance of the biopsychosocial model for understanding that mental disorders arise through systemic interactions
- the value of the diathesis-stress and gene-environment-correlation models as ways of understanding the etiology of mental disorders
- to identify and explain the theoretical models based on parts of the biopsychosocial model that have been used to explain how symptoms develop and methods that can be used to treat mental disorders
- the range of modalities that exist to treat mental disorders in various environments and the larger environments where these treatments are used
- to explain why the sociocultural perspective is important in therapy, the importance of cultural competence in practice, and some of the types of cultural barriers that prevent some people from receiving mental health services

This module covered a lot of ground. You learned that overly simplistic unidimensional explanations of mental disorders, like saying that they are caused by a "chemical imbalance" or by how your mother treated you when you were little, are not only inadequate to explain how these disorders occur, but they are also mostly inaccurate on their own. Mental disorders are the outcome of systemic effects that occur inside our body and brain and that interact with the external environment including social and cultural forces. There is evidence that epigenetic processes are also involved meaning that the environment actually influences which genes in our DNA are turned on or off.

A systemic model that gives a better understanding of the complexity involved is the biopsychosocial model that integrates biological (including genetics, neural transmission, and other aspects), psychological (thoughts, attitudes, beliefs, emotions, behaviors, learning), and sociocultural elements (prejudice, discrimination, stress, abuse, poverty, healthcare, etc.) to help us better understand how disorders arise. Remember that these elements are *interdependent*, meaning that they influence each other—there is no one-way street here. You also learned about the diathesis-stress model, an older explanation that describes how a person may have a genetic vulnerability or predisposition towards developing a disorder, and that if that person experiences sufficient stress (from any of the biopsychosocial elements) to exceed a threshold, they will develop the disorder. The more modern version of this is the gene-environment-correlation model (rGE) that describes how a person's genome (set of genes in their DNA) may not only make someone vulnerable to developing a disorder, but may actually contribute to generating the environmental stress that exceeds a threshold and triggers the disorder.

You then reviewed the various theoretical models that developed to explain the separate *parts* of the biopsychosocial model and that emerged either independently or in reaction to one another as scientists and clinicians tried to figure out how mental disorders arise. You started with the biological model that explained how mental disorders are polygenic, arising through the interactions of multiple genes. You found out about heritability and concordance rates that are used by scientists to estimate how much genetic variables contribute to a disorder versus the environment. You also learned about the structure of neurons, our primary brain cells, and how they communicate through producing neurotransmitters and releasing those into the synapse between neurons when they receive a signal of sufficient strength and how action potentials are all or none. You learned about receptor binding and reuptake when the presynaptic neurons "recycle" or repackage neurotransmitters for use again in a future signal. You also learned about the major neurotransmitters that are related to mental disorders: dopamine, norepinephrine, serotonin, GABA, and glutamate. You also learned about brain structures and the relationships between the brain and the endocrine system.

You learned about biomedical treatment approaches. These include the use of psychotropic medications for different mental disorders, an approach that is widely used today and that can be helpful for some, but not all, individuals. Most medications can create side effects that can be problematic and patients often relapse if medications are stopped. You also learned that combined therapy (medications and psychotherapy) can be helpful, especially when symptoms are more severe. For example, an individual with schizophrenia who is bombarded with visual or auditory hallucinations may find it difficult to focus in psychotherapy; with medication, the individual's hallucinations can be eliminated or reduced to a level that allows the individual to benefit from

psychotherapy. Other biomedical treatment included various forms of brain stimulation treatments such as electroconvulsive therapy (ECT) and repetitive transcranial magnetic stimulation; some of these therapies are still experimental and are being tested.

We then reviewed a wide range of psychotherapeutic models or theoretical explanations for how symptoms develop. These were paired with the treatment approaches or orientations that were developed based on these theories. These explanations included the following:

- **psychodynamic approaches**: based originally on the work of Freud, his models of personality, defense mechanisms, and unconscious conflicts, many different approaches or orientations to treatment have been developed. Many of Freud's ideas have not been supported by modern research, although a few have found support and have been integrated into later approaches. Freud developed psychoanalysis, the modern descendants of which are a range of psychodynamic therapies. The primary focus is to reveal the unconscious content of a client's psyche in an effort to alleviate psychic tension. Modern psychodynamic therapy tends to be briefer and less intensive than traditional psychoanalysis. Play therapy also originated in psychodynamic thought but has now been expanded into other forms of treatment as well.
- **humanistic approaches**: reacting to Freud's ideas, Carl Rogers and Abraham Maslow were the primary developers of humanistic therapy which was grounded in a more holistic model of human beings that emphasized the potential for good in all people; accepted and encouraged human choice and will; and emphasized human desires for meaning, value, and creativity. Related treatment orientations and methods included existential therapy and person-centered therapy. Some of the foundational ideas of Rogers' person-centered therapy, including unconditional positive regard and active listening, have been adopted in almost all subsequent forms of therapy and represent one of the common factors that appear to be related to the effectiveness of therapy regardless of orientation.
- **behavioral approaches**: rejecting ideas of that biology determined outcomes as well as Freud's observational theories of human functioning, behaviorists emphasized a more objective scientific approach based on research of observable behaviors. Grounded in the primary work of Pavlov, Watson, Thorndike, and Skinner, behaviorists uncovered important aspects of human learning such as classical conditioning, operant conditioning, and observational learning. Remember that negative reinforcement *increases* a behavior (usually avoidant) and is the *opposite* of punishment! In working on observational learning, Bandura began to integrate ideas of how human cognition interact with behavioral principles to influence behavior. These models led to a wide range of effective methods for treating a variety of mental disorders such as exposure and extinction therapies, counterconditioniing, and more.
- **cognitive and cognitive behavioral approaches (CBT)**: The cognitive model was grounded in the work of Adler, Ellis, and Beck, who explored the role that a person's thought patterns played in generating intense emotions and problem behaviors. The core idea at the center of the cognitive revolution in treatment was that maladaptive thought patterns based on irrational or non-realistic thinking that was absolute or judgmental influenced and intensified emotions and then shaped behaviors that also influenced or reinforced behaviors. Ellis proposed the A-B-C model that explained this sequence. Beck also proposed a triangle of core belief systems with negative perspectives on oneself, the world, and the future. Cognitive therapy seeks to identify these maladaptive cognitions (thoughts), appraisals, beliefs, and reactions, with the aim of reducing negative emotions, freeing the client to explore more healthy and adaptive behaviors and thought processes. CBT combines cognitive therapy and behavioral therapy to address maladaptive cognitions as well as dysfunctional behaviors using techniques from both models; this approach has demonstrated significant effectiveness for a wide variety of mental disorders and is in wide use today.
- **common factors and eclectic and integrative therapies**: although there is ongoing discussion and arguments about the effectiveness of different forms of therapy, decades of research have identified some common core features that seem to assist with healing and positive outcomes regardless of the theory or methods used. These include a positive and trusting relationship with a therapist, relevant and focused treatment approaches, enhancing hope and the client's belief that change is possible, and use of novel methods that the client has not used before. Many practitioners today are using an eclectic approach, meaning they combine aspects of multiple types of therapeutic methods targeting specific symptoms. Technical eclecticism does this without adopting any coherent or integrated ways of explaining a theory behind the patient's symptoms. On the other hand, integrative therapies have been specifically designed to blend different therapy models, as well as their methods, with the result of a coherent approach to treatment that uses methods from many different approaches.



Figure 3.20.1: Group therapy is a great way for individuals to connect with others who may share similar experiences.

We also explored different treatment modalities or treatment environments where psychotherapy models and techniques are used. All these involve, at minimum, an intake session where the clinician assesses the client's background and concerns and comes up with a treatment plan; this often involves a referral to another therapist who will carry out the actual treatment. These modalities include the following:

- **individual therapy**: one-on-one sessions between a client and a therapist
- **group therapy**: in this type of social therapy, one or more therapists treat a small group of clients together as a group (Figure 3.20.1)
- **psychoeducation groups**: a specialized form of group therapy that involves presentation of educational information to assist in coping and adapting to stressful situations
- **couples therapy**: two people in an intimate relationship working with a single therapist, focusing on enhancing and improving their relationship or deciding to terminate the relationship
- **family therapy**: a specialized form of group therapy focusing on a family unit where all members of the family are involved; the goal is to enhance relationships, improve communication, and clarify boundaries and structures within the family.
- **larger treatment contexts or environments**: we reviewed multiple contexts in which psychotherapies of various modalities are used, including inpatient hospitalization, partial hospitalization, residential treatment centers, outpatient therapy, and forensic units.]

Last, we explored the significant impact of sociocultural influences on a person's functioning. This included understanding that each person's identity consists of individual, group, and universal characteristics and how effective treatment must be grounded in the therapist understanding their own identity and biases as well as seeking to understand the client's identity and how cultural variables might shape their decisions, emotions, and behaviors. This includes recognizing individualist and collectivist elements of culture and requires specialized training for therapists to be culturally competent. Expectations of professional cultural competence now are part of the ethical codes of clinical disciplines. It is also important to understand and seek to reduce structural social barriers to treatment including racism, discrimination, access to care, health insurance, and the impact of social stigma on people's desire to seek treatment.

Having completed this module, you now have a much better understanding of the complexity of how mental disorders develop as well as the variety of theories and their connected treatment methods that exist today. You have an understanding of where those treatments are given and the importance of cultural competence. You are ready to start learning about specific disorders.

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CHAPTER OVERVIEW

4: Anxiety Disorders

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4.13: Putting It Together- Anxiety Disorders

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4.1: Why It Matters- Anxiety Disorders

Why learn about anxiety disorders?

What is anxiety? Most of us feel some anxiety almost every day of our lives. Maybe you have an important test coming up for school. Or maybe there's that big game next Saturday, or that first date with someone new you are hoping to impress. Anxiety can be defined as a negative mood state that is accompanied by bodily symptoms such as increased heart rate, muscle tension, a sense of unease, and apprehension about the future (APA, 2013; Barlow, 2002).



Figure **4.1.1**: While everyone may experience some level of anxiety at one time or another, those with anxiety disorders experience it consistently and so intensely that it has a significantly negative impact on their quality of life. [Image: Bada Bing, https://goo.gl/aawyLi, CC BY-NC-SA 2.0, https://goo.gl/Toc0ZF]

Although anxiety is closely related to fear, the two states possess important differences. Fear involves an instantaneous reaction to an imminent threat, whereas anxiety involves apprehension, avoidance, and cautiousness regarding a potential threat, danger, or other negative event (Craske, 1999). While anxiety is unpleasant to most people, it is important to our health, safety, and well-being. Anxiety motivates us to take actions—such as preparing for exams, watching our weight, showing up to work on time—that enable us to avert potential future problems. Anxiety also motivates us to avoid certain things—such as running up debts and engaging in illegal activities—that could lead to future trouble. Most individuals' level and duration of anxiety approximates the magnitude of the potential threat they face. For example, suppose a student who came to the United States as a "Dreamer" (someone whose parents didn't lawfully immigrate) is concerned about the possibility of being unable to continue in the university program or of losing access to academic financial aid due to changes and litigation around the Deferred Action for Childhood Arrivals (DACA) program. This person likely would experience anxiety of greater intensity and duration than would a 21-year-old junior who entered college as a birthright citizen.

Some people experience anxiety that is excessive, persistent, and greatly out of proportion to the actual threat; if one's anxiety has a disruptive influence on one's life, this is a strong indicator that the individual is experiencing an anxiety disorder. Those with an anxiety disorder may become so overwhelmed and distracted by anxiety that they actually fail their test, fumble the ball, or spend the whole date fidgeting and avoiding eye contact. If anxiety begins to interfere in a person's life in a significant way, it is considered a disorder.

As a group, anxiety disorders are common: approximately 25–30% of the U.S. population meets the criteria for at least one anxiety disorder during their lifetime (Kessler et al., 2005). Also, these disorders appear to be much more common in women than they are in men; within a 12-month period, around 23% of women and 14% of men will experience at least one anxiety disorder (National Comorbidity Survey, 2007). Anxiety disorders are the most frequently occurring class of mental disorders and are often comorbid with each other and with other mental disorders (Kessler, Ruscio, Shear, & Wittchen, 2009).

The *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5), classifies anxiety disorders as panic disorder, agoraphobia, generalized anxiety disorder, specific phobia, social anxiety disorder (social phobia), separation anxiety disorder, selective mutism, substance/medication-induced anxiety disorder, and anxiety disorder due to another medical condition. Read on to learn more about these anxiety disorders.

Watch It

This CrashCourse video provides an overview of the major anxiety disorders we will learn about in this module. Note that the video also mentions obsessive-compulsive disorder, which we will cover in a separate section.



You can view the transcript for "OCD and Anxiety Disorders: Crash Course Psychology #29" here (opens in new window).

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4.2: Introduction to Generalized Anxiety Disorder and Panic Disorder

What you'll learn to do: describe the features and characteristics of generalized anxiety disorder and panic disorder



In this section, you will learn about anxiety disorders, which are a group of disorders in which a person experiences excessive, persistent, and distressing fear and anxiety that interferes with normal functioning. Anxiety disorders can be categorized as generalized anxiety, panic disorder, phobia, agoraphobia, social anxiety, and separation anxiety. Each category of anxiety has its own unique presentation of anxiety through a person's behavior and set of anxiety symptoms. In this first section, we'll learn about generalized anxiety disorder (a relatively continuous state of tension, apprehension, and dread) and panic disorder (when a person is suddenly overwhelmed by panic).

Watch It

Anxiety is ever-prevalent in today's world and often exacerbated by external factors such as the global pandemic during Covid-19. This video from MedCircle provides more information about anxiety, including what it is and what it isn't.



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4.3: Generalized Anxiety Disorder

Learning Objectives

- Describe the symptoms of generalized anxiety disorder
- Distinguish between a normal fear response and an anxiety disorder

Generalized Anxiety Disorder



Figure **4.3.1**: Worry is a defining feature of generalized anxiety disorder. (credit: Freddie Peña)

What makes a person with generalized anxiety disorder (GAD) worry more than the average person? Research shows that individuals with generalized anxiety disorder (GAD) are more sensitive and vigilant toward possible threats than people who are not anxious (Aikins & Craske, 2001; Barlow, 2002; Bradley, Mogg, White, Groom, & de Bono, 1999). This sensitivity may be related to early stressful experiences, which can lead to a view of the world as an unpredictable, uncontrollable, and even dangerous place. Some have suggested that people with generalized anxiety disorder (GAD) worry as a way to gain some control over these otherwise uncontrollable or unpredictable experiences and against uncertain outcomes (Dugas, Gagnon, Ladouceur, & Freeston, 1998). By repeatedly going through all the possible "what if?" scenarios in their mind, the person might feel less vulnerable to an unexpected outcome, giving him or her the sense that he or she has some control over the situation (Wells, 2002).

Others have suggested people with GAD worry as a way to avoid feeling distressed (Borkovec, Alcaine, & Behar, 2004). For example, Borkovec and Hu (1990) found that those who worried when confronted with a stressful situation had less physiological arousal than those who didn't worry, maybe because the worry distracted them in some way. The problem is, all this "what if?"-ing doesn't get the person any closer to a solution or an answer and, in fact, might take them away from important things they should be paying attention to in the moment, such as finishing an important project. Many of the catastrophic outcomes people with GAD worry about are very unlikely to happen, so when the catastrophic event doesn't materialize, the act of worrying gets reinforced (Borkovec, Hazlett-Stevens, & Diaz, 1999). For example, if a mother spends all night worrying about whether her teenage daughter will get home safe from a night out and the daughter returns home without incident, the mother could easily attribute her daughter's safe return to her successful "vigil." What the mother hasn't learned is that her daughter would have returned home just as safe if she had been focusing on the movie she was watching, rather than being preoccupied with worries. In this way, the cycle of worry is perpetuated, and, subsequently, people with GAD often miss out on many otherwise enjoyable events in their lives.

Symptoms of Generalized Anxiety Disorder

People with GAD display excessive anxiety or worry on most days for at least six months about a number of things such as personal health, work, social interactions, and everyday routine life circumstances. The fear and anxiety can cause significant problems in areas of their life, such as social interactions, school, and work.

GAD symptoms include

- feeling restless, wound-up, or on-edge
- being easily fatigued
- having difficulty concentrating; mind going blank
- being irritable
- having muscle tension
- difficulty controlling feelings of worry
- having sleep problems, such as difficulty falling or staying asleep, restlessness, or unsatisfying sleep

? Alex: A narrative of worry

Alex was always worried about many things. He worried that his children would drown when they played at the beach. Each time he left the house, he worried that an electrical short circuit would start a fire in his home. He worried that his wife would lose her job at a prestigious law firm. He worried that his daughter's minor staph infection could turn into a massive life-threatening condition. These and other worries constantly weighed heavily on Alex's mind, so much so that they made it difficult for him to make decisions and often left him feeling tense, irritable, and worn out. One night, Alex's wife was to drive their son home from a soccer game. However, his wife stayed after the game and talked with some of the other parents, resulting in her arriving home 45 minutes late. Alex had tried to call her cell phone three or four times, but he could not get through because the soccer field did not have a signal. Extremely worried, Alex eventually called the police, convinced that his wife and son had not arrived home because they had been in a terrible car accident.

Alex suffers from **generalized anxiety disorder (GAD):** a relatively continuous state of excessive, uncontrollable, and pointless worry and apprehension. People with generalized anxiety disorder often worry about routine, everyday things, even though their concerns are unjustified. For example, an individual may worry about her health and finances, the health of family members, the safety of her children, or minor matters (e.g., being late for an appointment) without having any legitimate reason for doing so (APA, 2013).

Risk Factors for Anxiety

Researchers are finding that both genetic and environmental factors contribute to the risk of developing an anxiety disorder. Although there have been few investigations aimed at determining the heritability of generalized anxiety disorder, a summary of available family and twin studies suggests that genetic factors play a modest role in the disorder (Hettema et al., 2001). The risk factors for each type of anxiety disorder can vary, some general risk factors for all types of anxiety disorders include the following:

- temperamental traits of shyness or behavioral inhibition in childhood



Figure **4.3.2**: Shyness is a risk factor for some anxiety disorders.

- exposure to stressful and negative life or environmental events in early childhood or adulthood
- a history of anxiety or other mental illnesses in biological relatives
- some physical health conditions, such as thyroid problems or heart arrhythmias, or caffeine or other substances/medications, can produce or aggravate anxiety symptoms; a physical health examination is helpful in the evaluation of a possible anxiety disorder

Diagnosis of Generalized Anxiety Disorder

A diagnosis of GAD requires that the diffuse worrying and apprehension characteristic of this disorder—what Sigmund Freud referred to as free-floating anxiety—is not part of another disorder, occurs more days than not for at least six months, and is accompanied by any three of the following symptoms: restlessness, difficulty concentrating, being easily fatigued, muscle tension, irritability, and sleep difficulties.

About 5.7% of the U.S. population will develop symptoms of GAD during their lifetime (Kessler et al., 2005), and females are twice as likely as males to experience the disorder (APA, 2013). GAD is highly comorbid with mood disorders and other anxiety disorders (Noyes, 2001), and it tends to be chronic. Also, generalized anxiety disorder appears to increase the risk for heart attacks and strokes, especially in people with pre-existing heart conditions (Martens et al., 2010).

Watch It

Watch this video to learn about the causes, symptoms, and treatments for GAD.



You can view the transcript for "Generalized anxiety disorder (GAD) – causes, symptoms & treatment" here (opens in new window).

Watch It

This video explains Tasha's experience with GAD and how treatment helped her to identify and cope with her anxiety.



You can view the transcript for "Generalized Anxiety Disorders" here (opens in new window).

Cognitive Theory

Explaining GAD Diagnosis

Some people who are cognitive theorists suggest that worry represents a mental strategy to avoid more powerful negative emotions that perhaps may stem from earlier unpleasant or traumatic experiences (Aikins & Craske, 2001). Indeed, one longitudinal study found that childhood maltreatment was strongly related to the development of this disorder during adulthood (Moffitt et al., 2007); worrying might distract people from remembering painful childhood experiences.

Treatment for GAD

Both psychotherapies, predominately cognitive-behavioral therapy (CBT), and medications (such as SSRIs) have been shown to be effective in reducing anxiety. A comparison of overall outcomes of CBT and medication on anxiety did not show statistically significant differences (i.e., they were equally effective in treating anxiety).

To combat the cognitive and emotional aspects of GAD, psychologists often include some of the following key treatment components in their intervention plan: self-monitoring, relaxation techniques, self-control desensitization, gradual stimulus control, cognitive restructuring, worry outcome monitoring, present-moment focus, expectancy-free living, problem-solving techniques, processing of core fears, socialization, discussion and reframing of worry beliefs, emotional skills training, experiential exposure, psychoeducation, mindfulness, and acceptance exercises.

? Daily experiences of a person with GAD

- **Before Treatment**: "I was worried all the time and felt nervous. My family told me that there were no signs of problems, but I still felt upset. I dreaded going to work because I couldn't keep my mind focused. I was having trouble falling asleep at night and was irritated at my family all the time."
- **After Treatmen**t: "I saw my doctor and explained my constant worries. My doctor sent me to someone who knows about GAD. Now I am working with a counselor to cope better with my anxiety. I had to work hard, but I feel better. I'm glad I made that first call to my doctor."

Glossary

generalized anxiety disorder (GAD): characterized by a continuous state of excessive, uncontrollable, and pointless worry and apprehension

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4.4: Panic Disorder

Learning Objectives

- Explain the signs and symptoms of panic disorder, including panic attacks
- Describe agoraphobia
- Examine the etiology of panic disorder

Have you ever gotten into a near-accident or been taken by surprise in some way? You may have felt a flood of physical sensations, such as a racing heart, shortness of breath, or tingling sensations. This type of physiological reaction is called the *fight-or-flight response* (Cannon, 1929) and is your body's natural reaction to fear, preparing you to either fight or escape in response to threat or danger. It is likely you are not too concerned with these sensations because you knew what was causing them. But imagine if this alarm reaction came out of the blue, for no apparent reason, or in a situation in which you did not expect to be anxious or fearful. This sudden onset is considered an unexpected panic attack or a false alarm. Because there is no apparent reason or cue for the alarm reaction, you might react to the sensations with intense fear, maybe thinking you are having a heart attack, going crazy, or even dying. You might begin to associate the physical sensations you felt during this attack with this fear and may start to go out of your way to avoid having those sensations again.

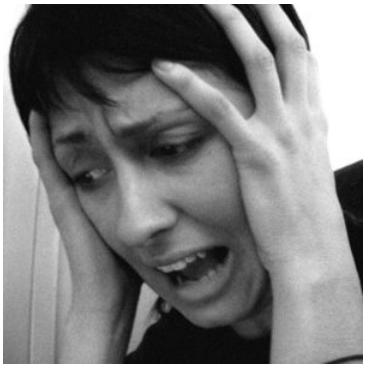


Figure **4.4.1**: Panic disorder is a debilitating condition that leaves sufferers with acute anxiety that persists long after a specific panic attack has subsided. When this anxiety leads to deliberate avoidance of particular places and situations a person may be given a diagnosis of agoraphobia. [Image: Nate Steiner, https://goo.gl/dUYWDf, Public Domain]

Panic Disorder

Unexpected panic attacks such as these are at the heart of **panic disorder (PD)**. However, to receive a diagnosis of PD, a person must not only have unexpected panic attacks but also must experience continued intense anxiety and avoidance related to the attack for at least one month, causing significant distress or interference in their lives. People with panic disorder tend to interpret even normal physical sensations in a catastrophic way, which triggers more anxiety and, ironically, more physical sensations, creating a vicious cycle of panic (Clark, 1986, 1996). The person may begin to avoid a number of situations or activities that produce the same physiological arousal that was present during the beginnings of a panic attack. For example, someone who experienced a racing heart during a panic attack might avoid exercise or caffeine. Someone who experienced choking sensations might avoid wearing high-necked sweaters or necklaces. Avoidance of these internal bodily or somatic cues for panic has been termed interoceptive avoidance (Barlow & Craske, 2007; Brown, White, & Barlow, 2005; Craske & Barlow, 2008; Shear et al., 1997).

Panic Attacks

People with panic disorder experience recurrent (more than one) and unexpected panic attacks, along with at least one month of persistent concern about additional panic attacks, worry over the consequences of the attacks, or self-defeating changes in behavior related to the attacks (e.g., avoidance of exercise or unfamiliar situations) (APA, 2013). As is the case with other anxiety disorders, the panic attacks cannot result from the physiological effects of drugs and other substances, a medical condition, or another mental disorder. A **panic attack** is defined as a period of extreme fear or discomfort that develops abruptly and reaches a peak within 10 minutes. Its symptoms include accelerated heart rate, sweating, trembling, choking sensations, hot flashes or chills, dizziness or lightheadedness, fears of losing control or going crazy, and fears of dying (APA, 2013). Sometimes panic attacks are expected,

occurring in response to specific environmental triggers (such as being in a tunnel); other times, these episodes are unexpected and emerge randomly (such as when relaxing). According to the DSM-5, the person must experience unexpected panic attacks to qualify for a diagnosis of panic disorder.

Experiencing a panic attack is terrifying. Rather than recognizing the symptoms of a panic attack merely as signs of intense anxiety, individuals with panic disorder often misinterpret them as a sign that something is intensely wrong internally (thinking, for example, that the pounding heart represents an impending heart attack). Panic attacks can occasionally precipitate trips to the emergency room because several symptoms of panic attacks are, in fact, similar to those associated with heart problems (e.g., palpitations, racing pulse, and a pounding sensation in the chest) (Root, 2000). Unsurprisingly, those with panic disorder fear future attacks and may become preoccupied with modifying their behavior in an effort to avoid future panic attacks. For this reason, panic disorder is often characterized as a fear of fear (Goldstein & Chambless, 1978).

Panic attacks themselves are not mental disorders and are fairly common in the United States. Approximately 23% of Americans experience isolated panic attacks in their lives without meeting the diagnostic criteria for panic disorder (Kessler et al., 2006). Panic disorder is, of course, much less common, afflicting 4.7% of Americans during their lifetime (Kessler et al., 2005). Many people with panic disorder develop agoraphobia, where they experience fear and avoidance of situations where it is difficult to or to access help during a panic attack. In addition, people with panic disorder often have another mental health disorder, a comorbid disorder, such as other anxiety disorders or a major depressive disorder (APA, 2013).

? Causes of Panic Disorder

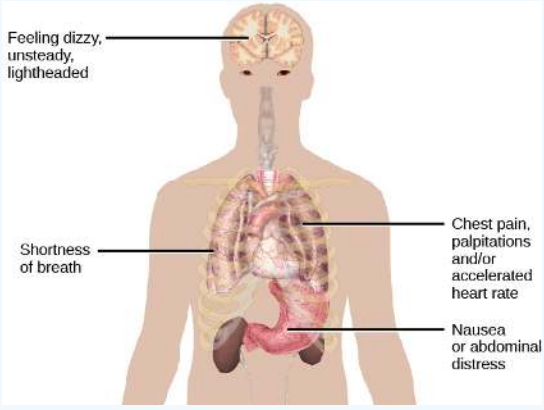


Figure 4.4.2: Some of the physical manifestations of a panic attack are shown. People may also experience sweating, trembling, feelings of faintness, or a fear of losing control, among other symptoms.

Researchers are not entirely sure what causes panic disorder. Children are at a higher risk of developing panic disorder if their parents have the disorder (Biederman et al., 2001), and family and twins studies indicate that the heritability of panic disorder is around 43% (Hettema, Neale, & Kendler, 2001). The exact genes and gene functions involved in this disorder, however, are not well understood (APA, 2013). Neurobiological theories of panic disorder suggest that a region of the brain called the locus coeruleus may play a role in this disorder. Located in the brainstem, the **locus coeruleus** is the brain's major source of norepinephrine, a neurotransmitter that triggers the body's fight-or-flight response. Activation of the locus coeruleus is associated with anxiety and fear, and research with nonhuman primates has shown that stimulating the locus coeruleus either electrically or through drugs produces panic-like symptoms (Charney et al., 1990). Previous findings have led to the theory that panic disorder may be caused by abnormal norepinephrine activity in the locus coeruleus (Bremner, Krystal, Southwick, & Charney, 1996).

Conditioning theories of panic disorder propose that panic attacks are classically conditioned responses to subtle bodily sensations resembling those normally occurring when one is anxious or frightened (Bouton, Mineka, & Barlow, 2001). For example, consider a child who has asthma. An acute asthma attack produces sensations, such as shortness of breath, coughing, and chest tightness, that typically elicit fear and anxiety. Later, when the child experiences subtle symptoms that resemble the frightening symptoms of earlier asthma attacks (such as shortness of breath after climbing stairs), he may become anxious, fearful, and then experience a panic attack. In this situation, the subtle symptoms would represent a conditioned stimulus, and the panic attack would be a conditioned response. The finding that panic disorder is nearly three times as frequent among people with asthma as it is among

people without asthma (Weiser, 2007) supports the possibility that panic disorder has the potential to develop through classical conditioning.

Cognitive factors may play an integral part in panic disorder. Generally, cognitive theories (Clark, 1996) argue that those with panic disorder are prone to interpret ordinary bodily sensations catastrophically, and these fearful interpretations set the stage for panic attacks. For example, a person might detect bodily changes that are routinely triggered by innocuous events such as getting up from a seated position (dizziness), exercising (increased heart rate, shortness of breath), or drinking a large cup of coffee (increased heart rate, trembling). The individual interprets these subtle bodily changes catastrophically ("Maybe I'm having a heart attack!"). Such interpretations create fear and anxiety, which trigger additional physical symptoms; subsequently, the person experiences a panic attack. Support of this contention rests with findings that people with more severe catastrophic thoughts about sensations have more frequent and severe panic attacks, and among those with panic disorder, reducing catastrophic cognitions about their sensations is as effective as medication in reducing panic attacks (Good & Hinton, 2009).

Watch It

Watch this video for an overview of panic disorder, including its causes, symptoms, and treatment.



You can view the transcript for "Panic disorder – panic attacks, causes, symptoms, diagnosis, treatment & pathology" here (opens in new window).

Agoraphobia



Figure 4.4.2: An ancient Roman agora in Tyre, Lebanon. This is one of the public spaces after which the condition agoraphobia is named.

An individual may also have experienced an overwhelming urge to escape during the unexpected panic attack. This can lead to a sense that certain places or situations—particularly situations where escape might not be possible—are not safe. These situations

become external cues for panic. If the person begins to avoid several places or situations, or still endure these situations but does so with a significant amount of apprehension and anxiety, then the person also has agoraphobia (Barlow, 2002; Craske & Barlow, 1988; Craske & Barlow, 2008). **Agoraphobia** can cause significant disruption to a person's life, causing them to go out of their way to avoid situations, such as adding hours to a commute to avoid taking the train or only ordering take-out to avoid having to enter a grocery store. In one tragic case seen by our clinic, a woman suffering from agoraphobia had not left her apartment for 20 years and had spent the past 10 years confined to one small area of her apartment, away from the view of the outside. In some cases, agoraphobia develops in the absence of panic attacks, and therefore is a separate disorder in DSM-5. However, agoraphobia often accompanies panic disorder.

Agoraphobia, which literally means "fear of the marketplace," is characterized by intense fear, anxiety, and avoidance of situations in which it might be difficult to escape or receive help if one experiences symptoms of a panic attack. These situations include public transportation, open spaces (parking lots), enclosed spaces (stores), crowds, or being outside the home alone (APA, 2013). About 1.4% of Americans experience agoraphobia during their lifetime (Kessler et al., 2005).

Treatment for Panic Disorder

Panic disorder is generally treated with psychotherapy, medication, or both. A type of psychotherapy called cognitive-behavioral therapy (CBT) is especially useful as a first-line treatment for panic disorder. CBT teaches different ways of thinking, behaving, and reacting to the feelings that come on with a panic attack, which can begin to disappear once a patient learns to react differently to the physical sensations of anxiety and fear that occur during panic attacks.

Some types of medications to help treat panic disorder include

- selective serotonin reuptake inhibitors (SSRIs),
- serotonin-norepinephrine reuptake inhibitors (SNRIs),
- beta-blockers, and
- benzodiazepines.

SSRIs and SNRIs are commonly used to treat depression, but they are also helpful for the symptoms of panic disorder. They may take several weeks to start working or may also cause side effects, such as headaches, nausea, or difficulty sleeping. Beta-blockers can help control some of the physical symptoms of panic disorder, such as rapid heart rate. Although doctors do not commonly prescribe beta-blockers for panic disorder, they may be helpful in certain situations that precede a panic attack. Benzodiazepines, which are sedative medications, are powerfully effective in rapidly decreasing panic attack symptoms, but they can cause tolerance and dependence if used continuously.

Narrative of Panic Disorder: Stress and the Loop of Anxiety

One day at a business conference, Colin Bien suddenly began to feel a surge of panic. He felt like he could not breathe, his body was on fire, and his heart was pounding. The sudden onset of these physiological symptoms was his first panic attack. As an ambitious graduate student, Colin was often stressed. He tried his best to be a part of a productive society that aimed to be "better, faster, and stronger." After the onset of his initial panic attack, Colin suffered from panic disorder for three years. He describes the experience of a panic attack as, "You typically realize them through a combination of three things: uncomfortable physical feelings, upsetting thoughts, and distressing emotions," which leads to anxiety and the fear that this panic attack will happen again (TED, 2017, 3:42). He describes this loop of anxiety and physical symptoms as a force of change in behavior due to "the fear of fear" (TED, 2017, 4:33). This change in behavior resulted in agoraphobia in which he avoided certain places or situations that could lead to a panic attack.

When Colin began behavioral therapy, he, alongside his therapist, examined the role of stress in his life, his routines and their contribution to his quality of life and stress level, and the loop of anxiety and stress. He looked at his routines through the lens of the patterns of his panic attacks and made positive lifestyle choices, like decreasing caffeine, eliminating alcohol and smoking, and starting to burn stress through running marathons (TED, 2017, 7:37). Now, Colin still has to monitor his routines, but with the combination of medication and behavioral therapy, he no longer experiences panic attacks.[1]

You can listen to Colin talk about his experiences in this TED talk.

Glossary

agoraphobia: anxiety disorder characterized by intense fear, anxiety, and avoidance of situations in which it might be difficult to escape if one experiences symptoms of a panic attack

anxiety disorder: characterized by excessive and persistent fear and anxiety and by related disturbances in behavior

locus coeruleus: area of the brainstem that contains norepinephrine, a neurotransmitter that triggers the body's fight-or-flight response; has been implicated in panic disorder

panic attack: period of extreme fear or discomfort that develops abruptly; symptoms of panic attacks are both physiological and psychological

panic disorder: anxiety disorder characterized by unexpected panic attacks, along with at least one month of worry about panic attacks or self-defeating behavior related to the attacks

1. TED. (2017, January 18). Colin Bien: Breaking The Loop of Anxiety [Video]. YouTube. https://www.youtube.com/watch?time_continue=215&v=DSLTuelMgRw&feature=emb_logo↵

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4.5: Introduction to Specific Phobias, Social Anxiety Disorder, and Separation Anxiety Disorder

What you'll learn to do: describe the features and characteristics of specific phobias, social anxiety disorder, and separation anxiety disorder



In this section, we'll examine more types of anxiety disorders, including specific phobias such as arachnophobia (fear of spiders) or ophidiophobia (fear of snakes) and anxiety connected to social situations (similar to agoraphobia) such as social anxiety and separation anxiety. We'll specifically learn about each of the following anxiety disorders:

- phobia: specific unrealistic fear
- social anxiety: extreme fear and avoidance of social situations
- separation anxiety: fear and discomfort when separated from an attached caregiver

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4.6: Specific Phobia

Learning Objectives

- Explain specific phobias
- Describe the etiology of specific phobias

Specific Phobia

Phobia is a Greek word that means "fear." A person diagnosed with a **specificphobia** (formerly known as simple **phobia**) experiences excessive, distressing, and persistent fear or anxiety about a specific object or situation (such as animals, enclosed spaces, elevators, or flying) (APA, 2013). Even though people realize their level of fear and anxiety in relation to the phobic stimulus is irrational, some people with a specific phobia may go to great lengths to avoid the phobic stimulus (the object or situation that triggers the fear and anxiety). Typically, the fear and anxiety a phobic stimulus elicits is disruptive to the person's life. For example, a person with a phobia of flying might refuse to accept a job that requires frequent air travel, thus negatively affecting their career. Clinicians who have worked with people who have specific phobias have encountered many kinds of phobias, some of which are shown in Table 1.

Table 1. Specific Phobias

Phobia	Feared Object or Situation
acrophobia	heights
aerophobia	flying
arachnophobia	spiders
claustrophobia	enclosed spaces
cynophobia	dogs
hematophobia	blood
ophidiophobia	snakes
taphophobia	being buried alive
trypanophobia	injections
xenophobia	strangers

Types of Phobias

The list of possible phobias is staggering, but four major subtypes of specific phobia are recognized: blood-injury-injection (BII) type, situational type (such as planes, elevators, or enclosed places), natural environment type for events one may encounter in nature (for example, heights, storms, and water), and animal type. A fifth category "other" includes phobias that do not fit any of the four major subtypes (for example, fears of choking, vomiting, or contracting an illness).

Most phobic reactions cause a surge of activity in the sympathetic nervous system, increased heart rate, and blood pressure, and possibly a panic attack. However, people with blood-injury-injection (BII) type phobias usually experience a marked drop in heart rate and blood pressure and may even faint. In this way, those with blood-injury-injection (BII) phobias almost always differ in their physiological reaction from people with other types of phobia (Barlow & Liebowitz, 1995; Craske, Antony, & Barlow, 2006; Hofmann, Alpers, & Pauli, 2009; Ost, 1992). BII phobia also runs in families more strongly than any phobic disorder we know (Antony & Barlow, 2002; Page & Martin, 1998). Specific phobia is one of the most common psychological disorders in the United States, with 12.5% of the population reporting a lifetime history of fears significant enough to be considered a phobia (Arrindell et al., 2003; Kessler, Berglund, et al., 2005) (see Table 1). Most people who suffer from a specific phobia tend to have multiple phobias of several types (Hofmann, Lehman, & Barlow, 1997).

Acquisition of Phobias through Learning

Many theories suggest that phobias develop through learning. Rachman (1977) proposed that phobias can be acquired through three major learning pathways. The first pathway is through **classical conditioning**. As you may recall, classical conditioning is a form of learning in which a previously neutral stimulus is paired with an unconditioned stimulus (UCS) that reflexively elicits an unconditioned response (UCR), eliciting the same response through its association with the unconditioned stimulus. The response is called a conditioned response (CR). For example, a child who has been bitten by a dog may come to fear dogs because of her past association with pain. In this case, the dog bite is the UCS and the fear it elicits is the UCR. Because a dog was associated with the bite, any dog may come to serve as a conditioned stimulus, thereby eliciting fear; the fear the child experiences around dogs, then, becomes a CR.

The second pathway of phobia acquisition is through vicarious learning, such as **modeling**. For example, a child who observes his cousin react fearfully to spiders may later express the same fears, even though spiders have never presented any danger to him. This phenomenon has been observed in both humans and nonhuman primates (Olsson & Phelps, 2007). A study of laboratory-reared monkeys readily acquired a fear of snakes after observing wild-reared monkeys react fearfully to snakes (Mineka & Cook, 1993).

The third pathway is through **verbal transmission** or information. For example, a child whose parents, siblings, friends, and classmates constantly tell her how disgusting and dangerous snakes are may come to acquire a fear of snakes.



Figure **4.6.1**: Even though the glass floor and glass walls prevent the child from falling, they might still be frightened by the height.

Interestingly, people are more likely to develop phobias of things that do not represent much actual danger to themselves, such as animals and heights, and are less likely to develop phobias toward things that present legitimate danger in contemporary society, such as motorcycles and weapons (Öhman & Mineka, 2001). Why might this be so? One theory suggests that the human brain is evolutionarily predisposed to more readily associate certain objects or situations with fear (Seligman, 1971). This theory argues that throughout our evolutionary history, our ancestors associated certain stimuli (e.g., snakes, spiders, heights, and thunder) with potential danger. As time progressed, the mind has become adapted to more readily develop fears of these things than of others. Experimental evidence has consistently demonstrated that conditioned fears develop more readily to fear-relevant stimuli (images of snakes and spiders) than to fear-irrelevant stimuli (images of flowers and berries) (Öhman & Mineka, 2001). Such prepared learning has also been shown to occur in monkeys. In one study (Cook & Mineka, 1989), monkeys watched videotapes of model monkeys reacting fearfully to either fear-relevant stimuli (toy snakes or a toy crocodile) or fear-irrelevant stimuli (flowers or a toy rabbit). The observer monkeys developed fears of the fear-relevant stimuli but not the fear-irrelevant stimuli.

Treatment

Cognitive-behavior therapy and exposure therapy are commonly used to treat specific phobias. CBT is a short-term, skills-focused therapy that aims to help people diffuse unhelpful emotional responses by helping people consider them differently or change their behavior and is effective in treating specific phobias. Exposure therapy is a particularly effective treatment method, as it consists of exposing a patient to the anxiety-inducing situation in manageable chunks. Medications to aid CBT have not been too promising, with the exception of adjunctive D-clycoserine.

Watch It

Watch this example of a woman dealing with specific phobias and her experience in facing her fears.



You can view the transcript for "Phobias" here (opens in new window).

Epidemiology

Specific phobias have a one-year prevalence of 8.7% in the United States with 21.9% of the cases being severe, 30.0% moderate, and 48.1% mild.[1] The usual age of onset is childhood to adolescence. Women are twice as likely to suffer from specific phobias than men.

Watch It

What's the difference between a specific phobia and panic disorder? This video with Dr. Judith Beck and Dr. Aaron Beck (the founder of cognitive therapy and CBT) describes the distinction between a specific phobia and panic disorder through the description of two patients. The first patient is afraid of flying and experiences a panic attack. The second patient is afraid of flying but also implements a **safety behavior**, an act designed to reduce anxiety in social situations by reducing the chance of negative outcomes.



You can view the transcript for "Specific Phobia Versus Panic Disorder in CBT" here (opens in new window).

Glossary

safety behavior: a coping strategy designed to reduce anxiety in social situations by reducing the chance of negative outcomes

specific phobia: an anxiety disorder characterized by excessive, distressing, and persistent fear or anxiety about a specific object or situation

1. Kessler, PhD, Ronald; Chiu, AM, Wai Tat; Demler, Olga; Walters, Ellen (2005). "Prevalence, Severity and Comorbidity of 12-Month DSM-IV Disorders in the National Comorbidity Survey Replication." Archives of General Psychiatry. 62 (6): 617–709.

doi:10.1001/archpsyc.62.6.617. PMC 2847357. PMID 15939839 ↵

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4.7: Social Anxiety Disorder

Learning Objectives

- Describe the symptoms of social anxiety disorder
- Examine the etiology of social anxiety disorder

Social Anxiety Disorder

Social anxiety disorder (formerly called social phobia) is characterized by extreme and persistent fear or anxiety and avoidance of social situations in which the person could potentially be evaluated negatively by others (APA, 2013). As with specific phobias, social anxiety disorder is common in the United States; a little over 12% of all Americans experience social anxiety disorder during their lifetime (Kessler et al., 2005).

Diagnosing Social Anxiety Disorder



Figure **4.7.1**: Social anxiety disorder can cause people to miss even the most important events.

Many people consider themselves shy, and most people find social evaluation uncomfortable at best, or giving a speech somewhat mortifying. Yet, only a small proportion of the population fear these types of situations significantly enough to merit a diagnosis of social anxiety disorder (APA, 2013). Social anxiety disorder is more than exaggerated shyness (Bogels et al., 2010; Schneier et al., 1996). To receive a diagnosis of social anxiety disorder, the fear and anxiety associated with social situations must be so strong that the person avoids them entirely, or if avoidance is not possible, the person endures them with a great deal of distress. Further, the fear and avoidance of social situations must get in the way of the person's daily life or seriously limit their academic or occupational functioning. For example, a patient at one clinic compromised her perfect 4.0 grade point average because she could not complete a required oral presentation in one of her classes, causing her to fail the course. Fears of negative evaluation might make someone repeatedly turn down invitations to social events or avoid having conversations with people, leading to greater and greater isolation.

The heart of the fear and anxiety for a person with social anxiety disorder is that they may act in a humiliating or embarrassing way, appear foolish; show symptoms of anxiety (blushing), or do or say something that might lead to rejection (such as offending others). The kinds of social situations in which individuals with social anxiety disorder usually have problems include public speaking, having a conversation, meeting strangers, eating in restaurants, and, in some cases, using public restrooms.

A person with social anxiety disorder fears any situation that could potentially draw attention to the person. For example, one patient went out of her way to avoid any situation in which she might have to use a public restroom for fear that someone would hear her in the bathroom stall and think she was disgusting. If the fear is limited to performance-based situations, such as public speaking, a diagnosis of social anxiety disorder-performance only is assigned.

Although many people become anxious in social situations like public speaking, the fear, anxiety, and avoidance experienced in social anxiety disorder are highly distressing and lead to serious impairments in life. For example, adults with social anxiety disorder are more likely to experience lower educational attainment and lower earnings (Katzelnick et al., 2001); perform more poorly at work; be unemployed (Moitra, Beard, Weisberg, & Keller, 2011); and report greater a dissatisfaction with their family lives, friends, leisure activities, and income (Stein & Kean, 2000).

Safety Behaviors

When people with social anxiety disorder are unable to avoid situations that provoke anxiety, they typically perform **safety behaviors**: mental or behavioral acts that reduce anxiety in social situations by reducing the chance of negative social outcomes.

Safety behaviors include, but are not limited to avoiding eye contact, rehearsing sentences before speaking, talking only briefly, and not talking about oneself (Alden & Bieling, 1998). Other examples of safety behaviors include the following (Marker, 2013):

- assuming roles in social situations that minimize interaction with others (e.g., taking pictures, setting up equipment, or helping prepare food)
- asking people many questions to keep the focus off of oneself
- selecting a position to avoid scrutiny or contact with others (sitting in the back of the room)
- wearing bland, neutral clothes to avoid drawing attention to oneself
- avoiding substances or activities that might cause anxiety symptoms (such as caffeine, warm clothing, and physical exercise)



Figure **4.7.2**: Panic attacks can occur abruptly and cause symptoms like heart palpitations, trembling, and shortness of breath.

Although these behaviors are intended to prevent the person with social anxiety disorder from doing something awkward that might draw criticism, these actions usually exacerbate the problem because they do not allow the individual to disconfirm his or her negative beliefs, often eliciting rejection and other negative reactions from others (Alden & Bieling, 1998).

A person might react so strongly to the anxiety provoked by a social situation that they have an unexpected panic attack. This panic attack then becomes associated (conditioned response) with the social situation, causing the person to fear he or she will panic the next time he or she is in that situation. This response is not considered panic disorder, however, because the person's fear is more focused on their social evaluation than having unexpected panic attacks, and the fear of having an attack is limited to social situations. As many as 12.1% of the general population suffer from a social phobia at some point in their lives (Kessler, Berglund, et al., 2005), making it one of the most common anxiety disorders, second only to specific phobia.

People with social anxiety disorder may resort to self-medication, such as drinking alcohol, as a means to avert the anxiety symptoms they experience in social situations (Battista & Kocovski, 2010). The use of alcohol, when faced with such situations, may become negatively reinforcing—encouraging individuals with social anxiety disorder to turn to the substance whenever they experience anxiety symptoms. The tendency to use alcohol as a coping mechanism for social anxiety, however, can come with a hefty price tag: a number of large-scale studies have reported a high rate of comorbidity between social anxiety disorder and alcohol use disorder (Morris, Stewart, & Ham, 2005).

Watch It

Watch this video for an overview of social anxiety disorder, including symptoms and common treatment methods such as CBT and medications such as antidepressants, benzodiazepines, and beta-blockers.



You can view the transcript for "Social Anxiety Disorder – causes, symptoms, diagnosis, treatment, pathology" here (opens in new window).

Etiology of Social Anxiety Disorder

As with specific phobias, it is highly probable that the fears inherent to social anxiety disorder can develop through conditioned experiences. For example, children who are subjected to early unpleasant social experiences (e.g., bullying at school) may develop negative social images of themselves that become activated later in anxiety-provoking situations (Hackmann, Clark, & McManus, 2000). Indeed, one study reported that 92% of a sample of adults with social anxiety disorder reported a history of severe teasing in childhood, compared to only 35% of a sample of adults with panic disorder (McCabe, Antony, Summerfeldt, Liss, & Swinson, 2003).

One of the most well-established risk factors for developing social anxiety disorder is *behavioral inhibition* (Clauss & Blackford, 2012). Behavioral inhibition is thought to be an inherited trait, and it is characterized by a consistent tendency to show fear and restraint when presented with unfamiliar people or situations (Kagan, Reznick, & Snidman, 1988). Behavioral inhibition is displayed very early in life; behaviorally inhibited toddlers and children respond with great caution and restraint in unfamiliar situations, and they are often timid, fearful, and shy around unfamiliar people (Fox, Henderson, Marshall, Nichols, & Ghera, 2005). A recent statistical review of studies demonstrated that behavioral inhibition was associated with more than a sevenfold increase in the risk of development of social anxiety disorder, demonstrating that behavioral inhibition is a major risk factor for the disorder (Clauss & Blackford, 2012).

Link to Learning

Watch this TedxYouth talk to hear the experiences from 9th-grader Marielle Cornes as she deals with social anxiety disorder.

Glossary

behavioral Inhibition: an inherited trait characterized by a consistent tendency to show fear and restraint when they are faced with a new social interaction or situation

social anxiety disorder: characterized by extreme and persistent fear or anxiety and avoidance of social situations in which one could potentially be evaluated negatively by others

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4.8: Separation Anxiety and Selective Mutism

Learning Objectives

- Describe separation anxiety disorder
- Explain selective mutism



Figure 4.8.1: A child with separation anxiety may refuse to be apart from his or her mother.

Separation anxiety disorder is a disorder in which an individual experiences excessive anxiety regarding separation from home and/or from people to whom the individual has a strong emotional attachment (e.g., a parent, a caregiver, a significant other, or siblings) called the **attachment figure**. It is most common in infants and small children, typically between the ages of six to seven months to three years, although it may pathologically manifest itself in older children, adolescents, and adults.

Separation anxiety is a natural part of the developmental process. Unlike separation anxiety disorder (indicated by excessive anxiety), normal separation anxiety indicates healthy advancements in a child's cognitive maturation and should not be considered a developing behavioral problem.

According to the APA, separation anxiety disorder is an excessive display of fear and distress when faced with situations of separation from the home and/or from a specific attachment figure. The anxiety that is expressed is categorized as being atypical of the expected developmental level and age. The severity of the symptoms ranges from anticipatory uneasiness to full-blown anxiety about separation. Individuals with separation anxiety disorder demonstrate avoidance behaviors. Individuals "typically exhibit excessive distress manifested by crying, repeated complaints of physical symptoms (e.g., stomachaches, headaches, etc.), avoidance (e.g., refusing to go to school, to sleep alone, to be left alone in the home, to engage in social events, to go to work, etc.), and engagement in safety behaviors (e.g., frequent calls to or from significant others, or primary caregivers)."[1]

Separation anxiety disorder may cause significant negative effects within areas of social and emotional functioning, family life, and physical health of the disordered individual.

Diagnosis of Separation Anxiety Disorder

The duration of this problem must persist for at least four weeks and must present itself before a child is eighteen years of age to be diagnosed as a separation anxiety disorder in children, but can now be diagnosed in adults with a duration typically lasting six months in adults as specified by the DSM-5.

As mentioned, separation anxiety is normal in young children, until they age three to four years, when children are left in a daycare or preschool away from their parent or primary caregiver. To be diagnosed with separation anxiety disorder, one must display at least three of the following criteria:

- recurrent excessive distress when anticipating or experiencing separation from home or from major attachment figures
- persistent and excessive worry about losing major attachment figures or about possible harm to oneself, such as illness, injury, disasters, or death
- persistent and excessive worry about experiencing an untoward event (e.g., getting lost, being kidnapped, having an accident, becoming ill) that causes separation from a major attachment figure

- persistent reluctance or refusal to go out, away from home, to school, to work, or elsewhere because of fear of separation
- persistent and excessive fear of or reluctance about being alone or without major attachment figures at home or in other settings
- persistent reluctance or refusal to sleep away from home or to go to sleep without being near a major attachment figure
- repeated nightmares involving the theme of separation
- repeated complaints of physical symptoms (e.g., headaches, stomachaches, nausea, vomiting) when separation from major attachment figures occurs or is anticipated

Epidemiology of Separation Anxiety Disorders

Anxiety disorders are the most common type of psychopathology to occur in today's youth, affecting from 5%-25% of children worldwide. Of these anxiety disorders, separation anxiety disorder accounts for a considerable proportion of diagnoses. Separation anxiety disorder may account for up to 50% of the anxiety disorders as recorded in referrals for mental health treatment.[2] Separation anxiety disorder is noted as one of the earliest-occurring of all anxiety disorders. Adult separation anxiety disorder affects roughly 7% of adults.

Research suggests that 4.1% of children will experience a clinical level of separation anxiety and 1.6% of adolescents. Of that 4.1%, it is calculated that nearly a third of all cases will persist into adulthood if left untreated. Research continues to explore the implications that early dispositions of separation anxiety disorder in childhood may serve as risk factors for the development of mental disorders throughout adolescence and adulthood. A higher percentage of children are presumed to suffer from a small amount of separation anxiety and are not actually diagnosed. Multiple studies have found higher rates of separation anxiety disorder in girls than in boys, and that paternal absence may increase the chances of separation anxiety disorder in girls.

One of the difficulties in the identification of separation anxiety disorder in children is that it is highly comorbid with other behavioral disorders, especially generalized anxiety disorder. Behaviors such as refusal or hesitancy in attending school or homesickness, for example, can easily reflect similar symptoms and behavioral patterns that are commonly associated with separation anxiety disorder but could be an overlap of symptoms. The prevalence of co-occurring disorders in adults with separation anxiety disorder is common and includes a much broader spectrum of diagnostic possibilities. Common comorbidities can include specific phobias, PTSD, panic disorder, obsessive-compulsive disorder, and personality disorders.[3]

Causes of Separation Anxiety Disorder: Risk Factors and Biological and Environmental Contributions

Factors that contribute to the disorder include a combination and interaction of biological, cognitive, environmental, child temperament, and behavioral factors.

Children are more likely to develop separation anxiety disorder if one or both of their parents was diagnosed with a psychological disorder.[4] Recent research by Daniel Schechter and colleagues have pointed to difficulties of mothers who have themselves had early adverse experiences such as maltreatment and disturbed attachments with their own caregivers, who then go on to develop responses to their infants' and toddlers' normative social bids in the service of social referencing, emotion regulation, and joint attention, which responses are linked to these mothers own psychopathology (i.e., maternal post-traumatic stress disorder (PTSD) and depression.)[5] These atypical maternal responses, which have been shown to be associated with separation anxiety, have been related to disturbances in maternal stress physiologic response to mother-toddler separation as well as lower maternal neural activity in the brain region of the medial prefrontal cortex when mothers with and without PTSD were shown video excerpts of their own and unfamiliar toddlers during mother-child separation versus free-play.[6]

Many psychological professionals have suggested that early or traumatic separation from a central caregiver in a child's life can increase the likelihood of them being diagnosed with separation anxiety disorder, school phobia, and depressive-spectrum disorders. Some children can be more vulnerable to separation anxiety disorder due to their temperament, for example, their level of anxiety when placed in new situations.

Environmental Causes

Most often, the onset of separation anxiety disorder is caused by a stressful life event, especially a loss of a loved one or pet, but can also include parental divorce, change of school or neighborhood, natural disasters, or circumstances that forced the individual to be separated from their attachment figure(s). In older individuals, stressful life experiences may include going away to college, moving out for the first time, or becoming a parent.

Genetic and Physiological Causes

There may be a genetic predisposition in children with separation anxiety disorder. Experts say, "Separation anxiety disorder in children may be heritable. . . . Heritability was estimated at 73% in a community sample of 6-year-old twins, with higher rates in girls."[7]

A child's temperament can also impact the development of separation anxiety disorder. Timid and shy behaviors may be referred to as "behaviorally inhibited temperaments" in which the child may experience anxiety when they are not familiar with a particular location or person.

Treatment

Non-Medication Based Treatment

Non-medication based treatments are the first choice when treating individuals diagnosed with separation anxiety disorder. Counseling tends to be the best replacement for drug treatments. There are two different non-medication approaches to treat separation anxiety. The first is a psychoeducational intervention, often used in conjunction with other therapeutic treatments. This specifically involves educating the individual and their family so that they are knowledgeable about the disorder, as well as parent counseling and guiding teachers on how to help the child. The second is a psychotherapeutic intervention when prior attempts are not effective. Psychotherapeutic interventions are more structured and include behavioral, cognitive-behavioral, contingency, psychodynamic psychotherapy, and family therapy.

Exposure and Behavioral Therapy

Behavioral therapies are types of non-medication based treatment that mainly consists of exposure-based techniques. These therapies include techniques such as systematic desensitization, emotive imagery, participant modeling, and contingency management. Behavioral therapies carefully expose individuals by small increments to slowly reduce their anxiety over time and mainly focuses on their behavior. Exposure-based therapy works under the principle of habituation that is derived from learning theory. The core concept of **exposure therapy** is that anxiety about situations, people, and things do not go away when people avoid the things that they fear, but rather, the uncomfortable feelings are simply kept at bay. In order to effectively diminish the negative feelings associated with the situation of fear, one must address them directly.

With separation anxiety disorder, the child may be encouraged to gradually separate from their caregiver (gradual exposure). They may begin by imagining this separation, work toward increasing separation within the therapy setting, and then progress to separating in real-world situations (e.g., school). In order to administer this treatment, the therapist and the anxious child might sit together and identify progressively intense situations. As each situation is dealt with masterfully, the child advances to the next phase of intensity. This pattern continues until the child is able to handle being away from their parent in a developmentally typical way that causes them and their caregiver(s) minimal amounts of stress. While there is some controversy about using exposure therapy with children, it is generally agreed upon that exposure therapy in the context of separation anxiety disorder is acceptable as it may be the most effective form of therapy in treating this disorder and there is minimal risk associated with the intervention in this context.



Figure **4.8.2**: The Anchors Away program utilizes a type of family-focused CBT to help children cope with anxiety, such as that experienced by military children with deployed parents.

Cognitive-Behavioral Therapy for Children

CBT focuses on helping children with separation anxiety disorder reduce feelings of anxiety through practices of exposure to anxiety-inducing situations and active metacognition to reduce anxious thoughts.

According to Kendall and colleagues, there are four components that must be taught to a child undergoing CBT:[8]

- recognizing anxious feelings and behaviors
- discussing situations that provoke anxious behaviors
- developing a coping plan with appropriate reactions to situations
- evaluating the effectiveness of the coping plan

In the application phase, individuals can take what they know and apply it in real-time situations for helpful exposure. The most important aspect of this phase is for the individuals to ultimately manage themselves throughout the process. In the relapse prevention phase, the individual is informed that continued exposure and application of what worked for them is the key to continual progress.

A study investigated the content of thoughts in anxious children who suffered from separation anxiety as well as from social anxiety or generalized anxiety. The results suggested that cognitive therapy for children suffering from separation anxiety (along with social phobia and generalized anxiety) should be aimed at identifying negative cognition of one's own behavior in the threat of anxiety-evoking situations and to modify these thoughts to promote self-esteem and ability to properly cope with the given situation.

Cognitive procedures are a form of treatment found to be ideal for older children with separation anxiety disorder. Cognitive procedures utilize techniques that the child's dysfunctional thoughts, attitudes, and beliefs are what lead to anxiety and cause anxious behavior. Children who are being treated with cognitive procedures are taught to ask themselves if there is evidence to support their anxious thoughts and behaviors. They are taught coping thoughts to replace previously distorted thoughts during anxiety-inducing situations, such as doing a reality check to assess the real danger of a situation and then to praise themselves for handling the situation bravely. Examples of such disordered thoughts include polarized thinking, overgeneralization, filtering (focusing on negative), jumping to conclusions, catastrophizing, emotional reasoning, labeling, "shoulds", and placing blame on self and others. Sometimes therapists will involve parents and teach them behavioral tactics such as contingency management.

Relaxation training is another way to combat anxiety. Similar to that in exposure-based treatment for phobias, prior to engaging in exposure training, the individual must learn a relaxation technique to apply during the onset of panic attacks. Deep breathing (control, slow, and purposeful breathing) and distraction (focusing on alternative things, grounding oneself to their senses) are commonly used strategies.

Contingency Management

Contingency management is a form of treatment found to be effective for younger children with separation anxiety disorder. Contingency management revolves around a reward system with verbal or tangible reinforcement requiring parental involvement. A contingency contract is written up between the parent and the child that entails a written agreement about specific goals that the child will try to achieve and the specific reward the parent will provide once the task is accomplished. When the child undergoing contingency management show signs of independence or achieve their treatment goals, they are praised or given their reward. Contingency management facilitates a new positive experience with what used to be filled with fear and anxiety. Children in preschool who show symptoms of separation anxiety disorder do not have the communicative ability to express their emotions or the self-control ability to cope with their separation anxiety on their own, so parental involvement is crucial in younger cases of separation anxiety disorder.

Medication

The use of medication is applied in extreme cases of separation anxiety disorder when other treatment options have been utilized and failed. However, research has been difficult to prove the benefits of drug treatment in patients with separation anxiety disorder because there have been many mixed results. Despite all the studies and tests, there has yet to be a specific medication for separation anxiety disorder. Medication prescribed for adults from the Food and Drug Administration (FDA) are often used and have been reported to show positive results for children and adolescents with separation anxiety disorder.

There are mixed results regarding the benefits of using tricyclic antidepressants (TCAs), which include imipramine and clomipramine. One study suggested that imipramine is helpful for children with school phobia who also had an underlying diagnosis of separation anxiety disorder. However, other studies have also shown that imipramine and clomipramine had the same effect of children who were treated with the medication and placebo. The most promising medication is the use of selective

serotonin reuptake inhibitors (SSRI) in adults and children. Several studies have shown that patients treated with fluvoxamine were significantly better than those treated with a placebo. Research has shown decreasing anxiety symptoms with short-term and long-term use of the medication.

Watch It

In the following video, Dr. Catchpole interviews fellow psychologist Dr. Jane Garland, specializing in separation anxiety phobias. She explains that when anxieties become too intense and too prolonged, they can cause problematic symptoms in three categories: body, mind, and behavior. Anxious children will have headaches, upset stomachs, muscle tension, and racing hearts. They will be more emotionally fragile, and maybe irritable, avoidant, and throw tantrums. As you watch it, consider how treatment for children with anxiety might differ from treatment for adults?



You can view the transcript for "Anxiety Disorders in Children and Teens" here (opens in new window).

Selective Mutism

Selective mutism (SM), also known as situational mutism, is an anxiety disorder in which a person normally capable of speech cannot speak in specific situations or to specific people if triggered. Selective mutism usually co-exists with social anxiety disorder. People with selective mutism stay silent even when the consequences of their silence include shame, social ostracism, or punishment.

Selective mutism is characterized by the following:[11]

- The consistent failure to speak in specific social situations (in which there is an expectation for speaking, e.g., at school) persists despite speaking in other situations.
- The disturbance interferes with educational or occupational achievement or with social communication.
- The duration of the disturbance is at least one month (not limited to the first month of school).
- The failure to speak is not due to a lack of knowledge of the spoken language required in the social situation.
- The disturbance is not better accounted for by a *communication* disorder (e.g., childhood-onset *fluency* disorder) and does not occur exclusively in people with autism spectrum disorders or psychotic disorders such as schizophrenia.

Watch It

In this video, Dr. Lindsey Bergman discusses the similarities differences between selective mutism and shyness in young children. The key difference between selective and shyness is that a child with selective mutism will not warm up over time, but a child with shyness will.



You can view the transcript for “Selective Mutism or is my child just shy? | R. Lindsey Bergman, PhD” here (opens in new window).

Selective mutism is strongly associated with other anxiety disorders, particularly social anxiety disorder. In fact, the majority of children diagnosed with selective mutism also have social anxiety disorder (100% of participants in two studies and 97% in another). Some researchers, therefore, speculate that selective mutism may be an avoidance strategy used by a subgroup of children with social anxiety disorder to reduce their distress in social situations.

Particularly in young children, selective mutism can sometimes be confused with an autism spectrum disorder diagnosis, especially if the child acts particularly withdrawn around their diagnostician, which can lead to incorrect diagnosis and treatment. Although people with autism may also be selectively mute, they often display other behaviors—hand flapping, repetitive behaviors, social isolation even among family members (not always answering to name, for example)—that set them apart from a child with selective mutism. Some people with autism may be selectively mute due to anxiety in social situations that they do not fully understand. If mutism is entirely due to autism spectrum disorder, it cannot be diagnosed as selective mutism as stated in the last item on the list above.

The former name *elective mutism* indicates a widespread misconception among psychologists that selective mute people choose to be silent in certain situations, while the truth is that they often wish to speak but are unable to do so. To reflect the involuntary nature of this disorder, the name was changed to *selective mutism* in 1994.

The incidence of selective mutism is not certain. Due to the poor understanding of this condition by the general public, many cases are likely undiagnosed. Based on the number of reported cases, the figure is commonly estimated to be one in 1,000, or around 0.1%.

Etiology

There are many theories to frame how children develop selective mutism across different psychological domains. Several are presented in the following table:[12]

Etiology	Description
psychodynamic theory	Mutism may be a coping mechanism caused by unresolved conflicts, possibly due to anger towards parents.
behavior theory	Mutism is a learned behavior and response to triggers in the environment.
social anxiety and phobia	Mutism could be an extreme form of excessive social anxiety.
family systems perspective	Mutism is caused by intense attachment and dependency that causes distrust of outsiders.
response to trauma	Mutism could be caused by PTSD or abuse.
dissociative identity disorder	Mutism could be a display of another identity.

Treatment

Treatment for selective mutism may rely on psychodynamic/play therapy, behavioral therapy, family therapy, or medications (most often antidepressants). Early treatment may help prevent the self-reinforcement of selective mutism. Self-modeling is a treatment method in which a child with the disorder is videotaped being asked questions by someone (such as a teacher), that they will not answer. Then they are videotaped and asked the same questions by a parent or someone they are comfortable speaking to, this time eliciting a verbal response. The two videos of the conversations are then edited together to show the child directly answering the questions posed by the teacher or other adult. This video is then shown to the child over a series of several weeks, and every time the child sees him- or herself verbally answering the teacher/another adult, the tape is stopped and the child is given positive reinforcement.

Watch It

Watch this video until the end of the first story, at the 4:13 mark. It highlights the experiences of Maya, a happy three-year-old girl who can speak but chooses to only speak to her immediate family. When other people are present, she only uses gestures and grunts. Starting at two years old, Maya started stopping talking to strangers, which gradually led to acquaintances and closer family, like grandparents. Maya illustrates how selective mutism affects her life where she can talk in some situations, but not others due to acute anxiety to talk.



You can view the transcript for "This Morning – Selective Mutism" here (opens in new window).

Link to Learning: Maya Angelou and Selective Mutism

Famous poet and author Maya Angelou wasn't always a prolific writer or speaker; in fact, as a child she experienced trauma that resulted in selective mutism. Read this article from Learning Lift-Off to read about Maya Angelou's experience.

Glossary

attachment figure: caregivers or adults to whom the individual is attached

contingency management: a form of treatment that revolves around a reward system with verbal or tangible reinforcement requiring parental involvement

relaxation training: techniques used to reduce anxiety that use strategies like deep breathing (control, slow, and purposeful breathing) and distraction (focusing on alternative things, grounding oneself to their senses)

separation anxiety disorder: a disorder that is characterized by excessive fear of separating from a caregiver

selective mutism: an anxiety disorder characterized by an absence of speech in particular social situations in which a person is expected to speak

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4.9: Introduction to Treating Anxiety Disorders

What you'll learn to do: examine various perspectives and treatment methods for anxiety disorders



In this section, we'll take a holistic look at the anxiety disorders we've learned about thus far and consider how the disorders are understood through the various psychological perspectives.

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4.10: Perspectives and Treatment for Anxiety Disorders

Learning Objectives

- Describe and compare viewpoints from the major psychological perspectives related to anxiety disorders
- Explain common treatment methods for anxiety disorders

Our understanding of anxiety disorders is shaped by multiple psychological perspectives. Researchers are finding that both genetic and environmental factors contribute to the risk of developing an anxiety disorder. Although the risk factors for each type of anxiety disorder can vary, some general risk factors for all types of anxiety disorders include

- temperamental traits of shyness or behavioral inhibition in childhood;
- exposure to stressful and negative life or environmental events in early childhood or adulthood;
- a history of anxiety or other mental illnesses in biological relatives; and
- some physical health conditions, such as thyroid problems, heart arrhythmias, or caffeine or other substances/medications, can produce or aggravate anxiety symptoms; a physical health examination is helpful in the evaluation of a possible anxiety disorder.

The Biological Perspective

The biological perspective seeks to understand the neurological and biological connections to anxiety. As mentioned above, some physical conditions can lead to anxiety. Researchers have concluded that several specific neurotransmitters are also connected with anxiety: gamma-hydroxybutyric acid (GABA), serotonin, and norepinephrine (noradrenalin).

When the balance of gamma-hydroxybutyric acid (GABA) is disrupted in the body, there is a correlated change in anxiety levels. Because of this, benzodiazepines, drugs that exert their effect through gamma-hydroxybutyric acid (GABA) receptors, are used to help people suffering from acute anxiety.

Serotonin is greatly important in anxiety. It is a monoamine neurotransmitter involved in controlling a wide range of behaviors by affecting the neural system, including emotions connected with fear and anxiety. Knockout studies on animals (called that because a certain receptor on the genetic code is knocked out) have shown that animals that have had their serotonin re-uptake transporters knocked out show abnormal responses to fear and anxiety in a number of behavioral conflict tests. These responses confirm the role of serotonin in modulating anxiety. Animals that had their serotonin receptor knocked out showed an increased heart rate and anxiety in a large variety of tasks such as eating and locomotion.

The best proof of the function of the serotonergic system in fear and anxiety is the pharmacological evidence. Drugs that change the function of serotonin have beneficial effects on various forms of anxiety. The best pharmacological treatment of anxiety is serotonin re-uptake inhibitors that allow greater levels of serotonin to accumulate and in that way help in treatment of anxiety.

Norepinephrine is also an important neurotransmitter in anxiety. Neurons which carry Norepinephrine rise from the locus coereuleus (LC), and these are also a center associated with warnings or alarms. The locus coereuleus (LC) secretes directly into the brain causing an immediate response. Increased levels of norepinephrine cause higher levels of anxiety. In pharmacology, norepinephrine blockers lower the levels of norepinephrine and do the same to a patient as do serotonin re-uptake blockers. Among adults, agents that alter noradrenergic functioning are powerful anxiolytics. Similarly, agents, such as yohimbine, that increase firing of the locus coeruleus (LC) are potent anxiogenic compounds.[1]

Due to these connections between anxiety and neurotransmitters, medication is commonly used to treat anxiety. In terms of treating anxiety with medication, it is important to note medication is one option of treatment and not always a one-stop treatment plan. Anti-anxiety medications are not cures for anxiety disorders, but they can help manage some of the symptoms. Medications (anti-anxiety drugs and antidepressants) have been found to be beneficial for disorders other than specific phobia, but relapse rates are high once medications are stopped (Heimberg et al., 1998; Hollon et al., 2005), and some classes of medications (minor tranquilizers or benzodiazepines) can be habit forming.



Figure 4.10.1: Medication such as benzodiazepines are commonly prescribed for anxiety disorders.

Anti-Anxiety Drugs

Anti-anxiety medications help reduce the symptoms of anxiety, such as panic attacks, or extreme fear and worry. The most common anti-anxiety medications are called benzodiazepines (such as Valium and Xanax). Benzodiazepines can treat generalized anxiety disorder. In the case of panic disorder or social phobia (social anxiety disorder), benzodiazepines are usually second-line treatments, behind SSRIs or other antidepressants.

Benzodiazepines used to treat anxiety disorders include the following:

- clonazepam
- alprazolam
- lorazepam

Short half-life (or short-acting) benzodiazepines (such as lorazepam) and beta-blockers are used to treat the short-term symptoms of anxiety. **Beta-blockers** help manage physical symptoms of anxiety, such as trembling, rapid heartbeat, and sweating, that people with phobias experience in difficult situations. Beta-blockers block the effects of epinephrine and reduce heart rate and blood pressure. Taking beta-blockers for a short period of time can help the person keep physical symptoms under control and can be used as needed to reduce acute anxiety.

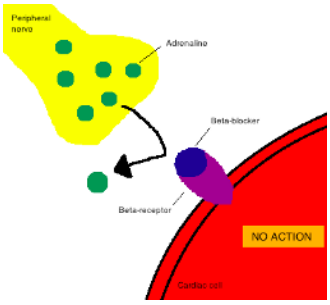


Figure 4.10.2: Beta-blockers block receptor sites for adrenaline, or epinephrine, so that it lessens the physical symptoms of anxiety.

Buspirone (which is unrelated to the benzodiazepines) is sometimes used for the long-term treatment of chronic anxiety. In contrast to the benzodiazepines, buspirone must be taken every day for a few weeks to reach its full effect. It is not useful on an as-needed basis.

How do people respond to anti-anxiety medications?

Anti-anxiety medications such as benzodiazepines are effective in relieving anxiety and take effect more quickly than the antidepressant medications (or buspirone) often prescribed for anxiety. However, people can build up a tolerance to benzodiazepines if they are taken over a long period of time and may need higher and higher doses to get the same effect. Some people may even become dependent on them. To avoid these problems, doctors usually prescribe benzodiazepines for short periods, a practice that is especially helpful for older adults, people who have substance abuse problems, and people who become dependent on medication easily. If people suddenly stop taking benzodiazepines, they may have withdrawal symptoms or their anxiety may return. Therefore, benzodiazepines should be tapered off slowly.

What are the possible side effects of anti-anxiety medications?

Like other medications, anti-anxiety medications may cause side effects. Some of these side effects and risks are serious. The most common side effects for benzodiazepines are drowsiness and dizziness. Other possible side effects include

- nausea,
- blurred vision,
- headache,
- confusion,
- tiredness, and
- nightmares.

Common side effects of beta-blockers include

- fatigue,
- cold hands,
- dizziness or light-headedness, and
- weakness.

Beta-blockers generally are not recommended for people with asthma or diabetes because they may worsen symptoms related to both conditions.

Possible side effects from buspirone include

- dizziness,
- headaches,
- nausea,
- nervousness,
- lightheadedness,
- excitement, and
- trouble sleeping.

Watch It

This video explains how beta-blockers work in treating anxiety.



You can view the transcript for "Why Can the Same Drug Treat Heart Attacks and Anxiety?" here (opens in new window).



Figure 4.10.3: Biofeedback testing.

Biofeedback is a technique that can be used to track and monitor physiological changes in the body in order to learn to control them. During biofeedback, a person may be connected to sensors to monitor heart rate, temperature, and other biological changes in order to practice controlling their reactions to various situations.

Cognitive and Behavioral Perspectives

Because anxiety disorders are connected to the way people perceive situations, cognitive therapies are particularly effective in thinking about and treating anxiety disorders. Some anxiety disorders, such as phobias, also stem from learned experiences, such as traumatic events. Cognitive theories suggest that anxiety disorders develop through thought patterns that overestimate threats to safety, therefore anxiety is caused by cognitive misinterpretations and can be treated by developing strategies to combat these thoughts. Therapies include cognitive behavioral therapy, acceptance and commitment therapy, intolerance of uncertainty therapy. Behavioral theories contend that anxiety disorders are mostly caused by conditioning, by modeling, or through experiences triggering their development, such as in specific phobias. Behavioral therapies often work in conjunction with cognitive therapies (such as in CBT), but they may also include behavior therapy, exposure treatment, systematic desensitization, and flooding.

Cognitive-Behavioral Therapy

CBT is an example of one type of psychotherapy that can help people with anxiety disorders. CBT teaches people different ways of thinking, behaving, and reacting to anxiety-producing and fearful objects and situations.

CBT has three phases: education, application, and relapse prevention. In the education phase, the individual is informed on the different effects anxiety can have physically and more importantly mentally. Understanding and being able to recognize their reactions will help the person to manage and eventually reduce his or her overall response.

CBT can also help people learn and practice social skills, which is vital for treating social anxiety disorder. CBT can be conducted individually or with a group of people who have similar difficulties. Often “homework” is assigned for participants to complete between sessions.

Exposure-based CBTs are effective psychosocial treatments for anxiety disorders, and many show greater treatment effects than medication in the long term (Barlow, Allen, & Basden, 2007; Barlow, Gorman, Shear, & Woods, 2000). In CBT, patients are taught skills to help identify and change problematic thought processes, beliefs, and behaviors that tend to worsen symptoms of anxiety, and practice applying these skills to real-life situations through exposure exercises. Patients learn how the automatic appraisals or thoughts they have about a situation affect both how they feel and how they behave. Similarly, patients learn how engaging in certain behaviors, such as avoiding situations, tends to strengthen the belief that the situation is something to be feared. A key aspect of CBT is exposure exercises, in which the patient learns to gradually approach situations they find fearful or distressing, in order to challenge their beliefs and learn new, less fearful associations about these situations.

Typically 50–80% of patients receiving drugs or CBT will show a good initial response, with the effect of CBT more durable. Newer developments in the treatment of anxiety disorders are focusing on novel interventions, such as the use of certain medications to enhance learning during CBT (Otto et al., 2010), and transdiagnostic treatments targeting core, underlying vulnerabilities (Barlow et al., 2011). As we advance our understanding of anxiety and related disorders, so too will our treatments advance, with the hopes that for the many people suffering from these disorders, anxiety can once again become something useful and adaptive, rather than something debilitating.

Among the cognitive-behavioral orientated psychotherapies the two main treatments are cognitive behavioral therapy and acceptance and commitment therapy (ACT). Intolerance of uncertainty therapy and motivational interviewing are two new treatments for GAD that are used as either stand-alone treatments or additional strategies that may enhance CBT.

Acceptance and Commitment Therapy

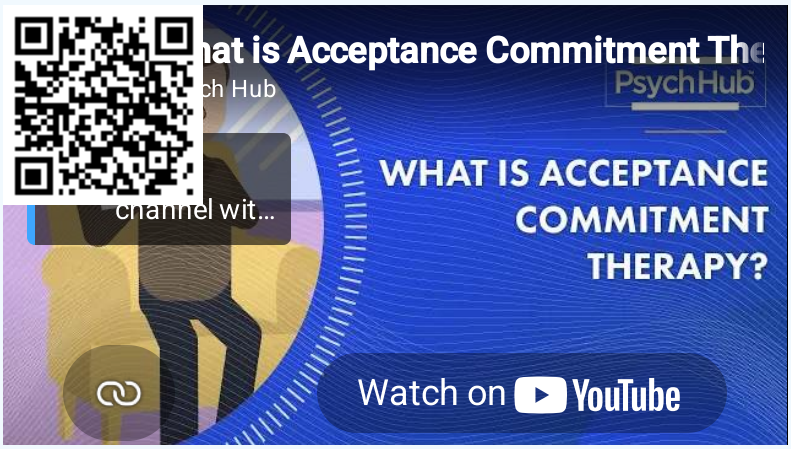
Acceptance and commitment therapy (ACT) is a behavioral treatment based on acceptance-based models. ACT is designed with the purpose to target three therapeutic goals:

- Accept emotions—reduce the use of avoiding strategies intended to avoid feelings, thoughts, memories, and sensations;
- Choose goals and directions—committing to these plans will decrease a person's literal response to their thoughts (e.g., understanding that thinking "I'm hopeless" does not mean that the person's life is truly hopeless), and
- Take action—increase the person's ability to keep commitments to changing their behaviors.

ACT's goals are attained by switching the person's attempt to control events to working towards changing their behavior and focusing on valued directions and goals in their lives as well as committing to behaviors that help the individual accomplish those personal goals. ACT psychological therapy teaches mindfulness (paying attention on purpose, in the present, and in a non-judgmental manner) and acceptance (openness and willingness to sustain contact) skills for responding to uncontrollable events and therefore manifesting behaviors that enact personal values.

Watch It

This video explains the experience of people who sometimes feel trapped in their situations. ACT includes mindfulness activities that help understand that thoughts or feelings can be distressing, and that is okay. Then a person with anxiety can choose the values they hold as important, and use these values to work towards change.



You can view the transcript for "What is Acceptance Commitment Therapy?" here (opens in new window).

Intolerance of Uncertainty Therapy

Intolerance of uncertainty therapy (IUT) refers to the idea that those with anxiety often experience consistent negative reactions to uncertain and ambiguous events regardless of their likelihood of occurrence. Central to the idea of intolerance of uncertainty is that someone with a high level of intolerance to uncertainty will feel that a new or uncertain situation is stressful and threatening. IUT focuses on helping patients in developing the ability to tolerate, cope with, and accept uncertainty in their life in order to reduce anxiety.

IUT is based on the psychological components of psychoeducation, awareness of worry, problem-solving training, re-evaluation of the usefulness of worry, imagining virtual exposure, recognition of uncertainty, and behavioral exposure. Past research has shown support for the efficacy of this therapy with GAD patients with continued improvements in follow-up periods.[2]

The Humanistic Perspective

According to the humanistic perspective, anxiety may develop if people do not see themselves honestly or do not practice self-acceptance. In this model, client-centered therapy is encouraged to help patients accept themselves and not be so self-judgemental. One humanistic type of treatment for anxiety is motivational interviewing.

Motivational Interviewing

A promising innovative approach to improving recovery rates for the treatment of GAD is to combine CBT with motivational interviewing (MI). **Motivational interviewing** (MI) is a strategy centered on the patient that aims to increase intrinsic motivation and decrease ambivalence about change due to the treatment. Motivational interviewing (MI) focuses on expressing empathy, encouraging self-efficacy, and heightening the dissonance between behaviors that are not desired and values that are not consistent with those behaviors.

Motivational interviewing (MI) is based on asking open-ended questions and listening carefully and reflectively to patients' answers, eliciting "change talk," and talking with patients about the pros and cons of change. Some studies have shown the combination of CBT with MI to be more effective than CBT alone.[3]

While there are as many differences in technique, the underlying spirit of the method remains the same and can be characterized in a few key points:

- Motivation to change is elicited from the client and is not imposed from outside forces.
- It is the client's task, not the counselor's, to articulate and resolve the client's ambivalence.
- Direct persuasion is not an effective method for resolving ambivalence.
- The counseling style is generally quiet and elicits information from the client.
- The counselor is directive, in that they help the client to examine and resolve ambivalence.
- Readiness to change is not a trait of the client, but a fluctuating result of interpersonal interaction.
- The therapeutic relationship resembles a partnership or companionship.

The Sociocultural Perspective

Globally, in 2010, approximately 273 million (4.5% of the population) had an anxiety disorder. It is more common in females (5.2%) than males (2.8%).

In Europe, Africa, and Asia, lifetime rates of anxiety disorders are between 9 and 16%, and yearly rates are between 4 and 7%. In the United States, the lifetime prevalence of anxiety disorders is about 29% and between 11 and 18% of adults have the condition in a given year. This difference is affected by the range of ways in which different cultures interpret anxiety symptoms and what they consider to be normative behavior. In general, anxiety disorders represent the most prevalent psychiatric condition in the United States, outside of substance use disorder.

Cultural Factors and Anxiety

Cultural factors that have been related to social anxiety disorder include a society's attitude towards shyness and avoidance, affecting the ability to form relationships or access employment or education, and shame. One study found that the effects of parenting are different depending on the culture: American children appear more likely to develop social anxiety disorder if their parents emphasize the importance of others' opinions and use shame as a disciplinary strategy (Leung *et al.*, 1994), but this association was not found for Chinese/Chinese-American children. In China, research has indicated that shy-inhibited children are more accepted than their peers and more likely to be considered for leadership and considered competent, in contrast to the findings in Western countries. Research indicates that Asian cultures have lower rates of social anxiety when compared with samples from the United States or Russia.[4]

Problems in developing social skills, or social fluency, may be a cause of some social anxiety disorder, through either inability or lack of confidence to interact socially and gain positive reactions and acceptance from others. The studies have been mixed, however, with some studies not finding significant problems in social skills while others have. What does seem clear is that the socially anxious perceive their own social skills to be low. It may be that the increasing need for sophisticated social skills in forming relationships or careers, and an emphasis on assertiveness and competitiveness, is making social anxiety problems more common, at least among the middle class. An interpersonal or media emphasis on "normal" or "attractive" personal characteristics

has also been argued to fuel perfectionism and feelings of inferiority or insecurity regarding negative evaluation from others. The need for social acceptance or social standing has been elaborated in other lines of research relating to social anxiety.

In contrast to the individualistic views of the Western culture, which emphasize individual thought, achievement, and well-being, Eastern cultures operate on collectivism, which focuses on the group and the interdependency of its members rather than any one person. The collectivism view of the self is that it can only be understood through one's social relationships and affiliations. These relationships and affiliations make up a network where the nuclear family shares the closest bonds, followed by the extended family and close friends.

Although the collectivism of Eastern cultures creates a strong support system for the group, it produces many stressors that can lead to depression and anxiety. For example, the overwhelming focus on the group rather than the individual can result in feelings of a lost individual identity and sense of self. Moreover, many rules and social norms exist in collective cultures than in individual cultures to maintain the harmony of the group. This combined with the cohesiveness of the group can make social mistakes very public and result in feelings of shame and embarrassment by the individual at fault. Research indicates that these feelings are associated with social anxiety within collectivistic societies. A bigger mistake that affects an individual's reputation or results in the loss of honor has more severe consequences because of the stress associated with maintaining that honor. *Taijin kyofusho*, a Japanese term for those who are embarrassed about their appearance or bodies or are fearful of displeasing others, is a possible culture-specific expression of social anxiety.[5]

Race and Anxiety

Racial trauma or race-based traumatic stress, is the cumulative effects of racism on an individual's mental and physical health. It has been linked to feelings of anxiety, depression, and suicidal ideation, as well as other physical health issues.

Racial trauma is not included in the most recent edition of the *Diagnostic and Statistical Manual on Mental Disorders* (DSM-5), since it does not meet the current criteria. However, researchers such as Robert T. Carter, Thema Bryant-Davis, and Carlota Ocampo have lobbied for its addition. According to them, racial trauma evokes symptoms similar to that of post-traumatic stress disorder (PTSD), hence the push for its recognition as a viable mental health concern. The effects of race-based traumatic stress on individuals depends on their experiences, and the ways in which it can manifest itself can vary significantly as well. Individuals who are exposed to race-based trauma or stress may experience dissociative symptoms following the event. Dissociative symptoms include depersonalization, in which an individual feels disconnected from their body or mind, and derealization, in which an individual has an unreal or distorted sense of experiences.

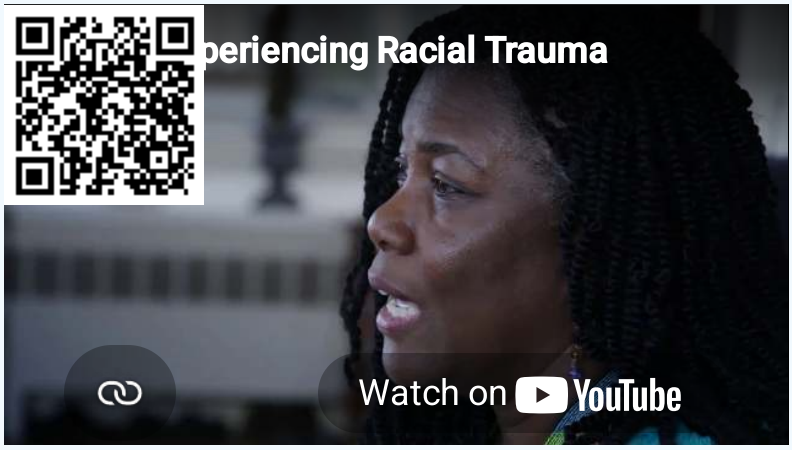
Race-based traumatic stress is a traumatic response to stress following a racial encounter. Robert T. Carter's (2007) theory of race-based traumatic stress implies that there are individuals of color who experience racially charged discrimination as traumatic, and often generate responses similar to post-traumatic stress. Race-based traumatic stress combines theories of stress, trauma, and race-based discrimination to describe a particular response to negative racial encounters.

Despite the limited research that examines race-based traumatic stress specifically, trauma research suggests that an individual's response to a stressor is highly dependent on that person's perception of the stressor; what one person may experience as traumatic, another person may not experience as such. These differing responses have been found to be strongly associated with each individual's ability to cope with the said stressor. According to Carter, a professor at Columbia University, race-based traumatic stress is an individual's response to racial discrimination as traumatic or outside of their ability to cope. Race-based traumatic stress can be experienced both directly and indirectly and can occur on an interpersonal level, institutional level, or cultural level. As such, research indicates that race-based traumatic stress can be demonstrated as a number of negative outcomes, including psychopathological symptoms, social inequities, and internalized racial oppression.

Research has indicated that children, as well as adults, can experience and be impacted by the reaches of race-based traumatic stress. Through direct experience from peers and/or authority figures, as well as indirectly through media exposure and/or bearing witness to the racial discrimination of their parents, research suggests children of color are particularly vulnerable to race-based traumatic stress.

Watch It

Watch the following video to learn more about racial trauma. In this video, Sheila Wise Rowe discusses how racial trauma in her family and early in her life affected her perceptions of self and her way of thinking about the world around her.



You can view the transcript for "Experiencing Racial Trauma" here (opens in new window).

Poverty and Anxiety

As of 2017, 12.3% of Americans were considered in poverty, according to the official poverty measure.[6] People who are in poverty have different health risks than those who are not considered in poverty, as well as different outcomes associated with those risks. People who are in poverty grapple with varying outcomes in physical health, mental health, and access to health care. Examining divergences in health between those above and below the poverty line gives insight into conditions for those who live in poverty.

Poverty has a complex relationship with mental health. Being in poverty may itself provoke a condition of elevated emotional stress, known as poverty distress. Poverty is also a precursor or risk factor for mental illness, particularly anxiety or mood disorders. Schizophrenia is also strongly associated with poverty, occurring most frequently in the poorest classes of people all over the world, especially in more unequal countries. In a sort of reciprocating relationship, having a mental illness is also a major risk factor for being in poverty, as having a mental illness may inhibit a person's ability to work or deter employees from hiring him or her.

A hypothesis known as the drift hypothesis posits that people with psychiatric disorders (primarily schizophrenia) tend to fall further down the socio-economic ladder as their condition reduces their functionality. The drift hypothesis is an effort to establish that people with profoundly limiting psychiatric symptoms are more likely to descend economically, not that the financially challenged are more likely to present severe psychiatric disorders. People experiencing less severe symptoms are less likely to be affected by "drift."

The social causation theory is an older theory with more evidence and research behind it. This hypothesis states that one's socioeconomic status (SES) is the cause of weakening mental functions. As Perry writes in *The Journal of Primary Prevention*, "members of the lower social classes experience excess psychological stress and relatively few societal rewards, the results of which are manifested in psychological disorder."[7] The excess stress that people with low socioeconomic status (SES) experience could be inadequate health care, job insecurity, and poverty, which can bring about many other psycho-social and physical stressors like crowding, discrimination, crime, etc. Thus, lower socioeconomic status (SES) predisposes individuals to the development of a mental illness.

Social risk factors for anxiety include a history of trauma (e.g., physical, sexual or emotional abuse or assault), bullying, early life experiences and parenting factors (e.g., rejection, lack of warmth, high hostility, harsh discipline, high parental negative affect, anxious childrearing, modelling of dysfunctional and drug-abusing behavior, discouragement of emotions, poor socialization, poor attachment, and child abuse and neglect), cultural factors (e.g., stoic families/cultures, persecuted minorities including the disabled), and socioeconomics (e.g., uneducated, unemployed, impoverished although developed countries have higher rates of anxiety disorders than developing countries). A 2019 comprehensive systematic review of over 50 studies showed that food

insecurity in the United States is strongly associated with depression, anxiety, and sleep disorders. Food-insecure individuals had an almost threefold risk increase of testing positive for anxiety when compared to food-secure individuals.

Contextual factors that are thought to contribute to anxiety include gender socialization and learning experiences. In particular, learning mastery (the degree to which people perceive their lives to be under their own control) and instrumentality, which includes such traits as self-confidence, self-efficacy, independence, and competitiveness fully mediate the relation between gender and anxiety. That is, though gender differences in anxiety exist, with higher levels of anxiety in women compared to men, gender socialization and learning mastery explain these gender differences.

Our review suggests that the prevalence and expression of social anxiety depend on the particular culture. Asian cultures typically show the lowest rates, whereas Russian and U.S. samples show the highest rates of social anxiety disorder. *Taijin kyofusho*, a Japanese term for those who are embarrassed about their appearance or bodies or are fearful of displeasing others, is a possible culture-specific expression of social anxiety, although the empirical evidence concerning the validity of this syndrome has been mixed. It is concluded that the individual's social concerns need to be examined in the context of the person's cultural, racial, and ethnic background in order to adequately assess the degree and expression of social anxiety and social anxiety disorder.

Link to Learning

Watch this video by pediatrician and researcher Nadine Burke Harris to learn more about the sociocultural implications of childhood trauma. Trauma and stress can have adverse effects on both physical and mental health, including anxiety, throughout the lifetime.

Glossary

acceptance and commitment therapy (ACT): a behavioral treatment based on accepting emotions, choosing goals, and taking action to keep commitments to change behaviors

anxiety: an emotion characterized by an unpleasant state of inner turmoil, often accompanied by nervous behavior such as pacing back and forth, somatic complaints, and rumination

generalized anxiety disorder (GAD): an anxiety disorder characterized by excessive, uncontrollable, and often irrational worry about events or activities

intolerance of uncertainty therapy: therapy focused on accepting uncertainty and developing the ability to tolerate and cope with uncertainty to reduce anxiety

motivational interviewing (MI): treatment approach centered on questioning the patient so that they increase intrinsic motivation and decrease ambivalence about change; MI focuses on expressing empathy, encouraging self-efficacy, and heightening the dissonance between behaviors that are not desired and values that are not consistent with those behaviors

racial trauma: characteristics similar to PTSD caused by racial discrimination or traumatic experiences connected to race

social anxiety disorder: also known as social phobia, is an anxiety disorder characterized by sentiments of fear and anxiety in social situations, causing considerable distress and impaired ability to function in at least some aspects of daily life

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4.11: Behavioral Treatments for Anxiety

Learning Objectives

- Explain how behavior therapy can be used to treat anxiety

Behavior Therapy and Anxiety

CBT and exposure therapy, a type of behavior therapy, are the two most commonly used interventions in the treatment of anxiety disorders. Exposure therapy has its roots in classical conditioning. In **exposure therapy**, patients make contact with the feared stimuli and this contact is maintained until the anxiety associated with the contact subsides. This process is termed **habituation** and it can only occur if the patients are prevented from using their usual escape or avoidance behavior (**extinction**). Several versions of exposure exist. **Systematic desensitization** (SD) is on the gentler part of the spectrum. Systematic desensitization (SD) is based on the idea of reciprocal inhibition proposing that two opposite emotions can not co-exist (e.g., fear and relaxation are mutually exclusive). In systematic desensitization (SD), relaxation training is followed by gradual (usually imaginary) exposure to the feared stimuli starting with the least feared stimulus. In contrast, **flooding** involves immediate exposure to the stimulus. Exposure therapy has been described as the most effective way to treat fear. Research indicates that the efficacy of exposure is optimal when it is graduated, repeated, and prolonged with practice tasks clearly specified.

Let's look at this case study with Jessica to see this type of therapy in practice.



Meet Jessica. She is smart, ambitious, creative, and full of energy. She is studying at a university, majoring in business. After she graduates, she wants to live in interesting places and get solid training and experience with a good corporation. Her dream is to start her own company, to be her own boss, and to do things that she can take pride in. For her, financial success and doing something worthwhile must go hand-in-hand.

But Jessica has a secret. She is terrified of speaking in front of people who are not her close friends. She has fought these fears for a long time, but she has never been able to conquer them. She is also aware of the fact that she will need to be able to speak to strangers comfortably and convincingly if she is going to meet her goals in business.

Now that you and your client have agreed upon your goals, it is time to choose a particular technique for the therapy. As a behavioral therapist, you are looking for a method to allow Jessica to learn a new response to the thought of public speaking. Now the idea terrifies her. After therapy is over, she should no longer be terrified and she may even look forward to the opportunity to speak in front of other people.

You know that everyone is not the same and different problems may call for different approaches to therapy. For these reasons, you have been trained in a variety of techniques that you can use to customize Jessica's therapy to meet her particular needs. It is time to decide how you are going to help Jessica.

Systematic Desensitization

SD works by gradually—step-by-step—exposing the person to situations that are increasingly more anxiety-producing. The anxiety-producing event is called "progressive exposure." By learning to cope with anxiety during less-threatening situations first, the person is better prepared to handle the more threatening situations. Even more important for treatment, the mind learns that nothing horrible happens. This retraining of the subconscious mind means that the situation actually becomes less threatening.



The first step in systematic desensitization is the development of a “hierarchy of fears.” The hierarchy of fears simply means that you must help Jessica create a list of situations related to her fear of public speaking. Then you create a hierarchy. The creation of the hierarchy means that you have her organize the situations from the least frightening to the most frightening.

For the next step in this exercise, you will need to take on Jessica’s role as the client. Imagine that you have developed a list of frightening situations, from ones that make you only slightly uncomfortable to ones that nearly make you sick with anxiety.

Remember that systematic desensitization works by putting the person in a series of situations. The early ones are not threatening or are only mildly threatening. However, as soon as your client learns to cope with each situation, you start working on the next most frightening situation.

So we’re ready to start, right? Wrong!

Behavior therapy teaches the client to cope with an anxiety-producing situation by replacing fear with an alternative response. A common alternative response is relaxation. The idea is that fear and anxiety cannot coexist with relaxation—if you are relaxed, you can’t be fully afraid.

However, most people are not very good at relaxing on command. So the behavioral therapist will teach the client how to relax effectively. The techniques are ones often used in meditation—slow breathing and focus on positive thoughts. Psychologist Kevin Arnold explains a deep breathing technique in this video.

Jessica’s Treatment

Jessica is an imaginary person, but behavior therapy is used by thousands of therapists with their clients every day. Review the following table to discover how Jessica’s therapy progressed. Her story is based on a fairly typical series of therapy sessions, though please understand that each person’s course of therapy is unique.

Jessica’s Treatment

Therapy Sessions	Session Description
Jessica's therapy: Preparation	Prior to starting progressive exposure, Jessica created her hierarchy of fears. She spent two sessions working on relaxation. She practiced relaxation at home several times a day until she and you, her therapist, agreed that she was ready to start treatment.
Jessica's therapy: Exposure Session 1	The bottom (lowest anxiety) of Jessica's fear hierarchy was chatting with friends about everyday topics. When asked to rate the fear level associated with doing this on a one to 10 scale, Jessica said one: no fear at all. Jessica brought two friends with her to the therapy session today. You had them sit in a comfortable part of your office, drinking tea and chatting for 15 minutes. Afterwards Jessica reported her fear level during the chat as a one on a 10-point scale: no fear. You then had her sit in a comfortable chair and think about giving a talk about the challenges of her job to a small, friendly audience. At the beginning of this task, she rated her anxiety as three on a 10-point scale. As she thought about it—with helpful suggestions from you—she also relaxed, using her relaxation training. After about 10 minutes, she reported her anxiety had dropped to one, the lowest level of anxiety on your scale. You gave Jessica homework—to repeat this exercise twice a day until the next session.
Jessica's therapy: Exposure Session 2	At the beginning of today's session, you had Jessica repeat the task from the previous session of thinking about talking about her job to a small, friendly group. At the beginning, she rated her fear at two, but it dropped to one within a few minutes. Now you take Jessica to the next level. You had her imagine telling a large audience of company executives about some technical problem she was working on at her job. At the beginning, just thinking about doing this led to a fear level of five. After 10 minutes, her fear level dropped to two. You repeated the exercise with a different topic and a different group, with similar results. Relaxation was practiced throughout the session. You gave Jessica homework again—to practice a similar situation at home.
Jessica's therapy: Exposure Session 3	You started this situation with a new scenario similar to the one Jessica did in the last session and practiced at home. She was quickly able to drop her anxiety level to one. You had a professional photography group create a video of someone very similar in appearance and manner to Jessica giving a talk in from of a small friendly audience on a topic similar to one Jessica might give. You asked her to watch this video and imagine herself in the place of the real speaker. She rated this a six on the anxiety scale. Over several repetitions, her rating dropped to two. For homework, Jessica watched the video several times a day. You instructed her in ways to make the video seem MORE REAL, so she could really feel the anxiety of being in front of people.
Jessica's therapy: Exposure Session 4	You have had Jessica arrange to give a talk NEXT SESSION to a small group of Jessica's co-workers. You also had Jessica prepare the talk. Today you practiced the talk with her. At the start of the

	practice session, with only you there, Jessica rated her anxiety level at nine out of 10. Over the course of the hour, her anxiety level dropped to five. Her homework was to continue to practice the talk and to work on relaxation.
Jessica's therapy: Exposure Session 5	Today, Jessica gave the talk to the small group. Her anxiety rating before she went in front of them was 10. Except for a little stumbling at the start, the 20-minute presentation went well. Jessica reported an anxiety level of four after the talk.
We'll skip a few sessions.	Hopefully, you have the basic idea.
Jessica's therapy: Exposure Session 5	In this last session, you have arranged for Jessica to be the introductory speaker at a literacy tutoring volunteer organization nearby. Jessica has done a small amount of volunteer work with the organization, but she knows very little about it. With the help of the staff, she prepares a talk during the week before this session. The audience comprises 45 people, all interested in doing literacy tutoring, who have come to the literacy center for an information session. Jessica knows none of them and none of them has ever heard of her. Jessica's introductory comments take about 15 minutes. She rates her anxiety level before going out at eight. After the talk, she rates her anxiety at . wotIn fact, she said it was almost fun.
After Therapy	Jessica continues to see you for a few more sessions. You give her additional homework and you help her develop a plan that includes arranging to give professional presentations for her job and continuing to give talks at the literacy volunteer organization. Jessica reports that none of these ideas create an anxiety level above three when she thinks about doing them.

Flooding

You just learned about systematic desensitization, a form of exposure therapy. Flooding is another type of exposure therapy. To understand how it works, let's review a few points from systematic desensitization.

In flooding therapy, you would skip the earliest situations described in systematic desensitization and you would move directly to highly threatening situations. Right after Jessica had mastered relaxation, your first session would require Jessica to give an actual talk. You would probably not start with the most extreme situation, but your goal would be to start Jessica in situations that she would immediately rate as nine or 10 on the anxiety scale.

Flooding has the potential to be more traumatic for Jessica (for your client), so it must be arranged carefully. But the same principles of learning work for flooding that work for systematic desensitization:

- The person consciously works to replace anxiety and fear with relaxation.
- The unconscious parts of the mind learn that the situation does not result in horrible outcomes. New expectations replace old fears.
- Learning does not just happen immediately. Homework and repeated practice reinforce the new positive response to situations that once produced fear.

Glossary

behavior therapy: a therapy focused on changing behavior; helping patients cope with an anxiety-producing situation by replacing fear with an alternative response

exposure therapy: being exposed with a feared stimulus until the anxiety associated with the contact subsides

flooding: a type of exposure therapy when a patient is exposed to high-anxiety inducing situations instead of gradual exposure

systematic desensitization: a type of exposure therapy consisting of gradual exposure to a feared stimulus that is paired with relaxation training

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4.12: Case Studies- Examining Anxiety

Learning Objectives

- Identify anxiety disorders in case studies

Case Study: Jameela

Jameela was a successful lawyer in her 40s who visited a psychiatrist, explaining that for almost a year she had been feeling anxious. She specifically mentioned having a hard time sleeping and concentrating and increased feelings of irritability, fatigue, and even physical symptoms like nausea and diarrhea. She was always worried about forgetting about one of her clients or getting diagnosed with cancer, and in recent months, her anxiety forced her to cut back hours at work. She has no other remarkable medical history or trauma.

Treatment

For a patient like Jameela, a combination of CBT and medications is often suggested. At first, Jameela was prescribed the benzodiazepine diazepam, but she did not like the side effect of feeling dull. Next, she was prescribed the serotonin-norepinephrine reuptake inhibitor venlafaxine, but first in mild dosages as to monitor side effects. After two weeks, dosages increased from 75 mg/day to 225 mg/day for six months. Jameela's symptoms resolved after three months, but she continued to take medication for three more months, then slowly reduced the medication amount. She showed no significant anxiety symptoms after one year.[1]

Case Study: Jane

Jane was a three-year-old girl, the youngest of three children of married parents. When Jane was born, she had a congenital heart defect that required multiple surgeries, and she continues to undergo regular follow-up procedures and tests. During her early life, Jane's parents, especially her mother, was very worried that she would die and spent every minute with Jane. Jane's mother was her primary caregiver as her father worked full time to support the family and the family needed flexibility to address medical issues for Jane. Jane survived the surgeries and lived a functional life where she was delayed, but met all her motor, communication, and cognitive developmental milestones.

Jane was very attached to her mother. Jane was able to attend daycare and sports classes, like gymnastics without her mother present, but Jane showed great distress if apart from her mother at home. If her mother left her sight (e.g., to use the bathroom), Jane would sob, cry, and try desperately to open the door. If her mother went out and left her with a family member, Jane would fuss, cry, and try to come along, and would continually ask to video-call her, so her mother would have to cut her outings short. Jane also was afraid of doctors' visits, riding in the car seat, and of walking independently up and down a staircase at home. She would approach new children only with assistance from her mother, and she was too afraid to take part in her gymnastics performances.

Jane also had some mood symptoms possibly related to her medical issues. She would intermittently have days when she was much more clingy, had uncharacteristically low energy, would want to be held, and would say "ow, ow" if put down to stand. She also had difficulty staying asleep and would periodically wake up with respiratory difficulties.[2]

---

1. Bandelow, B., Michaelis, S., & Wedekind, D. (2017). Treatment of anxiety disorders. Dialogues in clinical neuroscience, 19(2), 93–107. ↵
2. Hirshfeld-Becker DR, Henin A, Rapoport SJ, et alVery early family-based intervention for anxiety: two case studies with toddlersGeneral Psychiatry 2019;32:e100156. doi: 10.1136/gpsych-2019-100156 ↵

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4.13: Putting It Together- Anxiety Disorders



In summary, anxiety disorders are often severe chronic conditions, which can be present from an early age or begin suddenly after a triggering event. Anxiety triggers are prone to flare up at times of high stress and are frequently accompanied by physiological symptoms such as headache, sweating, muscle spasms, tachycardia, palpitations, and hypertension, which in some cases lead to fatigue.

In casual discourse the words *anxiety* and *fear* are often used interchangeably; in clinical usage, they have distinct meanings: *anxiety* is defined as an unpleasant emotional state for which the cause is either not readily identified or perceived to be uncontrollable or unavoidable, whereas *fear* is an emotional and physiological response to a recognized external threat. As you learned in this module, the umbrella term *anxiety disorder* refers to a number of specific disorders that include fears (phobias) or anxiety symptoms. The prognosis varies on the severity of each case and utilization of treatment for each individual.

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CHAPTER OVERVIEW

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5.3: Obsessive-Compulsive Disorder
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5.1: Why It Matters- Obsessive Compulsive Disorder and Stressor-Related Disorders

Why Learn about Obsessive-Compulsive Disorders and Stressor-Related Disorders?

We all have behaviors we perform out of habit. Sometimes it's ritualistic. Think of baseball: a batter tapping the bat several times in a pattern around the base prior to readying for the pitch or wearing the "lucky hat" on game day. Players may believe these rituals keep them on task, focused, and prepared for the win. This level of superstitious behavior appears *almost* like obsessive-compulsive disorder (OCD), and though they are unlikely enough to warrant a diagnosis of obsessive-compulsive disorder, they do provide an illustration for how these thoughts may be related to perfectionistic traits ("If I *don't* wear this hat, my team will lose"), or an idea that somehow, performing this ritual is a *responsibility* ("If I don't do this, I won't be as prepared for the game").

Watch it: Baseball Rituals

Watch this video to see some examples of unusual superstitious rituals among professional baseball players

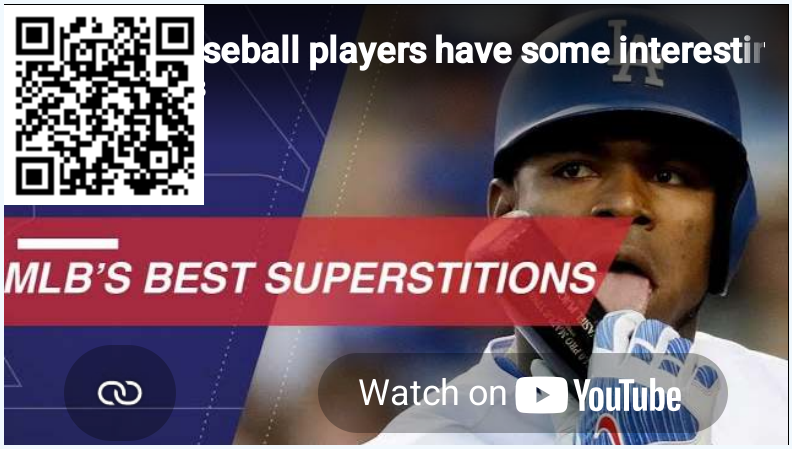




Figure **5.1.1**: An organized person or a superstitious person is usually not a person suffering from obsessive-compulsive disorder. Someone with OCD will feel *obsessions* and *compulsions* to act on certain behaviors.

The difference between an athlete prepping for a game and someone suffering from obsessive-compulsive disorder (OCD) is how it affects their normal lives. A baseball player isn't going to live life interrupted and poorly affected because he taps his bat a certain way or kisses his bat before the pitch. But someone with obsessive-compulsive disorder (OCD) may live a very disrupted life, trying to make it through the day despite a constant urge to give in to compulsions (repeated actions) that are triggered by obsessions (unwanted, uncontrollable thoughts).

Imagine you have a belief that if you don't vacuum the floors enough your family could get cancer. You may understand that, logically, there's no connection to dirty floors and cancer, but the unwanted thoughts are overwhelming and so is the urge to constantly vacuum your floor. Perhaps you begin vacuuming in a certain pattern, creating straight lines, and doing so twice a day. What if suddenly, you had a new urge to tap the vacuum several times before starting it and after stopping it? Then another urge to vacuum the floor again but in a different pattern? Or to touch a door as you passed it?

Of course, not everyone with OCD has thoughts and urges like those described above, but unfortunately, their reality is one that is plagued by a flurry of undesirable, unruly thoughts and follow-on behaviors that they perform as a way of relieving some of the stress. In this module, we'll take a closer look at OCD and other stressor-related disorders.

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5.2: Introduction to Obsessive-Compulsive and Related Disorders

What you'll learn to do: describe obsessive-compulsive and related disorders



In this section, you will learn about obsessive-compulsive disorder and other disorders related to this category. These are a group of disorders in which a person experiences both an *obsession* (uncontrollable, reoccurring thoughts) and a *compulsion* (behaviors that create an urge to repeat an action over and over). OCD and its related grouping of disorders are chronic, often caused by a dysfunctional response to stress, and interfere with a person's normal daily functioning. These OCD-related disorders include body dysmorphic disorder, hearing disorder, trichotillomania, and excoriation. Each one of these disorders has a unique presentation of symptoms that we'll examine more closely in the following pages.

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5.3: Obsessive-Compulsive Disorder

Learning Objectives

- Describe the main features and the development of obsessive-compulsive disorder

Obsessive-compulsive and related disorders are a group of overlapping disorders that generally involve intrusive, unpleasant thoughts, and repetitive behaviors. Many of us experience unwanted thoughts from time to time (e.g., craving double cheeseburgers when dieting), and many of us engage in repetitive behaviors on occasion (e.g., pacing when nervous). However, obsessive-compulsive disorders elevate unwanted thoughts and repetitive behaviors to a status so intense that these cognitions and activities disrupt daily life. Included in this category are **obsessive-compulsive disorder (OCD)**, body dysmorphic disorder, hoarding disorder, trichotillomania, and excoriation disorder.

Obsessive-Compulsive Disorder

People with OCD experience thoughts and urges that are intrusive and unwanted (obsessions) and/or the need to engage in repetitive behaviors or mental acts (compulsions). A person with this disorder might, for example, spend hours each day washing his or her hands or constantly checking and rechecking to make sure that a stove, faucet, or light has been turned off.



Figure **5.3.1**: (a) Repetitive hand washing and (b) checking (e.g., that a door is locked) are common compulsions among those with obsessive-compulsive disorder. (credit a: modification of work by the USDA; credit b: modification of work by Bradley Gordon)

Obsessions are more than just unwanted thoughts that seem to randomly jump into our head from time to time, such as recalling an insensitive remark a coworker made recently, and they are more significant than day-to-day worries we might have, such as justifiable concerns about being laid off from a job. Rather, **obsessions** are characterized as persistent, unintentional, and unwanted thoughts and urges that are highly intrusive, unpleasant, and distressing (APA, 2013). Common obsessions include concerns about germs and contamination, doubts (“Did I turn the water off?”), order and symmetry (“I need all the spoons in the tray to be arranged a certain way”), and urges that are aggressive or lustful. Usually, the person knows that such thoughts and urges are irrational and thus tries to suppress or ignore them, but has an extremely difficult time doing so. These obsessive symptoms sometimes overlap, such that someone might have both contamination and aggressive obsessions (Abramowitz & Siqueland, 2013).

Compulsions are repetitive and ritualistic acts that are typically carried out primarily as a means to minimize the distress that obsessions trigger or to reduce the likelihood of a feared event (APA, 2013). Compulsions often include such behaviors as repeated and extensive hand washing, cleaning, checking (e.g., that a door is locked) and ordering (e.g., lining up all the pencils in a particular way), and they also include mental acts such as counting, praying, or reciting something to oneself (Figure 1). Compulsions characteristic of OCD are not performed out of pleasure nor are they connected in a realistic way to the source of the distress or feared event. Approximately 2.3% of the U.S. population will experience OCD in their lifetime (Ruscio, Stein, Chiu, & Kessler, 2010) and, if left untreated, OCD tends to be a chronic condition creating lifelong interpersonal and psychological problems (Norberg, Calamari, Cohen, & Riemann, 2008).

Watch It

Watch this video to understand why people who are simply orderly or meticulous are probably not suffering from OCD.



You can view the transcript for "Debunking the myths of OCD – Natasha M. Santos" here (opens in new window).

Causes of OCD

The results of family and twin studies suggest that OCD has a moderate genetic component. OCD is five times more frequent in the first-degree relatives of people with OCD than in people without the disorder (Nestadt et al., 2000). Additionally, the **concordance rate** (the probability that a person, usually a twin or family member, will show symptoms if another has the disorder) of OCD among identical twins is around 57%; however, the concordance rate for fraternal twins is 22% (Bolton, Rijsdijk, O'Connor, Perrin, & Eley, 2007). Studies have implicated about two dozen potential genes that may be involved in OCD. These genes regulate the function of three neurotransmitters: serotonin, dopamine, and glutamate (Pauls, 2010). Many of these studies included small sample sizes and have yet to be replicated, thus additional research needs to be done in this area.

A brain region that is believed to play a critical role in OCD is the **orbitofrontal cortex** (Kopell & Greenberg, 2008), an area of the frontal lobe involved in learning and decision-making (Rushworth, Noonan, Boorman, Walton, & Behrens, 2011) (Figure 2). In people with OCD, the orbitofrontal cortex becomes especially hyperactive when they are provoked with tasks in which, for example, they are asked to look at a photo of a toilet or of pictures hanging crookedly on a wall (Simon, Kaufmann, Müsch, Kischkel, & Kathmann, 2010). The orbitofrontal cortex is part of a series of brain regions that, collectively, is called the OCD circuit; this circuit consists of several interconnected regions that influence the perceived emotional value of stimuli and the selection of both behavioral and cognitive responses (Graybiel & Rauch, 2000). As with the orbitofrontal cortex, other regions of the OCD circuit show heightened activity during symptom provocation (Rotge et al., 2008), which suggests that abnormalities in these regions may produce the symptoms of OCD (Saxena, Bota, & Brody, 2001). Consistent with this explanation, people with OCD show a substantially higher degree of connectivity of the orbitofrontal cortex and other regions of the OCD circuit than do those without OCD (Beucke et al., 2013). Additionally, researchers have discovered that people diagnosed with OCD actually show *under-activation* of brain areas responsible for stopping habitual behavior.[1]

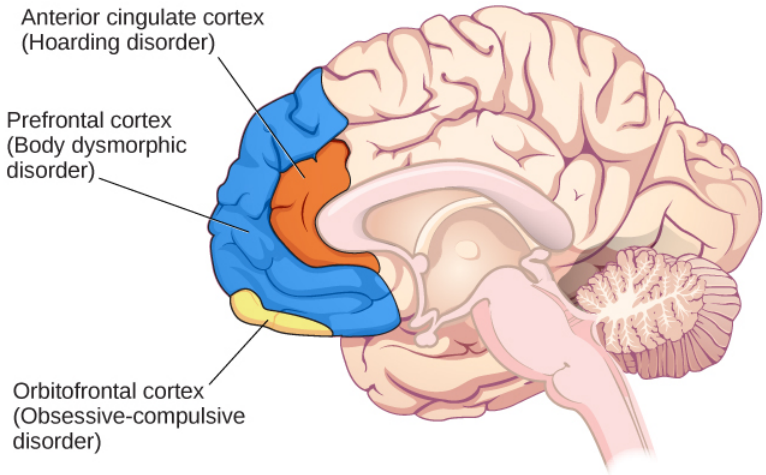


Figure **5.3.2**: Different regions of the brain may be associated with different psychological disorders.

Past research discussed above was based on imaging studies, which highlight the potential importance of brain dysfunction in OCD. However, one important limitation of these findings is the inability to explain differences in obsessions and compulsions. Another limitation is that the correlational relationship between neurological abnormalities and OCD symptoms cannot imply causation (Abramowitz & Siqueland, 2013).



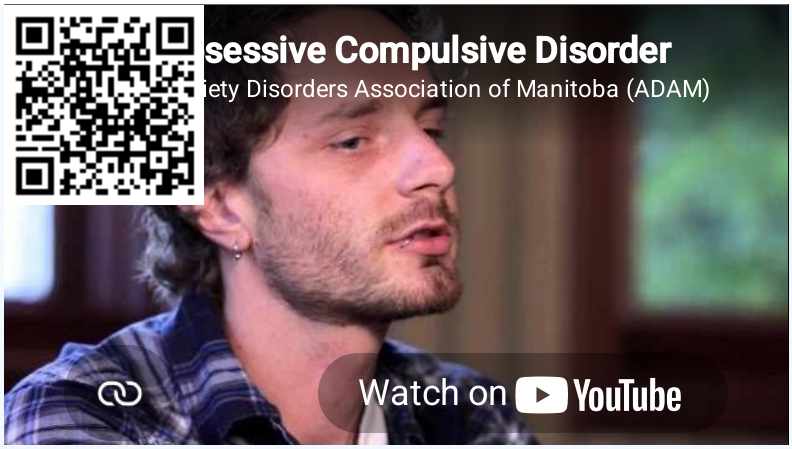
Figure **5.3.3**: PANDAS affects 1 in 200 children US-wide.

Another cause for OCD, specifically in children and adolescents, is PANDAS (short for pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections). This diagnosis occurs when strep triggers a misdirected immune response and results in inflammation on a child's brain. Usually, children with PANDAS have a very sudden onset or worsening of their symptoms, followed by slow, gradual improvement. If children with PANDAS get another strep infection, their symptoms suddenly worsen again. The increased symptom severity usually persists for at least several weeks but may last for several months or longer. [2]

Though the actual prevalence of PANDAS is not known, a conservative estimate from the PANDAS Research Network suggests that 1 in 200 children are affected by this condition in the United States alone.[3] Though symptoms often fade over time, low-level anxiety and OCD/TIC issues may remain permanently, and there are times where the exacerbation can take four to six months to remit. Treatment for this diagnosis includes antibiotics, IVIG (an intravenous pooled blood product comprising immunoglobulins that is used in treating immune deficiencies), plasmapheresis or plasma exchange (PEX) (a process during which the harmful auto-antibodies are removed from the blood system), and others such as cognitive-behavioral therapy (CBT), tonsillectomy, and probiotics. Generally, the best treatment for acute symptoms is antibiotics, and CBT to help manage neuropsychiatric symptoms.

Watch It

Watch this video to learn about Kyle's experience with OCD.



You can view the transcript for "Obsessive Compulsive Disorder" here (opens in new window).

Conditioning and OCD

The symptoms of OCD have been theorized to be learned responses, acquired and sustained as the result of a combination of two forms of learning: classical conditioning and operant conditioning (Mowrer, 1960; Steinmetz, Tracy, & Green, 2001). Specifically, the acquisition of OCD may occur first as the result of classical conditioning, whereby a neutral stimulus becomes associated with an unconditioned stimulus that provokes anxiety or distress. For example, someone may enter a library (neutral stimulus) and experience an unprompted panic attack (unconditioned stimulus). Though being in the library had nothing to do with the panic attack, the person who experiences this may begin to feel distressed upon entering the library in the future. If this continues (i.e., more panic attacks while in the library), the person may avoid the library place altogether. When an individual has acquired this association, subsequent encounters with the neutral stimulus trigger anxiety, including obsessive thoughts. The anxiety and obsessive thoughts (which are now a conditioned response) may persist until the person identifies some strategy to relieve it. Relief may take the form of a ritualistic behavior or mental activity that, when enacted repeatedly, reduces the anxiety. Such efforts to relieve anxiety constitute an example of negative reinforcement (a form of operant conditioning). Negative reinforcement involves the strengthening of behavior through its ability to remove something unpleasant or aversive. Hence, compulsive acts observed in OCD may be sustained because they are negatively reinforcing, in the sense that they reduce anxiety triggered by a conditioned stimulus. (For a quick review of classical and operant conditioning, you can watch this TedEd video.)

Suppose an individual with OCD experiences obsessive thoughts about germs, contamination, and disease whenever encountering a doorknob. What might have constituted a viable unconditioned stimulus? Also, what would constitute the conditioned stimulus, unconditioned response, and conditioned response? What kinds of compulsive behaviors might we expect and how do they reinforce themselves? What is decreased? Additionally, and from the standpoint of learning theory, how might the symptoms of OCD be treated successfully?

Glossary

body dysmorphic disorder: involves an excessive preoccupation with an imagined defect in physical appearance

concordance rate: the probability that an identical twin will show symptoms or traits of a disorder if one twin has the disorder

obsessive-compulsive and related disorders: a group of overlapping disorders listed in the DSM-5 that involves intrusive, unpleasant thoughts and/or repetitive behaviors

obsessive-compulsive disorder: characterized by the tendency to experience intrusive and unwanted thoughts and urges (obsession) and/or the need to engage in repetitive behaviors or mental acts (compulsions) in response to the unwanted thoughts and urges

orbitofrontal cortex: area of the frontal lobe involved in learning and decision-making

1. University of Cambridge. (2008, July 18). Obsessive-Compulsive Disorder Linked To Brain Activity. ScienceDaily. Retrieved August 11, 2020 from http://www.sciencedaily.com/releases/2008/07/080717140456.htm↵
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5.4: Body Dysmorphic Disorder

Learning Objectives

- Describe symptoms associated with body dysmorphic disorder

Body Dysmorphic Disorder

An individual with **body dysmorphic disorder (BDD)** is preoccupied with a perceived flaw in their physical appearance that is either nonexistent or barely noticeable to other people (APA, 2013). Body dysmorphic disorder's (BDD's) perceived physical defects cause the person to think they are unattractive, ugly, hideous, or deformed. These preoccupations can focus on any bodily area, but they typically involve the skin, face, or hair. The preoccupation with imagined physical flaws drives the person to engage in repetitive and ritualistic behavioral and mental acts, such as constantly looking in the mirror; trying to hide the offending body part; comparisons with others; and in some extreme cases, cosmetic surgery (Phillips, 2005). An estimated 2.4% of the adults in the United States meet the criteria for body dysmorphic disorder, with slightly higher rates in women than in men (APA, 2013).

Clinical Presentation



Figure **5.4.1**: Body dysmorphic disorder is characterized by a preoccupation with appearance and imagined physical flaws

Body dysmorphic disorder (BDD) is often associated with low quality of life and frequently is comorbid with major depressive disorder, substance use disorders, obsessive-compulsive disorder (OCD), and social phobias. Patients often are unaware that effective treatments are available and will hide symptoms because of feelings of shame or guilt. BDD is usually associated with increased suicidal ideation, delusional ideas, and poor or absent insight. The delusional variant of BDD is considered more severe. Both delusional and non-delusional variants present challenges in treatment compliance; many patients seek unnecessary dermatologic, dental, and other cosmetic interventions in hopes of removing their perceived flaws. These procedures typically have poor outcomes and lead to patient distress, often worsening symptoms and leading to patient dissatisfaction and loss of self-esteem. Some patients undergo repeated surgeries without achieving the expected outcome and thus have increased risk for depression and suicide.

Epidemiology



Figure **5.4.2**: Those with BDD often seek plastic surgery but remain dissatisfied with the results.

The prevalence of BDD in the general population is approximately 2% and is strongly associated with a history of cosmetic surgery and higher rates of suicidal ideation and suicide attempts. Patients who present for cosmetic surgery treatment are also affected by BDD at rates markedly higher than in the general population, ranging from 3%-53%. BDDs prevalence is markedly increased in the inpatient psychiatric setting, at approximately 16%. Prevalence in those seeking outpatient care for OCD, social phobia, and other disorders ranged from 10% to 40%. BDD is often initially undetected, suggesting the importance of BDD-specific screening practices and their role in achieving better outcomes.

Demographics

Body dysmorphic disorder has been reported to occur in children as young as five and in adults as old as 80. Regarding gender ratio, the two largest population-based studies of BDD (one conducted in the United States, the other in Germany) found a point prevalence of 2.5% of women versus 2.2% of men, and 1.9% of women and 1.4% of men, respectively. Thus, BDD may be *somewhat* more common in women, but it clearly affects many men as well.

Past research also found that individuals with BDD are less likely to be married than those without BDD, more likely to be divorced, and significantly more likely to be unemployed than the general population.[1]

Etiology

The cause of BDD is unknown. However, research shows that a number of things may be connected in influencing BDD:

- *the chemicals in the brain:* Low serotonin, one of the brain's neurotransmitters that gives us mood and pain, may help cause body dysmorphic disorder. Scientists can not explain this chemical problem in the brain, but it may be hereditary (passed down from parents to child).
- *OCD:* Many people with BDD also have OCD, where the person does things without wanting to. If people have had or have a genetic link to OCD, the person may be more likely to have BDD.
- *generalized anxiety disorder:* Many people with BDD also have generalized anxiety disorder. This disorder causes people to worry about things a lot, which makes them have anxiety about things in their life, for example, about their body as in BDD.
- *childhood abuse or trauma:* There is preliminary evidence that sexual, emotional, and physical abuse in childhood may be associated with BDD. One study found that 38% of 50 BDD patients reported some form of abuse during childhood as compared to 14% of a comparison group of 50 OCD patients. In a study of 75 BDD subjects who completed a childhood trauma questionnaire, 78.7% reported a history of childhood maltreatment. It could be that these individuals are already predisposed to BDD and with the additional factors (childhood trauma, bullying, etc.) they are more likely to experience chronic symptoms, which untreated, lead to a full-blown diagnosis; however, these studies don't establish a causative role for childhood trauma in the development of BDD.[2]

BDD usually develops in adolescence, a time when people usually worry about their appearance the most. However, many people with BDD suffer for years before they look for help. When they do look for professional help (i.e., seeking treatment via doctor, counselor, or psychiatrist), people with the problem often say they have other issues, for example, depression, social anxiety, or OCD, but do not admit the real, underlying problem, which is the way they see themselves. Not looking for professional help may be due to shame or guilt, though in many cases, individuals with this disorder lack insight to understand that the issue is with their perception and not a reality. Most patients cannot be convinced that the problem they have with their body is only imagined, and that they are seeing a changed view of themselves.

BDD is often wrongly thought of as a vanity obsession, but it is actually the opposite because people with BDD believe they are unattractive. A person with BDD can spend hours looking in the mirror, but they are not being vain; rather they are fretting about their appearance. A person with BDD may alternatively avoid mirrors or reflective surfaces at all costs so as not to see their appearance.

? Problem Areas and BDD

In researching BDD, Dr. Katherine Philips found that of more than 500 people with BDD, patients were most unhappy with the following body parts:[3]

- hair (56%)
- nose (37%)

- weight (22%)
- stomach (22%)
- breasts/chest/nipples (21%)
- eyes (20%)
- thighs (20%)
- teeth (20%)
- legs (overall) (18%)
- body shape/bone shape (16%)
- all of face (14%)
- lips (12%)
- buttocks (12%)
- chin (11%)
- fingers (11%)
- eyebrows (11%)

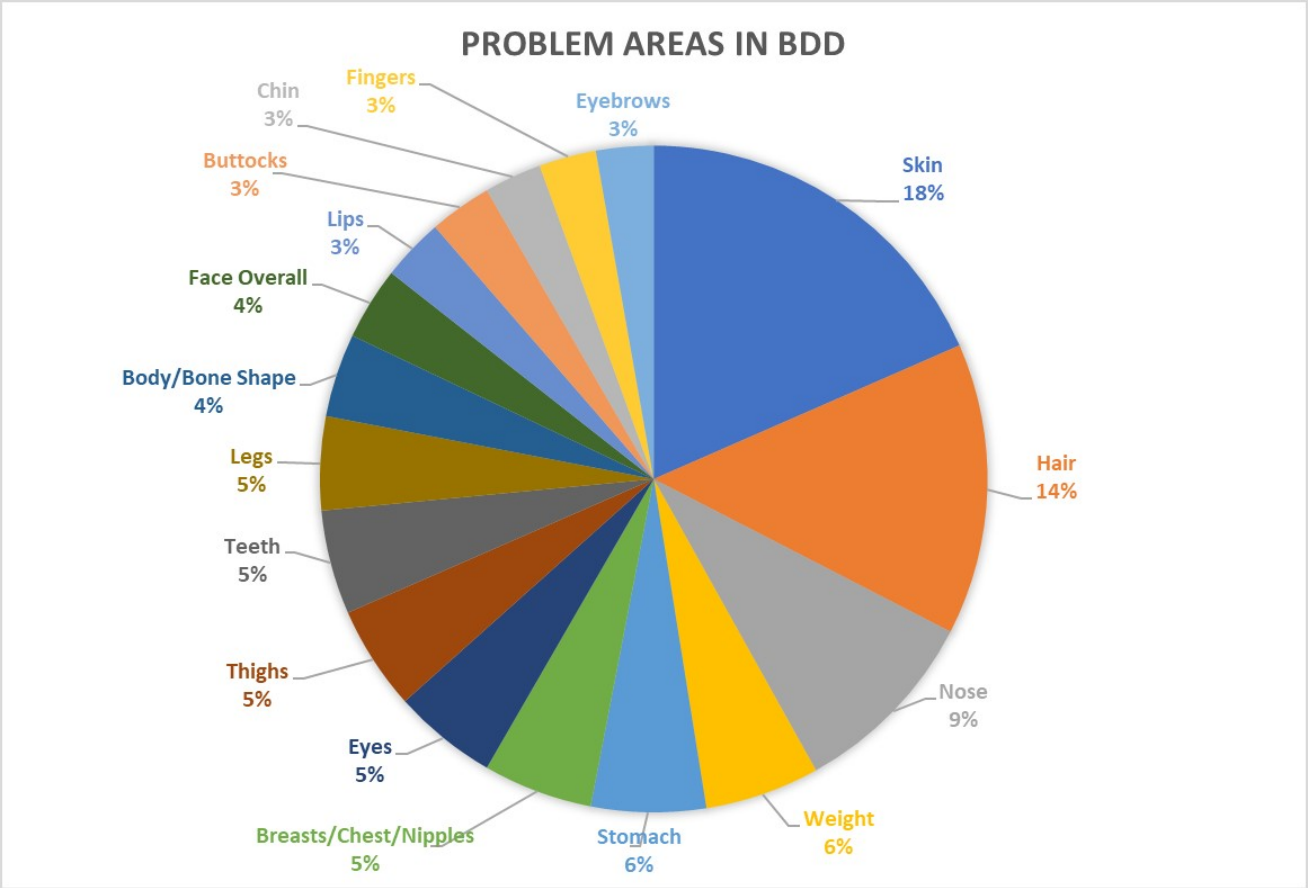


Figure **5.4.3**: Common problem areas associated with BDD.

Watch It

This clip shows examples of real children and adolescents with body dysmorphic disorder.



You can view the transcript for "Special Report: Imperfect Me – the impact of Body Dysmorphia" here (opens in new window).

To read more personal stories, visit the BDD Foundation website.

BDD and Suicide

Research by Phillips & Menard concluded that the percentage of people in the United States with BDD who commit suicide

- is 45 times higher than the percentage of all the U.S. population who commit suicide.
- is more than two times higher than the percentage of people in the Untied States with depression who commit suicide.
- is three times higher than the percentage of people in the United States with bipolar disorder who commit suicide.[4][/footnote]



Figure **5.4.4**: People with BDD are at risk for committing suicide.

Another condition, **gender dysphoria (GD)**, where the person finds his or her gender as a male or female uncomfortable, often has BDD-like feelings that are only directed at the primary and/or secondary sex characteristics on their body, which disagree with the person's perception of their own gender since birth. People with gender dysphoria (GD) may also have BDD-like traits or their condition may be misdiagnosed as BDD. Most studies reported a pre-transition gender dysphoria (GD) suicide attempt rate of 20% or more. The suicide attempt rate for patients with BDD is 15%. No studies are available showing overlap between GD and BDD suicide rate because BDD is excluded as a condition prior to GD diagnosis. GD is discussed in more detail in another module.

Glossary

body dysmorphic disorder (BDD): a disorder that manifests as an excessive concern with minor or wholly nonexistent defects in physical appearance; patients believe themselves to be unacceptably deformed and unattractive when actually they remain normal in appearance

gender dysphoria (GD): the distress a person feels due to a mismatch between gender identity and biological sex

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5.5: Hoarding Disorder

Learning Objectives

- Describe the main symptoms and development of hoarding disorder

Hoarding Disorder



Figure **5.5.1**: Those who suffer from hoarding disorder have great difficulty in discarding possessions, usually resulting in an accumulation of items that clutter living or work areas. (credit: "puuikibeach"/Flickr)

Although hoarding was traditionally considered to be a symptom of OCD, considerable evidence suggests that hoarding represents an entirely different disorder (Mataix-Cols et al., 2010). People with **hoarding disorder (HD)** cannot bear to part with personal possessions, regardless of how valueless or useless these possessions are. Hoarders accumulate excessive amounts of usually worthless items that clutter their living areas (Figure 2). Often, the quantity of cluttered items is so excessive that the person is unable to use their kitchen, or sleep in their bed. People who suffer from this disorder have great difficulty parting with items because they believe the items might be of some later use or because they form a sentimental attachment to the items (APA, 2013). Importantly, a diagnosis of hoarding disorder (HD) is made only if the hoarding is not caused by another medical condition and if the hoarding is not a symptom of another disorder (e.g., schizophrenia) (APA, 2013).

Prevalence rates have been estimated from two to 5% in adults, though the condition typically manifests in childhood with symptoms worsening in advanced age, at which point collected items have grown excessive and family members who would otherwise help to maintain and control the levels of clutter have either died or moved away. Hoarding appears to be more common in people with psychological disorders such as depression, anxiety, and attention deficit hyperactivity disorder (ADHD). Other factors often associated with hoarding include alcohol dependence and paranoid, schizotypal, and avoidance traits.

Clinical Presentation

People with hoarding disorder (HD), also referred to as *pathological collecting*, have severe anxiety over discarding possessions. While hoarders have an urge to collect and save items, they often have difficulty organizing the items, resulting in hazardous clutter. Homes of individuals with HD may present a health hazard to themselves and others due to health code violations, fire risk, infestations, or community expenses incurred from the cost of cleaning out the home or eviction from the home. Those with HD are also at risk for health problems such as food contamination, malnutrition, or falls. Causes for HD include experiencing strong emotional attachment to items, deficits in executive functioning, behavioral avoidance, or early developmental factors.[1]

As a result of hoarding, those with the disorder often

- do not allow visitors in, such as family and friends, or repair and maintenance professionals because the clutter embarrasses them.
- are reluctant or unable to return borrowed items.
- keep the shades drawn so that no one can look inside.
- have arguments with family members regarding the clutter.
- are at risk of fire, falling, infestation, or eviction.
- feel depressed or anxious due to the clutter.
- have suspicions of other people touching items.

- fear running out of an item or needing it in the future.
- check the garbage for accidentally discarded objects.

Many individuals with hoarding disorder do not recognize their hoarding as a problem. In one study, 42% of individuals with hoarding disorder found their behavior problematic compared to the 63% of their family and friends who saw the behavior as problematic. Low insight often leads to poor treatment outcomes. HD is problematic for many reasons, but one of the most frustrating aspects for friends and family members of those with this diagnosis is the fact that they often believe the individual can just "clean up" when in reality, the idea of getting rid of these items causes the most distress; often, it's the intervention that causes the most anxiety, *not* simply the fact they have so much clutter.

Link to Learning

For a historical case of severe hoarding, read this article about the Collyer brothers.

Watch It

Researchers have recognized compulsive hoarding as a phenomenon since at least the 1980s, but only recently began to study hoarding. It was first defined as a mental disorder in the fifth edition of the DSM in 2013. It is not clear whether compulsive hoarding is a separate, isolated disorder or rather a symptom of another condition, such as OCD**,** but the current DSM lists hoarding disorder as both a mental disability and a possible symptom for OCD.



You can view the transcript for "Hoarding: Designated Discrete Disorder in New Psychiatry 'Bible'" here (opens in new window).

Epidemiology

Compulsive hoarding affects about 2%-5% of the population. Hoarding behaviors usually start at a subclinical level in early adolescence and worsens with each decade so that it affects around 6% of those above age 70.[2] While most start hoarding in adolescence, often the disorder becomes clinically significant only in middle-aged patients, when individuals have accumulated enough things to cause alarm and or when the intervention of others, such as relatives or local authorities, causes greater stress. Stressful or traumatic events may be associated with the onset of hoarding symptoms. More education resulted in a slower increase in hoarding symptoms, and having a parent with hoarding tendencies showed a higher association in developing HD.[3]

Other psychiatric disorders often co-occur with HD. The most frequent comorbidity is a major depression, which can be present in up to 50% of the cases. Attention deficit/hyperactive disorder (ADHD) is also a common comorbidity with HD, with some studies suggesting association with inattentive symptoms of the disorder. Poorer general health is also associated with HD.[4]

In a study of 742 participants, the prevalence of hoarding was nearly 4% and was greater in older than younger age groups, greater in men than women (though generally, both are affected at an almost equal rate), and inversely related to household income (the odds of hoarding was over four times as great in the poorest, compared to the wealthiest, households). Hoarding was also correlated

with alcohol dependence, excessive physical discipline before age 16, parental psychopathology, insecurities caused by home break-ins, and some personality disorders (paranoid, schizotypal, avoidant, and obsessive-compulsive).[5]

Etiology

As is the case for most mental health disorders, the underlying cause has yet to be proven; however, it is thought that there is a strong genetic component to HD. Neuropsychological research has shown that cognitive deficits play some role in the onset and maintenance of hoarding symptoms and that these deficits are associated with issues in attention, memory, and executive functioning, although more research needs to be done to fully understand these connections.

Dysfunction in the anterior cingulate cortex (ACC), including the dorsal ACC involved in decision-making, error-monitoring, and reward-based learning as well as the ventral ACC connected to limbic structures that aid in assigning emotional salience to stimuli are associated with HD.[6]

Treatment

Many individuals with hoarding disorder do not recognize their hoarding as a problem. In one study, 42% of individuals with hoarding disorder found their behavior problematic compared to the 63% of their family and friends who saw the behavior as problematic.[7] Low insight leads to poor treatment outcomes. Most investigations have found that only a quarter of patients who hoard show an adequate response to these medications and therapeutic interventions.

Medication

Although no medication has received FDA approval for the treatment of compulsive hoarding, some monoamine reuptake inhibitors have been moderately successful in a small number of clinical studies. In patients where compulsive hoarding is secondary to or comorbid with OCD, serotonergic antidepressants such as SSRIs or the tricyclic antidepressant clomipramine are indicated, although the presence of hoarding predicts relatively poor treatment response. When examined, concurrent pharmacological and psychotherapeutic treatment appeared more effective than either alone.

Counseling

Cognitive-behavioral therapy (CBT) is a commonly implemented therapeutic intervention for compulsive hoarding. As part of CBT, the therapist may help the patient to

- discover why he or she is compelled to hoard.
- learn to organize possessions in order to decide what to discard.
- develop decision-making skills.
- declutter the home during in-home visits by a therapist or professional organizer.
- gain and perform relaxation skills.
- attend family and/or group therapy.
- be open to trying psychiatric hospitalization if the hoarding is serious.
- have periodic visits and consultations to keep a healthy lifestyle.



Figure **5.5.2**: Meditation is one way to achieve relaxation.

This modality of treatment usually involves exposure and response prevention to situations that cause anxiety and cognitive restructuring of beliefs related to hoarding. Furthermore, research has also shown that certain CBT protocols have been more effective in treatment than others. CBT programs that specifically address the motivation of the sufferer, organization, acquiring new clutter, and removing current clutter from the home have shown promising results. This type of treatment typically involves in-

home work with a therapist combined with between-session homework, the completion of which is associated with better treatment outcomes.

Other therapeutic approaches that have been found to be helpful include motivational interviewing, harm reduction, and group CBT.

Case Study: Hoarding Disorder

Read the following case study presented by the Life and Health Sciences Research Institute, School of Medicine at the University of Minho in Braga, Portugal.[8] Notice how the treatment plan utilizes both psychotherapy and medications, and that they also acknowledge comorbid symptoms of depression and insomnia.

“We present a case of a 52-year-old married man who worked as a mechanic was referred to a psychiatry department by his family doctor. He has no previous history of medical conditions. As a psychiatric background, he reported depressive symptoms following the death of his only son 22 years ago.

The patient collected various objects, predominantly stones, papers, and damaged pieces of cars because he thought they were valuable and/or usable in the future, but recognized that the collection was exaggerated. He could not discard any of these items, even if he never used them when needed. He reported to get anxious when someone else discarded some of these items. This behavior had started about 20 years earlier and it worsened with time. Despite, the thoughts of the possible use of these items were the reason why he could not throw out the possessions, these thoughts were not considered intrusive, repetitive, or egodystonic. The garage, attic, and surroundings of his house were cluttered with these objects (Figure **5.5.3**).



Figure **5.5.3**: Image from the patient’s garage before seeking treatment.

A psychotherapeutic plan for the patient was designed. The goals were to understand and educate the patient on his symptoms and beliefs, so he could understand the need for treatment. Cognitive restructuring of the beliefs and exposure to non-acquire and discard the objects saved were also explored. Along with the psychotherapeutic plan, a pharmacological treatment with fluvoxamine 100 mg id and trazodone 150 mg id was made, to treat the coexistent depressive symptoms and insomnia. During the first 6 months of treatment, the patient continued to buy and collect objects. Then, fluvoxamine was gradually augmented to 100 mg and quetiapine 200 mg was added to the treatment plan, with progressive improvement in the symptoms and the patient being able to sell/recycle most of the items after 9 months of this treatment (Figure 3). The patient is currently medicated with quetiapine 100 mg id for insomnia and organized the cluttered spaces. The patient and his wife were very gratified with the results.”

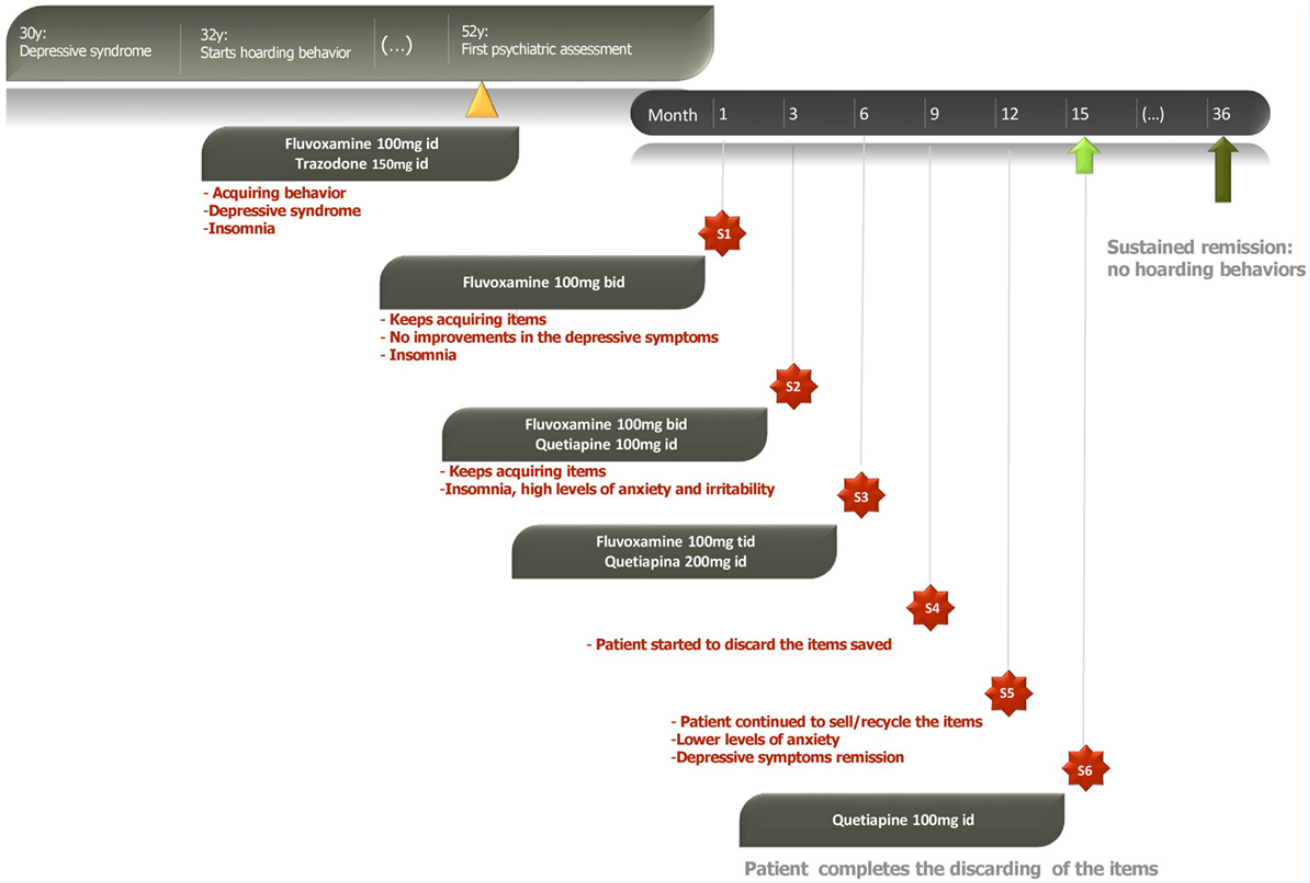


Figure **5.5.4**: The treatment plan took 15 months and frequent adjustments to help the patient lower his anxiety and discard his hoarded things.

Link to Learning

Watch clips from this Real Stories episode to learn more about hoarding disorder.

Glossary

hoarding disorder: characterized by persistent difficulty in parting with possessions, regardless of their actual value or usefulness

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5.6: Trichotillomania (Hairpulling Disorder)

Learning Objectives

- Describe the symptoms and diagnosis of trichotillomania

Trichotillomania (**TTM**), also known as hairpulling disorder or compulsive hairpulling, is a psychological disorder characterized by a long-term urge that results in the pulling out of one's hair. This occurs to such a degree that hair loss can be seen. A brief positive feeling may occur as hair is removed and efforts to stop pulling hair typically fail. Hair removal may occur anywhere; however, the head and around the eyes are most common. The hairpulling is to such a degree that it results in distress.

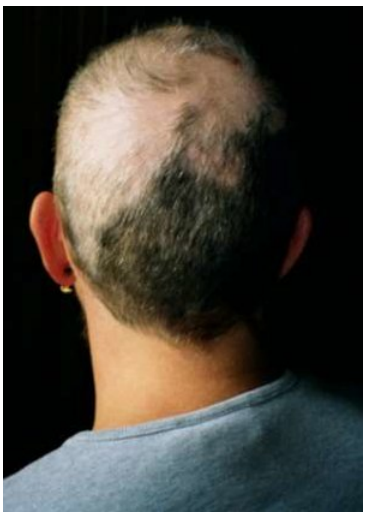


Figure **5.6.1**: A pattern of incomplete hair loss on the scalp of a person with trichotillomania.

TTM may run in families. The disorder occurs more commonly in those with obsessive-compulsive disorder or anxiety disorders, and episodes of pulling may be triggered by anxiety. People usually acknowledge that they pull their hair and on examination, broken hairs may be seen. Other conditions that may be present include body dysmorphic disorder; however, in that condition, people remove hair to try to improve what they see as a problem in how they look.

Trichotillomania is estimated to affect between 1%-4% of people. The lifetime prevalence of trichotillomania is estimated to be between 0.6% and 4.0% of the overall population. With a 1% prevalence rate, 2.5 million people in the United States may have trichotillomania at some time during their lifetimes. Trichotillomania most commonly begins in childhood or adolescence. Women are affected about 10 times more often than men.

Signs and Symptoms

Trichotillomania is usually confined to one or two areas of the body, but can involve multiple sites. The scalp is the most common pulling site, followed by the eyebrows, eyelashes, face, arms, and legs. Some less common areas include the pubic area, underarms, beard, and chest. The classic presentation is the *FriarTuck* form of vertex and crown alopecia. Children are less likely to pull from areas other than the scalp.

People who suffer from trichotillomania often pull only one hair at a time and these hairpulling episodes can last for hours at a time. Trichotillomania can go into remission-like states where the individual may not experience the urge to pull for days, weeks, months, and even years.

Individuals with trichotillomania exhibit hair of differing lengths; some are broken hairs with blunt ends, some new growth with tapered ends, some broken mid-shaft, or some uneven stubble. Scaling on the scalp is not present, overall hair density is normal, and a hair-pull test is negative (the hair does not pull out easily). Hair is often pulled out leaving an unusual shape. Individuals with trichotillomania may be secretive or shameful of the hairpulling behavior. If patients attempt to disguise their symptoms, it can make diagnosis difficult as symptoms are not always immediately obvious, or have been deliberately hidden to avoid disclosure.

An additional psychological effect can be low self-esteem, often associated with being shunned by peers and the fear of socializing, due to appearance and negative attention they may receive. Some people with trichotillomania wear hats, wigs, false eyelashes, eyebrow pencil, or style their hair in an effort to avoid such attention. TTM seems to have a strong stress-related component. In low-stress environments, some exhibit no symptoms (known as *pulling*) whatsoever. This pulling often resumes upon leaving the low-stress environment. Some individuals with trichotillomania may feel they are the only person with this problem due to low rates of reporting.

For some people, trichotillomania is a mild problem, merely a frustration. But for many, shame and embarrassment about hairpulling causes painful isolation and results in a great deal of emotional distress, placing them at risk for a co-occurring psychiatric disorder, such as a mood or anxiety disorder. Hairpulling can lead to great tension and strained relationships with family members and friends. Family members may need professional help in coping with this problem.



Figure **5.6.2**: Children may not consciously remember pulling their hair.

Trichotillomania is often not a focused act, but rather hairpulling occurs in a trance-like state; hence, trichotillomania is subdivided into “automatic” versus “focused” hairpulling. Children are more often in the automatic, or unconscious subtype, and may not consciously remember pulling their hair. Other individuals may have focused, or conscious, rituals associated with hairpulling, including seeking specific types of hairs to pull, pulling until the hair feels “just right,” or pulling in response to a specific sensation. Knowledge of the subtype is helpful in determining treatment strategies.

Epidemiology

Trichotillomania (TTM), or human compulsive hairpulling, is one of the most common psychiatric disorders, affecting approximately 3.5% of women, or 3.7 million people in the United States. TTM patients experience pronounced psychological distress with considerable negative impact in their quality of life.[1]

Trichotillomania has an estimated lifetime prevalence of approximately 0.5%-1%, and peak age of onset in adolescence (12–13 years of age) and is typically associated with impairment across domains of social, occupational, academic, and psychological functioning.[2]

Trichotillomania is more common in women than in men, and is currently classified as an OCD-related disorder. However, unlike the repetitive compulsive acts observed in OCD, repetitive behaviors in trichotillomania are not generally driven by intrusive thoughts.[3]

Patients with trichotillomania can come across a range of medical settings, including family doctors, dermatologists, neurologists, psychiatrists, pediatricians, endocrinologists, and geneticists.[4]

Etiology

Very little is known about the neurobiological basis of this disorder in humans, but in one study that reviewed available clinical and imaging studies of trichotillomania, previous work suggested an ABC model of trichotillomania, emphasizing the dysfunction of pathways involved in affect regulation, behavioral control, and cognition. In keeping with this, studies have found that trichotillomania is associated with impairment on response inhibition tests and phenomenological studies have found relationships between emotional states (dysphoria, anxiety) and the severity of the hairpulling symptoms.[5]

One study suggests that hormones may be a potential cause. In a study of adolescent girls with trichotillomania, researchers found that lower progesterone was associated with worse hairpulling severity (NIMH Trichotillomania Symptom Severity Scale, NIMH-

TSS), and that lower levels of all three hormones were associated with greater psychosocial dysfunction (Sheehan Disability Scale, SDS). Progesterone is believed to modulate the adaptive response to stress, mainly through the effect of its neurosteroid metabolite allopregnanolone on GABAA receptor activity.[6]

Some studies which included neuroimaging showed that individuals with trichotillomania had increased grey matter density in several brain regions involving affect regulation, motor habits, and cognition**.** Additionally, other studies examine the negative reinforcement people have when pulling; when emotional regulation is measured, those with trichotillomania have a harder time with emotional control that those without the disorder. Triggers for pulling include boredom, perfectionism, and frustration (to name a few) and hairpulling may be a way of relieving some of the stress and tension associated with these negative feelings.[7]

Causes and Comorbidity

Environment is a large factor that affects hairpulling. Sedentary activities such as being in a relaxed environment, or bored, are conducive to hairpulling. A common example of a sedentary activity promoting hairpulling is lying in a bed while trying to rest or fall asleep. An extreme example of automatic trichotillomania is found when some patients have been observed to pull their hair out while asleep. This is called sleep-isolated trichotillomania.

Anxiety, depression, and OCD are more frequently encountered in people with trichotillomania. Trichotillomania also has a high overlap with post-traumatic stress disorder, and some cases of trichotillomania may be triggered by stress. Another school of thought emphasizes hairpulling as addictive or negatively reinforcing, as it is associated with rising tension beforehand and relief afterward. A neurocognitive model—the notion that the basal ganglia plays a role in habit formation and that the frontal lobes are critical for normally suppressing or inhibiting such habits—sees trichotillomania as a habit disorder.

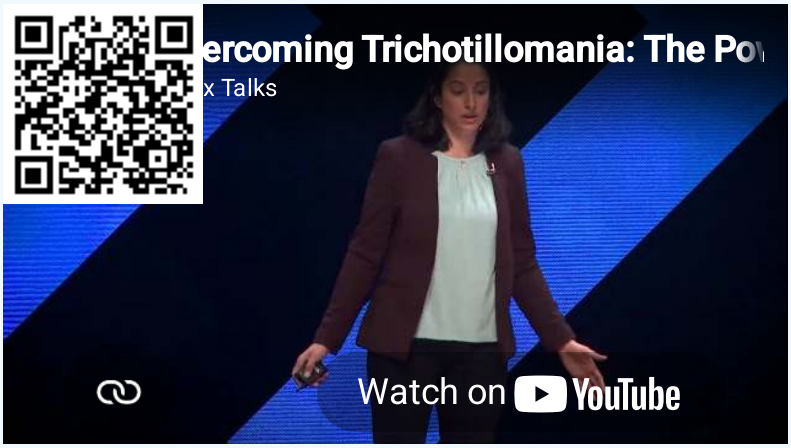
The most common age of onset of trichotillomania is between ages nine and 13, and a notable peak at ages 12–13. In this age range, trichotillomania is usually chronic and continues into adulthood. Trichotillomania that begins in adulthood most commonly arises from underlying psychiatric causes.

Psychotherapy

Treatment is based on a person’s age. Most preschool age children outgrow the condition if it is managed conservatively. In young adults, establishing the diagnosis and raising awareness of the condition is an important reassurance for the family and patient. Non-pharmacological interventions, including behavior modification programs, are often the first-line mediation; referrals to psychologists or psychiatrists may be considered when other interventions fail. When trichotillomania begins in adulthood, it is often associated with other psychological disorders, and referral to a psychologist or psychiatrist for evaluation or treatment is considered best. The hairpulling may resolve when other conditions are treated.

Watch It

Watch at least the first four minutes of this video (or the whole thing if you have time) to learn more about Aneela Idnani’s experience with a body-focused repetitive behavior.



You can view the transcript for “Overcoming Trichotillomania: The Power of Awareness | Aneela Idnani | TEDxFargo” here (opens in new window).

Glossary

habit reversal training (HRT): multicomponent behavioral treatment package originally developed to address a wide variety of repetitive behavior disorders

trichotillomania (TTM): also known as hairpulling disorder or compulsive hairpulling**;** a psychological disorder characterized by a long-term urge that results in pulling one’s hair out

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- Preventing, treating, and predicting barbering: A fundamental role for biomarkers of oxidative stress in a mouse model of Trichotillomania. **Authored by**: Giovana de L. T. Vieira,1 Amy C. Lossie, Donald C. Lay, Jr., John S. Radcliffe, and Joseph P. Garner. **Provided by**: PLoS One. **Located at**: . **License**: *CC0: No Rights Reserved*
- Cortical thickness abnormalities in trichotillomania: international multi-site analysis. **Authored by**: Samuel R. Chamberlain, Michael Harries, Sarah A. Redden, Nancy J. Keuthen, Dan J. Stein, Christine Lochner, and Jon E. Grant. **Provided by**: Brain Imaging Behavior. **Located at**: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5640149/. **License**: *CC BY: Attribution*
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5.7: Excoriation (Skin-Picking) Disorder

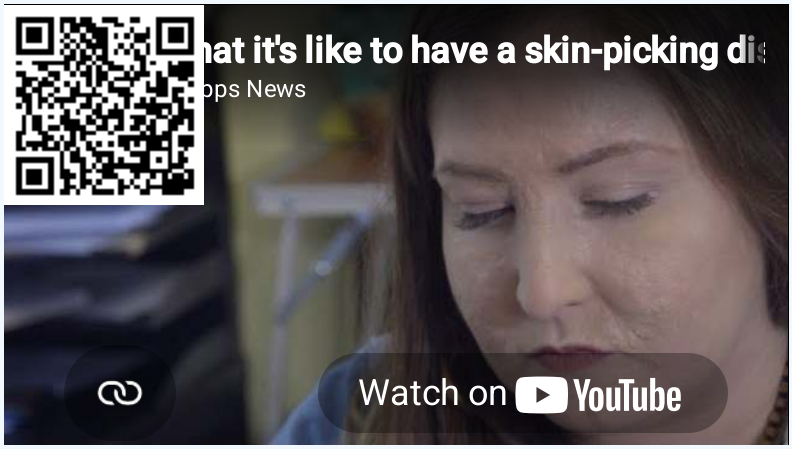
Learning Objectives

- Describe some of the main features and factors in the development of excoriation disorder

Excoriation disorder is an obsessive-compulsive spectrum disorder that is characterized by the repeated urge or impulse to pick at one's own skin to the extent that either psychological or physical damage is caused.

Watch It

Watch this video to better understand the experience of living with a skin-picking disorder.



You can view the transcript for "What it's like to have a skin-picking disorder" here (opens in new window).

Signs and Symptoms



Figure 5.7.1: Compulsive picking of the knuckles (via mouth) illustrating potentially temporary disfiguration of the distal and proximal joints of the middle and little fingers.

Episodes of skin picking are often preceded or accompanied by tension, anxiety, or stress. In some cases, following picking, the affected person may feel depressed. During these moments, there is commonly a compulsive urge to pick, squeeze, or scratch at a surface or region of the body, often at the location of a perceived skin defect. When picking, one may feel a sense of relief or satisfaction.

The region most commonly picked is the face, but other frequent locations include the arms, legs, back, gums, neck, shoulders, scalp, abdomen, chest, and extremities such as the fingernails, cuticles, and toenails. Most patients with excoriation disorder report having a primary area of the body that they focus their picking on, but they will often move to other areas of the body to allow their primary picking area to heal. Individuals with excoriation disorder vary in their picking behavior; some do it briefly multiple times a day while others can do one picking session that can last for hours. The most common way to pick is to use the fingers although a significant minority of people use tools such as tweezers or needles.

Skin picking often occurs as a result of some other triggering cause. Some common triggers are feeling or examining irregularities on the skin and feeling anxious or other negative feelings.

Complications arising from excoriation disorder include infection at the site of picking, tissue damage, and sepsis. Damage from picking can be so severe as to require skin grafting. Severe picking can cause epidermal abscesses. Severe cases of excoriation disorder can cause life-threatening injuries. For example, in one reported case, a female picked a hole through the bridge of her nose, which required surgery to fix, and a 48-year-old female picked through the skin on her neck exposing the carotid artery. Pain in the neck or back can arise due to prolonged bent-over positions while engaging in the behavior. Besides physical injuries, excoriation disorder can cause severe physical scarring and disfigurement.



Figure **5.7.2**: Compulsive picking of face using nail pliers and tweezers.

Excoriation disorder can cause feelings of intense helplessness, guilt, shame, and embarrassment in individuals, and this greatly increases the risk of self-harm. Studies have shown that excoriation disorder presented suicidal ideation in 12% of individuals with this condition, suicide attempts in 11.5% of individuals with this condition, and psychiatric hospitalizations in 15% of individuals with this condition.

Causes

There have been many different theories regarding the causes of excoriation disorder including biological and environmental factors. A common hypothesis is that excoriation disorder is often a coping mechanism to deal with elevated levels of turmoil, arousal, or stress within the individual, and that the individual has an impaired stress response. A review of behavioral studies found support in this hypothesis in that skin picking appears to be maintained by automatic reinforcement within the individual. In contrast to neurological theories, there are some psychologists who believe that picking behavior can be a result of repressed rage felt toward authoritarian parents. A similar theory holds that overbearing parents can cause the behavior to develop in their children.

Clinical studies have posited that there is a strong link between traumatic childhood events and excoriation disorder. Those with self-injurious disorders of the skin are found to also frequently report childhood sexual abuse. While attempting to produce diagnostic criteria for excoriation disorder, researchers conversed with 10 patients with the disorder and found that a majority reported personal problems before the picking began, and four reported on abuse they suffered in childhood or adolescence.[1]

Neurological

Studies have shown a linkage between dopamine and the urge to pick. Drugs such as cocaine and methamphetamine, which increase the pharmacological effects of dopamine, have been shown to cause uncontrollable picking in users. Central nervous system stimulants may create the sensation of **formication**, which feels like something is crawling on or under the skin. Thus, excoriation disorder could result from a dysfunction in the dopamine reward functions.

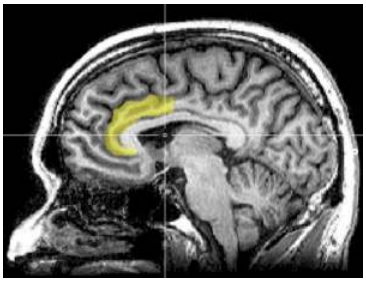


Figure **5.7.3**: The anterior cingulate cortex, part of the right-lateralized frontostriatal circuit, is shown here. We know that it plays a role in motor-inhibitory control and may be associated with excoriation disorder.

There may be another neurological explanation for excoriation disorder: individuals with the condition have less motor-inhibitory control, but show no sign of difference in cognitive flexibility, when compared to individuals without the condition. Motor-inhibitory control is a function of the right-lateralized frontostriatal circuit, which includes the right inferior frontal and bilateral anterior cingulate cortices. The impairment of motor-inhibitory control is similar to the neurological conditions of those who have problems suppressing inappropriate behaviors, such as abusing methamphetamine.

Epidemiology

The prevalence of excoriation disorder is not well understood. Estimates of prevalence of the condition range from 1.4 to 5.4% in the general population. One U.S. telephone survey found that 16.6% of respondents "picked their skin to the point of noticeable tissue damage" and that 1.4% would qualify as meeting the requirements of excoriation disorder. Another community survey found a rate of 5.4% had excoriation disorder. A survey of college students found a rate of 4%.[2] One study found that among non-disabled adults, 63% of individuals engaged in some form of skin picking and 5.4% engaged in serious skin picking.[3] Lastly, a survey of dermatology patients found that 2% suffered from excoriation disorder. Excoriation disorder is statistically more common in females than in males.

In some patients, excoriation disorder begins with the onset of acne in adolescence, but the compulsion continues even after the acne has gone away. Skin conditions such as keratosis pilaris, psoriasis, and eczema can also provoke the behavior. In patients with acne, the grooming of the skin is disproportionate to the severity of the acne. Certain stressful events including marital conflicts, deaths of friends or family, and unwanted pregnancies have been linked to the onset of the condition. If excoriation disorder does not occur during adolescence, another common age of onset is between the ages of 30 to 45. Additionally, many cases of excoriation disorder have been documented to begin in children under the age of 10. One small survey of patients with excoriation disorder found that 47.5% of them had an early onset of excoriation disorder that began before age 10. Traumatic childhood events may initiate the behavior.

Coborbidity

Excoriation disorder has a high rate of comorbidity with other psychiatric conditions, especially with mood and anxiety disorders. One survey of patients with excoriation disorder found that 38% also had alcohol- or drug-abuse problems. Studies have shown the following rates of psychiatric conditions found in patients with excoriation disorder: trichotillomania (38.3%), substance abuse (38%), major depressive disorder (approximately 31.7% to 58.1%), anxiety disorders (approximately 23% to 56%), OCD (approximately 16.7% to 68%), and body dysmorphic disorder (approximately 26.8% to 44.9%). There are also higher rates of excoriation disorder in patients in psychiatric facilities; a study of adolescent psychiatric inpatients found that excoriation disorder was present in 11.8% of patients. It is also present at high rates with some other conditions: 44.9% of patients with body dysmorphic disorder also have excoriation disorder; 8.9% of patients with OCD have excoriation disorder; and 8.3% of patients with trichotillomania have excoriation disorder.

Excoriation disorder has been the subject of several episodes of *Obsessed*, a television documentary series that focuses on treatment of anxiety disorders. Skin picking is shown as a symptom of Nina Sayers' anxiety and OCD in the movie *Black Swan*.

Link to Learning

Watch this Ted talk by artist Liz Atkin and listen to her tell her own story of struggle and recovery in dealing with excoriation disorder.

Glossary

excoriation disorder: an obsessive-compulsive spectrum disorder that is characterized by the repeated urge or impulse to pick at one's own skin to the extent that either psychological or physical damage is caused

formication: the sensation of something that is crawling on or under the skin

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5.8: Introduction to Trauma and Stressor-Related Disorders

What you'll learn to do: describe trauma and stressor-related disorders



Figure **5.8.1**: We all experience stress from time to time, but how we cope, adjust, and react will vary.

In this section, you will learn about trauma and stressor-related disorders. These are a group of disorders in which a person usually experiences a trauma or major life change. The disorders you'll learn about include post-traumatic stress disorder, acute stress disorder, adjustment disorder, and attachment disorders.

We all experience stress from time to time—from moving to a new home or getting involved in a car accident—but how we cope, adjust, and react will be different depending on prior experiences, personality, and level of resilience (adaptability to change). Those who are unable to adjust and cope with the stress of their experiences, to the point that it affects them emotionally, socially, etc., will likely be diagnosed with one of the disorders you'll read about over the next few pages.

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5.9: Stressors

Learning Objectives

- Describe different types of possible stressors, including major life readjustments and trauma

For an individual to experience stress, they must first encounter a potential stressor. In general, stressors can be placed into one of two broad categories: chronic and acute. **Chronic stressors** include events that persist over an extended period of time, such as caring for a parent with dementia, long-term unemployment, or imprisonment. **Acute stressors** involve brief focal events that sometimes continue to be experienced as overwhelming well after the event has ended, such as falling on an icy sidewalk and breaking your leg (Cohen, Janicki-Deverts, & Miller, 2007). Whether chronic or acute, potential stressors come in many shapes and sizes. They can include major traumatic events, significant life changes, daily hassles, as well as other situations in which a person is regularly exposed to threat, challenge, or danger.

Traumatic Events

Some stressors involve traumatic events or situations in which a person is exposed to actual or threatened death or serious injury. Stressors in this category include exposure to military combat, threatened or actual physical assaults (e.g., physical attacks, sexual assault, robbery, and childhood abuse), terrorist attacks, natural disasters (e.g., earthquakes, floods, and hurricanes), and automobile accidents. Men, non-Whites, and individuals in lower socioeconomic status (SES) groups report experiencing a greater number of traumatic events than do women, Whites, and individuals in higher SES groups (Hatch & Dohrenwend, 2007). Some individuals who are exposed to stressors of extreme magnitude develop post-traumatic stress disorder (PTSD): a chronic stress reaction characterized by experiences and behaviors that may include intrusive and painful memories of the stressor event, jumpiness, persistent negative emotional states, detachment from others, angry outbursts, and avoidance of reminders of the event (American Psychiatric Association [APA], 2013).

Life Changes

Most stressors that we encounter are not nearly as intense as the ones described above. Many potential stressors we face involve events or situations that require us to make changes in our ongoing lives and require time as we adjust to those changes. Examples include death of a close family member, marriage, divorce, and moving (Figure **5.9.1**).



Figure **5.9.1**: Some fairly typical life events, such as moving, can be significant stressors. Even when the move is intentional and positive, the amount of resulting change in daily life can cause stress. (credit: “Jellaluna”/Flickr)

In the 1960s, psychiatrists Thomas Holmes and Richard Rahe wanted to examine the link between life stressors and physical illness, based on the hypothesis that life events requiring significant changes in a person’s normal life routines are stressful, whether these events are desirable or undesirable. They developed the **Social Readjustment Rating Scale (SRRS)**, consisting of 43 life events that require varying degrees of personal readjustment (Holmes & Rahe, 1967). Many life events that most people would consider pleasant (e.g., holidays, retirement, marriage) are among those listed on the Social Readjustment Rating Scale (SRRS); these are examples of **eustress**. Holmes and Rahe also proposed that life events can add up over time, and that experiencing a cluster of stressful events increases one’s risk of developing physical illnesses.

In developing their scale, Holmes and Rahe asked 394 participants to provide a numerical estimate for each of the 43 items; each estimate corresponded to how much readjustment participants felt each event would require. These estimates resulted in mean value scores for each event—often called life change units (LCUs) (Rahe, McKeen, & Arthur, 1967). The numerical scores ranged

from 11 to 100, representing the perceived magnitude of life change each event entails. Death of a spouse ranked highest on the scale with 100 life change units (LCUs), and divorce ranked second highest with 73 life change units (LCUs). In addition, personal injury or illness, marriage, and job termination also ranked highly on the scale with 53, 50, and 47 LCUs, respectively. Conversely, change in residence (20 LCUs), change in eating habits (15 LCUs), and vacation (13 LCUs) ranked low on the scale (Table 1). Minor violations of the law ranked the lowest with 11 LCUs. To complete the scale, participants checked yes for events experienced within the last 12 months. LCUs for each checked item are totaled for a score quantifying the amount of life change. Agreement on the amount of adjustment required by the various life events on the SRRS is highly consistent, even cross-culturally (Holmes & Masuda, 1974).

Table 1. Some Stressors on the Social Readjustment Rating Scale (Holmes & Rahe, 1967)

Life event	Life change units
Death of a close family member	63
Personal injury or illness	53
Dismissal from work	47
Change in financial state	38
Change to different line of work	36
Outstanding personal achievement	28
Beginning or ending school	26
Change in living conditions	25
Change in working hours or conditions	20
Change in residence	20
Change in schools	20
Change in social activities	18
Change in sleeping habits	16
Change in eating habits	15
Minor violation of the law	11

Extensive research has demonstrated that accumulating a high number of life change units within a brief period of time (one or two years) is related to a wide range of physical illnesses (even accidents and athletic injuries) and mental health problems (Monat & Lazarus, 1991; Scully, Tosi, & Banning, 2000). In an early demonstration, researchers obtained LCU scores for U.S. and Norwegian Navy personnel who were about to embark on a six-month voyage. A later examination of medical records revealed positive (but small) correlations between LCU scores prior to the voyage and subsequent illness symptoms during the ensuing six-month journey (Rahe, 1974). In addition, people tend to experience more physical symptoms, such as backache, upset stomach, diarrhea, and acne, on specific days in which self-reported LCU values are considerably higher than normal, such as the day of a family member's wedding (Holmes & Holmes, 1970).

The Social Readjustment Rating Scale (SRRS) provides researchers a simple, easy-to-administer way of assessing the amount of stress in people's lives, and it has been used in hundreds of studies (Thoits, 2010). Despite its widespread use, the scale has been subject to criticism. First, many of the items on the SRRS are vague; for example, death of a close friend could involve the death of a long-absent childhood friend that requires little social readjustment (Dohrenwend, 2006). In addition, some have challenged its assumption that undesirable life events are no more stressful than desirable ones (Derogatis & Coons, 1993). However, most of the available evidence suggests that, at least as far as mental health is concerned, undesirable or negative events are more strongly associated with poor outcomes (such as depression) than are desirable, positive events (Hatch & Dohrenwend, 2007). Perhaps the most serious criticism is that the scale does not take into consideration respondents' appraisals of the life events it contains. As you

recall, appraisal of a stressor is a key element in the conceptualization and overall experience of stress. Being fired from work may be devastating to some but a welcome opportunity to obtain a better job for others. The SRRS remains one of the most well-known instruments in the study of stress, and it is a useful tool for identifying potential stress-related health outcomes (Scully et al., 2000).

Link to Learning

Go to this site to complete the SRRS scale and determine the total number of LCUs you have experienced over the last year.

Connect the Concepts: Correlational Research

The Holmes and Rahe Social Readjustment Rating Scale (SRRS) uses the correlational research method to identify the connection between stress and health. That is, respondents' LCU scores are correlated with the number or frequency of self-reported symptoms indicating health problems. These correlations are typically positive—as LCU scores increase, the number of symptoms increase. Consider all the thousands of studies that have used this scale to correlate stress and illness symptoms: If you were to assign an average correlation coefficient to this body of research, what would be your best guess? How strong do you think the correlation coefficient would be? Why can't the SRRS show a causal relationship between stress and illness? If it were possible to show causation, do you think stress causes illness or illness causes stress?

Hassles

Potential stressors do not always involve major life events. **Daily hassles**—the minor irritations and annoyances that are part of our everyday lives (e.g., rush-hour traffic, lost keys, obnoxious coworkers, inclement weather, arguments with friends or family)—can build on one another and leave us just as stressed as life change events (Figure **5.9.2**) (Kanner, Coyne, Schaefer, & Lazarus, 1981).



(a) (b)

Figure **5.9.2**: Daily commutes, whether (a) on the road or (b) via public transportation, can be hassles that contribute to our feelings of everyday stress. (credit a: modification of work by Jeff Turner; credit b: modification of work by "epSos.de"/Flickr)

Researchers have demonstrated that the frequency of daily hassles is actually a better predictor of both physical and psychological health than are life change units. In a well-known study of San Francisco residents, the frequency of daily hassles was found to be more strongly associated with physical health problems than were life change events (DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982). In addition, daily minor hassles, especially interpersonal conflicts, often lead to negative and distressed mood states (Bolger, DeLongis, Kessler, & Schilling, 1989). Cyber hassles that occur on social media may represent a new source of stress. In one investigation, undergraduates who, over a 10-week period, reported greater Facebook-induced stress (e.g., guilt or discomfort over rejecting friend requests and anger or sadness over being unfriended by another) experienced increased rates of upper respiratory infections, especially if they had larger social networks (Campisi et al., 2012). Clearly, daily hassles can add up and take a toll on us both emotionally and physically.

Watch It

Watch this Ted talk from pediatrician Nadine Burke Harris as she explains the impact of childhood trauma across a lifespan. Trauma and intense stressors during childhood can have both physical and psychological effects that last decades.



You can view the transcript for “How childhood trauma affects health across a lifetime | Nadine Burke Harris” here (opens in new window).

Other Stressors

Stressors can include situations in which one is frequently exposed to challenging and unpleasant events, such as difficult, demanding, or unsafe working conditions. Although most jobs and occupations can at times be demanding, some are clearly more stressful than others (Figure **5.9.3**). For example, most people would likely agree that a firefighter’s work is inherently more stressful than that of a florist. Equally likely, most would agree that jobs containing various unpleasant elements, such as those requiring exposure to loud noise (heavy equipment operator), constant harassment and threats of physical violence (prison guard), perpetual frustration (bus driver in a major city), or those mandating that an employee work alternating day and night shifts (hotel desk clerk), are much more demanding—and thus, more stressful—than those that do not contain such elements. Table 2 lists several occupations and some of the specific stressors associated with those occupations (Sulsky & Smith, 2005).



(a)



(b)

Figure **5.9.3**: (a) Police officers and (b) firefighters hold high-stress occupations. (credit a: modification of work by Australian Civil-Military Centre; credit b: modification of work by Andrew Magill)

Table 2. Occupations and Their Related Stressors

Occupation	Stressors Specific to Occupation (Sulsky & Smith, 2005)
Police officer	physical dangers, excessive paperwork, red tape, dealing with court system, coworker and supervisor conflict, lack of support from the public
Firefighter	uncertainty over whether a serious fire or hazard awaits after an alarm
Social worker	little positive feedback from jobs or from the public, unsafe work environments, frustration in dealing with bureaucracy, excessive paperwork, sense of personal responsibility for clients, work overload
Teacher	Excessive paperwork, lack of adequate supplies or facilities, work overload, lack of positive feedback, vandalism, threat of physical violence
Nurse	Work overload, heavy physical work, patient concerns (dealing with death and medical concerns), interpersonal problems with other medical staff (especially physicians)
Emergency medical worker	Unpredictable and extreme nature of the job, inexperience
Air traffic controller	Little control over potential crisis situations and workload, fear of causing an accident, peak traffic situations, general work environment
Clerical and secretarial work	Little control over job mobility, unsupportive supervisors, work overload, lack of perceived control
Managerial work	Work overload, conflict, and ambiguity in defining the managerial role, difficult work relationships

Although the specific stressors for these occupations are diverse, they seem to share two common denominators: heavy workload and uncertainty about and lack of control over certain aspects of a job. Both of these factors contribute to **job strain**, a work situation that combines excessive job demands and workload with little discretion in decision making or job control (Karasek & Theorell, 1990). Clearly, many occupations other than the ones listed in Table 2 involve at least a moderate amount of job strain in that they often involve heavy workloads and little job control (e.g., inability to decide when to take breaks). Such jobs are often low-status and include those of factory workers, postal clerks, supermarket cashiers, taxi drivers, and short-order cooks. Job strain can have adverse consequences on both physical and mental health; it has been shown to be associated with increased risk of hypertension (Schnall & Landsbergis, 1994), heart attacks (Theorell et al., 1998), recurrence of heart disease after a first heart attack (Aboa-Éboulé et al., 2007), significant weight loss or gain (Kivimäki et al., 2006), and major depressive disorder (Stansfeld, Shipley, Head, & Fuhrer, 2012). A longitudinal study of over 10,000 British civil servants reported that workers under 50 years old who earlier had reported high job strain were 68% more likely to later develop heart disease than were those workers under 50 years old who reported little job strain (Chandola et al., 2008).

Some people who are exposed to chronically stressful work conditions can experience **job burnout**, which is a general sense of emotional exhaustion and cynicism in relation to one's job (Maslach & Jackson, 1981). Job burnout occurs frequently among those in human service jobs (e.g., social workers, teachers, therapists, and police officers). Job burnout consists of three dimensions. The first dimension is exhaustion—a sense that one's emotional resources are drained or that one is at the end of his or her rope and has nothing more to give at a psychological level. Second, job burnout is characterized by depersonalization: a sense of emotional detachment between the worker and the recipients of his or her services, often resulting in callous, cynical, or indifferent attitudes toward these individuals. Third, job burnout is characterized by diminished personal accomplishment, which is the tendency to evaluate one's work negatively by, for example, experiencing dissatisfaction with one's job-related accomplishments or feeling as though one has categorically failed to influence others' lives through one's work.

Job strain appears to be one of the greatest risk factors leading to job burnout, which is most commonly observed in workers who are older (ages 55–64), unmarried, and whose jobs involve manual labor. Heavy alcohol consumption, physical inactivity, being overweight, and having a physical or lifetime mental disorder are also associated with job burnout (Ahola, et al., 2006). In addition, depression often co-occurs with job burnout. One large-scale study of over 3,000 Finnish employees reported that half of the participants with severe job burnout had some form of depressive disorder (Ahola et al., 2005). Job burnout is often precipitated by feelings of having invested considerable energy, effort, and time into one's work while receiving little in return (e.g., little respect or support from others or low pay) (Tatris, Peeters, Le Blanc, Schreurs, & Schaufeli, 2001).

As an illustration, consider Kianna, a nursing assistant who worked in a nursing home. Kianna worked long hours for little pay in a difficult facility. Her supervisor was domineering, unpleasant, and unsupportive; he was disrespectful of Kianna's personal time, frequently informing her at the last minute she must work several additional hours after her shift ended or that she must report to work on weekends. Kianna had very little autonomy at her job. She had little say in her day-to-day duties and how to perform them, and she was not permitted to take breaks unless her supervisor explicitly told her that she could. Kianna did not feel as though her hard work was appreciated, either by supervisory staff or by the residents of the home. She was very unhappy over her low pay, and she felt that many of the residents treated her disrespectfully.

After several years, Kianna began to hate her job. Kianna dreaded going to work in the morning, and she gradually developed a callous, hostile attitude toward many of the residents. Eventually, Kianna began to feel as though she could no longer help the nursing home residents. Kianna's absenteeism from work increased, and one day she decided that she had had enough and quit. Kianna now has a job in sales, vowing never to work in nursing again.

Finally, our close relationships with friends and family—particularly the negative aspects of these relationships—can be a potent source of stress. Negative aspects of close relationships can include adverse exchanges and conflicts, lack of emotional support or confiding, and lack of reciprocity. All these aspects can be overwhelming, threatening to the relationship, and thus stressful. Such stressors can take a toll both emotionally and physically. A longitudinal investigation of over 9,000 British civil servants found that those who at one point had reported the highest levels of negative interactions in their closest relationship were 34% more likely to experience serious heart problems (fatal or nonfatal heart attacks) over a 13–15 year period, compared to those who experienced the lowest levels of negative interaction (De Vogli, Chandola & Marmot, 2007).

Glossary

acute stressors: these are stressful, brief events that may cause a person to experience overwhelming feelings even after the event has ended, though the symptoms usually resolve within a month or so

chronic stressors: these are events that persist over an extended period of time (i.e., long-term unemployment, or imprisonment)

daily hassles: minor irritations and annoyances that are part of our everyday lives and are capable of producing stress

eustress: moderate or normal psychological stress that is perceived to be positive or beneficial

job burnout: a general sense of emotional exhaustion and cynicism in relation to one's job; consists of three dimensions: exhaustion, depersonalization, and sense of diminished personal accomplishment

job strain: work situation involving the combination of excessive job demands and workload with little decision-making latitude or job control

Social Readjustment Rating Scale (SRRS): popular scale designed to measure stress; consists of 43 potentially stressful events, each of which has a numerical value quantifying how much readjustment is associated with the event

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5.10: Post-Traumatic Stress Disorder

Learning Objectives

- Describe the diagnosis and symptoms associated with post-traumatic stress disorder
- Describe risk factors and possible causes of post-traumatic stress disorder

Extremely stressful or traumatic events, such as combat, natural disasters, and terrorist attacks, place the people who experience them at an increased risk for developing psychological disorders such as **post-traumatic stress disorder (PTSD)**. Throughout much of the 20th century, this disorder was called *shell shock* and *combat neurosis* because its symptoms were observed in soldiers who had engaged in wartime combat. By the late 1970s, information had become clear that people who had experienced sexual traumas (e.g., rape, domestic battery, and incest) often experienced the same set of symptoms as did soldiers (Herman, 1997). The term *post-traumatic stress disorder* was developed given that these symptoms could happen to anyone who experienced psychological trauma.



Figure 5.10.1: PTSD is caused by extremely stressful or threatening situations that can happen to anyone.

A Broader Definition of Post-Traumatic Stress Disorder

Post-traumatic stress disorder (PTSD) was listed among the anxiety disorders in previous DSM editions. In DSM-5, it is now listed among a group called trauma-and-stressor-related disorders. For a person to be diagnosed with post-traumatic stress disorder (PTSD), they be must exposed to, witness, or experience the details of a traumatic experience (e.g., a first responder), one that involves "actual or threatened death, serious injury, or sexual violence" (APA, 2013, p. 271). These experiences can include such events as combat, threatened or actual physical attack, sexual assault, natural disasters, terrorist attacks, and automobile accidents. This criterion makes PTSD the only disorder listed in the DSM in which a cause (extreme trauma) is explicitly specified.

Symptoms of PTSD include intrusive and distressing memories of the event, **flashbacks** (states that can last from a few seconds to several days, during which the individual relives the event and behaves as if the event were occurring at that moment [APA, 2013]), avoidance of stimuli connected to the event, persistently negative emotional states (e.g., fear, anger, guilt, and shame), feelings of detachment from others, irritability, proneness toward outbursts, and an exaggerated startle response (jumpiness). For PTSD to be diagnosed, these symptoms must occur for at least one month.



Figure 5.10.2: PTSD, originally called *shell shock*, was diagnosed in wartime soldiers but eventually noted that it can happen to anyone exposed to an extremely stressful or threatening situation(s).

Roughly 7% of adults in the United States, including 9.7% of women and 3.6% of men, experience PTSD in their lifetime (National Comorbidity Survey, 2007), with higher rates among people exposed to mass trauma and people whose jobs involve

duty-related trauma exposure (e.g., police officers, firefighters, and emergency medical personnel) (APA, 2013). Nearly 21% of residents of areas affected by Hurricane Katrina suffered from PTSD one year following the hurricane (Kessler et al., 2008), and 12.6% of Manhattan residents were observed as having PTSD two to three years after the 9/11 terrorist attacks (DiGrande et al., 2008).

Diagnosing PTSD

To diagnose PTSD, an individual must experience at least some symptoms among four different categories for at least a month after a traumatic event: intrusion symptoms (recurrent/distressing memories), avoidance of stimuli related to the event, negative changes in mood, and changes in arousal/reactivity.

1. Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s) occurred:

- recurrent, involuntary, and intrusive distressing memories of the traumatic event(s); in children older than six, there may be frightening dreams without recognizable content.
- recurrent distressing dreams in which the content and/or effect of the dream are related to the traumatic event(s)
- dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the trauma event(s) were recurring (such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings); in children, trauma-specific reenactment may occur in play
- intense or prolonged psychological distress at exposure to internal or external cues symbolizing or resembling an aspect of the traumatic event(s)
- marked physiological reactions to internal or external cues that symbolize or resemble an aspect of the traumatic event(s)

2. Persistent avoidance of stimuli associated with the traumatic event(s), beginning after the traumatic event(s) occurred, as evidenced by one or both of the following:

- avoidance of or efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s)
- avoidance of or efforts to avoid external reminders (people, places, conversations, activities, objects, or situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s)

3. Negative alterations in cognitions and mood associated with the traumatic events(s), beginning or worsening after the traumatic event(s), as evidenced by two (or more) of the following:

- inability to remember an important aspect of the traumatic events(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs)
- persistent and exaggerated negative beliefs or expectations about oneself, others, or the world (e.g., “I am bad,” “no one can be trusted,” “the world is completely dangerous,” “my whole nervousness system is permanently ruined”)
- persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead individuals to blame themselves or others
- persistent negative emotional state (e.g., fear, horror, anger, guilt, or shame)
- markedly diminished interest or participation in significant activities
- feelings of detachment or estrangement from others
- persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings)

4. Marked alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

- irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects
- reckless or self-destructive behavior
- hypervigilance
- exaggerated startle response
- problems with concentration
- sleep disturbances

Risk Factors for PTSD

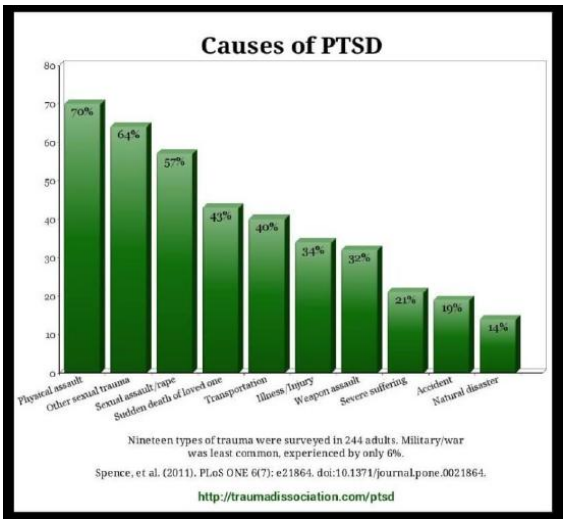


Figure **5.10.3**: Causes of PTSD by type of trauma infographic. Data from Australian adults, 2011.

Of course, not everyone who experiences a traumatic event will go on to develop PTSD; several factors strongly predict the development of PTSD: trauma experience, greater trauma severity, lack of immediate social support, and more subsequent life stress (Brewin, Andrews, & Valentine, 2000). Traumatic events that involve harm by others (e.g., combat, rape, and sexual molestation) carry greater risk than do other traumas (e.g., natural disasters) (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Women are more likely to have been traumatized because of sexual trauma, childhood neglect, and childhood physical abuse. Men are more likely to have been traumatized by natural disaster, life-threatening accident, and physical violence, either witnessed or directed at them. Adolescent boys are more likely to experience accident, physical assault, and witness death/injury; adolescent girls are more likely to experience rape/sexual assault, intimate partner violence, or unexpected death or injury of a loved one. Assaultive violence and witnessing trauma to others is more prevalent among non-Whites when compared to Whites. African-American males are more likely to be exposed to and be victims of violence than males of other races (Kilpatrick, Badour, & Resnick, 2017). A 2012 study found that 27% of corrections officers reported experiencing symptoms of PTSD in the past 30 days. Rates were higher for males (31%) than females (22%) (Spinaris, Denhof, & Kellaway, 2012). A study conducted by Jaegers et al (2019) found that 53.4% of jail correctional officers screened positively for PTSD. PTSD is more prevalent in prison populations than in the general public, with prevalence estimates of 6% in male prisoners and 21% in female prisoners (Facer-Irwin et al, 2019).

Factors that increase the risk of PTSD include female gender (based on biological differences), low SES, low intelligence, personal history of mental disorders, history of childhood adversity (abuse or other trauma during childhood), and family history of mental disorders (Brewin et al., 2000). Personality characteristics such as neuroticism and somatization (the tendency to experience physical symptoms when one encounters stress) have been shown to elevate the risk of PTSD (Bramsen, Dirkzwager, & van der Ploeg, 2000). People who experience childhood adversity and/or traumatic experiences during adulthood are at significantly higher risk of developing PTSD if they possess one or two short versions of a gene that regulates the neurotransmitter serotonin (Xie et al., 2009). This suggests a possible diathesis-stress interpretation of PTSD: its development is influenced by the interaction of psychosocial and biological factors.

Support for Sufferers of PTSD

Research has shown that social support following a traumatic event can reduce the likelihood of PTSD (Ozer, Best, Lipsey, & Weiss, 2003). Social support is often defined as the comfort, advice, and assistance received from relatives, friends, and neighbors. Social support can help individuals cope during difficult times by allowing them to discuss feelings and experiences and providing a sense of being loved and appreciated. A 14-year study of 1,377 American Legionnaires who had served in the Vietnam War found that those who perceived less social support when they came home were more likely to develop PTSD than were those who perceived greater support. In addition, individuals who became involved in the community were less likely to develop PTSD, and they were more likely to experience a remission of PTSD than were those who were less involved (Koenen, Stellman, Stellman, & Sommer, 2003).



Figure **5.10.4**: PTSD was first recognized in soldiers who had engaged in combat. Research has shown that strong social support decreases the risk of PTSD. This person stands at the Vietnam Traveling Memorial Wall. (credit: Kevin Stanchfield)

Behaviorism and Development of PTSD

PTSD learning models suggest that some symptoms are developed and maintained through classical conditioning. The traumatic event may act as an unconditioned stimulus that elicits an unconditioned response characterized by extreme fear and anxiety. Cognitive, emotional, physiological, and environmental cues accompanying or related to the event are conditioned stimuli. These traumatic reminders evoke conditioned responses (extreme fear and anxiety) similar to those caused by the event itself (Nader, 2001). A person who was in the vicinity of the Twin Towers during the 9/11 terrorist attacks and who developed PTSD may display excessive hypervigilance and distress when planes fly overhead; this behavior constitutes a conditioned response to the traumatic reminder (conditioned stimulus of the sight and sound of an airplane). Differences in how conditionable individuals are, help to explain differences in the development and maintenance of PTSD symptoms (Pittman, 1988). Conditioning studies demonstrate facilitated acquisition of conditioned responses and delayed extinction of conditioned responses in people with PTSD (Orr et al., 2000).

Cognition and PTSD

Cognitive factors are important in the development and maintenance of PTSD. One model suggests that two key processes are crucial: disturbances in memory for the event, and negative appraisals of the trauma and its aftermath (Ehlers & Clark, 2000). According to this theory, some people who experience traumas do not form coherent memories of the trauma; memories of the traumatic event are poorly encoded and, thus, are fragmented, disorganized, and lacking in detail. Therefore, these individuals are unable to remember the event in a way that gives it meaning and context. A rape victim who cannot coherently remember the event may remember only bits and pieces (e.g., the attacker repeatedly telling her she is stupid); because she was unable to develop a fully integrated memory, the fragmentary memory tends to stand out. Although unable to retrieve a complete memory of the event, she may be haunted by intrusive fragments involuntarily triggered by stimuli associated with the event (e.g., memories of the attacker's comments when encountering a person who resembles the attacker). This interpretation fits previously discussed material concerning PTSD and conditioning. The model also proposes that negative appraisals of the event ("I deserved to be raped because I'm stupid") may lead to dysfunctional behavioral strategies (e.g., avoiding social activities where men are likely to be present) that maintain PTSD symptoms by preventing both a change in the nature of the memory and a change in the problematic appraisals.

Watch It

Watch this video to review the symptoms of PTSD and consider some possible treatment options.



You can view the transcript for "Posttraumatic stress disorder (PTSD) – causes, symptoms, treatment & pathology" here (opens in new window).

Glossary

flashback: psychological state lasting from a few seconds to several days, during which one relives a traumatic event and behaves as though the event were occurring at that moment

post-traumatic stress disorder (PTSD): experiencing a profoundly traumatic event leads to a constellation of symptoms that include intrusive and distressing memories of the event, avoidance of stimuli connected to the event, negative emotional states, feelings of detachment from others, irritability, proneness toward outbursts, hypervigilance, and a tendency to startle easily; these symptoms must occur for at least one month

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5.11: Acute Stress Disorder and Adjustment Disorders

Learning Objectives

- Describe the diagnostic symptoms of acute stress disorder
- Explain the symptoms and treatment options for adjustment disorders

Acute Stress Disorder

Acute stress disorder is similar to post-traumatic stress disorder (PTSD), but describes a disorder that lasts between three days and one month after a traumatic event. After one month, a diagnosis of acute stress disorder would be considered PTSD. The diagnostic symptoms are similar to that of PTSD, although diagnosis requires at least nine of the following symptoms from any of the five categories of intrusion, negative mood, dissociation, avoidance, and arousal, beginning or worsening after the traumatic event(s) occurred.

Intrusion symptoms



Figure **5.11.1**: Symptoms of PTSD that occur for less than a month are considered acute stress disorder. Children may experience this following any type of stressful event, such as a death, natural disaster, injury, or incidence of abuse.

- recurrent, involuntary, and intrusive distressing memories of the traumatic event(s) (Note: in children, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.)
- recurrent distressing dreams in which the content and/or affect of the dream are related to the events(s) (Note: In children older than six, there may be frightening dreams without recognizable content.)
- dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring; such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings (Note: in children, trauma-specific reenactment may occur in play.)
- intense or prolonged psychological distress or marked physiological reactions in response to internal or external cues that symbolize or resemble an aspect of the traumatic events

Negative Mood

- persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings)

Dissociative Symptoms



Figure **5.11.2**: Avoidance behaviors are typical of someone dealing with acute stress disorder.

- an altered sense of the reality of one's surroundings or oneself (e.g., seeing oneself from another's perspective, being in a daze, time slowing)

- inability to remember an important aspect of the traumatic events(s), typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs

Avoidance symptoms

- efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).
- efforts to avoid external reminders (people, places, conversations, activities, objects, or situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s)

Arousal symptoms

- sleep disturbance (e.g., difficulty falling or staying asleep, restless sleep)
- irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects
- hypervigilance
- problems with concentration
- exaggerated startle response

Watch It

Watch this video to learn more about how acute stress disorder differs from PTSD.



You can view the transcript for "Acute Stress Disorder | Mood Disorders" here (opens in new window).

? Adjustment Disorder

Adjustment disorder (AD) represents an abnormal stress response that is different from normal adaptive reactions (Casey, 2014) that occurs within three months of the onset of a stressor. Adjustment disorder (AD) usually follows a stressful event such as losing a job, ending a relationship, financial conflict, changing environments, feeling overwhelming school or job stress, living in a dangerous situation, death of friends or family, illness, etc. According to DSM-5, adjustment disorder (AD) is characterized by

- (A) emotional or behavioral symptoms in response to an identifiable stressor that
- (B) are of clinical significance and
- (C) do not meet the criteria for another mental disorder, and
- (D) do not represent normal bereavement.

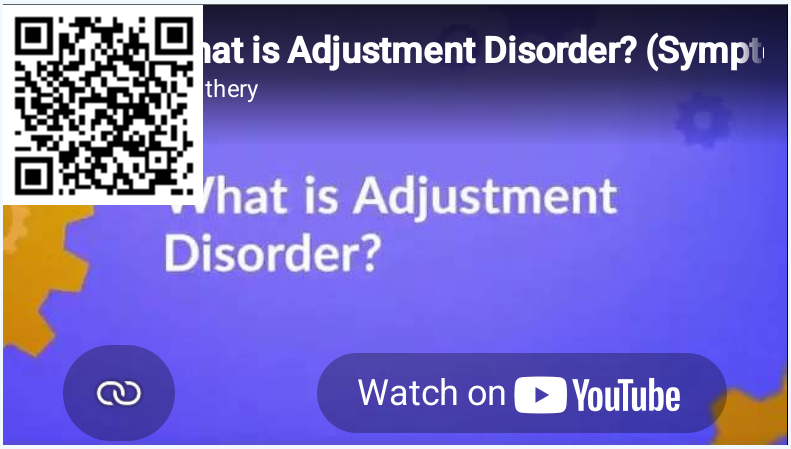
Typically, AD remits within six months if the stressor is terminated; however, a persistent form of AD has been described if the stressor persists (First, 2013; Maercker and Lorenz, 2018). Furthermore, untreated AD poses the risk of persistent AD, and may pave the way for psychiatric disorders other than AD, particularly major depressive disorder and anxiety disorders (O'Donnell et al., 2016).[1]

Some emotional signs of adjustment disorder are sadness, hopelessness, lack of enjoyment, crying spells, nervousness, anxiety, desperation, feeling overwhelmed, thoughts of suicide, performing poorly in school/work, etc. Common characteristics of AD

include mild depressive symptoms, anxiety symptoms, and traumatic stress symptoms or a combination of the three. According to the DSM-5, there are six types of AD, which are characterized by the following predominant symptoms: depressed mood, anxiety, mixed depression and anxiety, disturbance of conduct, mixed disturbance of emotions and conduct, and unspecified. However, the criteria for these symptoms are not specified in greater detail.

Watch It

This video summarizes the six main types of adjustment disorders and discusses some of the symptoms and treatment options for adjustment disorders.



You can view the transcript for "What is Adjustment Disorder? (Symptoms Occurring from a Stressful Life Event)" here (opens in new window).

Unlike major depression, adjustment disorder is caused by an outside stressor and generally resolves once the individual is able to adapt to the situation. The condition is different from an anxiety disorder, which lacks the presence of a stressor, or post-traumatic stress disorder and acute stress disorder, which usually are associated with a more intense stressor and involve different experiences, such as flashbacks. AD is a result of subjective and emotional distress triggered as consequences of a meaningful change in life.[2]

Case Study: Adjustment Disorder

Selena recently moved several states away from home to live on campus as a freshman. She grew up in a tight-knit family in a small, rural community where she had close friends and positive relationships. She'd chosen this university because it hadn't seemed overwhelmingly large but now that she's here, in a new living situation, surrounded by people she doesn't know, she feels overwhelmed anyway. Always a straight-A student, Selena is halfway through the semester and having difficulty with her courses. She had a panic attack right before a mid-term exam and was referred to counseling by her professor. At the counseling center, she talks to a therapist about her feelings of sudden depression, anxiety, and the physical symptoms that she can't explain: chest pain, fatigue, and frequent headaches. Her therapist explains she is likely having some difficulty adapting to and coping with all the adjustments she's experienced (moving, starting college, etc.). After working for a while with her therapist, learning new coping strategies, and getting more involved in the campus community, Selena begins to regain confidence. By spring term, her grades are back up and her symptoms have nearly resolved themselves.

Treatment for Adjustment Disorders

There has been little systematic research regarding the best way to manage individuals with an adjustment disorder. Because natural recovery is the norm, it has been argued that there is no need to intervene unless levels of risk or distress are high. However, for some individuals treatment may be beneficial. AD sufferers with depressive or anxiety symptoms may benefit from treatments usually used for depressive or anxiety disorders. One study found that AD sufferers received similar interventions to those with other psychiatric diagnoses, including psychological therapy and medication.

Like many of the items in the DSM, adjustment disorder receives criticism from a minority of the professional community as well as those in semi-related professions outside the health-care field. First, there has been criticism of its classification. The DSM has been criticized for its lack of specificity of symptoms, behavioral parameters, and close links with environmental factors. Relatively little research has been done on this condition.[3]

An editorial in the *British Journal of Psychiatry* described adjustment disorder as being so "vague and all-encompassing . . . as to be useless,"[4] but it has been retained in the DSM-5 because of the belief that it serves a useful clinical purpose for clinicians seeking a temporary, mild, non-stigmatizing label, particularly for patients who need a diagnosis for insurance coverage of therapy.

Glossary

acute stress disorder: a disorder occurring for three days or one month following a traumatic experience marked by intrusion, negative mood changes, dissociation, avoidance, and changes in arousal

adjustment disorder: a disorder involving a significant stress response occurring within three months of a stressor and marked by significant impairment but not meeting criteria for other disorders

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5.12: Reactive Attachment Disorder and Disinhibited Social Engagement Disorder

Learning Objectives

- Describe healthy attachment and identify the four types of attachment styles
- Differentiate between reactive attachment disorder and disinhibited social engagement disorder

Understanding Attachment

In general public dialogue, there is a lack of consensus about the precise meaning of the term *attachment disorder*, although there is general agreement that such disorders only arise following early adverse caregiving experiences. Some use the term as a blanket term to apply to all complications stemming from underdeveloped attachments in early childhood, but the DSM-5 specifically identifies two attachment disorders—reactive attachment disorder (RAD) and disinhibited social engagement disorder (DSED)—that indicate the absence of either or both the main aspects of **proximity seeking** to an identified attachment figure. This absence can occur either in institutions, or with repeated changes of caregiver, or from extremely neglectful primary caregivers who show persistent disregard for the child's basic attachment needs after the age of six months.

The words **attachment style** or pattern refer to the various types of attachment arising from early care experiences, known as either secure, insecure-avoidant, resistant, or disorganized. Some of these styles are more problematic than others, and, although they are not disorders in the clinical sense, are sometimes discussed under the term *attachment disorder*. For example, disorganized attachment styles are sometimes called attachment disorders because disorganized attachment is seen as the beginning of a developmental trajectory that will take the individual ever further from the normal range, culminating in actual disorders of thought, behavior, or mood. Early intervention for disorganized attachment, or other problematic styles, is directed toward changing the trajectory of development to provide a better outcome later in the person's life.

Attachment Styles

Building on the work of Harry Harlow and his work with monkeys displaying a preference for "cloth mothers" made of soft materials rather than just a "wire mother," John Bowlby developed the concept of **attachment theory**. He defined *attachment* as the affectional bond or tie that an infant forms with the mother (Bowlby, 1969). He believed that an infant must form this bond with a primary caregiver in order to have normal social and emotional development. In addition, Bowlby proposed that this attachment bond is powerful and continues throughout life. He used the concept of a secure base to define a healthy attachment between parent and child (1988). A secure base is a parental presence that gives children a sense of safety as they explore their surroundings. Bowlby said that two things are needed for a healthy attachment: the caregiver must be responsive to the child's physical, social, and emotional needs and the caregiver and child must engage in mutually enjoyable interactions (Bowlby, 1969). For a brief video explanation of attachment theory, watch this video.



Figure **5.12.1**: Mutually enjoyable interactions promote the mother-infant bond. (credit: Peter Shanks)

While Bowlby thought attachment was an all-or-nothing process, Mary Ainsworth's (1970) research showed otherwise. Ainsworth wanted to know if children differ in the ways they bond, and if so, how. To find the answers, she used the **Strange Situation** procedure to study attachment between mothers and their infants (1970). In the Strange Situation, the mother (or primary caregiver) and the infant (age 12–18 months) are placed in a room together. There are toys in the room, and the

caregiver and child spend some time alone in the room. After the child has had time to explore their surroundings, a stranger enters the room. The mother then leaves her baby with the stranger. After a few minutes, she returns to comfort her child.

Based on how the infants responded to the separation and reunion, Ainsworth identified three types of parent-child attachments: secure, avoidant, and resistant (Ainsworth & Bell, 1970). A fourth style, known as disorganized attachment, was later described (Main & Solomon, 1990).

The most common type of attachment—also considered the healthiest—is called **secure attachment**. In this type of attachment, the toddler prefers their parent over a stranger. The attachment figure is used as a secure base to explore the environment and is sought out in times of stress. Securely attached children were distressed when their caregivers left the room in the Strange Situation experiment, but when their caregivers returned, the securely attached children were happy to see them. Securely attached children have caregivers who are sensitive and responsive to their needs.



Figure 5.12.2: In secure attachment, the parent provides a secure base for the toddler, allowing him to securely explore his environment. (credit: Kerry Ceszyk)

With **avoidant attachment (or insecure-avoidant)**, the child is unresponsive to the parent, does not use the parent as a secure base, and does not care if the parent leaves. The toddler reacts to the parent the same way they react to a stranger. When the parent does return, the child is slow to show a positive reaction. Ainsworth theorized that these children were most likely to have a caregiver who was insensitive and inattentive to their needs (Ainsworth, Blehar, Waters, & Wall, 1978).

In cases of **resistant attachment (or insecure-resistant/ambivalent)**, children tend to show clingy behavior, but then they reject the attachment figure's attempts to interact with them (Ainsworth & Bell, 1970). These children do not explore the toys in the room, appearing too fearful. During separation in the Strange Situation, they become extremely disturbed and angry with the parent. When the parent returns, the children are difficult to comfort. Resistant attachment is thought to be the result of the caregivers' inconsistent level of response to their child.

Finally, children with **disorganized attachment** behaved oddly in the Strange Situation. They freeze, run around the room in an erratic manner, or try to run away when the caregiver returns (Main & Solomon, 1990). This type of attachment is seen most often in kids who have been abused or severely neglected. Research has shown that abuse disrupts a child's ability to regulate their emotions.

While Ainsworth's research has found support in subsequent studies, it has also met criticism. Some researchers have pointed out that a child's temperament may have a strong influence on attachment (Gervai, 2009; Harris, 2009), and others have noted that attachment varies from culture to culture, a factor that was not accounted for in Ainsworth's research (Rothbaum, Weisz, Pott, Miyake, & Morelli, 2000; van Ijzendoorn & Sagi-Schwartz, 2008).

Attachment styles vary in the amount of security and closeness felt in the relationship and they can change with new experiences. The type of attachment fostered in parenting styles varies by culture as well. For example, German parents value independence and Japanese mothers are typically by their children's sides. As a result, the rate of insecure-avoidant attachments is higher in Germany and insecure-resistant attachments are higher in Japan. These differences reflect cultural variation rather than true insecurity, however (van Ijzendoorn and Sagi, 1999). Keep in mind that methods for measuring attachment styles have been based on a model that reflects middle-class, U.S. values and interpretation. Newer methods for assessing attachment styles involve using a Q-sort technique in which a large number of behaviors are recorded on cards and the observer sorts the cards in a way that reflects the type of behavior that occurs within the situation.

Watch It

Watch this video to better understand Mary Ainsworth's research and to see examples of how she conducted the experiment.



You can view the transcript for "The Strange Situation | Mary Ainsworth, 1969 | Developmental Psychology" here (opens in new window).

Attachment Disorders

The DSM-5, classifies **reactive attachment disorder (RAD)** as a stressor-related disorder caused by social neglect during childhood (meaning a lack of adequate caregiving). Reactive attachment disorder (RAD) is characterized by markedly disturbed and developmentally inappropriate ways of relating socially in most contexts. It can take the form of a persistent failure to initiate or respond to most social interactions in a developmentally appropriate way, known as the "inhibited form." The uninhibited form, which manifests as a lack of inhibitions or externalizing behavior are consistent with **disinhibited social engagement disorder (DSED).** The DSM-4 listed disinhibited social engagement disorder (DSED) as a subtype of RAD, but the latest version lists it as a distinct disorder. The disorders have similar patterns of insufficient care during early development, but those diagnosed with disinhibited social engagement disorder (DSED) appear more outgoing, as compared to the internalizing, withdrawn behavior, and depressive symptoms present in RAD.

Key Takeaways: RAD and DSED

Table 1. Comparing Reactive Attachment Disorder and Disinhibited Social Engagement Disorder

Reactive Attachment Disorder	Disinhibited Social Engagement Disorder
A. A consistent pattern of inhibited, emotionally withdrawn behavior toward adult caregivers, manifested by both of the following: • The child rarely or minimally seeks comfort when distressed. • The child rarely or minimally responds to comfort when distressed. B. A persistent social and emotional disturbance characterized by at least two of the following: • minimal social and emotional responsiveness to others • limited positive affect • episodes of unexplained irritability, sadness, or fearfulness that are evident even during nonthreatening interaction with adult caregivers	A. A pattern of behavior in which a child actively approaches and interacts with unfamiliar adults and exhibits at least two of the following: • reduced or absent reticence in approaching and interacting with unfamiliar adults • overly familiar verbal or physical behavior (that is not consistent with culturally sanctioned and with age-appropriate social boundaries) • diminished or absent checking back with adult caregiver after venturing away, even in unfamiliar settings • willingness to go off with an unfamiliar adult with minimal or no hesitation B. The behaviors in Criterion A are not limited to impulsivity (as in attention-deficit/hyperactivity disorder) but include socially disinhibited behavior.

Understanding Attachment Disorders

RAD and DSED arise from a failure to form normal attachments to primary caregivers in early childhood. Such a failure could result from severe early experiences of neglect, abuse, abrupt separation from caregivers between the ages of six months and three years, frequent changes of caregivers, or a lack of caregiver responsiveness to a child's communicative efforts. Not all, or even a majority of such experiences, result in the disorder. The diagnosis is differentiated from pervasive developmental disorder or developmental delay and from possible comorbid conditions such as intellectual disability, all of which can affect attachment behavior. The criteria for a diagnosis of a reactive attachment disorder are very different from the criteria used in the assessment or categorization of attachment styles such as insecure or disorganized attachment.

Signs and Symptoms

Children with RAD are presumed to have grossly disturbed internal working models of relationships that may lead to interpersonal and behavioral difficulties in later life. There are few studies of long-term effects, and there is a lack of clarity about the presentation of the disorder beyond the age of five years.[1] However, the opening of orphanages in Eastern Europe following the end of the Cold War in the early 1990s provided opportunities for research on infants and toddlers brought up in very deprived conditions. Such research broadened the understanding of the prevalence, causes, mechanism, and assessment of disorders of attachment and led to efforts from the late 1990s onwards to develop treatment and prevention programs and better methods of assessment. Mainstream theorists in the field have proposed that a broader range of conditions arising from problems with attachment should be defined beyond current classifications.

Pediatricians are often the first health professionals to assess and raise suspicions of RAD in children with the disorder. The initial presentation varies according to the child's developmental and chronological age, although it always involves a disturbance in social interaction. Infants up to about 18–24 months *may* present with **non-organic failure to thrive** and display abnormal responsiveness to stimuli. Laboratory investigations will be unremarkable barring possible findings consistent with malnutrition or dehydration, while serum growth hormone levels will be normal or elevated.

The core feature is severely inappropriate social relating by affected children. This can manifest itself in three ways:

1. Indiscriminate and excessive attempts to receive comfort and affection from any available adult, even relative strangers (older children and adolescents may also aim attempts at peers). This may oftentimes appear as a denial of comfort from anyone as well.
2. Extreme reluctance to initiate or accept comfort and affection, even from familiar adults, especially when distressed.

3. Actions that otherwise would be classified as conduct disorder, such as mutilating animals, harming siblings or other family members, or harming themselves intentionally.

While RAD occurs in relation to neglectful and abusive treatment, automatic diagnoses on this basis alone cannot be made, as children can form stable attachments and social relationships despite marked abuse and neglect. However, the instances of that ability are rare.

There is as yet no universally accepted diagnostic protocol for reactive attachment disorder. Often a range of measures is used in research and diagnosis. Recognized assessment methods of attachment styles, difficulties or disorders include the Strange Situation Procedure (devised by developmental psychologist Mary Ainsworth), the separation and reunion procedure and the Preschool Assessment of Attachment,[16] the Observational Record of the Caregiving Environment, the Attachment Q-sort and a variety of narrative techniques using stem stories, puppets or pictures. For older children, actual interviews such as the Child Attachment Interview and the Autobiographical Emotional Events Dialogue can be used. Caregivers may also be assessed using procedures such as the Working Model of the Child Interview.,

Other research also uses the Disturbances of Attachment Interview (DAI) developed by Smyke and Zeanah (1999). The Disturbances of Attachment Interview (DAI) is a semi-structured interview designed to be administered by clinicians to caregivers. It covers 12 items, namely “having a discriminated, preferred adult,” “seeking comfort when distressed,” “responding to comfort when offered,” “social and emotional reciprocity,” “emotional regulation,” “checking back after venturing away from the caregiver,” “reticence with unfamiliar adults,” “willingness to go off with relative strangers,” “self-endangering behavior,” “excessive clinging,” “vigilance/hypercompliance,” and “role reversal.” This method is designed to pick up not only RAD but also the proposed new alternative categories of disorders of attachment.

Comorbidity

In children ages zero to three, research has shown high rates of comorbidity with RAD and speech/language developmental disorders as well as autism. Additionally, ADHD was found to be common among those with RAD, in children aged seven to nine and for non-psychological disorders, respiratory and digestive diseases were found to be fairly common as well.

Causes of RAD

Although increasing numbers of childhood mental health problems are being attributed to genetic defects, reactive attachment disorder is by definition based on a problematic history of care and social relationships. Abuse can occur alongside the required factors, but on its own does not explain attachment disorder. It has been suggested that types of **temperament,** or constitutional response to the environment, may make some individuals susceptible to the stress of unpredictable or hostile relationships with caregivers in the early years. In the absence of available and responsive caregivers, it appears that most children are particularly vulnerable to developing attachment disorders.

While similar abnormal parenting may produce the two distinct forms of the disorder, inhibited (RAD) and disinhibited (DSED), studies show that the abuse and neglect was far more prominent and severe in the cases of disinhibited social engagement disorder.

Treatment

Mainstream treatment and prevention programs that target RAD and other problematic early attachment behaviors are based on attachment theory and concentrate on increasing the responsiveness and sensitivity of the caregiver, or if that is not possible, placing the child with a different caregiver. Play therapy and expression therapy, even nonverbal therapy, can be used to help children form attachments.

Mainstream practitioners and theorists have presented significant criticism of the diagnosis and treatment of attachment disorders within the controversial form of psychotherapy commonly known as attachment therapy. **Attachment therapy** has a scientifically unsupported theoretical base and uses diagnostic criteria or symptom lists markedly different from criteria under ICD-10 or DSM-5, or to attachment behaviors. A range of treatment approaches are used in attachment therapy, some of which are physically and psychologically coercive, and considered to be antithetical to attachment theory.

The Controversy of Attachment Therapy

Attachment therapy is not the same as attachment-based family therapy. These are two different concepts that must not be confused.

In brief, when children are unable to securely attach to a primary caregiver, they may develop an attachment disorder, or at the very least, have difficulty understanding proper relationships. These issues need to be addressed in a sensitive therapeutic setting and treatment such as ABFT can be helpful. Therapists trained in ABFT understand attachment theory and work with families to improve their bonding via psychotherapy. For more information on ABFT, watch this short video.

Attachment therapy (also known as Coercive Restraint Therapy) is much different. In sum, it is used by therapists to treat children diagnosed with RAD in an attempt to repair attachment issues that began with earlier caregivers. Rising in popularity in the 80s and 90s, it's often used to treat adoptive and foster care children and still in practice today. The idea behind it is this: the tantrums; rage; and inappropriate, uncontrollable behavior often seen in children with RAD is a result from early neglect and mistreatment, thus they must be provoked in order to "release" this rage and form better attachments, which result in affection and obedience.

Three main techniques are holding, rebirthing, and therapeutic foster parenting. In holding therapy, the therapist (or trained parent) restrains the child in an attempt to trigger the rage. In rebirthing, the child is wrapped and/or covered in pillows and held down until they can force themselves out, hence the term "rebirth." The latter is illegal in Colorado after 10-year-old Candace Newmaker suffocated to death during a "rebirthing." Therapy may also involve placing children in a totally controlled and structured environment, like a boot-camp, in attempts to break them into becoming more obedient.

Unfortunately, attachment therapy has resulted in injuries—even death—for many children who've been through this treatment. For a list of real examples of victims, visit Advocates for Children in Therapy. Research into its efficacy has concluded that there's no empirical evidence showing this type of intervention to be valid.[2]

In 2005, the American Academy of Child and Adolescent Psychiatry laid down guidelines (devised by N.W. Boris and C.H. Zeanah) based on its published parameters for the diagnosis and treatment of RAD.[3] Recommendations in the guidelines include the following:

1. Clinicians should ensure the child has an emotionally available attachment figure.
2. While RAD is a diagnosis for symptoms displayed by a child, it's also important to assess the caregiver's attitude about the child.
3. Treatment should focus on creating positive interactions with caregivers.
4. Children with RAD should also display aggressive or oppositional behavior should be provided with additional treatment.

Undoubtedly, identifying RAD and DSED while overlooking other common disorders may be detrimental due to missed treatment of other treatable disorders. Yet, neglecting to properly diagnose RAD or DSED may be equally damaging, as an incomplete or incorrect case formulation may reduce the likelihood of adequate developmental support for a child. In cases with RAD or DSED, caregivers may need specialized interventions, aiming to enhance their sensitivity, emotional availability, and commitment to the child.[4]

Pharmacological interventions are under scrutiny and there is little supporting evidence to show efficacy. For one, it is difficult to assess in very young children with ongoing development of the liver and the kidneys. It has been observed that psychotropic medications in children have shorter half-lives (Kearns et al.), which would require use of higher doses for body weight in comparison to adults for same plasma level. Unfortunately, that in turn significantly increases the likelihood and severity of potential side effects. There is also a question on effects of early exposure to antipsychotics on neurodevelopment. In particular, in the first three years of life, there are many changes in developing brains, such as an increase in synaptic density, pruning, and an increase in neuronal myelination to list just a few.[5]

If at all possible, prevention is key. Once a diagnosis of RAD or DSED is established, then measures should be taken to provide as much emotional stability as possible, while considering appropriate therapeutic interventions.

Research and RAD

Research by Rachel Pritchett and others took a closer look at the nuanced nature of RAD and its comorbidity with other disorders. They described their results as follows:[6]

> This study was part of a population-based study investigating the prevalence of reactive attachment disorder (RAD) in 6–8 years old children from a sector of a UK city characterized by high levels of deprivation.
>
> We described the characteristics of 22 children with a suspected, borderline, or definite diagnosis of RAD. We found that they have a high level of comorbidity with other disorders, had lower IQs than population norms, had a higher level of disorganized attachment than has been found in general population studies, more problem behaviors, and had lower social skills than would be found in the general population. These findings are in line with previous research about children coming from institutions. This study shows that those children in the general population with RAD also have these additional problems, providing further evidence that a multidisciplinary approach is needed when working with these children.

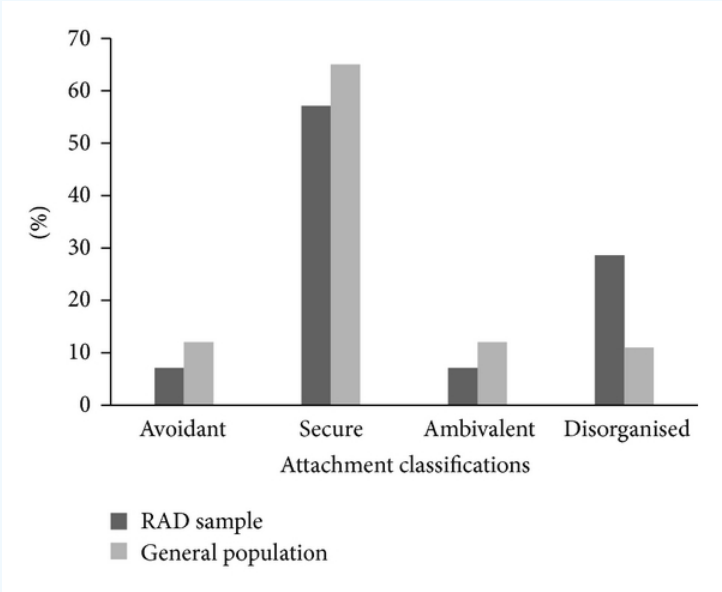


Figure **5.12.3**: Results from a study of those with RAD showing that many display secure attachment, while a larger percentage have a history of disorganized attachment when compared to the general population.

> We found that over half of our sample had a secure attachment; this offers support to the growing body of research showing that RAD and insecure attachment are not the same thing. We did, however, also find that there was a higher rate of disorganized attachment than would be found in the general population. This is not surprising as research has previously shown that those with a history of maltreatment have a greater chance of having a disorganized attachment in later development.

Glossary

attachment style: these refer to the various types of attachment arising from early care experiences, which are secure, insecure-avoidant, resistant, or disorganized

attachment theory: the belief that young children need to develop a secure and/or healthy bond with at least one primary caregiver for normal social and emotional development

attachment therapy: a pseudoscientific child mental health intervention intended to treat attachment disorders through techniques like holding or rebirthing

proximity seeking: these are behaviors (by the infant) that are intended to keep the caregiver close (or whoever the infant is attached to); this is usually exhibited by actions such as crawling/walking to the person or climbing in their lap, lifting their arms to be held, etc.

avoidant attachment (or insecure-avoidant): the child is unresponsive to the parent, does not use the parent as a secure base, and does not care if the parent leaves

disinhibited social engagement disorder (DSED): a stressor-related disorder caused by childhood neglect and considered the "uninhibited form" of RAD, which manifests as a lack of inhibitions or externalizing behavior

disorganized attachment: this type of attachment is seen most often in kids who have been abused or severely neglected; research has shown that abuse disrupts a child's ability to regulate their emotions

reactive attachment disorder (RAD): a stressor-related disorder caused by social neglect during childhood (meaning a lack of adequate caregiving) that manifests as inhibited, emotionally withdrawn behavior toward adult caregivers

resistant attachment (or insecure-resistant/ambivalent): children tend to show clingy behavior, but then they reject the attachment figure's attempts to interact with them

secure attachment: considered the "healthiest" style, a child with this attachment has a caregiver that is sensitive and responsive to their needs; the parent is a "secure base" for them and the child will seek them when in stress

strange situation: a study conducted in 1970 in which researchers observed attachment between mothers and their infants

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5.13: Introduction to Treating Obsessive-Compulsive Disorder and Stressor-Related Disorders

What you'll learn to do: explain psychological perspectives and treatment methods for OCD and PTSD



There is hope for those who suffer from a diagnosis of OCD, PTSD, or their related disorders. Various treatments exist to help improve quality of life, both psychotherapeutic interventions, as well as pharmaceuticals to help balance brain chemistry. In this section, we'll take a closer look at some of the perspectives and methods used to read OCD and PTSD.

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5.14: Psychological Perspectives on OCD and Trauma-Related Disorders

Learning Objectives

- Describe views on OCD from the major psychological perspectives
- Describe views on stress-related disorders from the major psychological perspectives

The Biological Perspective

Past research of family and twin studies suggest that OCD has a moderate genetic component. OCD is five times more frequent in the first-degree relatives of people with OCD than in people without the disorder (Nestadt et al., 2000); the concordance rate of OCD among identical twins is around 57% and 22% for fraternal twins (Bolton, Rijsdijk, O'Connor, Perrin, & Eley, 2007). Studies have implicated about two dozen potential genes that may be involved in OCD; these genes regulate the function of three neurotransmitters: serotonin, dopamine, and glutamate (Pauls, 2010).

Furthermore, research also suggests that there may be differences in the brain structures of those with OCD versus those without. The orbitofrontal cortex is part of a series of brain regions that, collectively, is called the OCD circuit; this circuit consists of several interconnected regions that influence the perceived emotional value of stimuli and the selection of both behavioral and cognitive responses (Graybiel & Rauch, 2000). People with OCD show more brain activity in these areas when asked to do things that provoke symptoms, such as looking at crookedly hung pictures or touching a dirty counter; because of this, it is believed that abnormalities in these regions may be the cause for OCD symptoms.

For PTSD, and other stress-related disorders, researchers have found that genetics *and* environment play an important role in one's risk for developing PTSD due to trauma; estimates for the genetic influences for PTSD account for 30–73% of vulnerability for the disorder. Some of the hereditary traits or genes that affect the increased risk for developing PTSD are common to those linked to a higher rate of susceptibility to major depression, anxiety, and panic disorder. One study actually identified 17 gene variants that were associated with PTSD.[1]

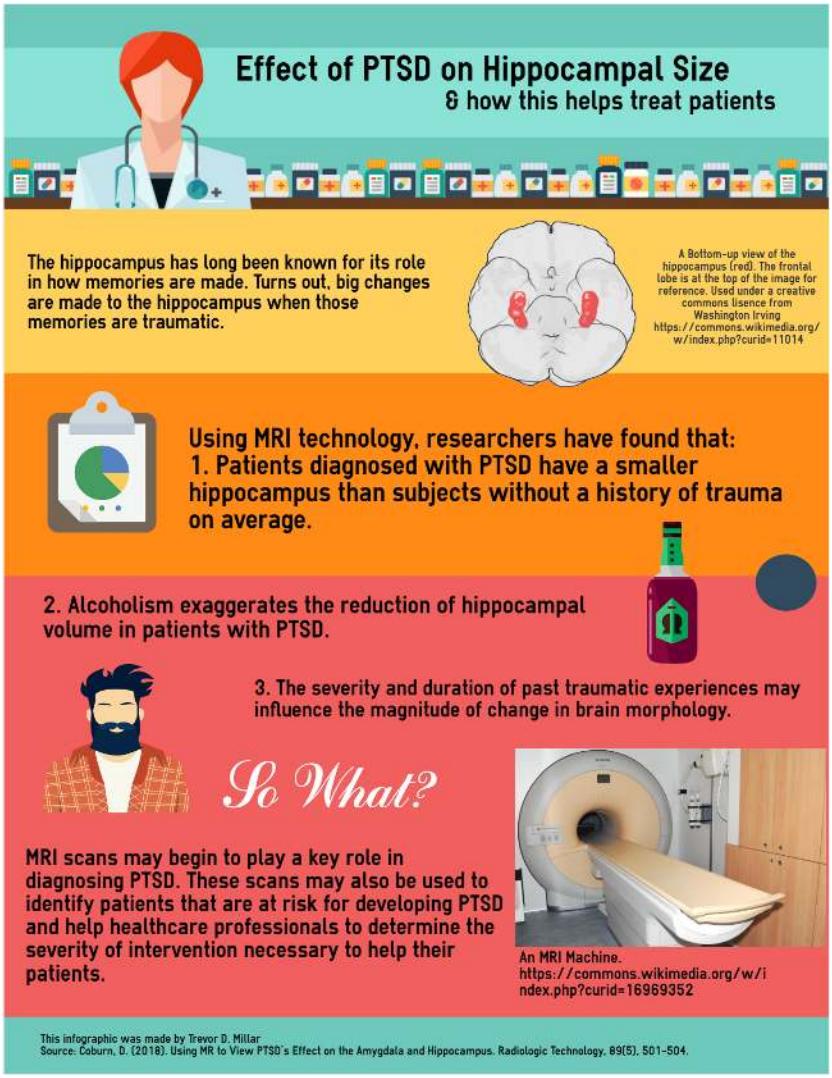


Figure 5.14.1: Recent studies have shown that smaller hippocampal volume has been correlated with PTSD.

Genes may not be the only thing affecting the risk factor for PTSD. In fact, the very structures of a person's brain, like in the case of OCD, may have something to do with this. Recent studies have shown that smaller hippocampal volume has been correlated with PTSD—the relationship between PTSD and smaller volume in this region of the brain is associated with negative effects of stress hormones; what's interesting is that smaller hippocampal volume was found not only in the brains of combat veterans diagnosed with PTSD but *also* in their twins who hadn't experienced combat, and thus never developed the disorder. This biological factor implies that someone could be predisposed to developing this disorder should they experience a traumatic event.[2]

The Sociocultural Perspective

The sociocultural perspective looks at a person's behaviors and symptoms in the context of their culture and background.

Some cultures in which religion and customs are held to a very high standard may be connected to OCD; these factors are so ingrained and so varied that they can influence the onset, outcome, and response to treatment for OCD. Among Egyptians, for example, there is an emphasis on religious rituals that involve repeated phrases—it appears that the prevalence of religious obsessions and compulsions are hard to distinguish between those with OCD and those simply practicing their religion. Types of obsessions among study participants in Egypt and Jerusalem were similar, mostly associated with religion and cleanliness or purity, whereas themes among Indian and British participants related more to being orderly and avoiding aggressiveness.[3] Understanding social influences on behavior is important; for example, how might you tell the difference between someone who is working hard to "purify" themselves via the strict adherence of religious rituals—possibly washing their hands frequently, praying, or performing specific tasks several times a day—and someone who has a diagnosis of OCD?

In regards to PTSD, different cultures and belief systems may help or hinder the prevalence of the disorder. Cultural beliefs may influence an individual's personal meanings of trauma and his or her attempts to come to terms with trauma memories in helpful and unhelpful ways. The meanings linked to PTSD show cultural variations: in individualistic cultures appraisals about a vulnerable or inadequate self are common; in collectivistic cultures appraisals about social functioning or evaluation by others. Cultural beliefs may also influence the reactions of significant others and the community and can thus facilitate or impede recovery from trauma.[4]

In a 2013 study that examined the prevalence and determinants of PTSD and post-traumatic stress symptoms following the 2004 tsunami in Indonesia and India, Rajkumar et al. credited the absence of avoidance symptoms among tsunami survivors to the cohesive social bonds and religious rituals that facilitated talking about the event and collective mourning in the community. Religious and spiritual beliefs may also influence trauma susceptibility.[5] Research has indicated that locus of control plays a role in susceptibility to PTSD. While an **internal locus of control**—the belief that the outcomes of events in your life are in your own control—seems to provide a protective role in people subjected to traumatic experiences, an external locus, in which events in one's life are believed to be controlled by outside forces, tends to instill a sense of helplessness and lack of control in victims of trauma. In India, where the role of religion and strong beliefs in karma are highly predominant, society is generally characterized by an external locus of control.[6]

The Psychodynamic Perspective

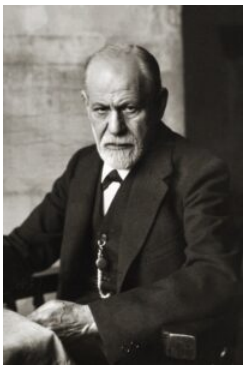


Figure 5.14.2: According to Freud, our personality develops from a conflict between two forces: our biological aggressive and pleasure-seeking drives versus our internal (socialized) control over these drives.

Psychodynamic theory suggests that there are three areas at work in determining a person's personality and behavior: the id, superego, and ego. The id represents our unconscious, primitive drives or urges. The superego is what we learn as we grow—societal rules, what is or isn't acceptable—our "moral compass" essentially, and when we fail to live up to the standards of the superego, we may experience shame or guilt. The ego is the most rational part of our personality that balances the two by satisfying the desires of the id in a realistic way.

When there is an imbalance of these three interacting parts of our personality, the psychodynamic approach says that we are likely suffering from neurosis (a tendency to experience negative emotions), anxiety disorders, or unhealthy behaviors. For example, a person who is dominated by their id might be narcissistic and impulsive. A person with a dominant superego might be controlled by feelings of guilt and deny themselves even socially acceptable pleasures; conversely, if the superego is weak or absent, a person might become a psychopath. An overly dominant superego might be seen in an over-controlled individual whose rational grasp on reality is so strong that they are unaware of their emotional needs, or, in a neurotic who is overly defensive (overusing ego defense mechanisms).

To view OCD in light of this perspective, obsessions and compulsions are, in actuality "unconscious conflict" that you may be trying to stop or cope with, and this conflict comes from a clash between the id (usually a sexual or aggressive urge) and the superego (the desire to do the right thing and follow socially acceptable behavior). A good way to illustrate this would be to picture in your mind's eye that common image of a devil and an angel on each shoulder, whispering in each ear. A person with OCD is stuck between "right" and "wrong," no longer able to function within normal limits. When these two (id and superego) are at odds, especially in extreme circumstances, a person is forced to manage them via repeated task(s), such as hand-washing or frequent checking.[7]

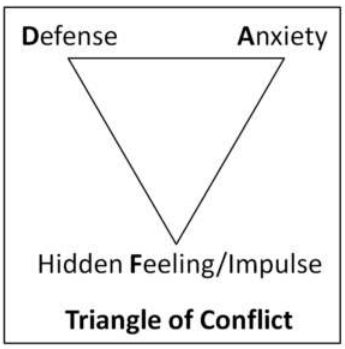


Figure 5.14.3: Researcher David Malan proposed the triangle of conflict, which holds that true feelings can be blocked by defenses and anxieties.

The psychodynamic perspective attempts to bring repressed traumatic experiences out of the unconscious, where they are perceived as more toxic, and address them by helping patients to understand the relationship between hidden impulses, anxiety, and defense mechanisms.[8]

The Humanistic Perspective

From the humanistic perspective, with its emphasis on the potential for good and self-actualization in all people, those suffering OCD and PTSD are likely undergoing an existential crisis. People with these disorders are attempting to work toward self-actualization but finding themselves "stuck" and unable to move forward. For those with OCD, it manifests in obsessions and compulsions, they are trapped in a vicious, never-ending cycle, unable to move higher on Maslow's hierarchy. These obsessions and complusions could stem from a basic need that wasn't met in the early years, or from a feeling of insecurity. Similarly, those with PTSD have had a traumatic experience, one in which a basic need was severely threatened (i.e., their life), and therefore, they may feel helpless to move forward because of the difficulty with never truly feeling safe.

The Cognitive Perspective

Cognitive psychology focuses on studying our thoughts and their relationship to our experiences and our actions. The cognitive model of OCD, as proposed by Paul Salkovskis, proposes that everyone will experience intrusive thoughts from time to time, but those with OCD falsely assume that "(i) thinking about an action is the same as doing it; (ii) failing to prevent harm is morally equivalent to causing harm; (iii) responsibility for harm is not diminished by extenuating circumstances; (iv) failing to ritualize in response to a thought about harm is the same as an intention to harm; and (v) one should exercise control over one's thoughts.[9] The idea that thinking about an action is the same as doing it, is known as thought-action fusion. If someone with OCD has the sudden thought of robbing a bank, their mind acts as if they've already done it—the thought, in turn, is just as bad as the action itself.[10] Cognitive behavioral therapy (CBT) attempts to address and end the patterns of thinking that connect a thought with a compulsion.

In regards to PTSD, cognitive theory takes the following approach: traumatic events disrupt a person's perspective on life and the world around them, which ultimately creates a negative viewpoint that has lasting damage, resulting in chronic stress symptoms seen in PTSD. When you can't help but view the world as an unpredictable, dangerous place, it's hard to move forward without high levels of stress. People with PTSD may have a hard time believing that anyone or anything can be trusted, to the point where they may even struggle with their own self-worth.[11]

The Behavioral Perspective

According to the behavioral approach, for someone with OCD, a neutral event comes to elicit fear or stress when it is paired with a stressful event (an unconditioned stimulus, or UCS). The conditioned stimulus (CS) could be a thought or something physical, such as a doorknob or a bathroom, and for someone with OCD, the stress caused by the conditioned stimulus is only reduced through compulsions.[12] Unfortunately, these rituals a person with OCD uses to neutralize the problem actually reinforce this notion that the obsession *is* bad and they must do something to stop it, which results in becoming "stuck" in the constant cycle of obsession compulsion.

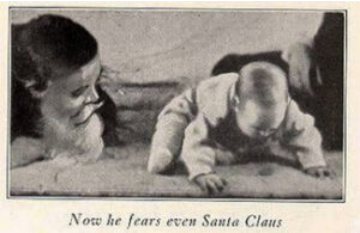


Figure **5.14.4**: The Little Albert Experiment: Through classical conditioning, Little Albert came to fear furry things, including Watson (the researcher) in a Santa Claus mask.

PTSD learning models suggest that some symptoms are developed and maintained through classical conditioning. The traumatic event may act as an unconditioned stimulus that elicits an unconditioned response characterized by extreme fear and anxiety. Cognitive, emotional, physiological, and environmental cues accompanying or related to the event are conditioned stimuli. These traumatic reminders evoke conditioned responses (extreme fear and anxiety) similar to those caused by the event itself (Nader, 2001). Differences in how conditionable individuals are help to explain differences in the development and maintenance of PTSD symptoms (Pittman, 1988).

For example, if a combat veteran witnessed the death of a fellow soldier due to a roadside bomb, he may have fear of even benign objects on the side of the road upon return home (where there is little danger of being exposed to roadside bombs); though this initial response may be a normal reaction to extreme stress/trauma, if the fear continues, (i.e., he swerves around trash in the road or avoids driving altogether) and/or if it remains heightened (every time he passes by objects in the road—a trash bag on the curb, a discarded box—he experiences flashbacks), then this persistent conditioned stimulus that is no longer part of a true environmental danger becomes PTSD symptoms.

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5.15: Treatment for OCD and Related Disorders

Learning Objectives

- Examine methods used in treating OCD and related disorders, including habit reversal training

Treating OCD

OCD is typically treated with medication, psychotherapy, or a combination of the two. Although most patients with OCD respond to treatment, some patients continue to experience symptoms.

Medication

Serotonin reuptake inhibitors (SRIs), which include selective serotonin reuptake inhibitors (SSRIs), are used to help reduce OCD symptoms. SRIs often require higher daily doses in the treatment of OCD than of depression and may take eight to 12 weeks to start working, but some patients experience more rapid improvement.

If symptoms do not improve with these types of medications, research shows that some patients may respond well to antipsychotic medication. Although research shows that an antipsychotic medication may help manage symptoms for people who have both OCD and a tic disorder, research on the effectiveness of antipsychotics to treat OCD is mixed.

Psychotherapy

Psychotherapy can be an effective treatment for adults and children with OCD. Research shows that certain types of psychotherapy, CBT and other related therapies (e.g., habit reversal training) can be as effective as medication for many individuals.

Watch It

This video shows how clinical psychologist and the clinical director of the Centre for Anxiety Disorders and Trauma (CADAT), Paul Salkovskis, worked with Karen Robinson in treating her OCD with CBT.



You can view the transcript for "A guide to Cognitive Behavioural Therapy (CBT)" here (opens in new window).

Exposure and Response Prevention

Research also shows that a type of CBT called **exposure and response prevention** is effective in reducing compulsive behaviors in OCD, even in people who did not respond well to SRI medication. In ERP, the patient first identifies the things that trigger obsessive thoughts, such as external situations like people, places, and thoughts. Therapists then examine the distress caused in those situations and what the feared outcome will be if they do not perform rituals to lower the distress. For example, a person may lock the door ten times after coming inside because they fear an intrusion. The patient will work with the clinician to discuss this fear, evaluate and rank its severity, and practice imagining ways to tolerate the distress. Eventually, the patient is exposed to the anxiety-provoking situation (keeping the doors unlocked) without performing a response and they learn strategies to cope with the

distress.[1] Ultimately, ERP means spending time in the very situation that triggers compulsions (e.g. touching dirty objects) but then being prevented from undertaking the usual resulting compulsion (e.g. handwashing).

As with most mental disorders, treatment is usually personalized and might begin with either medication or psychotherapy, or with a combination of both. For many patients, ERP is the add-on treatment of choice when SRIs or SSRIs medication does not effectively treat OCD symptoms or vice versa for individuals who begin treatment with psychotherapy.

Link to Learning

Visit this site to watch a video explanation of ERP.

Treating Hoarding Disorder

Cognitive-behavioral therapy (CBT) is a commonly implemented therapeutic intervention for compulsive hoarding. As part of cognitive behavior therapy, the therapist may help the patient to

- discover why he or she is compelled to hoard.
- learn to organize possessions in order to decide what to discard.
- develop decision-making skills.
- declutter the home during *in-home visits* by a therapist or professional organizer.
- gain and perform relaxation skills.
- attend family and/or group therapy.
- be open to trying psychiatric hospitalization if the hoarding is serious.
- have periodic visits and consultations to keep a healthy lifestyle.

CBT usually involves exposure and response prevention to situations that cause anxiety and cognitive restructuring of beliefs related to hoarding. Furthermore, research has also shown that certain CBT protocols have been more effective in treatment than others. CBT programs that specifically address the motivation of the sufferer, organization, acquiring new clutter, and removing current clutter from the home have shown promising results. CBT typically involves in-home work with a therapist combined with between-session homework, the completion of which is associated with better treatment outcomes. Research on internet-based CBT treatments for the disorder (where participants have access to educational resources, cognitive strategies, and chat groups) has also shown promising results both in terms of short- and long-term recovery.

Habit Reversal Training

Habit reversal training (HRT) has the highest rate of success in treating trichotillomania and other BFRRs. **Habit reversal training** (**HRT**) is a "multicomponent behavioral treatment package originally developed to address a wide variety of repetitive behavior disorders."[2] It consists of five components: awareness training, competing response training, contingency management, relaxation training, and generalization training. HRT has also been shown to be a successful adjunct to medication as a way to treat trichotillomania. With HRT, the individual is trained to learn to recognize their impulse to pull and also teach them to redirect this impulse. In comparisons of behavioral versus pharmacologic treatment, CBT (including HRT) has shown significant improvement over medication alone. HRT has also proven effective in treating children. Biofeedback**,** cognitive-behavioral methods, and hypnosis may improve symptoms. Acceptance and Commitment Therapy (ACT) is also demonstrating promise in trichotillomania treatment. A systematic review from 2012 found tentative evidence for "movement decoupling," a self-help variant of HRT.[3]

Glossary

exposure and response prevention: a type of CBT treatment in which a patient first identifies the things that trigger obsessive thoughts, such as external situations like people or places and thoughts, then spending time facing those fears to lower stress levels

habit reversal training: a multicomponent behavioral treatment package developed to address a wide variety of repetitive behavior disorders that focuses on re-training responses to habits

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5.16: Treatments for PTSD

Learning Objectives

- Describe cognitive behavioral therapy treatment methods for PTSD, including eye movement desensitization and reprocessing

Treating PTSD

Common therapeutic methods for PTSD include psychotherapy, CBT, and drug therapy. Treatments for PTSD aim to decrease stress and hyperarousal when recalling highly traumatic events. Several therapies reduce positive and negative symptoms by altering pathways involved in memory reconsolidation or by interfering with the hypothalamus-pituitary-adrenal axis. Detection and intervention techniques usually involve monitoring and stabilizing cortisol levels to control stress responses in patients. Many treatments have shown varied results among patients with different subtypes of PTSD.

Cognitive-Behavioral Therapy for PTSD

Perhaps the most widely practiced approach for treating PTSD is cognitive behavioral therapy (CBT). The goal is that patients identify the sources of their trauma and cope with them thereby eliminating negative emotional reactions associated with the memories.[1] In general, around eight to 12 sessions are done with the patients prolonging about 60 to 90 minutes each, during which therapists ask challenging questions about the source of the stress-inducing memory. CBT has been shown to be successful in a wide variety of PTSD subtypes of different age groups.

Clients usually meet with their therapists for sessions once or twice per week. Each session begins with the individual closing their eyes and recalling the trauma as if the event is presently occurring. The clients retell all their emotions and physical senses while the event happened. Clients are also given homework assignments to complete, usually watching video footage of previous sessions. Studies have observed symptom reduction after about two weeks of intervention therapy.[2]

Eye Movement Desensitization & Reprocessing (EMDR)

Eye movement desensitization and reprocessing (EMDR) is a form of psychotherapy developed by Francine Shapiro starting in 1988 in which the person being treated is asked to recall distressing images. The therapist then directs the patient in one type of bilateral stimulation, such as side-to-side eye movements or hand tapping. According to the 2013 World Health Organization practice guideline, “This therapy [EMDR] is based on the idea that negative thoughts, feelings, and behaviors are the result of unprocessed memories. The treatment involves standardized procedures that include focusing simultaneously on (a) spontaneous associations of traumatic images, thoughts, emotions and bodily sensations and (b) bilateral stimulation that is most commonly in the form of repeated eye movements.”[3]

Eye movement desensitization and reprocessing (EMDR) is included in several evidence-based guidelines for the treatment of PTSD. As of 2020, the American Psychological Association lists eye movement desensitization and reprocessing (EMDR) as an evidence-based treatment for PTSD, but stresses that “the available evidence can be interpreted in several ways” and notes there is debate about the precise mechanism by which EMDR appears to relieve PTSD symptoms with some evidence EMDR may simply be variety of exposure therapy.[4]

Watch It

Watch this video to learn more about EMDR.



You can view the transcript for "Can Moving Your Eyes Re-Code Your Memories?" here (opens in new window).

This form of treatment is a hybrid of many different types of interventions such as imaginal exposure as well as free association. The EMDR session commences with the patient remembering their traumatic memories while simultaneously looking at external visual or auditory cues, often with the therapist moving their finger across the individual's visual field. The patients' eyes move in the direction of the cue. However, several studies have suggested that the eye movements or sensory stimulation is not essential to the effectiveness of the therapy. Further research is necessary to discover the influence and importance of the bilateral stimulation in eliminating symptoms.

As a result, current research studies are finding new therapies that cater to specific types of PTSD patients. For instance, **virtual reality therapy (VRT)** is being specifically used for treating combat-related PTSD .

Virtual Reality Therapy



Figure **5.16.1**: VRT is a way to successfully treat combat-related PTSD.

Virtual reality therapy (VRT) is currently being used as treatment for various psychological disorders. Ranging from anxiety disorders to stroke rehabilitation, virtual reality therapy (VRT) is being utilized to eliminate debilitating symptoms associated with each of these situations. VRT consists of viewing a computerized combat or war scene on a visor screen. The computerized scenes closely resemble a real combat environment experienced by soldiers during deployment. Common among soldiers and military personnel arriving from overseas missions, combat-related PTSD is efficiently treated with this computerized therapy. After making a script of their traumatic memories, veterans undergo around six sessions of VRT. In contrast to other therapeutic methods, VRT has been shown to be extremely efficient in reducing negative symptoms such as avoidance and guilt. Since drug strategies and other therapies mainly treat positive symptoms and are inefficient in decreasing the negative symptoms, VRT is revolutionary for PTSD treatment. As a result, the patients are able to converse with the therapist while actively re-experiencing their memories. Due to this study, computerized intervention strategies have become increasingly common for treating PTSD.

Even though PTSD was only added to the DSM thirty years ago, the disease had been documented in literature many years before. For instance, Henry IV, in Shakespeare's historical play of the same name, displayed several symptoms of someone experiencing PTSD. More recently, numerous films in popular culture feature main characters with PTSD. In particular, Rambo, the famous protagonist played by Sylvester Stallone in the *First Blood* film series, suffers from PTSD. In the 2009 movie *Brothers*, actor

Tobey Maguire portrays a young soldier who was a prisoner of war while in Afghanistan. The character displays avoidance, withdrawal, and paranoia, which are all indicators of the disease. Over the years, CBT accompanied with drug therapy has been accepted as the first-line intervention for the disease.

Watch It

Virtual Iraq allows soldiers to re-experience and cope with their traumatic memories. The scenes, sounds, and even odors transmitted through the different gadgets closely resemble those experienced in Iraq.



You can view the transcript for “PTSD Therapy Session at VA using Virtual Iraq” here (opens in new window).

Glossary

eye movement desensitization and reprocessing: a person is asked to recall distressing events or images while a therapist then directs the patient in one type of bilateral stimulation, such as side-to-side eye movements or hand tapping, with the goal of reprocessing and coping with the stress

virtual reality therapy (VRT): a type of exposure therapy that relies on virtual reality or technology to create simulations to help confront and treat mental disorders, such as PTSD

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5.17: Memory Reconsolidation for PTSD and Anxiety Disorders

Learning Objectives

- Explain how memory reconsolidation is used to treat PTSD or anxiety disorders

Problems with memory are at the core of many psychological disorders. For example, people suffering from both clinical-level depression and PTSD often have difficulty remembering details of specific memories, especially for happy experiences. The memory distress is called overgeneralized autobiographical memory (OGM). A therapist might ask a depressed person showing overgeneralized autobiographical memory (OGM) to recall a recent happy experience. The depressed person might answer, "When I was visiting my friends last weekend," but then be unable to recall or describe any particular events or interactions during that visit that were enjoyable or rewarding. For another example, people suffering from OCD experience less confidence in the accuracy of memories they retrieve than people without the disorder. This uncertainty about memory can lead to obsessive thoughts about whether they turned off the stove or paid the electric bill when it was due. People with OCD also tend to show a bias to retrieve threatening memories. Nearly every major psychological disorder you will study in this course has some aspect of memory that is either a symptom or a process that maintains the disorder or more often both.

You may also have learned by now that remembering and thinking about past events—either recent or long ago—is the basis of most forms of psychotherapy. The psychodynamic therapy developed by Sigmund Freud is almost entirely based on remembering actual experiences or recent dreams. Even newer forms of therapy, like Cognitive-Behavioral Therapy (CBT), involve a great deal of memory work.

It may seem that research laboratories in universities and medical centers are a long way from psychotherapists' offices, but professional therapists keep up with new developments in basic research and they often collaborate with researchers in bridging the gap between new theories and the application of those theories in the real world. A great example of the basic research-applied research connection is the development of therapies that can change the emotional impact of some memories without erasing or otherwise distorting them.

Memory Consolidation



Figure **5.17.1**: Older theories on memory said that memories were stored like printed books, but new research suggests that they are not so set.

Until the early part of the 21st century, most people thought of memories—particularly memories of personal events, technically known as autobiographical memories—as mental representations that become relatively stable and unchangeable very quickly. We knew that memories do not stabilize immediately, however, because brain trauma (e.g., a concussion) or certain drugs could interfere with people's ability to recall events immediately before the trauma or administration of the drug. The neural processes that occur between an experience and the stabilization of the memory for that experience is called **consolidation**. Consolidation is complex, with some consolidation processes taking minutes to hours and other consolidation processes taking weeks, months, or even years. For the rest of this reading, we will concern ourselves with the quick part of consolidation that occurs in the hours and days immediately after an experience.

The idea of consolidation does not rule out forgetting. Memories can fade—that is**,** lose details—or become impossible to retrieve. In the reading on memory, you also learned that misinformation that a person hears shortly after an event can be incorporated into the memory. But the idea is that the final version of the memory is fixed once it has consolidated within a few hours. This late-20th century theory says that memory is like a book. When it is first printed, the ink must dry (the consolidation process that takes up to

a few hours), but when that has occurred, the contents of the book don't change. The ink may fade over time or you may have trouble finding it in your library, but the contents of the book never change, no matter how often you pull it out to read it.

Reconsolidation

Around the beginning of the current century, our understanding of memory was shaken by new research, first in animal labs, but later with humans.[1] The study that initially caught the attention of memory scientists was a study using rats as subjects by Karim Nader, Glenn Schafe, and Joseph Le Doux of New York University in the year 2000. The research taught their animals a fear memory by pairing a particular sound with a mild, but unpleasant shock using classical conditioning.[2] The researchers found that they could change a memory that had already been consolidated if they did just the right things at just the right time.

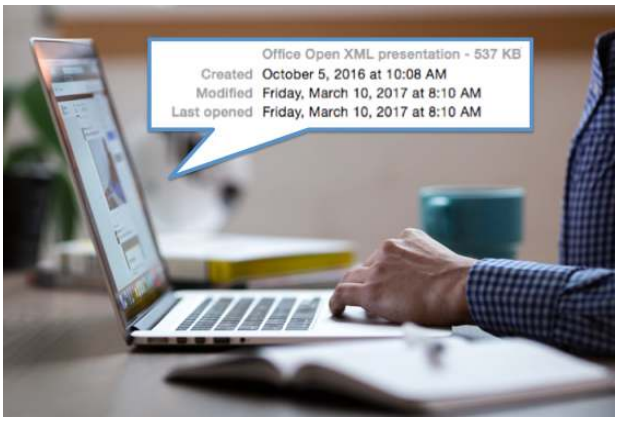


Figure **5.17.2**: Research on consolidation supports the idea that memory is saved somewhat like a computer file: the original file is there, but that file can be modified and re-saved.

What Nader and his colleagues found was that memories become open to changes for a brief period of time when they are retrieved. For a few hours, the changed memories can be disrupted (e.g., by trauma to the brain, by drugs, and by other means), but once they have **reconsolidated**, they become the new version of the memory. This newer theory of memory says that our memories are not really like books, which don't change after the print has dried. Now **memory is more like a computer file that is updated without saving the original**. You originally create the memory (consolidation) and store it away. When you retrieve the memory, you can change some information in the file, but this new version now becomes the memory. Many researchers believe we don't have a backup version of the original memory. All we have is the new, modified memory of the event.

Reconsolidation: In the Basic Research Lab

The theory of reconsolidation has changed the way we think about the stability and accuracy of memories, but a scientific theory must be more than interesting or novel: it must be supported by careful research. There is now an impressive body of research about reconsolidation. We have already mentioned experiments with rats by Karim Nader and his colleagues, but we will go into more detail on a study by Elizabeth Phelps, a highly respected psychologist who is one of the leaders on modern neuroscience of emotion and cognition. The study we will discuss is by Dr. Phelps, Daniella Schiller (now an associate professor of psychiatry at Mt. Sinai hospital in New York), and some of their colleagues.

You may remember learning about classical conditioning. Ivan Pavlov discovered how classical conditioning works when he trained dogs to salivate when they heard a bell (click **HERE** to review classical conditioning). Dr. Phelps and her colleagues classically conditioned volunteer research participants to fear a shock. They allowed this learning (i.e., the conditioned fear response) to consolidate, and then figured out the way to eliminate the fear response.

To start, we are going to look at what happened in one of the control conditions, which will give you an idea about what normally happens with this kind of fear learning.

Note: we used emoticons in the exercise above, but the actual dependent variable in the study was a physiological measure of fear: skin conductance. When we are scared, our sweat glands respond by producing sweat, sometimes a lot, sometimes a little, but always some. The perspiration on our skin changes the way that electricity moves across the skin, and these changes can be detected and measured, even if the changes are very subtle. This is the skin conductance response (called SCR). Detection of changes in skin conductance is simple, requiring only some detectors on your fingers, and it is painless.

Day 2 has been successful. The person is no longer afraid of the yellow box. But, we're still not quite done. We need to test for spontaneous recovery. Let's go to Day 3.

Spontaneous recovery is one of the big problems with extinction training. You can get rid of a response for a while, but the response can return over and over again. According to the researchers—Dr. Phelps and Dr. Schiller—the problem may be that the person has two memories: one where the yellow box means a shock is coming, and another that means the yellow box equates to no shock. These two memories are both available, so when a yellow box happens to retrieve the first memory (yellow box = shock), the fear response returns.

So how can we change the first memory without creating a new memory? Here is a second condition in the experiment. We're going to call this group the "10-Minute Group," and we'll explain why shortly.

The first step involves the same process as in the control group and involves conditioning the subject to "fear" a yellow box.

Now let's go to Day 2. Remember from the control group that Day 2 involves extinction, which is the process of unlearning the fear response. But for this new group, we're going to try something different to see if we can replace their original memory without creating a new memory.

Memory Reactivation

This time, before we begin the process of extinction, we are going to get the person to *think* about the shock experience—that is, we want them to *retrieve the full fear memory*—before they start extinction. Once the full memory is reactivated, there is a 10-minute delay, and then the subjects go through the same extinction trials that the Control Group subjects experienced on Day 2.

This reintroduction of the yellow box on Day 2 is the one event that did not happen in the control condition you read about earlier. It turns out that this reactivation step is crucial to preventing spontaneous recovery.

> After the extinction process has been completed on Day 2, the question is this: will the person show spontaneous recovery of the fear response on Day 3? If they do show spontaneous recovery, then our new procedure (reinstatement of the memory on Day 2) has failed to produce the change in memory that we hoped for.

> The last step is to again test for spontaneous recovery.

> So far, the experimenters have shown that fear can be learned (Day 1), extinguished (Day 2), and then spontaneously recover (Day 3) for the control condition. By contrast, the reactivation condition shows that, if the full memory is activated on Day 2 just before extinction, then the fear response does not spontaneously recover.

However, our journey is not quite complete. The experimenters claim that a reactivated memory acts like a new memory: it is open to change for only a brief time and then it becomes stable again. So the Day 2 extinction process should only work to change the original memory for a short while—at most, a few hours. If the memory is reactivated, but extinction is delayed for a few hours, then the memory should not be changed because it has had time to reconsolidate.

The final experiment tests this idea. The only difference between this new group and the last group is the time delay on the second day. Rather than waiting 10 minutes between reactivating the memory and extinction, the experimenters waited six hours. After six hours, the fear memory should no longer be active and extinction should not change the memory.

This experiment is important because it serves as a control to help us determine if "rewriting a memory" is actually the correct interpretation of the results. In this experiment, the memory is reactivated (just like in the 10-minute group), but the memory is then allowed to deactivate over a six-hour delay. If there is no spontaneous recovery in this condition, then rewriting memory is not a particularly convincing explanation for the results. If there is spontaneous recovery of fear, then the theory that we are actually rewriting a memory is more convincing.

So let's see what happens.

> The procedure on Day 3 is the same for all three groups, but the responses are different. Participants in the two control conditions (control group and six-hour group) both act the same: they both show spontaneous recovery of the fear response.

Those in the reconsolidation treatment condition (the 10-minute group), however, show no spontaneous recovery of the fear response.

Interpreting Results

Let's take another look at the results of the study by Schiller, Phelps, and their colleagues. The *y*-axis on the graph below shows the skin conductance response of the subjects. Higher values indicate higher levels of fear.[3] You will be adjusting the lines, so move them up to indicate more fear and down to indicate less fear. The *x*-axis shows the end of Day 1, after successful fear conditioning, and the first trial on Day 3, when spontaneous recovery is being measured.

We have placed the circles for Day 1 in their correct positions. The fact that they sit high on the graph reflects the fact that all three groups of participants were successfully conditioned on Day 1 to fear the yellow box. The differences among the three lines are not statistically significant.[4] Your task is to grab the circles on the right and move them to the appropriate positions for the results of the experiment. You can move them up or down or leave them where they are. When you have entered your solution, you can look at the actual results.

Remember, spontaneous recovery means that the person returns to the fear level they had learned earlier, on Day 1. No spontaneous recovery means that the fear response (high levels of skin conductance) had been eliminated. Lower fear is shown if the dots get closer to the *x*-axis.

Watch It

This video shows the experimenters you have been reading about (Daniella Schiller and Elizabeth Phelps) discussing their work and you will even see a reenactment of part of the study. The video does not include many of the technical details you just went through, but it shows some of the procedures and the researchers give you some idea of the implications of their work.



You can view the transcript for "Erasing fear memories" here (opens in new window).

What is the practical value of this research?

At the very end of the video, you heard Dr. Phelps (from an interview in 2009) explain the potential for turning this research into a useful procedure for therapists:

So, you know, at this point, how this works in the clinic is going to be all speculation. But what this data suggests might happen in the future is: if you come into the clinic with a fear-related disorder, like a phobia or PTSD, if we can understand how these memories are re-stored when they are retrieved, much as we did in this study, we then may be able to time our therapeutic

interventions in such a way where we aren't creating new learning that's overriding those earlier memories but actually rewriting them, in a sense. If we can time that correctly so we can target these mechanisms, perhaps we'd have a more effective, long-lasting outcome.

One of the goals of this research, then, is to give therapists a way of working with memory disorders. Of course, rather than creating a fear as the researchers did, therapists work with people who experience debilitating fear-related memories that came from experiences, often traumatic ones, in their lives. The therapist's job is to help the person overcome the disabling experiences of fear. In most cases, they would like to reduce the emotional impact of the experience, which is part of the memory itself, without actually changing the facts that are remembered.

This application of reconsolidation theory to therapy is already underway. Here are the basic steps in this therapy:

- REINSTATEMENT: Have the person retrieve the memory. Be sure that the retrieval is emotionally powerful. If the person avoids fully reactivating the memory in its complete painful form, then reduction of the emotional impact will be impossible. The emotion may be fear or anxiety or some other strong negative response.
- REDUCTION OF EMOTIONAL IMPACT: While the memory is active and painful, the therapist acts to reduce its impact. There are two approaches to this, using the example of a phobia (irrational fear) to illustrate the method:
  - EXTINCTION OF THE FEAR RESPONSE: In a therapy session, a person with a phobia (e.g., fear of spiders or dogs or heights) might (a) have the fear response reactivated (have them stand near a spider or dog or on a high perch) and then, (b) through continuous or repeated exposure to the source of fear with support from the therapist and experience of no bad consequences (not getting bitten or not falling), show a reduction of the fear response.
  - DRUGS THAT BLOCK FEAR MEMORY: In a therapy session, a person with a phobia (e.g., fear of spiders or dogs or heights) might (a) have the fear response reactivated (have them stand near a spider or dog or on a high perch) and then, (b) the person is given propranolol, a drug that inhibits the storage of emotional aspects of a memory.
- REPETITION ACROSS DAY OR WEEKS: For a deep-seated problem, it is very unlikely that a single session will eliminate or even substantially reduce the automatic negative emotional response. The process of reinstatement followed by either extinction or drug intervention is necessary for effective treatment.

Watch It

This video explains some of the remarkable work of Merel Kindt, a therapist and memory researcher. Dr. Kindt uses the drug propranolol, which interferes with the reconsolidation of the fear aspect of a memory, though it does not prevent the person from feeling fear during the training session nor does it interfere with the person's memory for the events that occurred.

As you can see from the video, therapists can now use the new insights coming from research on reconsolidation of memory to help in their treatment of people with disorders that include memory dysfunctions. The video showed treatment of a phobia, but reconsolidation therapy has also been used with some success with people suffering from PTSD.

The reconsolidation research discussed is this exercise is just one example of the relationship between basic research taking place in scientific laboratories and practical application of discoveries about the mind and brain in the real world. Psychology in the 21st century owes a great deal to researchers in the 20th century, but old dogma is constantly being updated and even overthrown in favor of better ideas that come from deeper understanding of the causes of human behavior.

Glossary

consolidation: the neural processes that occur between an experience and the stabilization of the memory

reconsolidation: the process of replacing or disrupting a stored memory with a new version of the memory

1. The basic idea of reconsolidation and some relevant research had been around for decades, but the idea did not grab hold and the supporting research was not sufficient until the last two decades. ↵
2. If you've forgotten what classical conditioning is, we will review it when we discuss a human version of Nader, Schafe, and Le Doux's study. ↵

3. The actual dependent variable was a bit more complicated than the simple measure of skin conductance suggested in the figure. Consult the original study if you need to know the exact way that skin conductance was measured. ↵
4. In real research, we seldom find exactly the same averages for different conditions. There is always some natural variability. We use statistical tests to be sure that these typical differences are not greater than we would expect by chance. ↵

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5.18: Case Studies- OCD and PTSD

Learning Objectives

- Identify OCD and PTSD in case studies

Case Study: Mauricio



As a teenager, Mauricio had always tried to live up to every standard (academic, religious, familial) that was placed upon him. Before every exam, he lined up his pencils, erasers, and notebooks exactly the same way, each two fingers apart. He felt a strong urge to complete this task because if he didn't, he would fail his exam. His classmates thought it was funny and one day, a boy sitting next to him purposely knocked his pencils to the floor. When this occurred, Mauricio was certain he would fail and became suddenly, very ill, excusing himself to the bathroom where he vomited his lunch. As the years progressed, his habits increased. He was too afraid of being a failure at work and the only thing that gave him confidence was arranging his desk the same every day, tying the same knot in his tie, and readjusting his seat ten times exactly. His dating life was a mess; no woman could deal with his oddities at home—rearranging cutlery, turning light switches on and off . . . and this left him lonely and sad. When he became too depressed to leave his home, a friend advised him to see a counselor and offered to drive him to his appointments.

Case Study: Cho



Cho was thirteen when her home caught fire during a terrible lightning storm. A firefighter managed to help her escape through the window but her mother was trapped in a room on the other side of the house. Almost two years later, Cho still has night terrors. She hears her mother's screams in her sleep and wakes up in a cold sweat. She barely sleeps for more than a couple hours at a time and constantly walks around the house, checking doors and windows and ensuring the stove is off. Whenever there's a lightning storm, she hides in her room until it's over. This tendency caused difficulty at school one day when a storm grew overhead; she ran to the bathroom in a state of panic. Her father has considered homeschooling her for her senior year because she is constantly exhausted, her grades are plummeting, and she refuses to socialize, often sitting in the farthest corner of the classroom and avoiding crowds.

? Treatment

Possible treatment considerations for Cho may include CBT or eye movement desensitization and reprocessing (EMDR). This could also be coupled with pharmaceutical treatment, such as anti-anxiety medication or anti-depressants to help alleviate symptoms. Cho will need a trauma therapist who is experienced in working with adolescents. Other treatment that may be helpful is starting family therapy as well to ensure everyone is learning to cope with the trauma and work together through the painful experience.

Link to Learning

To read more about the ongoing issues of PTSD in violent-prone communities, read this article about a mother and her seven-year-old with PTSD.

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5.19: Putting It Together- Obsessive Compulsive Disorder and Stressor Related Disorders

In this chapter, you covered OCD and related disorders, such as hoarding disorder and trichotillomania. You've also reviewed PTSD and other stressor disorders, including attachment disorders. By this point, you should have a basic, yet firm grasp on understanding how each is unique in its presentation of symptoms, how they affect people, and common treatment methods. You also covered a section on various psychological viewpoints of these disorders. Take some time to consider what resonated with you the most. Is there a perspective that you felt closely aligned with your personal beliefs or ideas regarding mental illness? Take a look at some of the popular treatment methods as well: what might be an underlying psychological perspective of a disorder that prompted the research into a now-popular method for treating it? It's important to take time to reflect on learning and think critically about concepts.

As you reflect on what you've gleaned from this module, spend a moment reviewing the disorders discussed in this module:

- **obsessive-compulsive disorder (OCD):** this disorder is characterized by the tendency to experience intrusive and unwanted thoughts and urges (obsession) and/or the need to engage in repetitive behaviors or mental acts (compulsions) in response to the unwanted thoughts and urges.
- body dysmorphic disorder (BDD): this disorder involves an excessive preoccupation with an imagined defect in physical appearance.
- hoarding disorder (HD): this disorder (also known as "pathological collecting") is marked by persistent difficulty in parting with possessions, regardless of their actual value or usefulness.
- trichotillomania: also known as hairpulling disorder or compulsive hairpulling; a psychological disorder characterized by a long-term urge that results in the pulling out of one's hair.
- excoriation disorder: an obsessive-compulsive spectrum disorder in which symptoms involve the repeated urge or impulse to pick at one's own skin to the extent that either psychological or physical damage is caused.
- post-traumatic stress disorder (PTSD): experiencing a profoundly traumatic event leads to a constellation of symptoms that include intrusive and distressing memories of the event, avoidance of stimuli connected to the event, negative emotional states, feelings of detachment from others, irritability, proneness toward outbursts, hypervigilance, and a tendency to startle easily; these symptoms must occur for at least one month, though often they last much longer.
- acute stress disorder (ASD): similar to PTSD, this disorder occurs following a traumatic experience marked by intrusion, negative mood changes, dissociation, avoidance, and changes in arousal. However, it often resolves within one month; if it lasts longer it becomes PTSD.
- adjustment disorders (AD): this disorder involves a significant stress response occurring within three months of a stressor and marked by significant impairment but not meeting criteria for other disorders.
- reactive attachment disorder (RAD): this is a stressor-related disorder caused by social neglect during childhood (meaning a lack of adequate caregiving) that manifests as inhibited, emotionally withdrawn behavior toward adult caregivers.
- disinhibited social engagement disorder (DSED): this stressor-related disorder is caused by childhood neglect and is considered the "uninhibited form" of RAD, which manifests as a lack of inhibitions or externalizing behavior.

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CHAPTER OVERVIEW

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6.2: Introduction to Dissociative Disorders
6.3: Understanding Dissociation
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6.5: Dissociative Identity Disorder
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6.1: Why It Matters- Dissociative and Somatic Symptom Disorders

Why learn about dissociative and somatic symptom disorders?



Figure **6.1.1**: Not all people who daydream suffer from dissociative disorder.

Think about the last time you were daydreaming. Perhaps it was while you were driving or attending class. Some portion of your attention was on the activity at hand, but most of your conscious mind was wrapped up in fantasy. Now imagine that you could not control your daydreams. What if they intruded your waking consciousness unannounced, causing you to lose track of reality or experience the loss of time. Imagine how difficult it would be for you. Imagining is similar to what people who suffer from dissociative disorders may experience. Of the many disorders listed in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) (American Psychiatric Association, 2013), dissociative disorders rank as among the most puzzling and controversial. Dissociative disorders encompass an array of symptoms ranging from memory loss (amnesia) for autobiographical events to changes in identity and the experience of everyday reality (American Psychiatric Association, 2013).

In psychopathology, dissociation happens when thoughts, feelings, and experiences of our consciousness and memory do not align well with each other. This module provides an overview of dissociative disorders, including the definitions of dissociation, its origins and competing theories, and their relation to traumatic experiences and sleep problems. We will also discuss somatic symptom disorders, which are disorders related to bodily illnesses and injuries that cannot be explained by medical conditions.

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6.2: Introduction to Dissociative Disorders

What you'll learn to do: examine and explain dissociative disorders



In this section, you will learn about the differences between dissociative fugue, dissociative amnesia, depersonalization/derealization, and dissociative identity disorder. You will consider possible reasons for how these conditions develop (exploring possible underlying causes such as trauma or sleep issues). Each one of these disorders has a unique presentation of symptoms.

Watch It

Watch this video for a comprehensive overview of the things we'll cover in this section.



You can view the transcript for "Dissociative disorders – causes, symptoms, diagnosis, treatment, pathology" here (opens in new window).

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6.3: Understanding Dissociation

Learning Objectives

- Define symptoms of dissociation

Defining Dissociation



Figure **6.3.1**: People often think of dissociation as multiple personality disorder. While multiple personality disorder, called dissociative identity disorder, is an example of identity confusion and alteration consistent with dissociation, dissociation also includes amnesia, fugue, depersonalization, and derealization.

The DSM-5 defines dissociation as “a disruption and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control and behavior” (American Psychiatric Association, 2013, p. 291). A distinction is often made between dissociative states and dissociative traits (e.g., Bremner, 2010; Bremner & Brett, 1997). State dissociation is viewed as a transient symptom, which lasts for a few minutes or hours (e.g., dissociation during a traumatic event). Trait dissociation is viewed as an integral aspect of personality. Dissociative symptoms occur in patients but also in the general population, like you and me. Therefore, dissociation has commonly been conceptualized as ranging on a continuum, from nonsevere manifestations of daydreaming to more severe disturbances typical of dissociative disorders (Bernstein & Putnam, 1986). The dissociative disorders include the following:

- **dissociative amnesia:** extensive forgetting typically associated with highly aversive events
  - **dissociative fugue:** short-lived reversible amnesia for personal identity, involving unplanned travel or “bewildered wandering.” Dissociative fugue is not viewed as a separate disorder but is a feature of some, but not all, cases of dissociative amnesia.
- **depersonalization/derealization disorder:** feeling as though one is an outside observer of one’s body
- **dissociative identity disorder (DID):** experiencing two or more distinct identities that recurrently take control over one’s behavior (American Psychiatric Association, 2000)

Although the concept of dissociation lacks a generally accepted definition, the Structural Clinical Interview for DSM-4 Dissociative Disorders (SCID-D) (Steinberg, 2001) assesses five symptom clusters that encompass key features of the dissociative disorders. These clusters (also found in the DSM-5) are

- depersonalization,
- derealization,
- dissociative amnesia,
- identity confusion, and
- identity alteration.

Depersonalization refers to a “feeling of detachment or estrangement from one’s self.” Imagine that you are outside of your own body, looking at yourself from a distance as though you were looking at somebody else. Maybe you can also imagine what it would be like if you felt like a robot, deprived of all feelings. These are examples of depersonalization. Derealization is defined as “an alteration in the perception of one’s surroundings so that a sense of reality of the external world is lost” (Steinberg, 2001, p. 101).

Imagine that the world around you seems as if you are living in a movie or looking through a fog. These are examples of derealization. Dissociative amnesia does not refer to permanent memory loss, similar to the erasure of a computer disk, but rather to the hypothetical disconnection of memories from conscious inspection (Steinberg, 2001). Thus, the memory is still there somewhere, but you cannot reach it. Identity confusion is defined by Steinberg as “thoughts and feelings of uncertainty and conflict a person has related to his or her identity” (Steinberg, 2001, p. 101), whereas identity alteration describes the behavioral acting out of this uncertainty and conflict (Bernstein & Putnam, 1986).

Dissociative disorders are not as uncommon as you would expect. Several studies in a variety of patient groups show that dissociative disorders are prevalent in a four to 29% range (Ross, Anderson, Fleischer, & Norton, 1991; Sar, Tutkun, Alyanak, Bakim, & Baral, 2000; Tutkun et al., 1998. For reviews see: Foote, Smolin, Kaplan, Legatt, & Lipschitz, 2006; Spiegel et al., 2011). Studies generally find a much lower prevalence in the general population, with rates in the order of 1%-3% (Lee, Kwok, Hunter, Richards, & David, 2010; Rauschenberger & Lynn, 1995; Sandberg & Lynn, 1992). Importantly, dissociative symptoms are not limited to the dissociative disorders. Certain diagnostic groups, notably patients with borderline personality disorder, post-traumatic stress disorder (PTSD), obsessive-compulsive disorder (OCD) (Rufer, Fricke, Held, Cremer, & Hand, 2006), and schizophrenia (Allen & Coyne, 1995; Merckelbach, à Campo, Hardy, & Giesbrecht, 2005; Yu et al., 2010) also display heightened levels of dissociation.

Measuring Dissociation

The Dissociative Experiences Scale (DES) (Bernstein & Putnam, 1986; Carlson & Putnam, 2000; Wright & Loftus, 1999) is the most widely used self-report measure of dissociation. A self-report measure is a type of psychological test in which a person completes a survey or questionnaire with or without the help of an investigator. The DES measures dissociation with items such as (a) “Some people sometimes have the experience of feeling as though they are standing next to themselves or watching themselves do something, and they actually see themselves as if they were looking at another person” and (b) “Some people find that sometimes they are listening to someone talk, and they suddenly realize that they did not hear part or all of what was said.”

The DES is suitable only as a screening tool. When somebody scores a high level of dissociation on this scale, this does not necessarily mean that he or she is suffering from a dissociative disorder. It does, however, give an indication to investigate the symptoms more extensively. This is usually done with a structured clinical interview, called the Structured Clinical Interview for DSM-4 Dissociative Disorders (Steinberg, 1994), which is performed by an experienced clinician. With the publication of the new DSM-5 there has been an updated version of this instrument.



Figure **6.3.2**: As with many disorders, it is widely thought that childhood neglect and/or abuse is partly to blame as an underlying cause of dissociative disorders.

Etiology

People with this disorder tend to report a history of childhood trauma, some cases having been corroborated through medical or legal records (Cardeña & Gleaves, 2006).

Research by Ross et al. (1990) suggests that in one study about 95% of people with DID were physically and/or sexually abused as children. Of course, not all reports of childhood abuse can be expected to be valid or accurate. However, there is strong evidence that traumatic experiences can cause people to experience states of dissociation, suggesting that dissociative states—including the adoption of multiple personalities—may serve as a psychologically important coping mechanism for threat and danger (Dalenberg et al., 2012).

Glossary

dissociation: a disruption in the usually integrated function of consciousness, memory, identity, or perception of the environment

dissociative identity disorder (DID): formerly known as multiple personality disorder, is at the far end of the dissociative disorder spectrum. It is characterized by at least two distinct, and dissociated personality states. These personality states or "alters" alternately control a person's behavior. The sufferer, therefore, experiences significant memory impairment for important information not explained by ordinary forgetfulness.

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6.4: Dissociative Amnesia, Fugue, and Depersonalization/Derealization Disorder

Learning Objectives

- Describe and differentiate between dissociative amnesia and fugue
- Explain depersonalization/derealization disorder



Figure 6.4.1: The most well-known dissociative disorder is dissociative identity disorder, in which people exhibit more than one identity.

Dissociative disorders are characterized by an individual becoming split off, or dissociated, from his or her core sense of self. Memory and identity become disturbed; these disturbances have a psychological rather than physical cause. Dissociative disorders listed in the DSM-5 include dissociative amnesia, depersonalization/derealization disorder, and dissociative identity disorder.

Dissociative Amnesia

Amnesia refers to the partial or total forgetting of some experience or event. An individual with **dissociative amnesia** is unable to recall important personal information, usually following an extremely stressful or traumatic experience such as combat, natural disasters, or being the victim of violence. The memory impairments are not caused by ordinary forgetting. One study of residents in communities in upstate New York reported that about 1.8% experienced dissociative amnesia in the previous year (Johnson, Cohen, Kasen, & Brook, 2006). Some individuals with dissociative amnesia will also experience **dissociative fugue**(from the word "to flee" in French), whereby they suddenly wander away from their home, experience confusion about their identity, and sometimes even adopt a new identity (Cardeña & Gleaves, 2006). Most fugue episodes last only a few hours or days, but some can last longer.



Figure 6.4.2: People with dissociative fugue temporarily lose their sense of personal identity and impulsively wander away from their homes or places of work.

People with dissociative fugue may travel far distances during the fugue, as far as several thousand miles, and could remain in the fugue state for a couple of days, several weeks or even months. Once they have returned to their pre-dissociative states, they do not remember any events that occurred during the fugue. Dissociative fugue is a rare disorder and data available indicate a prevalence of 0.2% in the general population. The onset is often in adolescence or early adulthood and onset is usually sudden, and often related to traumatic or stressful life events. Dissociative fugue has also been noted to be associated with a previous history of child abuse. Other factors that could predispose someone to dissociative reactions include neuropsychological cognitive dysfunctions and genetic factors. Recovery is usually sudden and often complete, although the fugue state may end gradually in some individuals.

Some have questioned the validity of dissociative amnesia (Pope, Hudson, Bodkin, & Oliva, 1998); it has even been characterized as a "piece of psychiatric folklore devoid of convincing empirical support" (McNally, 2003, p. 275). Notably, scientific publications regarding dissociative amnesia rose during the 1980s and reached a peak in the mid-1990s, followed by an equally sharp decline by 2003; in fact, only 13 cases of individuals with dissociative amnesia worldwide could be found in the literature that same year (Pope, Barry, Bodkin, & Hudson, 2006). Further, no description of individuals showing dissociative amnesia following a trauma exists in any fictional or nonfictional work prior to 1800 (Pope, Poliakoff, Parker, Boynes, & Hudson, 2006). However, a study of 82 individuals who enrolled for treatment at a psychiatric outpatient hospital found that nearly 10% met the criteria for dissociative amnesia, perhaps suggesting that the condition is underdiagnosed, especially in psychiatric populations (Foote, Smolin, Kaplan, Legatt, & Lipschitz, 2006).

? Depersonalization/Derealization Disorder

Depersonalization/derealization disorder is characterized by recurring episodes of depersonalization, derealization, or both. Depersonalization is defined as feelings of "unreality or detachment from, or unfamiliarity with, one's whole self or from aspects of the self" (APA, 2013, p. 302). Individuals who experience **depersonalization** might believe their thoughts and feelings are not their own; they may feel robotic as though they lack control over their movements and speech; they may experience a distorted sense of time and, in extreme cases, they may sense an "out-of-body" experience in which they see themselves from the vantage point of another person. **Derealization** is conceptualized as a sense of "unreality or detachment from, or unfamiliarity with, the world, be it individuals, inanimate objects, or all surroundings" (APA, 2013, p. 303). A person who experiences derealization might feel as though he is in a fog or a dream, or that the surrounding world is somehow artificial and unreal. Individuals with depersonalization/derealization disorder often have difficulty describing their symptoms and may think they are going crazy (APA, 2013).

✔ Watch It

This video explains depersonalization and derealization.



You can view the transcript for "What Are Derealization & Depersonalization Disorder?" here (opens in new window).

Etiology



Figure **6.4.3**: Pierre Janet was one of the first people to allege a connection between events in a subject's past life and his or her present-day trauma, and coined the words "dissociation" and "subconscious."

One prominent interpretation of the origins of dissociative disorders is that they are the direct result of exposure to traumatic experiences. The exposure is known as the *post-traumatic model* or PTM. According to the post-traumatic model *(*PTM), dissociative symptoms can best be understood as mental strategies to cope with or avoid the impact of highly aversive experiences. Within post-traumatic model *(*PTM), individuals rely on dissociation to escape from painful memories. Once individuals have learned to use this defensive coping mechanism, it can become automatic and habitual, even emerging in response to minor stressors. The idea that dissociation can serve a defensive function can be traced back to Pierre Janet (1899–1973), one of the first scholars to link dissociation to psychological trauma.

The empirical evidence that trauma *leads to* dissociative symptoms is the subject of intense debate. Most important, however, is that the PTM does not tell us *how* trauma produces dissociative symptoms. Therefore, workers in the field have searched for other explanations. Clinicians and researchers proposed that due to their dreamlike character, dissociative symptoms such as derealization, depersonalization, and absorption are associated with sleep-related experiences. Clinicians and researchers further noted that sleep-related experiences can explain the relationship between highly aversive events and dissociative symptoms.

Treatment

Overall, the most common form of treatment for dissociative disorders is psychotherapy, which generally focuses on the dissociative psychopathology and associated trauma or stressor. The types of psychotherapy can include psychodynamic, cognitive-behavioral, supportive, hypnotherapeutic, free association, and drug-assisted. A therapist has to be flexible in the approach and technique applied because of the challenging symptoms people experience with these disorders.

Applying skill-building interventions at the beginning stages of treatment helps stabilize the patient and improve the disabling dissociative symptoms, allowing treatment to progress and help patients to cope with painful affect and recollections of the traumatic experience. Patients with dissociative amnesia and dissociative fugue generally recover more quickly, especially when the dissociative event is of short duration, and their symptoms may even resolve spontaneously when the person is removed from the previous trauma or stressor. Treatment of dissociative amnesia is aimed at the restoration of missing memories while treatment of dissociative fugue is focused on the recovery of memory for identity and events preceding the fugue. Cognitive and psychodynamic therapy are the most common techniques applied in the treatment of dissociative amnesia and dissociative fugue; however, hypnotherapy and drug-assisted interviews are frequently necessary techniques to assist with memory recovery.

Overall, the use of pharmacotherapy in the treatment of dissociative disorders is limited and controversial, as most medications (such as antidepressants and anxiolytics) are initiated to alleviate comorbid anxiety and mood symptoms, but do not treat the actual dissociative symptoms. Currently, no pharmacological treatment has been found to reduce dissociation, and no medication has been shown to be efficacious to date; research has been limited, and thus no definitive medication treatment guidelines exist.

Glossary

depersonalization/derealization disorder: dissociative disorder in which people feel detached from the self (depersonalization), and the world feels artificial and unreal (derealization)

dissociative amnesia: dissociative disorder characterized by an inability to recall important personal information, usually following an extremely stressful or traumatic experience

dissociative disorders: group of DSM-5 disorders in which the primary feature is that a person becomes dissociated, or split off, from his or her core sense of self, resulting in disturbances in identity and memory

dissociative fugue: symptom of dissociative amnesia in which a person suddenly wanders away from one's home and experiences confusion about his or her identity

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6.5: Dissociative Identity Disorder

Learning Objectives

- Explain the symptoms, diagnosis, and possible causes of dissociative identity disorder

Is It Real?

Let's start with a little history. Multiple personality disorder, or **dissociative identity disorder (DID)** as it is known now, used to be a mere curiosity. This is a disorder in which people present with more than one personality. For example, at times they might act and identify as an adult while at other times they might identify and behave like a child. dissociative identity disorder (DID) was rarely diagnosed until the 1980s. That is when multiple personality disorder became an official diagnosis in the DSM-3. From then on, the numbers of "multiples" increased rapidly. In the 1990s, there were hundreds of people diagnosed with multiple personality in every major city in the United States (Hacking, 1995). How could this "epidemic" be explained?

One possible explanation might be the media attention that was given to the disorder. It all started with the book *TheThree Faces of Eve* (Thigpen & Cleckley, 1957). This book, and later the movie, was one of the first to speak of multiple personality disorder. However, it wasn't until years later, when the fictional, as-told-to book of *Sybil* (Schreiber, 1973) became known worldwide, that the prototype of what it was like to be a "multiple personality" was born. Sybil tells the story of how a clinician—Cornelia Wilbur—unravels the different personalities of her patient Sybil during a long course of treatment (over 2,500 office hours!). Wilbur was one of the first to relate multiple personality to childhood sexual abuse. Probably, this relation between childhood abuse and dissociation has fueled the increase of numbers of multiples from that time on. It motivated therapists to actively seek for clues of childhood abuse in their dissociative patients, well within the mindset of the 1980s, as childhood abuse was a sensitive issue then in psychology as well as in politics (Hacking, 1995).

From then on, many movies and books were made on the subject of multiple personality, and nowadays, we see patients with dissociative identity disorder as guests visiting the Oprah Winfrey show, as if they were our modern-day circus acts.

Despite its controversial nature, dissociative identity disorder (DID) is clearly a legitimate and serious disorder, and although some people may fake symptoms, others suffer their entire lives with it. People with this disorder tend to report a history of childhood trauma, some cases having been corroborated through medical or legal records (Cardeña & Gleaves, 2006). Research by Ross et al. (1990) suggests that in one study about 95% of people with DID were physically and/or sexually abused as children. Of course, not all reports of childhood abuse can be expected to be valid or accurate. However, there is strong evidence that traumatic experiences can cause people to experience states of dissociation, suggesting that dissociative states—including the adoption of multiple personalities—may serve as a psychologically important coping mechanism for threat and danger (Dalenberg et al., 2012).

Sadly, more than 70% of people with DID have attempted suicide, and self-injurious behavior is common among this population. Treatment is crucial to improving quality of life and preventing suicide attempts. Cognitive Behavioral Therapy (CBT) could be used as a mode of treatment for these patients.[1]

Dissociative Identity Disorder

By far, the most well-known dissociative disorder is dissociative identity disorder. People with dissociative identity disorder exhibit two or more separate personalities or identities, each well-defined and distinct from one another. Those with DID also experience memory gaps for the time during which another identity is in charge (e.g., one might find unfamiliar items in her shopping bags or among her possessions), and in some cases may report hearing voices, such as a child's voice or the sound of somebody crying (APA, 2013). The study of upstate New York residents mentioned above (Johnson et al., 2006) reported that 1.5% of their sample experienced symptoms consistent with dissociative identity disorder in the previous year. The estimated DID prevalence around the globe is about 5% among the inpatient psychiatric population, 2%-3% among outpatients, and 1% in the general population.[2]

The DSM-5 gives the following diagnostic criteria for DID:[3]



Figure 6.5.1: A person diagnosed with DID will generally feel a discontinuity in their sense of self and experience changes in identifies—each with their own personalities, names, stories, and mannerisms.

- "Disruption of identity characterized by two or more distinct personality states marked by the discontinuity in the sense of self and agency and accompanied by related alterations in affect, behavior, consciousness, memory, perception, cognition, and/or sensory-motor functioning.
- Recurrent gaps in the recall of everyday events, important personal information, and/or traumatic events that are inconsistent with ordinary forgetting.
- The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- The disturbance is not a normal part of a broadly accepted cultural or religious practice. Note: In children, the symptoms are not better explained by imaginary playmates or other fantasy play.
- The symptoms are not attributable to the physiological effects of a substance (e.g., blackouts or chaotic behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures)."

Sense of Self and Agency

The terms *sense of self* and *sense of agency* are used in the DSM as it discusses the presence of distinct personality states, better known as alter personalities. Distinct personality states are the discontinuities (switches) between alters, as well as their presence that this criterion describes. A discontinuity in a person's sense of self can affect any part of someone's functioning. Attitudes, outlooks, and personal preferences (like preferred foods or clothes) may change suddenly and inexplicably and then change back again. This happens because alter personalities have different attitudes, outlooks, and preferences, so a very sudden change without explanation occurs when an alter has either taken control or is strongly influencing the person. When that alter is no longer active, everything changes back (until the next time the same alter is active). During these shifts, a person may find they have bought clothes they would never choose to wear, or a very outgoing person may suddenly become shy and introverted with no apparent reason.

Discontinuity in a person's sense of agency means not feeling in control of, or as if you don't "own" your feelings, thoughts or actions. For example, experiencing thoughts, feelings or actions that seem as if they are "not mine" or "belong to someone else." This is not the delusional belief that you belong to an outside person; it is the perception that your own speech, thoughts, and/or behavior do not feel like they belong to them and may make no sense to you. Emotions and impulses are often described as puzzling to the person.

In DID some of the thoughts, feelings, or actions of alter personalities intrude into the person's conscious awareness, even when the person is not aware of having any alter personalities or has amnesia for his or her actions. This is known as passive influence or partially dissociated intrusions of alter identities into conscious awareness. A person with DID may also experience a fully dissociated intrusion, and may say things like

- "I have no control;, I watch what happens, but can't stop it."
- "I find myself coming to in the downtown area where I live, but I won't remember where I parked the car."
- "I have found myself crying uncontrollably and sucking my thumb, but I can't explain why."
- "Sometimes I've had people call me by a name I don't recognize, and I don't know who they are."

Some people describe this combined change of “sense of self” and “sense of agency” as feeling like an experience of possession, in a non-religious sense, or having their body “hijacked.” A person with DID may find that their body feels totally different during this time (e.g., like a small child, the opposite gender, huge and muscular), or may feel as if they are suddenly younger or older.

Causes of DID

Dissociation and Trauma

The most widely held perspective on dissociative symptoms is that they reflect a defensive response to highly aversive events, mostly trauma experiences during the childhood years (Bremner, 2010; Spiegel et al., 2011; Spitzer, Vogel, Barnow, Freyberger, & Grabe, 2007).

As mentioned previously, one prominent interpretation of the origins of dissociative disorders is that they are the direct result of exposure to traumatic experiences, called the post-traumatic model (PTM). According to the PTM, dissociative symptoms can best be understood as mental strategies to cope with or avoid the impact of highly aversive experiences (e.g., Spiegel et al., 2011).

The post-traumatic model casts the clinical observation that dissociative disorders are linked to a trauma history in straightforward, causal terms, that is, one causes the other (Gershuny & Thayer, 1999). For example, Vermetten and colleagues (Vermetten, Schmahl, Lindner, Loewenstein, & Bremner, 2006) found that the DID patients in their study all suffered from PTSD and concluded that DID should be conceptualized as an extreme form of early abuse–related PTSD (Vermetten et al., 2006).

Causality and Evidence

The empirical evidence that trauma leads to dissociative symptoms is the subject of intense debate (Kihlstrom, 2005; Bremner, 2010; Giesbrecht, Lynn, Lilienfeld & Merckelbach, 2010). Three limitations of the PTM will be described below.

First, the majority of studies reporting links between self-reported trauma and dissociation are based on cross-sectional designs. Cross-sectional approaches mean that the data are collected at one point in time. When analyzing this type of data, one can only state whether scoring high on a particular questionnaire (for example, a trauma questionnaire) is indicative of also scoring high on another questionnaire (for example, the DES). This makes it difficult to state if one thing led to another, and therefore if the relation between the two is causal. Thus, the data that these designs yield do not allow for strong causal claims (Merckelbach & Muris, 2002).

Second, whether somebody has experienced trauma is often established using a questionnaire that the person completes himself or herself. This is called a self-report measure. Herein lies the problem. Individuals suffering from dissociative symptoms typically have high fantasy proneness. High fantasy proneness is a character trait to engage in extensive and vivid fantasizing. The tendency to fantasize a lot may increase the risk of exaggerating or understating self-reports of traumatic experiences (Merckelbach et al., 2005; Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008).

Third, high dissociative individuals report more cognitive failures than low dissociative individuals. Cognitive failures are everyday slips and lapses, such as failing to notice signposts on the road, forgetting appointments, or bumping into people. This can be seen, in part, in the DSM-5 criteria for DID, in which people may have difficulty recalling everyday events as well as those that are traumatic. People who frequently make such slips and lapses often mistrust their own cognitive capacities. They also tend to overvalue the hints and cues provided by others (Merckelbach, Horselenberg, & Schmidt, 2002; Merckelbach, Muris, Rassin, & Horselenberg, 2000). The overvaluation of hints and clues make them vulnerable to suggestive information, which may distort self-reports, and thus limits conclusions that can be drawn from studies that rely solely on self-reports to investigate the trauma-dissociation link (Merckelbach & Jelicic, 2004).

Most important, however, is that the PTM does not tell us how trauma produces dissociative symptoms. Therefore, workers in the field have searched for other explanations. Clinicians proposed that due to their dreamlike character, dissociative symptoms such as derealization, depersonalization, and absorption are associated with sleep-related experiences. Clinicians further noted that sleep-related experiences can explain the relationship between highly aversive events and dissociative symptoms (Giesbrecht et al., 2008; Watson, 2001).

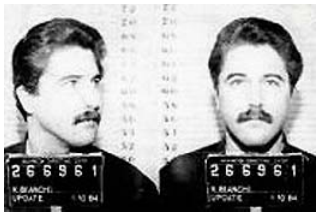


Figure **6.5.2**: Known as the Hillside Strangler, Kenneth Bianchi faked multiple personality disorder, pleading innocence due to insanity. He was found guilty and sentenced to life in prison.

DID is highly controversial. Some believe that people fake symptoms to avoid the consequences of illegal actions (e.g., "I am not responsible for shoplifting because it was my other personality"). In fact, it has been demonstrated that people are generally skilled at adopting the role of a person with different personalities when they believe it might be advantageous to do so. As an example, Kenneth Bianchi was an infamous serial killer who, along with his cousin, murdered over a dozen females around Los Angeles in the late 1970s. Eventually, he and his cousin were apprehended. At Bianchi's trial, he pled not guilty by reason of insanity, presenting himself as though he had DID and claiming that a different personality ("Steve Walker") committed the murders. When these claims were scrutinized, he admitted faking the symptoms and was found guilty (Schwartz, 1981).

A second reason DID is controversial is because rates of the disorder suddenly skyrocketed in the 1980s. More cases of DID were identified during the five years prior to 1986 than in the preceding two centuries (Putnam, Guroff, Silberman, Barban, & Post, 1986). Although this increase may be due to the development of more sophisticated diagnostic techniques, it is also possible that the popularization of DID—helped in part by *Sybil*, a popular 1970s book (and later film) about a woman with 16 different personalities—may have prompted clinicians to overdiagnose the disorder (Piper & Merskey, 2004). Casting further scrutiny on the existence of multiple personalities or identities is the recent suggestion that the story of Sybil was largely fabricated, and the idea for the book might have been exaggerated (Nathan, 2011).

? Case Study: Virginia

In 2018, a 55-year-old Caucasian female, *Virginia*, with a history of substance use disorder and a comorbid bipolar disorder, came to the local general hospital with a history of the fragmentation of a single personality into different personalities under emotional stress and under the influence of a drug. Multiple aspects of her personalities were reported, including the following: a personality of a seven-year-old child, a personality that would behave as a teenager, and another that acted like a male person in addition to her normal 55-year-old personality. Virginia reported that she had been constantly dominated by her alternate personalities and became aware of their existence when people around her informed her, usually after a situation ended. She reported that stressful situations and substance abuse could aggravate the fragmentation of her personality. This was found to be mostly an involuntary phenomenon with seldom memory of the event.

While transitioning between these personalities, Virginia was found to be violent even to people who were close to her. This could range from being suicidal to homicidal, for which she was arrested twice in the past. She had to be isolated and restrained by being locked in a room while the police were called. As a result, she was hospitalized in a mental institution for a significant period at least two to three times in the past few years. Under the influence of stress or substances like marijuana or cocaine, her personality would split into various personalities. These states were very different from one another in terms of age or gender.

One of Virginia's alternate personalities behaved as a seven-year-old child and would show the same interests and choices that included becoming moody or a self-arrogant personality. While in these states, she could hurt herself or had weeping spells if her wants were not met.

Another personality acted as a teenager with some sharp choices and dressing. Increases in substance abuse, alcohol use, and smoking would lead to multiple cases of fights or homicidal attacks, with some incidents of self-harming events. Multiple scars were found on the dorsal side of her right hand. Her speech was found to be pressured and she would repeat the same words/conversations.

The next personality was diagnosed to be a temporary transition to the opposite gender (a male). There was a change in voice and behavior. This included male dressing, language, a perception of male body parts, choices of friends, and attraction

towards females, including sexual behavior.

The normal state of a 55-year aged female was the default personality that made Virginia feel most comfortable. She reported that she had anxiety during a personality state transition, as it could occur at any time, and involuntarily, but mostly in stressful situations and during substance abuse.

Virginia's treatment included psychotherapy with cognitive behavioral therapy addressing stress and substance use disorder. The psychotherapeutic treatment lasted for at least six months. The dual treatment of drug therapy was also involved to calm her down. She was prescribed escitalopram to reduce her anxiety symptoms, and Virginia believed that the anxiety pills were really helpful. After six months, her condition was not drastically different. However, she believed her stress was getting better and her coping mechanisms were improving.[4]

Watch It

Start at the 08:20 mark of this video to learn about dissociative disorders.



You can view the transcript for "Schizophrenia and Dissociative Disorders: Crash Course Psychology #32" here (opens in new window).

Glossary

dissociative identity disorder (DID): formerly known as multiple personality disorder, is at the far end of the dissociative disorder spectrum. It is characterized by at least two distinct, and dissociated personality states. These personality states—or alters—alternately control a person's behavior. The sufferer, therefore, experiences significant memory impairment for important information not explained by ordinary forgetfulness.

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6.6: Dissociation and Sleep

Learning Objectives

- Explain the sleep-dissociation model

A Little History



Figure **6.6.1**: Those who have fallen asleep in class or on the bus have likely experienced those "micro-dreams" – that moment or two where reality kind of blends in with your dreams. For a long time, scientists thought dissociative disorders were simply this confusion of waking and dreaming states. [Image: kooklanekookla, https://goo.gl/Cn3xul, CC BY 2.0, https://goo.gl/BRvSA7]

Researchers (Watson, 2001) have proposed that dissociative symptoms, such as absorption, derealization, and depersonalization originate from sleep. This idea is not entirely new. In the 19th century, double consciousness (or dédoublement), the historical precursor of dissociative identity disorder (DID; formerly known as multiple personality disorder), was often described as "somnambulism," which refers to a state of sleepwalking. Patients suffering from this disorder were referred to as "somnambules" (Hacking, 1995). Many 19th-century scholars believed that these patients were switching between a "normal state" and a "somnambulistic state." Hughlings Jackson, a well-known English neurologist from this era, viewed dissociation as the uncoupling of normal consciousness, which would result in what he termed "the dreamy state" (Meares, 1999). Interestingly, a century later, Levitan (1967) hypothesized that "depersonalization is a compromise state between dreaming and waking" (p.157). Arlow (1966) observed that the dissociation between the "experiencing self" and the "observing self" serves as the basis of depersonalized states, emphasizing its occurrence, especially in dreams. Likewise, Franklin (1990) considered dreamlike thoughts, the amnesia one usually has for dreams, and the lack of orientation of time, place, and person during dreams to be strikingly similar to the amnesia DID patients often report for their traumas. Related, Barrett (1994, 1995) described the similarity between dream characters and "alter personalities" in DID, with respect to cognitive and sensory abilities, movement, amnesia, and continuity with normal waking. The many similarities between dreaming states and dissociative symptoms are also a recurrent theme in the more recent clinical literature (e.g., Bob, 2004).

Sleep Problems and Dissociative Disorders

Anecdotal evidence supports the idea that sleep disruptions are linked to dissociation. For example, in patients with depersonalization, symptoms are worst when they are tired (Simeon & Abugel, 2006). Interestingly, among participants who report memories of childhood sexual abuse, experiences of **sleep paralysis** typically are accompanied by raised levels of dissociative symptoms (McNally & Clancy, 2005; Abrams, Mulligan, Carleton, & Asmundson, 2008).

Patients with mood disorders, anxiety disorders, schizophrenia, and borderline personality disorder—conditions with relatively high levels of dissociative symptoms—as a rule exhibit sleep abnormalities. Recent research points to fairly specific relationships between certain sleep complaints (e.g., **insomnia**, nightmares) and certain forms of psychopathology (e.g., depression, PTSD) (Koffel & Watson, 2009).

Studying the relationship between dissociation and sleep

In the general population, both dissociative symptoms and sleep problems are highly prevalent. For example, 29% of American adults report sleep problems (National Sleep Foundation, 2005). Thus researchers study the relationship between dissociation and

sleep not only in patients, but also in the general population. In a pioneering study, Watson (2001) showed that dissociative symptoms—measured by the Dissociative Experiences Scale (DES)—are linked to self-reports of vivid dreams, nightmares, recurrent dreams, and other unusual sleep phenomena. This relationship has been studied extensively ever since, leading to three important statements.

First, Watson's (2001) basic findings have been reproduced time and again. This means that the same results (namely that dissociation and sleep problems are related) have been found in lots of different studies, using different groups, and different materials. All lead to the conclusion that unusual sleep experiences and dissociative symptoms are linked.

Second, the connection between sleep and dissociation is specific. It seems that unusual sleep phenomena that are difficult to control, including nightmares and waking dreams, are related to dissociative symptoms, but **lucid dreaming**—dreams that are controllable—are only weakly related to dissociative symptoms. For example, dream recall frequency was related to dissociation (Suszek & Kopera, 2005). Individuals who reported three or more nightmares over a three-week period showed higher levels of dissociation compared to individuals reporting two nightmares or less (Levin & Fireman, 2002), and a relation was found between dream intensity and dissociation (Yu et al., 2010).

Third, the sleep-dissociation link is apparent not only in general population groups—people such as you and me—but also in patient groups. Accordingly, one group of researchers reported nightmare disorder in 17 out of 30 DID patients (Agargun et al., 2003). They also found a 27.5% prevalence of nocturnal dissociative episodes in patients with dissociative disorders (Agargun et al., 2001). Another study investigated a group of borderline personality disorder patients and found that 49% of them suffered from nightmare disorder. Moreover, the patients with nightmare disorder displayed higher levels of dissociation than patients not suffering from nightmare disorder (Semiz, Basoglu, Ebrinc, & Cetin, 2008). Additionally, Ross (2011) found that patients suffering from DID reported higher rates of sleepwalking compared to a group of psychiatric outpatients and a sample from the general population.

To sum up, there seems to be a strong relationship between dissociative symptoms and unusual sleep experiences that is evident in a range of phenomena, including waking dreams, nightmares, and sleepwalking.

Inducing and Reducing Sleep Problems

Sleep problems can be induced in healthy participants by keeping them awake for a long duration of time. The result is called **sleep deprivation**. If dissociative symptoms are fueled by a labile **sleep-wake cycle**, then sleep loss would be expected to intensify dissociative symptoms. Some evidence that this expectation might be true was already found in 2001, when soldiers who underwent a U.S. Army survival training, which included sleep deprivation, showed increases in dissociative symptoms (Morgan et al., 2001). Other researchers conducted a study that tracked 25 healthy volunteers during one day and one night of sleep loss. They found that dissociative symptoms increased substantially after one night of sleep loss (Giesbrecht, Smeets, Leppink, Jelicic, & Merckelbach, 2007).

To further examine the causal link between dissociative experiences and sleep, a group of researchers (van der Kloet, Giesbrecht, Lynn, Merckelbach, & de Zutter, 2011) investigated the relationship between unusual sleep experiences and dissociation in a patient group at a private clinic. They completed questionnaires upon arrival at the clinic and again when they departed eight weeks later. During their stay, they followed a strict program designed to improve sleep problems. And it worked! In most patients, sleep quality was improved after eight weeks. The researchers found a robust link between sleep experiences and dissociative symptoms and determined that sleep normalization was accompanied by a reduction in dissociative symptoms.

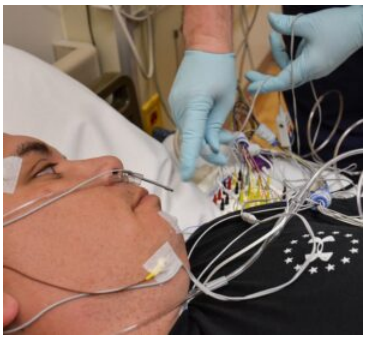


Figure 6.6.2: No longer are dissociative disorders untreatable illnesses. With the new methods developed by researchers, there is hope for curing or at least improving the lives of those with this debilitating disorder. [Image: CC0 Public Domain, https://goo.gl/m25gce]

An exciting interpretation of the link between dissociative symptoms and unusual sleep phenomena (see also, Watson, 2001) may be this: a disturbed sleep-wake cycle may lead to dissociative symptoms. However, we should be cautious. Although studies support a causal arrow leading from sleep disruption to dissociative symptoms, the associations between sleep and dissociation may be more complex. For example, causal links may be bi-directional, such that dissociative symptoms may lead to sleep problems and vice versa, and other psychopathology may interfere in the link between sleep and dissociative symptoms (van der Kloet et al., 2011).

Implications and Conclusions

The sleep-dissociation model offers a fresh and exciting perspective on dissociative symptoms. This model may seem remote from the PTM. However, both models can be integrated into a single conceptual scheme in which traumatic childhood experiences may lead to disturbed sleep patterns, which may be the final common pathway to dissociative symptoms. Accordingly, the sleep-dissociation model may explain both (a) how traumatic experiences disrupt the sleep–wake cycle and increase vulnerability to dissociative symptoms and (b) why dissociation, trauma, fantasy proneness, and cognitive failures overlap.

Future studies can also discern which characteristic sleep disruptions in the sleep-wake cycle are most reliably related to dissociative disorders, and then establish training programs, including medication regimens, to address these problems. This would constitute an entirely novel and exciting approach to the treatment of dissociative symptoms.

In closing, the sleep-dissociation model can serve as a framework for studies that address a wide range of fascinating questions about dissociative symptoms and disorders. We now have good reason to be confident that research on sleep and dissociative symptoms will inform psychiatry, clinical science, and psychotherapeutic practice in meaningful ways in the years to come.

Glossary

insomnia: a sleep disorder in which there is an inability to fall asleep or to stay asleep as long as desired. Symptoms also include waking up too early, experiencing many awakenings during the night and not feeling rested during the day

lucid dreams: any dream in which one is aware that one is dreaming

sleep deprivation: a sufficient lack of restorative sleep over a cumulative period so as to cause physical or psychiatric symptoms and affect routine performances of tasks

sleep paralysis: sleep paralysis occurs when the normal paralysis during REM sleep manifests when falling asleep or awakening, often accompanied by hallucinations of danger or a malevolent presence in the room

sleep-wake cycle: a daily rhythmic activity cycle, based on 24-hour intervals, that is exhibited by many organisms

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6.7: Introduction to Somatic Symptom and Related Disorders

What you'll learn to do: explain somatic symptom and related disorders



In this section, you'll review and learn to identify the somatic symptom disorders (previously known as "somataform disorders"). These include somatic symptom disorder, illness anxiety disorder, conversion disorder, and fictitious disorder. You'll read about the various symptoms, epidemiology, how individuals present with these problems, and a brief overview of possible causes.

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6.8: Somatic Symptom Disorder and Illness Anxiety Disorder

Learning Objectives

- Describe somatic symptom disorder
- Explain illness anxiety disorder

Somatic Symptom and Related Disorders



Figure 6.8.1: Those with somatic symptom disorders often seek continued medical care despite no medical explanations for the symptoms.

A **somatic symptom disorder**, formerly known as a somatoform disorder, is any mental disorder that manifests as physical symptoms that suggest illness or injury, but cannot be explained fully by a general medical condition or by the direct effect of a substance, and are not attributable to another mental disorder (e.g., panic disorder).

In people who have been diagnosed with a somatic symptom disorder, medical test results are either normal or do not explain the person's symptoms, and history and physical examination do not indicate the presence of a known medical condition that could cause them, though the DSM-5 cautions that this alone is not sufficient for diagnosis. The patient must also be excessively worried about their symptoms, and this worry must be judged to be out of proportion to the severity of the physical complaints themselves. A diagnosis of somatic symptom disorder requires that the subject have recurring somatic complaints for at least six months.

Symptoms are sometimes similar to those of other illnesses and may last for years. Usually, the symptoms begin appearing during adolescence, and patients are diagnosed before the age of 30 years. Symptoms may occur across cultures and gender. Other common symptoms include anxiety and depression. However, since anxiety and depression are also very common in persons with confirmed medical illnesses, it remains possible that such symptoms are a consequence of the physical impairment, rather than a cause. Somatic symptom disorders are not the result of conscious malingering (fabricating or exaggerating symptoms for secondary motives) or factitious disorders (deliberately producing, feigning, or exaggerating symptoms). Somatic symptom disorder is difficult to diagnose and treat.

Watch It

This video explains some of the key diagnostic features of somatoform disorders.



You can view the transcript for "Somatic symptom disorder – causes, symptoms, diagnosis, treatment, pathology" here (opens in new window).

Illness Anxiety Disorder



Figure **6.8.2**: Formerly known as hypochondria, illness anxiety disorder is characterized by a preoccupation and concern with developing a medical condition.

The excessive worries and thoughts of a presumed and nonexistent illness, which was known as hypochondriasis in DSM-4, is now under the umbrella of the somatic symptom disorders. Diagnosis of somatic symptom disorder requires at least one somatic symptom that is causing a significant disruption of the patient's life with significant actions and emotions that result in high anxiety level or excessive time consumption. **Illness anxiety disorder (IAD)** does not typically involve somatic symptoms, meaning that individuals do not experience any specific physical ailment, but instead, they are preoccupied with concern about developing a severe medical condition.

The DSM-5 encompasses two types of patients with illness anxiety disorder: care-seeking type and care-avoidant type. Since patients with illness anxiety disorder (IAD) emanate their distress and anxiety not primarily from the physical complaint itself but rather from his or her anxiety about the meaning, significance, and cause of the complaint, they remain unsatisfied with the reassurance of the physicians. Illness anxiety disorder (IAD) can cause a huge burden on the resources of health care facilities and on health care providers.[1][/footnote]

In one case study published in 2016, a 73-year-old Saudi man started visiting the primary health care center around 25 years ago. With concerns about having cancer, the patient continuously visited the hospital, which ended up costing over $170,000. Throughout this period, the patient was exposed to extensive unnecessary imaging studies and laboratory tests that had effects on his life in all aspects with such concerns. Five years ago, a family doctor put an end to that by directing the patient on the right path. The doctor made several actions; most importantly, he directed the patient to CBT, which significantly improved a range of hypochondriacal beliefs and attitudes. This patient's case demonstrates the fundamental importance of a proper health system that limits such patients from abusing the health system and depleting medical resources.[2]

Glossary

illness anxiety disorder: a disorder in which a person does not typically have physical symptoms, rather they are preoccupied with a fear of developing a severe medical condition. The two categories of patients in this group are the care-seeking type and care-avoidant type.

somatic symptom disorder: a mental disorder that manifests as physical symptoms that suggest illness or injury, but cannot be explained fully by a general medical condition

1. [footnote]Almalki, M., Al-Tawayjri, I., Al-Anazi, A., Mahmoud, S., & Al-Mohrej, A. (2016, May 25). A Recommendation for the Management of Illness Anxiety Disorder Patients Abusing the Health Care System. Case Reports in Psychiatry. https://www.hindawi.com/journals/crips/2016/6073598/. ↵
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6.9: Conversion Disorder and Other Somatic Symptom Disorders

Learning Objectives

- Describe conversion disorder
- Describe factitious disorder and factitious disorder imposed on another

Conversion Disorder

Conversion disorder, also known as **functional neurological symptom disorder,** is a type of somatic disorder that is sometimes applied to patients who present with neurological symptoms, such as numbness, blindness, paralysis, or fits, which are not consistent with a well-established organic cause. The symptoms cause significant distress and can be traced back to a psychological trigger. It is thought that these symptoms arise in response to stressful situations affecting a patient's mental health or an ongoing mental health condition such as depression.



Figure **6.9.1**: Conversion disorder occurs when physical symptoms result from psychological triggers.

Conversion disorder begins with some stressor, trauma, or psychological distress. Usually, the physical symptoms of the syndrome affect the senses or movement. Common symptoms of conversion disorder include blindness, partial or total paralysis, inability to speak, deafness, numbness, difficulty swallowing, incontinence, balance problems, seizures, tremors, and difficulty walking. These symptoms are attributed to conversion disorder when a medical explanation for the afflictions cannot be found. Symptoms of conversion disorder usually occur suddenly. Conversion disorder is typically seen in individuals aged 10 to 35 and affects between 0.011% and 0.5% of the general population.

Conversion disorder can present with motor or sensory symptoms including any of the following:

- motor symptoms or deficits:
  - impaired coordination or balance
  - weakness/paralysis of a limb or the entire body (hysterical paralysis or motor conversion disorders)
  - impairment or loss of speech (hysterical aphonia)
  - difficulty swallowing (dysphagia) or a sensation of a lump in the throat
  - urinary retention
  - psychogenic non-epileptic seizures or convulsions
  - persistent dystonia
  - tremor, myoclonus or other movement disorders
  - gait problems (astasia-abasia)
  - loss of consciousness (fainting)
- sensory symptoms or deficits:
  - impaired vision (hysterical blindness), double vision
  - impaired hearing (deafness)
  - loss or disturbance of touch or pain sensation

Factitious Disorder

A **factitious disorder (FD)** is a condition in which a person, without a malingering motive, acts as if they have an illness by deliberately producing, feigning, or exaggerating symptoms purely to attain (for themselves or for another) a patient's role. Individuals with a factitious disorder may produce symptoms by contaminating urine samples, taking hallucinogens, injecting fecal material to produce abscesses, and similar behavior.

Factitious disorder imposed on self (also called Munchausen syndrome) was for some time the umbrella term for all such disorders. **Factitious disorder imposed on another** (also called Munchausen syndrome by proxy, Munchausen by proxy, or factitious disorder by proxy) is a condition in which a person deliberately produces, feigns, or exaggerates the symptoms of someone in their care. In either case, the perpetrator's motive is to perpetrate factitious disorders, either as a patient or by proxy as a caregiver, in order to attain (for themselves or for another) a patient's role. Malingering differs fundamentally from factitious disorders in that the malingerer simulates illness intending to obtain a material benefit or avoid an obligation or responsibility. Somatic symptom disorders, though also diagnoses of exclusion, are characterized by physical complaints that are not produced intentionally.

Link to Learning

This article highlights some famous cases of Munchausen by proxy in the news.

Glossary

conversion disorder (functional neurological symptom disorder): a type of somatic disorder that is sometimes applied to patients who present with neurological symptoms which are not otherwise explained by a well-established organic cause, and often can be traced back to a psychological trigger

factitious disorder (FD): a condition in which a person, without a malingering motive, acts as if they have an illness by deliberately producing, feigning, or exaggerating symptoms, purely to attain (for themselves or for another) a patient's role; presents in two ways: factitious disorder imposed on self occurs when someone feigns illness so *they* experience being the patient, or by others (factitious disorder imposed on another)

factitious disorder imposed on another: also known as Munchausen by proxy, this is a condition in which a person deliberately produces, feigns, or exaggerates the symptoms of someone in their care

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1. Jaghab, K., Skodnek, K. B., & Padder, T. A. (2006). Munchausen's Syndrome and Other Factitious Disorders in Children: Case Series and Literature Review. *Psychiatry* (Edgmont (Pa. : Township)), 3(3), 46–55. ↵

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6.10: Introduction to Treating Dissociative and Somatic Symptom Disorders

What you'll learn to do: examine various perspectives and treatment methods for dissociative and somatic symptom disorders



Unfortunately, dissociative and somatic symptom disorders can be difficult to diagnose, and often, even more difficult to treat. Though researchers have found various treatments to help improve quality of life, both psychotherapeutic interventions, as well as an eclectic approach that could include alternative medicine such as physical or occupational therapy. One's theoretical framework (i.e., their way of thinking about a disorder) will depend on the type of treatment they may prescribe (or wish to receive). We will delve more into theoretical framework as well so you have a good understanding of the different ways of thinking about these mental health disorders.

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6.11: Perspectives on Dissociative and Somatic Symptom Disorders

Learning Objectives

- Describe and compare viewpoints from the major psychological perspectives related to dissociative and somatic symptom disorders

Dissociative disorders and somatic symptom disorders are influenced by a variety of factors, including biological, cognitive, sociocultural, and psychodynamic influences, with the strongest explanation for the disorder being the psychodynamic perspective, which emphasizes dissociation as a type of defense mechanism used to cope with trauma or abuse.

The Biological Perspective



Figure **6.11.1**: The science behind a psychological disorder is complex.

Though researchers have not found a specific genetic link for these disorders, there are genetic links to dissociation as it relates to childhood adversity. Biological factors may include trauma-induced responses.[1]

Some studies have found that heritability rates for dissociation rage from 50 to 60% but it is often believed that the mix of genetic and environment plays a larger role in developing dissociative disorders than solely a biological explanation.[2]

The Psychodynamic Perspective

Freud's model suggested the emotional charge deriving from painful experiences would be consciously repressed as a way of managing the pain, but that the emotional charge would be somehow converted into neurological symptoms. Freud later argued that the repressed experiences were of a sexual nature. Pierre Janet, the other great theoretician of hysteria, argued that symptoms arose through the power of suggestion, acting on a personality vulnerable to dissociation. In this hypothetical process, the subject's experience of their leg, for example, is split off from the rest of their consciousness, resulting in paralysis or numbness in that leg.

Some support for the Freudian model comes from findings of high rates of childhood sexual abuse in conversion patients. Support for the dissociation model comes from studies showing heightened suggestibility in conversion patients.

Essentially, the idea behind the psychodynamic perspective is that someone experiencing dissociation, either of identity or in conversion disorder, is using a type of defense mechanism to guard against negative feelings because of inadequate coping skills; this type of emotional conflict becomes overwhelming and results in the symptoms one might experience.

The Cognitive Perspective

Cognitive psychologists have identified all manners of different kinds of memory—iconic, haptic, echoic, short-term, working, long-term, declarative, non-declarative, procedural, semantic, episodic, implicit, explicit, and more. These kinds of memory, or memory systems, can be thought of as (collections of) different kinds of specific memories, and they are distinguished in terms of their content, durability, and the way in which they are acquired and accessed. The concepts of *retrieval* and *forgetting* are, of course, central to any discussion of memory and memory-related disorders. Dissociative amnesia, flashbacks and other dissociative phenomena have frequently been observed not only as a result of traumatic stress, but also as a result of the use of dissociative drugs such as PCP, ketamine, or LSD.[3]

Additionally, imaging of the prefrontal cortex in those with conversion disorder shows that a person with paralysis due to the disorder *does* attempt to move the paralyzed area of the body but is unable to—thus proving that the patient is not faking their issues; rather they are prohibited from completing the action they wish due to a disconnect in the brain regions responsible for making a connection between conscious will and the ability to act it out.[4]

The Sociocultural Perspective

It has been found that those with lower socioeconomic status (SES), education, and history of abuse are more likely to develop DID and other somatic disorders. Many authors have found the occurrence of conversion to be more frequent in rural, lower socio-economic groups, where technological investigation of patients is limited and individuals may be less knowledgeable about medical and psychological concepts.



Figure **6.11.2**: Familial, societal, and cultural factors may contribute to trauma and people's response to it, which can lead to disorders such as DID.

DID can be found in all cultural settings. Even though it is common thought that childhood abuse or trauma is a prerequisite to experiencing this disorder, there still needs to be suitable environmental factors in which this disorder can evolve, usually one defined by a home context in which there is denial, boundary violations, reality distortions, paranoia, and narcissism; in many cases, these negative traits are what makes up the family dynamics, including tyrannical and/or unfair traditions that further the issues. However, it is important to note that familial systems are only a small part of a greater context with society and culture. For example, in South Africa, some communities may find that dissociation is necessary for survival when these areas are plagued with mixed messages and there is major cultural and societal conflict (versus inner psychological conflict).[5]

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6.12: Treating Dissociative Disorders and Somatic Symptom Disorders

Learning Objectives

- Recommend effective treatment methods for dissociative disorders and somatic symptom disorders

Therapists may prescribe different types of treatment for patients with dissociative disorders or somatic symptom disorders based on their viewpoints on the disability and various theories or frameworks. For example, a psychotherapist with a background in psychodynamic theory who studied Freud may approach a client differently by focusing on trying to assist them with overcoming unhealthy defense mechanisms. Whereas someone who has a strong CBT background may focus on psychological techniques to apply and medication management.

Treating Dissociative Disorders

Dissociative disorders are generally treated with long-term psychotherapy. Common treatment methods include an eclectic mix of psychotherapy techniques, including cognitive-behavioral therapy (CBT), insight-oriented therapies, dialectical behavioral therapy (DBT), hypnotherapy, and eye movement desensitization and reprocessing (EMDR).



Figure **6.12.1**: In the case of DID, most people with the disorder seek treatment for other psychological problems, like depression, and DID is often difficult to detect. It may take many years for a therapist to notice symptoms of DID.

Although there are multiple approaches for treating dissociative disorders, the common element of these treatments addresses the dissociative pathology and exploring prior traumatic events. Treatment of dissociative disorders is associated with improvements in symptoms of dissociation, depression, general distress, anxiety, and PTSD, as well as decreased use of medications and improved work and social functioning (Brand et al. 2009). Duration of treatment varies depending on the particular dissociative disorder being treated, with dissociative amnesia and dissociative fugue recovering more quickly and having a better outcome as compared to dissociative identity disorder and depersonalization disorder. However, a significant proportion of patients' improvement during initial treatment may not remain stable over time, indicating the need for additional follow-up for contingent intervention in the case of recurrent dissociative symptoms or other psychopathological states.

There is no consensus in the treatment of DID, and research on treatment effectiveness focuses mainly on clinical approaches described in case studies. General treatment guidelines suggest a phased, eclectic approach with more concrete guidance and agreement on early stages; however, no systematic, empirically supported approach exists and later stages of treatment have no consensus. Even highly experienced therapists have few patients that achieve a unified identity.

Unlike DID, the length of an event of dissociative amnesia may be a few minutes or several years. If an episode is associated with a traumatic event, the amnesia may clear up when the person is removed from the traumatic situation. Similarly, once dissociative fugue is discovered and treated, many people recover quickly.

Medications can be used for comorbid disorders and/or targeted symptom relief. Medications (such as antidepressants, anti-anxiety medications, or tranquilizers) can help control the mental health symptoms associated with the disorders; however, there are no medications that specifically treat dissociative disorders.

Treating Somatic Symptom Disorders

Treatment for conversion disorder can include hypnosis, psychotherapy, physical therapy, occupational therapy, stress management, and transcranial magnetic stimulation (TMS). Transcranial magnetic stimulation (TMS) is a form of brain stimulation in which a changing magnetic field is used to cause an electric current at a specific area of the brain through electromagnetic induction.

In treating somatic symptoms (such as conversion disorder, factitious disorder, etc.), the provider may need to avoid **colluding**with the patient, but also avoid denying the reality of the symptoms. Research has shown that empowering explanations are the most beneficial for patients with medically unexplained symptoms. Psychoeducation is also very important. Patients with medically unexplained symptoms often appear to be seeking reassurance, but this assurance can be difficult to deliver effectively. It is counterproductive to tell a patient that "there is nothing wrong" when their symptoms are proof that there is. On the other hand, it is important to counter specific illness fears that the patient may hold (e.g., "My symptoms mean I've got cancer," "This rash shows that I have HIV," "If I do too much I will permanently damage my spine") if that is not the case.



Figure **6.12.2**: Copy and Paste Caption here. (Copyright; author via source)

If there is evidence of anxiety or depression, this should be treated in the usual way. Doing so will often, although not always, lead to a significant improvement in the patient's somatic symptoms. A doctor that sees a patient with medically unexplained symptoms for follow-up has an important role to play in managing that patient's interaction with medical services. Even if the doctor does not perceive themselves to be providing active therapy, they can provide regular follow-up that is not contingent on the patient being symptomatic, thereby discouraging the need for the patient to complain of symptoms in order to elicit care. It is sometimes possible to agree beforehand that only a certain proportion of the session will be devoted to discussing symptoms, and leave it to the patient to decide the content of the second half of the interview.

Overall CBT and similar therapies have shown specific usefulness in the treatment of somatic symptom disorders. Though CBT can be adapted for use in any of these disorders, like most medical treatments, it relies on the patient being sufficiently motivated to participate. One of the first goals in CBT is for the therapist and patient to come to a shared understanding of the patient's problems using a CBT framework. Additionally, psychopharmacology can be useful in the treatment of somatic symptom disorders, especially if there's comorbid depression or anxiety. In this case, antidepressants may be prescribed.

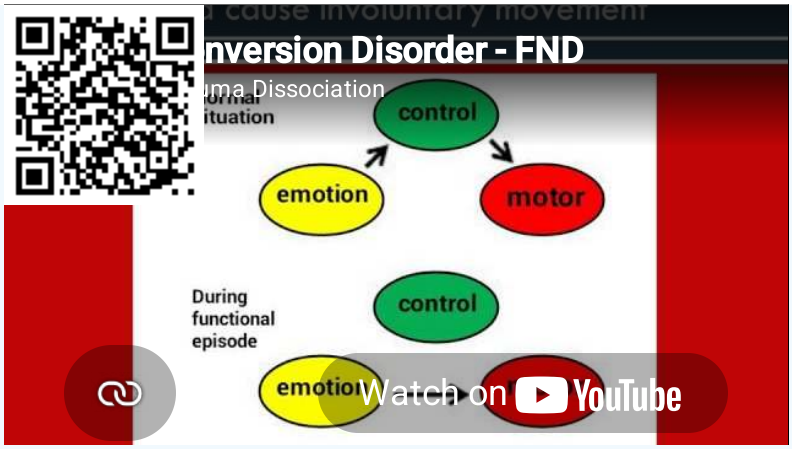
In clinical practice, it is common to combine a psychotherapeutic and pharmacological approach to management. The patient may have strong feelings about treatment and these should be taken into consideration.

Treating Conversion Disorder: A Case Study

Even when a clear psychological cause is known, some physical treatment may be helpful in combination with *talking therapy*, as described in this case of a 13-year old girl with hand tremors.

Watch It

Watch this video to see the hand tremors in a girl and the improvement following treatment (note that there is no sound).



You can view a descriptive transcript for "Conversion Disorder – FND" here (opens in new window).

The conversion disorder hand tremor shown in the video occurred in a 13-year-old girl, who was treated with both a customized wrist brace and psychological therapy using cognitive behavioral therapy.

Her severe psychogenic tremor was diagnosed as a dissociative motor disorder and was videotaped at different stages during treatment. The videos were useful in showing the girl clear evidence of her improvements. The cause was psychological: severe performance and exam anxiety at school, combined with her parents' difficult relationship. Her hand tremor prevented writing and attending school, increasing her avoidance of school.

"Stop pretending" made things worse

The tremor was inconsistent, reducing when her concentration was on something else and would stop when she grasped something, e.g., clasping her hands together. Finding no neurological or physical cause for the tremor, a physiotherapist told her to "stop pretending," which led to prolonged sadness and anger in the girl and an increase in the tremor. Secondary gain is sometimes referred to in conversion disorder despite not being part of the diagnostic criteria. This means the person's conversion disorder causes an indirect benefit: any secondary gain from not needing to attend school was clearly canceled out by her inability to sleep or eat normally due to the tremor, plus the distress it caused her. She was also hospitalized and agreed to attend the psychiatric unit. Even in cases where secondary gain is present, this should not be used as a basis for diagnosis.

Treatment

The patient had anxiety-reduction sessions and learned progressive muscle relaxation. Her customized wrist brace to help her write, and she practiced writing daily. This reduced her school anxiety and avoidance, and she began attending again. She also had private lessons in the subjects she struggled with the most. In her treatment, it was key for her to understand what triggered the tremor and prevented it from diminishing. From the girl's perspective, she found what helped most was being more active rather than listening to music alone, physical and social activity, identifying and discussing her feelings with others, eventually mastering the relaxation methods to reduce stress, and attending physical therapy aimed at symptom reduction. The combination of treatment reduced her tremor by 80% during her time in the hospital, and two weeks after discharge it was gone.

Glossary

colluding: when a therapist participates *with* a client, buying into what they are saying either consciously or unconsciously, and by doing so avoids an issue that needs to be addressed: for patients with DID, this may involve the therapist asking to reveal the different personalities and to ask questions out of curiosity instead of helping to address specific concerns

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6.13: Case Studies- Somatic Symptom and Dissociative Disorders

Learning Objectives

- Identify somatic disorders in case studies

Case Study: Samuel



The patient, named “Samuel,” is a 28-year-old, final year medical student from the south-eastern region of Nigeria in sub-Saharan Africa. He was declared missing for 10 days then later seen in a city in south-western Nigeria, a distance of about 634 km from south-eastern Nigeria where he lived and schooled. Ten days before his presentation, while studying in his room alone at night, Samuel suddenly saw a full human skeleton reading at the same table with him, sitting at the opposite side. At the same time, he claimed he felt uneasy and quite uncomfortable. He saw the whole room turning with everything inside becoming unstable and unreal. After this experience, Samuel had overwhelming fears and did not know when he left the room. Two days later, he discovered he was with his younger sibling in south-western Nigeria. Samuel had no knowledge of how he made the journey that takes approximately eight hours by road. He equally could not remember where he slept the night he left his room, how he raised money for the journey, or the buses and routes he took. Samuel denied all memory of events for the two days from when he left his room at the university to the time he suddenly realized he was at his brother’s house, hours away. The brother, however, reported that Samuel appeared unkempt, looked exhausted but was fully conscious and alert on arrival at his house without any assistance.

Prior to this episode, Samuel had been under severe economic and academic pressures. The younger brother, who paid the patient’s bills, had threatened to withdraw his sponsorship because of Samuel’s prolonged stay in school beyond the stipulated duration of training due to his repeats of examinations and classes. Samuel had been worried that he might also fail in his final qualifying examinations scheduled to be held in three months. He subsequently became involved in several religious activities to obviate his perception of impending doom.

Samuel admitted to having a low mood, loss of interest in usually pleasurable activities, and poor appetite. He had lost weight and most often preferred being alone. He had also been feeling weak, especially in the morning hours, but had managed to grudgingly carry on with the day’s activities. He had suicidal ideation but never attempted suicide. Samuel slept poorly at night. His sleep had been marked by early morning wakefulness and waking up not feeling refreshed.

There were no symptoms suggestive of seizure, manic episode, schizophrenia, anxiety, or organic disorders. Samuel never drank alcohol or abused any psychoactive substances. He denied a history of head trauma or loss of consciousness in the past. Past medical, psychiatric, family, and personal histories revealed no significant findings.

Examination of his mental state revealed a young man who was clean, appropriately dressed, and mildly emaciated with poor eye contact. His mood was depressed. He had no thought disorders. Samuel was oriented in time, place, and person but had impaired attention and concentration at the time of the examination. Immediate recall, short- and long-term memory were intact. However, there was amnesia for the two days he wandered away from school. Judgment and insight were not impaired.

His physical examination was unremarkable. Neurological assessment and basic laboratory testing revealed no significant abnormalities.[/footnote]Igwe, M.N. *Dissociative fugue symptoms in a 28-year-old male Nigerian medical student: a case report*. J Med Case Reports 7, 143 (2013). https://doi.org/10.1186/1752-1947-7-143[/footnote]

Treatment

In regards to the case study about Samuel written above, the patient engaged in psychotherapy by clinical psychologists, and his depression was treated with paroxetine. He responded very well and was able to write his final qualifying examinations three months later. He, however, did not pass either of the two subjects examined. At a six-month follow up, Samuel could still not recall events for the two days from when he left school to the time he was seen in his brother's house, 634 km away. He reported no further periods of amnesia or wandering away from his place of residence.[/footnote]Igwe, M.N. *Dissociative fugue symptoms in a 28-year-old male Nigerian medical student: a case report*. J Med Case Reports 7, 143 (2013). https://doi.org/10.1186/1752-1947-7-143[/footnote]

Case Study: Ali

A 73-year-old Saudi man, "Ali" started visiting the primary health care center in our institution 25 years ago. He was concerned with having cancer that would give him only a few days to live. In the beginning, Ali was evaluated medically through a detailed history and documentation of his symptoms and then a management plan was created accordingly to exclude cancer. Full history, physical examinations, and radiological and pathological investigations were ordered and the results were all negative for cancer. The physician explained the results of the investigations to Ali, but he refused them and continued to insist that he had cancer regardless of the results. He was then referred to the Psychiatry Department to be evaluated but he could not realize that his symptoms might be of a nonorganic cause, either psychological or mental.

Ali continued to visit the general hospital, emergency department, and the primary health care in the institution and was still occupied with the idea of cancer. Although he was seen by many physicians, Ali was never satisfied with their conclusions. Eventually, a physician reported the case to the department of medical eligibility, addressing the issue of continuous primary health care center visits with very variable symptoms, nonconclusive diagnosis, and an unconvinced patient. The department of medical eligibility in the hospital administration decided to temporarily limit Ali's file to the psychiatry department to drive Ali to visit the psychiatrist to be evaluated psychologically in order to make his file eligible again. Ali was unhappy at the beginning but he had to visit the psychiatrist. So, an appointment with the psychiatrist was booked and a full psychological and social evaluation was performed by taking a thorough history from Ali. The family and social history evaluation revealed Ali's very low SES and similar conditions in the family. Following this extensive detailed history and evaluation, the treating psychiatrist contacted the department of medical eligibility to make Ali's file eligible again as promised by them with unlimited eligibility to all specialties and the primary care center. For the past 25 years of continuous hospital visiting, Ali had almost weekly and even daily visits to the primary care facility. Ali kept complaining of variable symptoms every week and specifically asking for certain lab tests and radiological studies. Also, he successfully convinced some physicians to order a biopsy for him.

Over the years, Ali's investigations cost approximately $178,200. His most requested investigations were mainly invasive radiological studies specifically abdomen and pelvis CT (18 times) and a wide range of other invasive and noninvasive studies like chest CT (11 times), brain CT (seven times), chest MRI (four times), lumbar spine MRI (six times), and other studies. In addition, he asked for a variety of lab tests and underwent several biopsies.[1]

Treatment

Ali was treated as follows:

Five years ago, an experienced board-certified family physician was shocked when he looked at Ali's file and decided to take some action to stop Ali's costly visits. First, they limited Ali's primary care visits to only one clinic where he would be referred to the supervising physician himself. Then, they worked to build strong and effective patient rapport based on trust and honesty. After negotiations, the patient agreed to enroll in CBT for six sessions at 90 minutes each. The patient was advised to have a scheduled sleeping time, ensure healthy eating habits with regular physical exercise, be involved in social activities, stay away from stressors like searching the web for symptoms, and avoid TV health shows and health magazines during active cycles of the disorder. After that, there was an agreement with the patient not to disturb the clinic and he would get to see the physician regularly for checkups every three months.

After five years of implementing the new rules, Ali is still visiting the clinic for regular follow-up and sometimes he breaks the rules and comes regularly to the clinic, especially in active cycles of the disorder, though there is a dramatic decrease in the total financial cost due to the relative decreasing number of visits, less time spent on arguing with the patient, and the filtration of his

requests. Moreover, there is a remarkable improvement in the patient's condition because of CBT and relative compliance to health advice.[2]

Case Study: Mia

A 15-year-old white female, "Mia" presented with flu-like symptoms that progressed two days later to throwing up blood. She was sent to the emergency room where she was diagnosed with possible appendicitis and was discharged the next day. That weekend, she was still vomiting, unable to hold food down, and nauseated. Mia was admitted to Arkansas Children's Hospital (ACH) for one to two weeks where she received extensive workup including MRI, CT scan, gastrointestinal imaging, and a spinal tap, and all the results came back normal. She was also evaluated by an ophthalmologist and a cardiologist, and no organic etiology was found for her symptoms. Mia was discharged home with an unknown diagnosis and started on symptomatic medical management to help relieve some of the symptoms. Soon after she was discharged from ACH, her legs started getting weak, and it was discussed that she may be having some neurological disorders and would need to be evaluated further. Presenting with symptoms of throwing up, unable to keep food down, leg numbness, and a constant migraine, Mia was readmitted at ACH for another two weeks where even more extensive workup was done again, including CT and MRI scans of the brain. All results came back normal, and she was sent home without a true diagnosis and was treated for depression and anxiety.

Mia was referred to a neurologist at the University of Arkansas for Medical Sciences (UAMS) two to three weeks later where she was evaluated and admitted to the hospital for one day to treat a migraine. The therapist uncovered that from the age of six to 12 years, Mia experienced two active addicted parents, and she received physical and mental abuse from her father. During that time, she was required to become the head of the household to ensure bills were being paid and that her parents were being taken care of. At the age of 12, her mom received de-addiction treatment for three to four months and has been clean and sober for the past five years. Between the ages of 12 and 13, her parents got divorced, and she moved with her mom. Mia attended high school for one year but then transferred to another city.[3]

Treatment

With no evidence of any organic etiology found on the extensive workup and no sign of relief with continued treatment, Mia was diagnosed with conversion disorder. She was discharged to receive physical therapy (three times a week) and CBT (once a week) and also seen by a therapist. Seven months later, she was referred and accepted into the Mayo Clinic as an outpatient for further management. At Mayo Clinic, she went to group therapy every day from 8 a.m. to 4 p.m. for four weeks with people who were experiencing other neurological disorders, not just conversion disorder. During the group therapy at Mayo Clinic, she not only went through physical therapy and occupational therapy but also learned how to stay on a schedule, how to live a normal life with conversion disorder, and how to manage and tolerate the pain. She believes that the group therapy at Mayo helped her the best in her recovery.

Mia states that there were no real triggers that caused this disorder, but her history could have been an underlying cause. She is now still in recovery, all symptoms have improved but not completely gone away. She still experiences numbness and weakness on the right side, headaches, nausea, and pain in the right leg. She learned, at Mayo Clinic, how to tolerate and deal with the pain, which she believes helps her. Mia is currently on Topamax, iron, vitamin D3, and calcium. She plans to graduate high school this year and plans to attend college as a premedical student.[4]

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6.14: Putting It Together- Somatic Symptom Disorders and Dissociative Disorders

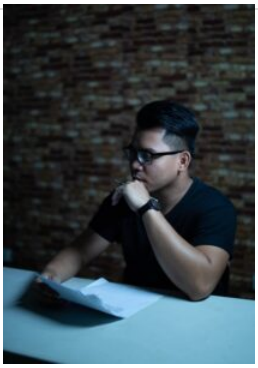


Figure **6.14.1**: Reflecting on studied material, and considering how it might be applied today or in your future vocation is an important part of learning.

In this chapter, you learned about dissociative disorders such as dissociative amnesia, DID, and depersonalization/derealization. You also reviewed somatic symptom and related disorders, including illness anxiety disorder, conversion disorder, and factitious disorder. By this point, you should have a basic, yet firm grasp on understanding how each is unique in its presentation of symptoms, how they affect people, and some of the common treatment methods. Take a look at some of the popular treatment methods. What might a treatment plan look like if you were to develop one based on a certain psychological perspective? Or how might one's theoretical background or way of thinking about a disorder prompt the research of a helpful method for treating it? How might you respond if you were a therapist treating someone with symptoms similar to those outlined in this module? Given how often these disorders may be misdiagnosed and/or undiagnosed for years, consider the challenge this presents to counselors. It's important to take time to reflect on learning and think critically about concepts.

As you reflect on what you've gleaned from this module, spend a moment reviewing the disorders discussed in this module:

- **conversion disorder:** a type of somatic disorder that is sometimes applied to patients who present with neurological symptoms which are not otherwise explained by a well-established organic cause, and often can be traced back to a psychological trigger
- **dissociative amnesia:** dissociative disorder characterized by an inability to recall important personal information, usually following an extremely stressful or traumatic experience
- **depersonalization/derealization disorder:** dissociative disorder in which people feel detached from the self (depersonalization), and the world feels artificial and unreal (derealization)
- **dissociative identity disorder (DID):** formerly known as multiple personality disorder, DID is at the far end of the dissociative disorder spectrum; characterized by at least two distinct, and dissociated personality states which alternately control a person's behavior; sufferer, therefore, experiences significant memory impairment for important information not explained by ordinary forgetfulness
- **dissociative fugue:** symptom of dissociative amnesia in which a person suddenly wanders away from one's home and experiences confusion about his or her identity
- **factitious disorder:** a condition in which a person, without a malingering motive, acts as if they have an illness by deliberately producing, feigning, or exaggerating symptoms, purely to attain (for themselves or for another) a patient's role; presents in two ways: factitious disorder imposed on self occurs when someone feigns illness so *they* experience being the patient and factitious disorder imposed on another is a condition in which a person deliberately produces, feigns, or exaggerates the symptoms of someone in their care
- **illness anxiety disorder:** a disorder in which a person does not typically have physical symptoms, rather they are preoccupied with a fear of developing a severe medical condition; two categories of patients in this group are the care-seeking type and care-avoidant type
- **insomnia:** a sleep disorder in which there is an inability to fall asleep or to stay asleep as long as desired; symptoms also include waking up too early, experience many awakenings during the night, and not feeling rested during the day
- **somatic symptom disorder:** a mental disorder that manifests as physical symptoms that suggest illness or injury, but cannot be explained fully by a general medical condition

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CHAPTER OVERVIEW

7: Mood Disorders

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7.4: Subtypes of Depression and Persistent Depressive Disorder
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7.1: Why It Matters- Mood Disorders

Why learn about mood disorders?



Everyone feels down or euphoric from time to time, but this is different from having a mood disorder such as major depressive disorder or bipolar disorder. Mood disorders are extended periods of depressed, euphoric, or irritable moods that in combination with other symptoms cause the person significant distress and interfere with his or her daily life, often resulting in social and occupational difficulties.

In this module, we describe major mood disorders, including their symptom presentations, general prevalence rates, and how and why the rates of these disorders tend to vary by age, gender, and race. In addition, biological and environmental risk factors that have been implicated in the development and course of mood disorders, such as heritability and stressful life events, are reviewed. Finally, we provide an overview of treatments for mood disorders, covering treatments with demonstrated effectiveness, as well as new treatment options showing promise.

Watch It

Watch this video for a good overview of the types of mood disorders covered in this module.



You can view the transcript for "Depressive and Bipolar Disorders: Crash Course Psychology #30" here (opens in new window).

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7.2: Introduction to Depressive Disorders

What you'll learn to do: describe the characteristic symptoms and risk factors of depressive disorders



Mood disorders are those in which the person experiences severe disturbances in mood and emotion. Depressive disorders include major depressive disorder, which is characterized by episodes of profound sadness and loss of interest or pleasure in usual activities and other associated features, and persistent depressive disorder, which is marked by a chronic state of sadness. Other depressive disorders include depressive disorder due to another medical condition, other specified or unspecified depressive disorders, and premenstrual dysphoric disorder (PMDD).

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7.3: Major Depressive Disorder

Learning Objectives

- Describe the symptoms and risk factors of major depressive disorder

Jacob cries all day and, feeling that he is worthless and his life is hopeless, he cannot get out of bed. Ava stays up all night, talks very rapidly, and went on a shopping spree in which she spent $3,000 on furniture, although she cannot afford it. Maria recently had a baby and she feels overwhelmed, teary, anxious, and panicked, and believes she is a terrible mother for practically every day since the baby was born. All these individuals demonstrate symptoms of a potential mood disorder.

Mood disorders (Figure **7.3.1**) are characterized by severe disturbances in mood and emotions—most often depression, but also mania and elation (Rothschild, 1999). All of us experience fluctuations in our moods and emotional states, and often these fluctuations are caused by events in our lives. We become elated if our favorite team wins the World Series and dejected if a romantic relationship ends or if we lose our job. At times, we feel fantastic or miserable for no clear reason. People with mood disorders also experience mood fluctuations, but their fluctuations are extreme, distort their outlook on life, and impair their ability to function.



Figure **7.3.1**: Mood disorders are characterized by massive disruptions in mood. Symptoms can range from the extreme sadness and hopelessness of depression to the extreme elation and irritability of mania. (credit: Kiran Foster)

The DSM-5 lists two general categories of mood disorders. Depressive disorders are a group of disorders in which depression is the main feature. Depression is a vague term that, in everyday language, refers to an intense and persistent sadness. Depression is a heterogeneous mood state—it consists of a broad spectrum of symptoms that range in severity. Depressed people feel sad, discouraged, and hopeless. These individuals lose interest in activities once enjoyed, often experience a decrease in drives such as hunger and sex, and frequently doubt personal worth. Depressive disorders vary by degree, but this module highlights the most well-known ones: major depressive disorder and persistent depressive disorder (which both are types of unipolar depression).

Bipolar and related disorders are a group of disorders in which mania is the defining feature. Mania is a state of extreme elation and agitation. When people experience mania, they may become extremely talkative, behave recklessly, or attempt to take on many tasks simultaneously. The most recognized of these disorders is bipolar disorder.

What Is Major Depressive Disorder?

According to the DSM-5, the defining symptoms of **major depressive disorder (MDD)** include “depressed mood most of the day, nearly every day” (feeling sad, empty, or hopeless or appearing tearful to others), and loss of interest and pleasure in usual activities (APA, 2013). In addition to feeling overwhelmingly sad most of each day, people with depression will no longer show interest or enjoyment in activities that previously were gratifying, such as hobbies, sports, sex, social events, time spent with family, and so

on. Friends and family members may notice that the person has completely abandoned previously enjoyed hobbies; for example, an avid tennis player who develops major depressive disorder no longer plays tennis (Rothschild, 1999).

To receive a diagnosis of major depressive disorder, one must experience a total of five symptoms for at least a two-week period; these symptoms must cause significant distress or impair normal functioning, and they must not be caused by substances or a medical condition. At least one of the two symptoms mentioned above must be present, plus any combination of the following symptoms (APA, 2013):

- significant weight loss (when not dieting) or weight gain and/or significant decrease or increase in appetite;
- difficulty falling asleep or sleeping too much;
- psychomotor agitation (the person is noticeably fidgety and jittery, demonstrated by behaviors like the inability to sit, pacing, hand-wringing, pulling or rubbing of the skin, clothing, or other objects) or psychomotor retardation (the person talks and moves slowly, for example, or talks softly, very little, or in a monotone voice);
- fatigue or loss of energy;
- feelings of worthlessness or guilt;
- difficulty concentrating and indecisiveness; and
- **suicidal ideation**: thoughts of death (not just fear of dying), thinking about or planning suicide, or making an actual suicide attempt.

Major depressive disorder (MDD) is considered episodic: its symptoms are typically present at their full magnitude for a certain period of time and then gradually abate. Approximately 50–60% of people who experience an episode of major depressive disorder (MDD) will have a second episode at some point in the future; those who have had two episodes have a 70% chance of having a third episode, and those who have had three episodes have a 90% chance of having a fourth episode (Rothschild, 1999). Although the episodes can last for months, a majority of people diagnosed with this condition (around 70%) recover within a year. However, a substantial number do not recover; around 12% show serious signs of impairment associated with major depressive disorder after five years (Boland & Keller, 2009). In the long term, many who do recover will still show minor symptoms that fluctuate in their severity (Judd, 2012).

Prevalence

Major depressive disorder affected approximately 163 million people in 2017 (2% of the global population).[1] The percentage of people who are affected at one point in their life varies from 7% in Japan to 21% in France. In most countries, the number of people who have depression during their lives falls within an eight to 18% range. In North America, the probability of having a major depressive episode within a year-long period is 3%-5% for males and eight to 10% for females. Major depression is about twice as common in women as in men, although it is unclear why this is so and whether factors unaccounted for are contributing to this. The relative increase in occurrence is related to pubertal development rather than chronological age, reaches adult ratios between the ages of 15 and 18, and appears associated with psychosocial more than hormonal factors. Depression is a major cause of disability worldwide.

People are most likely to develop their first depressive episode between the ages of 30 and 40, and there is a second, smaller peak of incidence between ages 50 and 60. The risk of major depression is increased with neurological conditions such as stroke, Parkinson's disease, or multiple sclerosis, and during the first year after childbirth. It is also more common after cardiovascular illnesses and is related more to those with a poor cardiac disease outcome than to a better one. Studies conflict on the prevalence of depression in the elderly, but most data suggest there is a reduction in this age group. Depressive disorders are more common in urban populations than in rural ones and the prevalence is increased in groups with poorer socioeconomic factors, e.g., homelessness.

Causes of Depression

MDD is believed to be a combination of genetic, environmental, and psychological factors. Risk factors include a family history of the condition, major life changes, certain medications, chronic health problems, and substance abuse. About 40% of the risk appears to be related to genetics.[2]

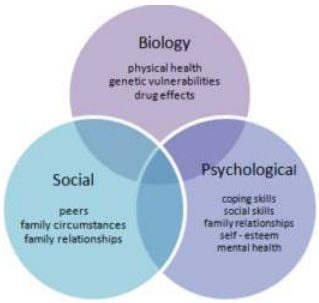


Figure **7.3.2**: The biopsychosocial model shows that a variety of factors may influence mental health.

The diathesis-stress model specifies that depression results when a preexisting vulnerability, or diathesis, is activated by stressful life events. The pre-existing vulnerability can be either genetic, implying an interaction between nature and nurture, or schematic, resulting from views of the world learned in childhood.

The biopsychosocial model is an interdisciplinary model that looks at the interconnection between biological, psychological, and social-environmental factors. The model specifically examines how these aspects play a role in topics ranging from health and disease models to human development. Childhood abuse, whether physical, sexual, or psychological, are all risk factors for depression, among other psychiatric issues that co-occur such as anxiety and drug abuse. Childhood trauma also correlates with the severity of depression, lack of response to treatment, and length of illness. However, some are more susceptible to developing mental illness such as depression after trauma, and various genes have been suggested to control susceptibility.

Watch It

Research shows us that the neurotransmitter serotonin plays a role in depression, but that role may not be a clear cut as once hypothesized. The monoamine theory, derived from the efficacy of monoaminergic drugs (such as SSRIs) in treating depression, was the dominant theory until recently. Studies have shown that depletion of tryptophan, a necessary precursor of serotonin, a monoamine, can cause depression in those in remission or relatives of depressed patients; this suggests that decreased serotonergic neurotransmission is important in depression. There is also a correlation between depression risk and polymorphisms in the 5-HTTLPR gene, which codes for serotonin receptors. This and other research suggests a link between serotonin and depression. However, the monamine theory is inconsistent with the fact that serotonin depletion does not cause depression in healthy persons, the fact that antidepressants instantly increase levels of monoamines but take weeks to work, and the existence of atypical antidepressants, which can be effective despite not targeting this pathway.

This video explains a little bit of the research and explains other theories that help to explain depression.



You can view the transcript for “Why Depression Isn’t Just a Chemical Imbalance” here (opens in new window).

Results of Major Depressive Disorder

Major depressive disorder is a serious and incapacitating condition that can have a devastating effect on the quality of one's life. The person suffering from this disorder lives a profoundly miserable existence that often results in unavailability for work or education, abandonment of promising careers, and lost wages; occasionally, the condition requires hospitalization. The majority of those with major depressive disorder report having faced some kind of discrimination and many report that having received such treatment has stopped them from initiating close relationships, applying for jobs for which they are qualified, and applying for education or training (Lasalvia et al., 2013). Major depressive disorder also takes a toll on health. Depression is a risk factor for the development of heart disease in healthy patients, as well as adverse cardiovascular outcomes in patients with preexisting heart disease (Whooley, 2006).

Risk Factors for Major Depressive Disorder

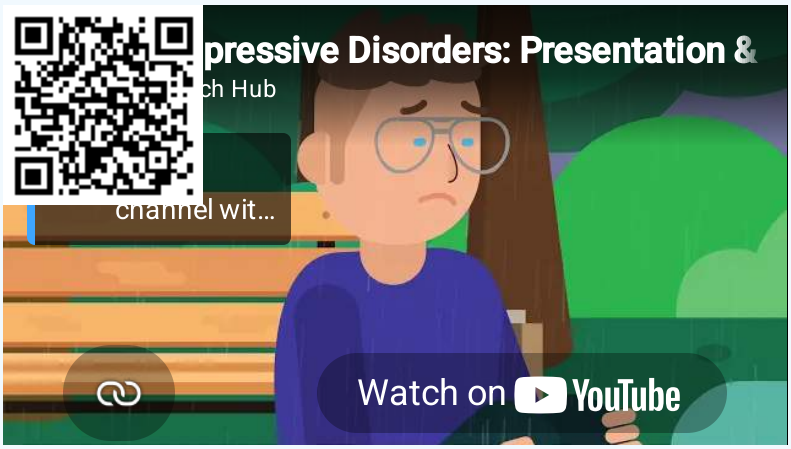
Major depressive disorder is often referred to as the common cold of psychiatric disorders. Around 6.6% of the U.S. population experiences major depressive disorder each year; 16.9% will experience the disorder during their lifetime (Kessler & Wang, 2009). It is more common among women than among men, affecting approximately 20% of women and 13% of men at some point in their lives (National Comorbidity Survey, 2007). The greater risk among women is not accounted for by a tendency to report symptoms or to seek help more readily, suggesting that gender differences in the rates of major depressive disorder may reflect biological and gender-related environmental experiences (Kessler, 2003).

Lifetime rates of major depressive disorder tend to be highest in North and South America, Europe, and Australia; they are considerably lower in Asian countries (Hasin, Fenton, & Weissman, 2011). The rates of major depressive disorder are higher among younger age cohorts than among older cohorts, perhaps because people in younger age cohorts are more willing to admit depression (Kessler & Wang, 2009).

A number of risk factors are associated with major depressive disorder: unemployment (including homemakers); earning less than $20,000 per year; living in urban areas; or being separated, divorced, or widowed (Hasin et al., 2011). Comorbid disorders include anxiety disorders and substance abuse disorders (Kessler & Wang, 2009).

Watch It

This video explains depression from the viewpoint of a clinical provider. Doctors, nurses, and other medical professionals should be aware of the signs of major depression (and other depressive disorders such as persistent depressive disorder) in order to ensure that individuals get the appropriate help they need.



You can view the transcript for "Depressive Disorders: Presentation & Treatment" here (opens in new window).

Glossary

depressive disorder: one of a group of mood disorders in which depression is the defining feature

flight of ideas: symptom of mania that involves abruptly switching in conversation from one topic to another

hopelessness theory: cognitive theory of depression proposing that a style of thinking that perceives negative life events as having stable and global causes leads to a sense of hopelessness and then to depression

major depressive disorder: commonly referred to as *depression* or *major depression*, characterized by sadness or loss of pleasure in usual activities, as well as other symptoms

mood disorder: one of a group of disorders characterized by severe disturbances in mood and emotions; the categories of mood disorders listed in the DSM-5 are bipolar and related disorders and depressive disorders

rumination: in depression, tendency to repetitively and passively dwell on one's depressed symptoms, their meanings, and their consequences

suicide: death caused by intentional, self-directed injurious behavior

1. GBD 2017 Disease and Injury Incidence and Prevalence Collaborators (10 November 2018). "Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017". Lancet. 392 (10159): 1789–1858. doi:10.1016/S0140-6736(18)32279-7. PMC 6227754. PMID 30496104. Retrieved 23 June 2020. ↵
2. American Psychiatric Association (2013), Diagnostic and Statistical Manual of Mental Disorders (5th ed.), Arlington: American Psychiatric Publishing, pp. 160–68, ISBN 978-0-89042-555-8, retrieved 22 July 2016 ↵

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7.4: Subtypes of Depression and Persistent Depressive Disorder

Learning Objectives

- Describe subtypes of depression, including seasonal pattern and peripartum onset depression

Subtypes of Depression

The DSM-5 lists several different subtypes of depression. These subtypes—what the DSM-5 refers to as specifiers—are not specific disorders; rather, they are labels used to indicate specific patterns of symptoms or to specify certain periods of time in which the symptoms may be present. These specifiers are listed following the description of major depressive disorder along with whether it is a single or recurrent episode. They add clarification about the type of depression and are designated as follows:

- **anxious distress**: a person experiences anxiety in the form of tenseness, restlessness, lack of focus, fear, or of losing control
- **mixed features**: a person experiences some manic symptoms alongside depression, such as elevated moods, increased energy, or talkativeness
- **melancholic features**: a person experiences lack of interest or pleasure in activities
- **atypical features**: a person dealing with depression may also experience elevated moods for certain periods of time or around particular people, show weight gain or increased appetite, or excessive sleep
- **mood-congruent psychotic features**: a person may also experience delusions or hallucinations
- **catatonia**: a person may show catatonic symptoms, such as staying still or immobilized for long periods of time
- **seasonal pattern:** (formerly known as seasonal affective disorder) applies to situations in which a person experiences the symptoms of major depressive disorder only during a particular time of year (e.g., fall or winter); in everyday language, often referred to as the winter blues
- **peripartum onset**: symptoms appear during pregnancy or in the four weeks following birth



Figure 7.4.1: Other catatonic symptoms can include lack of speech and strange movements.

Watch It

Major depressive disorder with season pattern, also known as season affective disorder, is described in more detail in the following video. One popular treatment method is light therapy, which involves sitting close to a bright UVB light that can mimic sunlight, which produces vitamin D.



You can view the transcript for “Seasonal affective disorder – causes, symptoms, diagnosis, treatment, pathology” here (opens in new window).

Peripartum Onset Depression

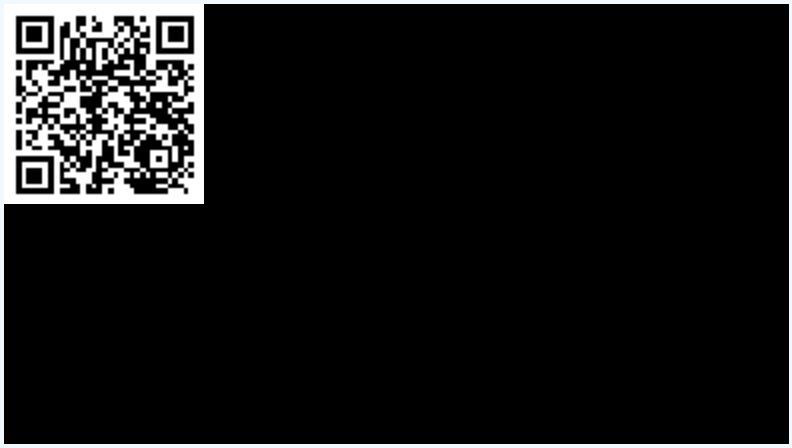
Another subtype, **peripartum onset** (commonly referred to as **postpartum depression**), applies to women who experience major depression during pregnancy or in the first four weeks following the birth of the child (APA, 2013). These women often feel very anxious and may even have panic attacks. They may feel guilty or agitated and be weepy. They may not want to hold or care for their newborn, even in cases in which the pregnancy was desired and intended. In extreme cases, the mother may have feelings of wanting to harm her child or herself. In a horrific illustration, a woman named Andrea Yates, who suffered from extreme peripartum-onset depression (as well as other mental illnesses), drowned her five children in a bathtub (Roche, 2002). Most women with peripartum-onset depression do not physically harm their children, but most do have difficulty being adequate caregivers (Fields, 2010). A surprisingly high number of women experience symptoms of peripartum-onset depression. A study of 10,000 women who had recently given birth found that 14% screened positive for peripartum-onset depression, and that nearly 20% reported having thoughts of wanting to harm themselves (Wisner et al., 2013).

Postpartum Depression

The actress Brooke Shields published a memoir titled *Down Came the Rain: My Journey through Postpartum Depression* in which she described her struggles with depression following the birth of her daughter. Despite the fact that about one in 20 women experience depression after the birth of a baby (American Psychiatric Association [APA], 2013), postpartum depression —recently renamed “perinatal depression”—continues to be veiled by stigma, owing in part to a widely held expectation that motherhood should be a time of great joy.

In an opinion piece in the New York Times, Shields revealed that entering motherhood was a profoundly overwhelming experience for her. She vividly describes experiencing a sense of “doom” and “dread” in response to her newborn baby. Because motherhood is conventionally thought of as a joyous event and not associated with sadness and hopelessness, responding to a newborn baby in this way can be shocking to the new mother as well as those close to her. It may also involve a great deal of shame for the mother, making her reluctant to divulge her experience to others, including her doctors and family.

This video from the National Institute on Mental Health explains more about the disorder.



You can view the transcript for “”Baby Blues” — or Postpartum Depression?” here (opens in new window).

Link to Learning

Dr. Fatimah Jackson-Best has done extensive research on the impact of postpartum depression and mental health on women in developing nations, specifically in areas of the Caribbean. She explains some of her research in this TED talk.

Persistent Depressive Disorder

People with **persistent depressive disorder** (previously known as **dysthymia**) experience mild to moderate depressed moods most of the day nearly every day for at least two years, as well as at least two of the other symptoms of major depressive disorder. People with persistent depressive disorder are chronically sad and melancholy, but do not meet all the criteria for major depression. However, episodes of full-blown major depressive disorder can occur during persistent depressive disorder (APA, 2013).

There are no known biological causes that apply consistently to all cases of dysthymia, which suggests diverse origins for the disorder. However, there are some indications that there is a genetic predisposition to dysthymia as depression is found in as many as 50% of family members of someone with dysthymia. In a study using identical and fraternal twins, results indicated that there is a stronger likelihood of identical twins both having depression than fraternal twins. This provides support for the idea that dysthymia is in part caused by heredity. Other factors linked with dysthymia include stress, social isolation, and lack of social support.

A combination of antidepressant medication and psychotherapy has consistently been shown to be the most effective line of treatment for people diagnosed with dysthymia. Working with a psychotherapist to address the causes and effects of the disorder, in addition to taking antidepressants to help eliminate the symptoms, can be extremely beneficial. This combination is often the preferred method of treatment for those who have dysthymia. Looking at various studies involving treatment for dysthymia, 75% of people responded positively to a combination of cognitive behavioral therapy (CBT) and pharmacotherapy, whereas only 48% of people responded positively to just CBT or medication alone.

Glossary

peripartum onset: subtype of depression that applies to women who experience an episode of major depression either during pregnancy or in the first four weeks following childbirth

persistent depressive disorder: depressive disorder characterized by a chronically sad and melancholy mood

seasonal pattern: subtype of depression in which a person experiences the symptoms of major depressive disorder only during a particular time of year

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7.5: Disruptive Mood Dysregulation Disorder

Learning Objectives

Describe symptoms, characteristics, and treatment methods related to disruptive mood dysregulation disorder

Disruptive mood dysregulation disorder (DMDD) is a childhood condition of extreme irritability, anger, and frequent, intense temper outbursts. Disruptive mood dysregulation disorder (DMDD) symptoms go beyond being a "moody" child—children with disruptive mood dysregulation disorder (DMDD) experience severe impairment that requires clinical attention. DMDD is a fairly new diagnosis, appearing for the first time in the DSM-5, published in 2013.

Signs and Symptoms



Figure **7.5.1**: Disruptive mood dysregulation disorder is more severe than being "moody."

DMDD symptoms typically begin before the age of 10, but the diagnosis is not given to children under six or adolescents over 18. A child with DMDD experiences

- irritable or angry mood most of the day, nearly every day;
- severe temper outbursts (verbal or behavioral) at an average of three or more times per week that are out of keeping with the situation and the child's developmental level; and
- trouble functioning due to irritability in more than one place (e.g., home, school, or with peers).

To be diagnosed with DMDD, a child must have these symptoms steadily for 12 or more months. The core features of DMDD—temper outbursts and chronic irritability—are sometimes seen in children and adolescents with other psychiatric conditions. Differentiating DMDD from these other conditions can be difficult. Three disorders that most closely resemble DMDD are attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and bipolar disorder in children. Additionally, in both community and clinical samples DMDD is highly comorbid with internalizing and externalizing disorders, particularly with ODD, and long-term functional outcome is likely poor.

There are not good estimates of the prevalence of DMDD, but as of 2015, primary studies have found a rate of 0.8 to 3.3%. Epidemiological studies show that approximately 3.2% of children in the community have chronic problems with irritability and temper, the essential features of DMDD. These problems are probably more common among clinic-referred youths. Parents report that approximately 30% of children hospitalized for psychiatric problems meet diagnostic criteria for DMDD; 15% meet criteria based on the observations of hospital staff.

Watch It

This video explains more about the history of the diagnosis of disruptive mood dysregulation disorder.



You can view the transcript for “Disruptive Mood Dysregulation Disorder” here (opens in new window).

Etiology

The causes of DMDD are unknown, but researchers are exploring risk factors and brain mechanisms of this disorder. Functional MRI studies suggest that under-activity of the amygdala, the brain area that plays a role in the interpretation and expression of emotions and novel stimuli, is associated with these deficits. Deficits in interpreting social cues may predispose children to instances of anger and aggression in social settings with little provocation. For example, youths with DMDD may selectively attend to negative social cues (e.g., others scowling or teasing) and minimize all other information about the events. They may also misinterpret the emotional displays of others, believing others’ benign actions to be hostile or threatening. Consequently, they may be more likely than their peers to act in impulsive and angry ways.

Youth with DMDD have difficulty attending, processing, and responding to negative emotional stimuli and social experiences in their everyday lives. For example, some studies have shown youths with DMDD to have problems interpreting the social cues and emotional expressions of others. These youths may be especially bad at judging others’ negative emotional displays, such as feelings of sadness, fearfulness, and anger.

Treatment and Therapies

DMDD is a new diagnosis. Therefore, treatment is often based on what has been helpful for other disorders that share the symptoms of irritability and temper tantrums. These disorders include attention deficit hyperactivity disorder (ADHD), anxiety disorders, oppositional defiant disorder, and major depressive disorder.

DMDD can impair a child’s quality of life and school performance and disrupt relationships with his or her family and peers. Children with DMDD may find it hard to participate in activities or make friends. Having DMDD also increases the risk of developing depression or anxiety disorders in adulthood.

While researchers are still determining which treatments work best, two major types of treatment are currently used to treat DMDD symptoms: medication and psychological treatments like psychotherapy, parent training, and computer-based training. Psychological treatments should be considered first, with medication added later if necessary, or psychological treatments can be provided along with medication from the beginning. It is important for parents or caregivers to work closely with the doctor to make a treatment decision that is best for their child.

Medication

Many medications used to treat children and adolescents with mental illness are effective in relieving symptoms. However, some of these medications have not been studied in depth and/or do not have U.S. Food and Drug Administration (FDA) approval for use with children or adolescents. All medications have side effects and the need for continuing them should be reviewed frequently with the child’s doctor.

For basic information about these and other mental health medications, you can visit the National Institute of Mental Health (NIMH) Mental Health Medications webpage. For the most up-to-date information on medications, side effects, and warnings, visit the FDA website.

Stimulants

Stimulants are medications that are commonly used to treat ADHD. There is evidence that, in children with irritability and ADHD, stimulant medications also decrease irritability.

Stimulants should not be used in individuals with serious heart problems. According to the FDA, people on stimulant medications should be periodically monitored for changes in heart rate and blood pressure.

Antidepressants

Antidepressant medication is sometimes used to treat the irritability and mood problems associated with DMDD. Ongoing studies are testing whether these medicines are effective for this problem. It is important to note that although antidepressants are safe and effective for many people, they carry a risk of suicidal thoughts and behavior in children and teens. A “black box” warning—the most serious type of warning that a prescription can carry—has been added to the labels of these medications to alert parents and patients to this risk. For this reason, a child taking an antidepressant should be monitored closely, especially when they first start taking the medication.

Atypical Antipsychotic

An atypical antipsychotic medication may be prescribed for children with very severe temper outbursts that involve physical aggression toward people or property. Risperidone and aripiprazole are FDA-approved for the treatment of irritability associated with autism and are sometimes used to treat DMDD. Atypical antipsychotic medications are associated with many significant side effects, including suicidal ideation/behaviors, weight gain, metabolic abnormalities, sedation, movement disorders, hormone changes, and others.

Psychological Treatments

Psychotherapy

CBT, a type of psychotherapy, is commonly used to teach children and teens how to deal with thoughts and feelings that contribute to their feeling depressed or anxious. Clinicians can use similar techniques to teach children to more effectively regulate their mood and to increase their tolerance for frustration. The therapy also teaches coping skills for regulating anger and ways to identify and re-label the distorted perceptions that contribute to outbursts. Other research psychotherapies are being explored at the National Institute of Mental Health (NIMH).

Parent Training



Figure **7.5.2**:Parent-child relationships may be strengthened through parent training.

Parent training aims to help parents interact with a child in a way that will reduce aggression and irritable behavior and improve the parent-child relationship. Multiple studies show that such interventions can be effective. Specifically, parent training teaches parents more effective ways to respond to irritable behavior, such as anticipating events that might lead a child to have a temper outburst and working ahead to avert the outburst. Training also focuses on the importance of predictability, being consistent with children, and rewarding positive behavior.

Computer-based training

Evidence suggests that irritable youth with DMDD may be prone to misperceiving ambiguous facial expressions as angry. There is preliminary evidence that computer-based training designed to correct this problem may help youth with DMDD or severe irritability.

Glossary

disruptive mood dysregulation disorder(DMDD): a childhood condition marked by extreme irritability; anger; and frequent, intense temper outbursts

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7.6: Premenstrual Dsyphoric Disorder

Learning Objectives

- Explain symptoms and treatments for premenstrual dysphoric disorder

Premenstrual dysphoric disorder (PMDD) is a health problem that is similar to premenstrual syndrome (PMS) but is more serious. Premenstrual dysphoric disorder (PMDD) causes severe irritability, depression, or anxiety in the week or two before your period starts. Symptoms usually go away two to three days after your period starts. Premenstrual dysphoric disorder (PMDD) causes a severe and disabling form of premenstrual syndrome affecting 1.8–5.8% of menstruating women. Anyone suffering from PMDD may need medicine or other treatment to help with your symptoms.

Symptoms of PMDD include the following:[1]

- lasting irritability or anger that may affect other people
- feelings of sadness or despair, or even thoughts of suicide
- feelings of tension or anxiety
- panic attacks
- mood swings or crying often
- lack of interest in daily activities and relationships
- trouble thinking or focusing
- tiredness or low energy
- food cravings or binge eating
- trouble sleeping
- feeling out of control
- physical symptoms, such as cramps, bloating, breast tenderness, headaches, and joint or muscle pain

Researchers do not know for sure what causes PMDD or premenstrual syndrome (PMS). Hormonal changes throughout the menstrual cycle may play a role. A brain chemical called serotonin may also play a role in PMDD since serotonin levels change throughout the menstrual cycle. Some women may be more sensitive to these changes.

Treatment

Treatments for PMDD include the following:

- Antidepressants called selective serotonin reuptake inhibitors (SSRIs) change serotonin levels in the brain. The FDA approved three SSRIs to treat PMDD:
  - Sertraline
  - Fluoxetine
  - Paroxetine HCI
- The FDA has approved a birth control pill containing drospirenone (droh-SPIR-uh-nohn) and ethinyl estradiol (ETH-uh-nil es-truh-DEYE-ohl), to treat PMDD.
- Over-the-counter pain relievers may help relieve physical symptoms, such as cramps, joint pain, headaches, backaches, and breast tenderness and include ibuprofen, naproxen, and aspirin.
- Stress management, such as relaxation techniques and spending time on enjoyable activities, may help.
- Making healthy changes, such as eating a healthy combination of foods across the food groups, cutting back on salty and sugary foods, and getting more physical activity, may also help relieve some PMDD symptoms.

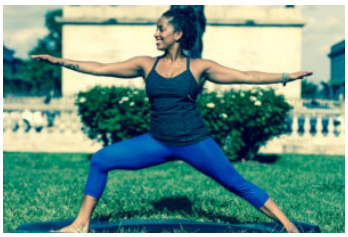


Figure 7.6.2: Yoga is a low impact, physical activity that can help you relax and release tension.

When drug-based treatments are ineffective or produce significant side effects, then removing the ovaries through oophorectomy can produce an immediate and permanent cure. Typically, the uterus is removed during the same surgery, and the woman is prescribed a low-dose estrogen patch to reduce the symptoms produced by surgically induced menopause.

Watch It

What's the difference between premenstrual syndrome (PMS) and PMDD? Dr. Lita Landry explains the differences in this clip from *The Doctors*.



You can view the transcript for "Could Your PMS Actually Be PPMD?" here (opens in new window).

Glossary

premenstrual dysphoric disorder (PMDD): a more serious form of PMS marked by severe irritability, depression, or anxiety in the week or two before your period starts

1. American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Washington, DC: The American Psychiatric Association. ↵

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7.7: Introduction to Bipolar and Related Disorders

What you'll learn to do: examine bipolar and related disorders



Bipolar disorder is characterized by mood states that vacillate between sadness and euphoria; a diagnosis of bipolar disorder requires experiencing at least one manic episode, which is defined as a period of extreme euphoria, irritability, and increased activity. Mood disorders appear to have a genetic component, with genetic factors playing a more prominent role in bipolar disorder than in depression.

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7.8: Bipolar Disorder

Learning Objectives

- Describe the symptoms and risk factors of bipolar disorder
- Examine the epidemiology and etiology of bipolar disorder

Bipolar Mood Disorders

Three major types of bipolar disorder are described by the DSM-5 (APA, 2013): Bipolar I, Bipolar II, and cyclothymia. **Bipolar I Disorder** (BD I), which was previously known as manic depression, is characterized by a single or recurrent manic episode. A depressive episode is not necessary but commonly present for the diagnosis of Bipolar I disorder (BD I).

Manic or Hypomanic Episode

The core criterion for a manic or hypomanic episode is a distinct period of abnormally and persistently euphoric, expansive, or irritable mood and persistently increased goal-directed activity or energy. The mood disturbance must be present for one week or longer in mania (unless hospitalization is required) or four days or longer in hypomania. Concurrently, at least three of the following symptoms must be present in the context of euphoric mood (or at least four in the context of irritable mood):

1. inflated self-esteem or grandiosity
2. increased goal-directed activity or psychomotor agitation
3. reduced need for sleep
4. racing thoughts or flight of ideas
5. distractibility
6. increased talkativeness
7. excessive involvement in risky behaviors

Manic episodes are distinguished from hypomanic episodes by their duration and associated impairment. Whereas manic episodes must last one week and are defined by a significant impairment in functioning, hypomanic episodes are shorter and not necessarily accompanied by impairment in functioning.

Bipolar II disorder is characterized by single (or recurrent) *hypomanic* episodes and depressive episodes. Another type of bipolar disorder is **cyclothymic disorder**, characterized by numerous and alternating periods of hypomania and depression, lasting at least two years. To qualify for cyclothymic disorder, the periods of depression cannot meet full diagnostic criteria for a diagnosis of major depressive disorder; the person must experience symptoms at least half the time with no more than two consecutive, symptom-free months, and the symptoms must cause significant distress or impairment.

A person with bipolar disorder often experiences mood states that vacillate between depression and mania; that is, the person's mood is said to alternate from one emotional extreme to the other (in contrast to unipolar, which indicates a persistently sad mood).



Figure **7.8.1**: Being abnormally talkative is different from just having an outgoing personality.

To be diagnosed with bipolar disorder, a person must have experienced a manic episode at least once in his life; although major depressive episodes are common in bipolar disorder, they are not required for a diagnosis (APA, 2013). According to the DSM-5, a **manic episode** is characterized as a "distinct period of abnormally and persistently elevated, expansive, or irritable mood and

abnormally and persistently increased activity or energy lasting at least one week" that lasts most of the time each day (APA, 2013, p. 124).

During a manic episode, some experience a mood that is almost euphoric and become excessively talkative, sometimes spontaneously starting conversations with strangers; others become excessively irritable and complain or make hostile comments. An individual may talk loudly and rapidly, exhibiting **flight of ideas**: abruptly switching from one topic to another. Manic episodes make individuals very distracted, which can make a conversation very difficult. Individuals with bipolar disorder (BD) may exhibit grandiosity, in which they experience inflated but unjustified self-esteem and self-confidence. For example, they might quit a job in order to "strike it rich" in the stock market, despite lacking the knowledge, experience, and capital for such an endeavor. One with bipolar disorder (BD) may take on several tasks at the same time (e.g., several time-consuming projects at work) and yet show little, if any, need for sleep; some may go for days without sleep. Patients may also recklessly engage in pleasurable activities that could have harmful consequences, including spending sprees, reckless driving, making foolish investments, excessive gambling, or engaging in sexual encounters with strangers (APA, 2013).

During a manic episode, individuals usually feel as though they are not ill and do not need treatment. However, the reckless behaviors that often accompany these episodes—which can be antisocial, illegal, or physically threatening to others—may require involuntary hospitalization (APA, 2013). Some patients with bipolar disorder will experience a rapid-cycling subtype, which is characterized by at least four manic episodes (or some combination of at least four manic and major depressive episodes) within one year.

Risk Factors for Bipolar Disorder

Bipolar disorder is considerably less frequent than major depressive disorder. In the United States, one out of every 167 people meets the criteria for bipolar disorder each year, and one out of 100 meet the criteria within their lifetime (Merikangas et al., 2011). The rates of BD are higher in men than in women, and about half of those with this disorder report onset before the age of 25 (Merikangas et al., 2011). Around 90% of those with bipolar disorder have a comorbid disorder, most often an anxiety disorder or a substance abuse problem. Unfortunately, close to half of the people suffering from bipolar disorder do not receive treatment (Merikangas & Tohen, 2011). Suicide rates are extremely high among those with bipolar disorder: around 36% of individuals with this disorder attempt suicide at least once in their lifetime (Novick, Swartz, & Frank, 2010), and between 15–19% complete suicide (Newman, 2004).

Epidemiology of Bipolar Disorders

Bipolar disorder is the sixth leading cause of disability worldwide and has a lifetime prevalence of about 1%-3% in the general population. However, a reanalysis of data from the National Epidemiological Catchment Area survey in the United States suggested that 0.8% of the population experience a manic episode at least once (the diagnostic threshold for Bipolar I) and a further 0.5% have a hypomanic episode (the diagnostic threshold for bipolar II or cyclothymia). Including sub-threshold diagnostic criteria, such as one or two symptoms over a short time-period, an additional 5.1% of the population, adding up to a total of 6.4%, were classified as having a bipolar spectrum disorder. A more recent analysis of data from a second U.S. National Comorbitity Survey found that 1% met lifetime prevalence criteria for Bipolar I, 1.1% for Bipolar II, and 2.4% for subthreshold symptoms.

The lifetime prevalence rate of bipolar spectrum disorders in the general U.S. population is estimated at approximately 4.4%, with BD I constituting about 1% of this rate (Merikangas et al., 2007). Prevalence estimates, however, are highly dependent on the diagnostic procedures used (e.g., interviews versus self-report) and whether or not sub-threshold forms of the disorder are included in the estimate. BD often co-occurs with other psychiatric disorders. Approximately 65% of people with BD meet diagnostic criteria for at least one additional psychiatric disorder, most commonly anxiety disorders and substance use disorders (McElroy et al., 2001). The co-occurrence of BD with other psychiatric disorders is associated with poorer illness course, including higher rates of suicidality (Leverich et al., 2003).

A recent cross-national study sample of more than 60,000 adults from 11 countries, estimated the worldwide prevalence of BD at 2.4%, with BD I constituting 0.6% of this rate (Merikangas et al., 2011). In this study, the prevalence of BD varied somewhat by country. Whereas the United States had the highest lifetime prevalence (4.4%), India had the lowest (0.1%). Variation in prevalence rates was not necessarily related to socioeconomic status (SES), as in the case of Japan, a high-income country with a very low prevalence rate of BD (0.7%).

With regard to ethnicity, data from studies not confounded by SES or inaccuracies in diagnosis are limited, but available reports suggest rates of BD among European Americans are similar to those found among African Americans (Blazer et al., 1985) and Hispanic Americans (Breslau, Kendler, Su, Gaxiola-Aguilar, & Kessler, 2005). Another large community-based study found that although prevalence rates of mood disorders were similar across ethnic groups, Hispanic Americans and African Americans with a mood disorder were more likely to remain persistently ill than European Americans (Breslau et al., 2005). Compared with European Americans with BD, African Americans tend to be underdiagnosed for BD (and over-diagnosed for schizophrenia) (Kilbourne, Haas, Mulsant, Bauer, & Pincus, 2004; Minsky, Vega, Miskimen, Gara, & Escobar, 2003), and Hispanic Americans with BD have been shown to receive fewer psychiatric medication prescriptions and specialty treatment visits (Gonzalez et al., 2007). Misdiagnosis of BD can result in the underutilization of treatment or the utilization of inappropriate treatment, and thus profoundly impact the course of illness.

As with major depressive disorder, adolescence is known to be a significant risk period for BD; mood symptoms start by adolescence in roughly half of BD cases (Leverich et al., 2007; Perlis et al., 2004). Longitudinal studies show that those diagnosed with BD prior to adulthood experience a more pernicious course of illness relative to those with adult-onset, including more episode recurrence; higher rates of suicidality; and profound social, occupational, and economic repercussions (e.g., Lewinsohn, Seeley, Buckley, & Klein, 2002). The prevalence of BD is substantially lower in older adults compared with younger adults (1% vs. 4%) (Merikangas et al., 2007).

Etiology of Bipolar Disorders

Although there have been important advances in research on the etiology, course, and treatment of BD, there remains a need to understand the mechanisms that contribute to episode onset and relapse. There is compelling evidence for biological causes of BD, which is known to be highly heritable (McGuffin, Rijsdijk, Andrew, Sham, Katz, & Cardno, 2003). Genetic influences are believed to account for 73–93% of the risk of developing the disorder. For Bipolar I, the rate at which identical twins (same genes) will both have Bipolar I (concordance) is around 40%, compared to about 5% in fraternal twins. The high rate of heritability demonstrates that BD could fundamentally be a biological phenomenon. However, variability is high in the course of BD, both within a person across time and across people (Johnson, 2005). The triggers that determine how and when this genetic vulnerability is expressed are not yet understood; however, there is evidence to suggest that psychosocial triggers may play an important role in BD risk (e.g., Johnson et al., 2008; Malkoff-Schwartz et al., 1998).



Figure **7.8.2**: While there is high hereditability in BD, psychosocial triggers are thought to play an important role in developing the disorder.

In addition to the genetic contribution, biological explanations of BD have also focused on brain function. Many of the studies using fMRI techniques to characterize BD have focused on the processing of emotional stimuli based on the idea that BD is fundamentally a disorder of emotion (APA, 2000). Findings have shown that regions of the brain thought to be involved in emotional processing and regulation are activated differently in people with BD relative to healthy controls (e.g., Altshuler et al., 2008; Hassel et al., 2008; Lennox, Jacob, Calder, Lupson, & Bullmore, 2004).

However, there is little consensus as to whether a particular brain region becomes more or less active in response to an emotional stimulus among people with BD compared with healthy controls. Mixed findings are in part due to samples consisting of participants who are at various phases of illness at the time of testing (manic, depressed, or inter-episode). Sample sizes tend to be relatively small, making comparisons between subgroups difficult. Additionally, the use of a standardized stimulus (e.g., facial expression of anger) may not elicit a sufficiently strong response. Personally engaging stimuli, such as recalling a memory, may be more effective in inducing strong emotions (Isacowitz, Gershon, Allard, & Johnson, 2013).

Within the psychosocial level, research has focused on the environmental contributors to BD. A series of studies show that environmental stressors, particularly severe stressors (e.g., loss of a significant relationship), can adversely impact the course of BD. People with BD have a substantially increased risk of relapse (Ellicott, Hammen, Gitlin, Brown, & Jamison, 1990) and suffer more depressive symptoms (Johnson, Winett, Meyer, Greenhouse, & Miller, 1999) following a severe life stressor. In surveys, 30–50% of adults diagnosed with bipolar disorder report traumatic/abusive experiences in childhood, which is associated with earlier onset, a higher rate of suicide attempts, and more co-occurring disorders such as post-traumatic stress disorder. Interestingly, positive life events can also adversely impact the course of BD. People with BD suffer more manic symptoms after life events involving the attainment of a desired goal (Johnson et al., 2008). Such findings suggest that people with BD may have a hypersensitivity to rewards.

Evidence from the life stress literature has also suggested that people with mood disorders may have a circadian vulnerability that renders them sensitive to stressors that disrupt their sleep or rhythms. According to social zeitgeber theory (Ehlers, Frank, & Kupfer, 1988; Frank et al., 1994), stressors that disrupt sleep or that disrupt the daily routines that entrain the biological clock (e.g., meal times) can trigger episode relapse. Consistent with this theory, studies have shown that life events that involve a disruption in sleep and daily routines, such as overnight travel, can increase bipolar symptoms in people with BD (Malkoff-Schwartz et al., 1998).

Watch It

This video addresses common myths about bipolar disorder:

1. There are only manic or depressive episodes in bipolar disorder.
2. Bipolar II is a “milder” form of bipolar disorder.
3. Mood swings during the day mean you have bipolar disorder.
4. All bipolar patients have mania.
5. People with bipolar disorder are unreliable



You can view the transcript for “5 misunderstandings about Bipolar Disorder” here (opens in new window).

Treatment for Bipolar Disorder

Psychotherapy aims to assist a person with bipolar disorder in accepting and understanding their diagnosis, coping with various types of stress, improving their interpersonal relationships, and recognizing prodomal symptoms before full-blown recurrence. Cognitive behavioral therapy, family-focused therapy, and psychoeducation have the most evidence for efficacy in regard to relapse prevention, while interpersonal and social rhythm therapy and CBT appear the most effective in regard to residual depressive symptoms. Most studies have been based only on Bipolar I, however, and treatment during the acute phase can be a particular challenge. Some clinicians emphasize the need to talk with individuals experiencing mania to develop a therapeutic alliance in support of recovery.

Medication

Lithium (Lithobid, Eskalith) is effective at stabilizing mood and preventing the extreme highs and lows of bipolar disorder. Periodic blood tests are required because lithium can cause thyroid and kidney problems. Common side effects include restlessness, dry mouth, and digestive issues. Lithium levels should be monitored carefully to ensure the best dosage and watch for toxicity. Lithium is used for continued treatment of bipolar depression and for preventing relapse. There is evidence that lithium can lower the risk of suicide but the FDA has not granted approval specifically for this purpose.

Other mood-stabilizing medications and anticonvulsants (generally used to treat seizures) are recommended for treating bipolar disorder. Common side effects include weight gain, dizziness, and drowsiness. But sometimes, certain anticonvulsants cause more serious problems, such as skin rashes, blood disorders or liver problems.

Valproic acid and carbamazepine are used to treat mania. These drugs, also used to treat epilepsy, were found to be as effective as lithium for treating acute mania. They may be better than lithium in treating the more complex bipolar subtypes of rapid cycling and dysphoric mania.

Lamotrigine is used to delay occurrences of Bipolar I. Lamotrigine does not have FDA approval for treatment of the acute episodes of depression or mania. Studies of lamotrigine for treatment of acute bipolar depression have produced inconsistent results.

Second-Generation Antipsychotics (SGAs)

Second-generation antipsychotics (SGAs) are commonly used to treat the symptoms of bipolar disorder and are often paired with other medications, including mood stabilizers. They are generally used for treating manic or mixed episodes. Second-generation antipsychotics (SGAs) are often prescribed to help control acute episodes of mania or depression. Finding the right medication is not an exact science; it is specific to each person. Currently, only quetiapine and the combination of olanzepine and fluoxetine (Symbax) are approved for treating bipolar depression. Regularly check with the FDA website, as side effects can change over time.

Standard Antidepressants

Antidepressants present special concerns when used in treating bipolar disorder, as they can trigger mania in some people. A National Institute of Mental Health (NIMH) study showed that taking an antidepressant in addition to a mood stabilizer is no more effective that using a mood stabilizer alone for Bipolar I. This is an essential area to review treatment risks and benefits.

The aim of management is to treat acute episodes safely with medication and work with the patient in long-term maintenance to prevent further episodes and optimize functioning using a combination of pharmological and psychotherapeutic techniques. Hospitalization may be required especially with the manic episodes present in Bipolar I. This can be voluntary or (local legislation permitting) involuntary. Long-term inpatient stays are now less common due to deinstitutionalism, although these stays can still occur. Following (or in lieu of) a hospital admission, support services available can include drop-in centers, visits from members of a community mental health team or an Assertive Community Treatment team, supported employment, patient-led support groups, and intensive outpatient programs. These are sometimes referred to as partial-inpatient programs.

Glossary

bipolar and related disorders: group of mood disorders in which mania is the defining feature

bipolar disorder: mood disorder characterized by mood states that vacillate between depression and mania

Bipolar I: previously known as manic-depression, characterized by a single or recurrent manic episode

Bipolar II: single (or recurrent) *hypomanic* episodes and depressive episodes; manic episodes are less severe than in Bipolar I

flight of ideas: symptom of mania that involves an abruptly switching in conversation from one topic to another

mania: state of extreme elation and agitation

manic episode: period in which an individual experiences mania, characterized by extremely cheerful and euphoric mood, excessive talkativeness, irritability, increased activity levels, and other symptoms

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7.9: Cyclothymic Disorder

Learning Objectives

- Describe cyclothymic disorder

Cyclothymia



Figure **7.9.1**: Cyclothymia is characterized by fluctuations between depressive symptoms and symptoms of hypomania, but it is less severe than bipolar disorder.

Cyclothymic disorder is a mental disorder that involves numerous periods of symptoms of depression and periods of symptoms of hypomania. Symptoms, however, are not sufficient to be a major depressive episode or a hypomanic episode. Symptoms must last for more than one year in children and two years in adults.

Cyclothymia is classified in DSM-5 as a subtype of bipolar disorder. The criteria are listed below:[1]

1. periods of elevated mood and depressive symptoms for at least half the time during the last two years for adults and one year for children and teenagers
2. periods of stable moods last only two months at most
3. symptoms create significant problems in one or more areas of life
4. symptoms do not meet the criteria for bipolar disorder, major depression, or another mental disorder
5. symptoms are not caused by substance use or a medical condition

People with cyclothymia experience both depressive phases and hypomanic phases (which are less severe than a full hypomanic episode). The depressive and manic symptoms in cyclothymia last for variable amounts of time due to the unstable and reactive nature of the disorder. The depressive phases are similar to major depressive disorder and are characterized by dulled thoughts and sensations and the lack of motivation for intellectual or social activities. Most people with cyclothymia are generally fatigued and tend to sleep frequently and for long periods of time. However, other people experience insomnia. Cyclothymia differs from bipolar in that major depression, mania, or hypomania have never occurred.

The cause of cyclothymia is unknown. Risk factors include a family history of bipolar disorder. First-degree relatives of people with cyclothymia have major depression disorder (MDD), Bipolar I disorder (BD I), and Bipolar II disorder (BD II) more often than the general population. Individuals may also have a history of higher risks for substance-related disorders within the family. First-degree relatives of a BD I individuals may have a higher risk of cyclothymic disorder than the general population.

Treatment is generally with counseling and mood stabilizers such as lithium. It is estimated that 0.4%-1% of people have cyclothymia at some point in their life. Onset is typically in late childhood to early adulthood and males and females are affected equally often.

Glossary

cyclothymic disorder: a mental disorder considered to be a less extreme form of bipolar disorder characterized by numerous periods of symptoms of depression and periods of symptoms of hypomania

1. *Diagnostic and statistical manual of mental disorders : DSM-5* (5th ed.). Washington, D.C.: American Psychiatric Association. 2013. ISBN 978-0-89042-554-1. ↵

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7.10: Introduction to Perspectives and Treatments Related to Mood Disorders

What you'll learn to do: examine various perspectives and treatment methods for mood disorders



Meta-analyses show that high scores on the personality domain neuroticism are a strong predictor for the development of mood disorders. A number of authors have also suggested that mood disorders are an evolutionary adaptation. A low or depressed mood can increase an individual's ability to cope with situations in which the effort to pursue a major goal could result in danger, loss, or wasted effort. In such situations, low motivation may give an advantage by inhibiting certain actions. This theory helps to explain why negative life incidents precede depression in around 80% of cases, and why they so often strike people during their peak reproductive years. These characteristics would be difficult to understand if depression were a dysfunction.

A depressed mood is a predictable response to certain types of life occurrences, such as loss of status, divorce, or death of a child or spouse. These are events that signal a loss of reproductive ability or potential, or that did so in humans' ancestral environment. A depressed mood can be seen as an adaptive response in the sense that it causes an individual to turn away from the earlier (and reproductively unsuccessful) modes of behavior.

A depressed mood is common during illnesses, such as influenza. It has been argued that this is an evolved mechanism that assists the individual in recovering by limiting his or her physical activity. The occurrence of low-level depression during the winter months, or seasonal affective disorder, may have been adaptive in the past, by limiting physical activity at times when food was scarce. It is argued that humans have retained the instinct to experience low mood during the winter months, even if the availability of food is no longer determined by the weather.

There are different types of treatments available for mood disorders, such as therapy and medications. Behavior therapy, CBT, and interpersonal therapy have all shown to be potentially beneficial in depression. Major depressive disorder medications usually include antidepressants, while bipolar disorder medications can consist of antipsychotics, mood stabilizers, anticonvulsants, and/or lithium. Lithium specifically has been proven to reduce suicide and all causes of mortality in people with mood disorders. If mitochondrial dysfunction or mitochondrial diseases are the cause of mood disorders like bipolar disorder, then it has been hypothesized that N-acetyl-cysteine (NAC), acetyl-L-carnitine (ALCAR), S-adenosylmethionine (SAMe), coenzyme Q10 (CoQ10), alpha-lipoic acid (ALA), creatine monohydrate (CM), and melatonin could be potential treatment options. In determining treatment, there are many types of depression scales that are used. One of the depression scales is a self-report scale called Beck Depression Inventory (BDI). Another scale is the Hamilton Depression Rating Scale (HAMD). HAMD is a clinical rating scale in which the patient is rated based on clinician observation. The Center for Epidemiologic Studies Depression Scale (CES-D) is a scale for depression symptoms that applies to the general population. This scale is typically used in research and not for self-reports. The PHQ-9 which stands for Patient-Health Questionnaire-9 questions, is a self-report as well. Finally, the Mood Disorder Questionnaire (MDQ) evaluates bipolar disorder.

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7.11: Psychological Perspectives on Mood Disorders

Learning Objectives

- Describe and compare viewpoints from the major psychological perspectives related to mood disorders
- Describe biological and genetic explanations for mood disorders

Biological Theories on Mood Disorders

Mood disorders have been shown to have a strong genetic and biological basis. Relatives of those with major depressive disorder have double the risk of developing major depressive disorder, whereas relatives of patients with bipolar disorder have over nine times the risk (Merikangas et al., 2011). The rate of concordance for major depressive disorder is higher among identical twins than fraternal twins (50% vs. 38%, respectively), as is that of bipolar disorder (67% versus 16%, respectively), suggesting that genetic factors play a stronger role in bipolar disorder than in major depressive disorder (Merikangas et al. 2011). People with mood disorders often have imbalances in certain neurotransmitters, particularly norepinephrine and serotonin (Thase, 2009). Specific neurotransmitters are important regulators of the bodily functions that are disrupted in mood disorders, including appetite, sex drive, sleep, arousal, and mood. Medications that are used to treat major depressive disorder typically boost serotonin and norepinephrine activity, whereas lithium—used in the treatment of bipolar disorder—blocks norepinephrine activity at the synapses (Figure 1).

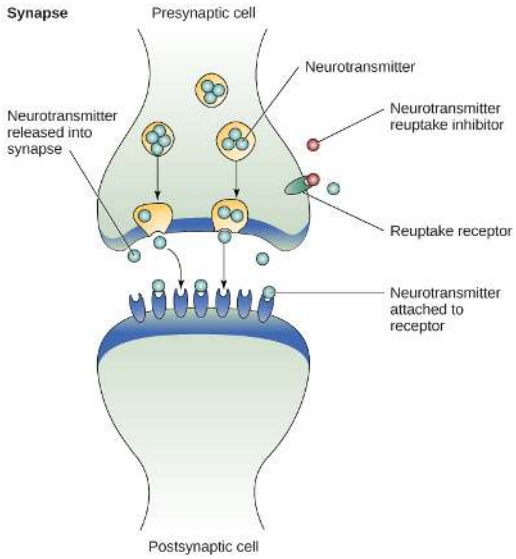


Figure **7.11.1**: Many medications designed to treat mood disorders work by altering neurotransmitter activity in the neural synapse.

Depression is linked to abnormal activity in several regions of the brain (Fitzgerald, Laird, Maller, & Daskalakis, 2008), including those important in assessing the emotional significance of stimuli and experiencing emotions (amygdala) and in regulating and controlling emotions (like the prefrontal cortex, or PFC) (LeMoult, Castonguay, Joormann, & McAleavey, 2013). Depressed individuals show elevated amygdala activity (Drevets, Bogers, & Raichle, 2002), especially when presented with negative emotional stimuli, such as photos of sad faces (Surguladze et al., 2005). Interestingly, heightened amygdala activation to negative emotional stimuli among depressed persons occurs even when stimuli are presented outside of conscious awareness (Victor, Furey, Fromm, Öhman, & Drevets, 2010), and it persists even after the negative emotional stimuli are no longer present (Siegle, Thompson, Carter, Steinhauer, & Thase, 2007). Additionally, depressed individuals exhibit less activation in the prefrontal, particularly on the left side (Davidson, Pizzagalli, & Nitschke, 2009). Because the prefrontal cortex (PFC) can dampen amygdala activation, thereby enabling one to suppress negative emotions (Phan et al., 2005), decreased activation in certain regions of the prefrontal cortex (PFC) may inhibit its ability to override negative emotions that might then lead to more negative mood states (Davidson et al., 2009). These findings suggest that depressed persons are more prone to react to emotionally negative stimuli, yet have greater difficulty controlling these reactions.



Figure **7.11.2**: Depressed individuals react to negative emotional stimuli, such as sad faces, with greater amygdala activation than do non-depressed individuals. (credit: Ian Munroe)

Since the 1950s, researchers have noted that depressed individuals have abnormal levels of cortisol, a stress hormone released into the blood by the neuroendocrine system during times of stress (Mackin & Young, 2004). When cortisol is released, the body initiates a fight-or-flight response in reaction to a threat or danger. Many people with depression show elevated cortisol levels (Holsboer & Ising, 2010), especially those reporting a history of early life trauma such as the loss of a parent or abuse during childhood (Baes, Tofoli, Martins, & Juruena, 2012). Such findings raise the question of whether high cortisol levels are a cause or a consequence of depression. High levels of cortisol are a risk factor for future depression (Halligan, Herbert, Goodyer, & Murray, 2007), and cortisol activates activity in the amygdala while deactivating activity in the PFC (McEwen, 2005)—both brain disturbances are connected to depression. Thus, high cortisol levels may have a causal effect on depression, as well as on its brain function abnormalities (van Praag, 2005). Also, because stress results in increased cortisol release (Michaud, Matheson, Kelly, Anisman, 2008), it is equally reasonable to assume that stress may precipitate depression.

Diathesis-Stress Model and Major Depressive Disorders

Indeed, it has long been believed that stressful life events can trigger depression, and research has consistently supported this conclusion (Mazure, 1998). Stressful life events include significant losses, such as death of a loved one; divorce or separation; and serious health and money problems; life events such as these often precede the onset of depressive episodes (Brown & Harris, 1989). In particular, exit events—instances in which an important person departs (e.g., a death, divorce or separation, or a family member leaving home)—often occur prior to an episode (Paykel, 2003). Exit events are especially likely to trigger depression if these happenings occur in a way that humiliates or devalues the individual. For example, people who experience the breakup of a relationship initiated by the other person develop major depressive disorder at a rate more than two times that of people who experience the death of a loved one (Kendler, Hettema, Butera, Gardner, & Prescott, 2003).

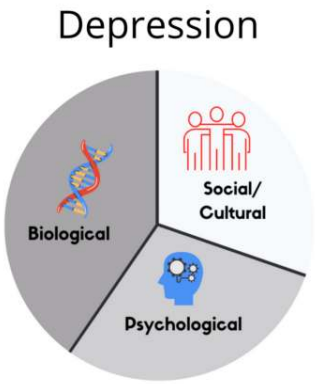


Figure **7.11.3**: Depression is influenced by biological, psychological, and social/cultural factors, though research indicates that there are strong genetic and biological ties to depression.

Likewise, individuals who are exposed to traumatic stress during childhood—such as separation from a parent, family turmoil, and maltreatment (physical or sexual abuse)—are at a heightened risk of developing depression at any point in their lives (Kessler, 1997). A recent review of 16 studies involving over 23,000 subjects concluded that those who experience childhood maltreatment are more than two times as likely to develop recurring and persistent depression (Nanni, Uher, & Danese, 2012).

Of course, not everyone who experiences stressful life events or childhood adversities succumbs to depression—indeed, most do not. Clearly, a **diathesis-stress** interpretation of major depressive disorder, in which certain predispositions or vulnerability factors

influence one's reaction to stress, would seem logical. If so, what might such predispositions be? Caspi et al. (2003) suggested that an alteration in a specific gene that regulates serotonin (the 5-HTTLPR gene) might be one culprit. Caspi et al. found that people who experienced several stressful life events were significantly more likely to experience episodes of major depression if they carried one or two short versions of this gene than if they carried two long versions. Individuals who carried one or two short versions of the 5-HTTLPR gene were unlikely to experience an episode, however, if they had experienced few or no stressful life events. Numerous studies have replicated these findings, including studies of people who experienced maltreatment during childhood (Goodman & Brand, 2009). In a recent investigation conducted in the United Kingdom (Brown & Harris, 2013), researchers found that childhood maltreatment before age nine elevated the risk of chronic adult depression (a depression episode lasting for at least 12 months) among those individuals having one (LS) or two (SS) short versions of the 5-HTTLPR gene. Childhood maltreatment did not increase the risk for chronic depression for those have two long (LL) versions of this gene. Thus, genetic vulnerability may be one mechanism through which stress potentially leads to depression.

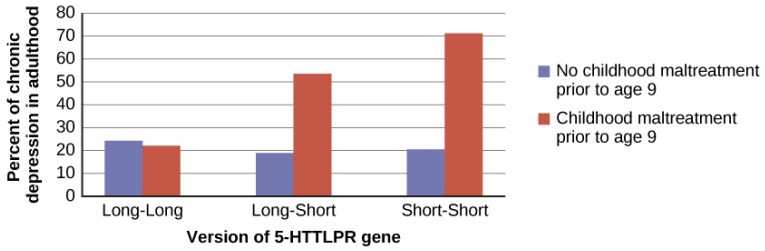


Figure **7.11.4**: A study on gene-environment interaction in people experiencing chronic depression in adulthood suggests a much higher incidence in individuals with a short version of the gene in combination with childhood maltreatment (Brown & Harris, 2013).

Cognitive Theories of Depression

Cognitive theories of depression take the view that depression is triggered by negative thoughts, interpretations, self-evaluations, and expectations (Joormann, 2009). These **diathesis-stress models** propose that depression is triggered by a "cognitive vulnerability" (negative and maladaptive thinking) and by precipitating stressful life events (Gotlib & Joormann, 2010).

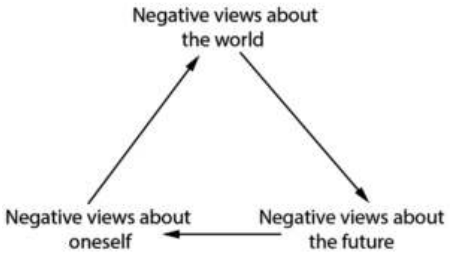


Figure **7.11.5**: Beck's cognitive triad assumes a pattern of negative thinking. For example, the self may think, "I'm worthless and ugly" or "I wish I was different," leading to beliefs from the world that "no one values me" or "people ignore me all the time," which may lead to thinking about the future as "I'm hopeless because things will never change" or "things can only get worse!"

Perhaps the most well-known cognitive theory of depression was developed in the 1960s by psychiatrist Aaron Beck, based on clinical observations and supported by research (Beck, 2008). Beck theorized that depression-prone people possess depressive schemas, or mental predispositions to think about most things in a negative way (Beck, 1976). Depressive schemas contain themes of loss, failure, rejection, worthlessness, and inadequacy, and may develop early in childhood in response to adverse experiences, then remain dormant until they are activated by stressful or negative life events. Depressive schemas prompt dysfunctional and pessimistic thoughts about the self, the world, and the future.

Beck believed that this dysfunctional style of thinking is maintained by cognitive biases, or errors in how we process information about ourselves, which lead us to focus on negative aspects of experiences, interpret things negatively, and block positive memories (Beck, 2008). A person whose depressive schema consists of a theme of rejection might be overly attentive to social cues of rejection (more likely to notice another's frown), and he might interpret this cue as a sign of rejection and automatically remember past incidents of rejection.

Longitudinal studies have supported Beck's theory, in showing that a pre-existing tendency to engage in this negative, self-defeating style of thinking—when combined with life stress—over time predicts the onset of depression (Dozois & Beck, 2008). Cognitive therapies for depression, aimed at changing a depressed person's negative thinking, were developed as an expansion of this theory (Beck, 1976).

? Cognitive Therapy in Action

Having made a mistake at work, a man may believe, "I'm useless and can't do anything right at work." He may then focus on the mistake (which he takes as evidence that his belief is true), and his thoughts about being "useless" are likely to lead to negative emotion (frustration, sadness, and hopelessness). Given these thoughts and feelings, he may then begin to avoid challenges at work, which is behavior that could provide even more evidence for him that his belief is true. As a result, any adaptive response and further constructive consequences become unlikely, and he may focus even more on any mistakes he may make, which serve to reinforce the original belief of being "useless." In therapy, this example could be identified as a self-fulfilling prophecy or "problem cycle," and the efforts of the therapist and patient would be directed at working together to explore and shift this cycle.

People who are working with a cognitive therapist often practice the use of more flexible ways to think and respond, learning to ask themselves whether their thoughts are completely true, and whether those thoughts are helping them to meet their goals. Thoughts that do not meet this description may then be shifted to something more accurate or helpful, leading to more positive emotion, more desirable behavior, and movement toward the person's goals. Cognitive therapy takes a skill-building approach, where the therapist helps the person to learn and practice these skills independently, eventually "becoming his or her own therapist."

Another cognitive theory of depression, **hopelessness theory**, postulates that a particular style of negative thinking leads to a sense of hopelessness, which then leads to depression (Abramson, Metalsky, & Alloy, 1989). According to this theory, hopelessness is an expectation that unpleasant outcomes will occur or that desired outcomes will not occur, and there is nothing one can do to prevent such outcomes. A key assumption of this theory is that hopelessness stems from a tendency to perceive negative life events as having stable ("It's never going to change"), global ("It's going to affect my whole life") causes, in contrast to unstable ("It's fixable"), and specific ("It applies only to this particular situation") causes, especially if these negative life events occur in important life realms, such as relationships, academic achievement, and the like.

Suppose a student who wishes to go to law school does poorly on an admissions test. If the student infers negative life events as having stable and global causes, she may believe that her poor performance has a stable and global cause ("I lack intelligence, and it's going to prevent me from ever finding a meaningful career"), as opposed to an unstable and specific cause ("I was sick the day of the exam, so my low score was a fluke"). Hopelessness theory predicts that people who exhibit this cognitive style in response to undesirable life events will view such events as having negative implications for their future and self-worth, thereby increasing the likelihood of hopelessness—the primary cause of depression (Abramson et al., 1989). One study testing hopelessness theory measured the tendency to make negative inferences for bad life effects in participants who were experiencing uncontrollable stressors. Over the ensuing six months, those individuals with scores reflecting high cognitive vulnerability were seven times more likely to develop depression compared to those with lower scores (Kleim, Gonzalo, & Ehlers, 2011).

A third cognitive theory of depression focuses on how people's thoughts about their distressed moods—depressed symptoms in particular—can increase the risk and duration of depression. This theory, which focuses on rumination in the development of depression, was first described in the late 1980s to explain the higher rates of depression in women than in men (Nolen-Hoeksema, 1987). Rumination is the repetitive and passive focus on the fact that one is depressed and dwelling on depressed symptoms, rather than distracting oneself from the symptoms or attempting to address them in an active, problem-solving manner (Nolen-Hoeksema, 1991). When people ruminate, they have thoughts such as "Why am I so unmotivated? I just can't get going. I'm never going to get my work done feeling this way" (Nolen-Hoeksema & Hilt, 2009, p. 393). Women are more likely than men to ruminate when they are sad or depressed (Butler & Nolen-Hoeksema, 1994), and the tendency to ruminate is associated with increases in depression symptoms (Nolen-Hoeksema, Larson, & Grayson, 1999), heightened risk of major depressive episodes (Abela & Hankin, 2011), and chronicity of such episodes (Robinson & Alloy, 2003).

The Sociocultural Perspective

Depression is more prevalent in women than in men in individualist and collective cultures. Some have hypothesized that this is due to their inferior positions in the culture, in which they may experience domestic violence, poverty, and inequality that can greatly contribute to depression. Moreover, research conducted in the United States, Chile, and Spain found that cultural differences on the expectations of individuals based on gender varied across cultures and that resulted in different levels of depression across individuals. Changes in gender roles across countries and cultures in this study served to explain varying levels of stress and responsibility that changed on the basis of gender. These ever-changing expectations and these additional responsibilities on the basis of gender increased levels of depression.

The differences in gender roles within a community also impact one's level of depression. According to Wichstrøm (1999), women tend to ruminate more and therefore symptoms of depression can be accentuated. Since women are encouraged to talk about and share their feelings, they can get caught talking about and staying in a negative state· On the other hand, boys are taught not to discuss their feelings with others, which then diminishes or even gets rid of rumination entirely. The way one is socialized to deal with their negative emotions will vary across cultures and may lead to different outcomes.

In many other non-Western societies, a stigma is associated with mental disorders despite the high prevalence of depression and anxiety. Many Easterners believe that symptoms of depression and anxiety are simply a part of life and do not require medical attention. Also, individuals from collective societies tend to be extremely careful in maintaining their reputations, and mental distress is usually interpreted as a weak faith in God and/or a weak self. Therefore, they do not seek treatment for their symptoms in order to avoid shame and gossip. This stigma stems in part from the limited knowledge many Easterners have about depression and anxiety. Multiple studies reveal that the majority of Easterners do not know the names of mental illnesses. Instead, they describe their symptoms through a series of physical complaints.

Considering the stigma associated with mental illness in the Arab culture, it is not surprising that many Easterners express depression and anxiety through somatic complaints. This is especially true with Eastern women, who may feel symptoms of depression as a result of inequality, but are taught not to express personal feelings. Instead, they complain of headaches, numbness, breathlessness, and chest pains to receive some form of medical attention. Somatization is also common among individuals with little knowledge of mental health who have no linguistic way to express their symptoms.

The causes of depression and anxiety in Arabs and Arab Americans are a combination of biological factors and psychological and social factors that are greatly influenced by the Arab culture and immigration. Research indicates that biological markers of depression are consistent between Arab and Western populations, as are many psychological factors.

Arabs who seek psychological help are more confident about the effectiveness of medication over therapy. But since many Arabs either refuse to meet with mental health professionals or do not have any knowledge of them, traditional treatment methods are still commonly used. Islamic therapy, for example, involves Quran recitation by an imam to repel moral deviance from the soul of the individual. The imam may also suggest old Islamic remedies, such as ice baths and herbal antidotes. Using Islam to simply cope with psychological distress is a more widely used method of treatment that is supported by research. A study completed on depressed Pakistani patients also reported that the participants believed that medicine could rid them of their symptoms but had little to no knowledge of psychotherapy.[1]

Latino adolescents tend to have higher levels of symptoms of depression than some of their Caucasian and African-American peers. An explanation for this difference is the increase in cultural stressors that in turn add to this form of cultural disparity. Although the cultural stressors have not yet been identified, the correlation between it and symptoms of and the prevalence of depression is still significant. Within communities, discrimination on the basis of race and ethnicity is also a strong contributor to the elevated number of depressive symptoms among Latino youth.

The Psychodynamic Perspective

According to the psychodynamic model, depression is caused by unresolved unconscious conflict or repressed anger towards others. A person may be so plagued with inner conflict, anxiety, and guilt that they are unable to perceive reality clearly or meet the ordinary demands of the environment in which they live.

The Humanistic Perspective

Humanistic psychology includes several approaches to counseling and therapy. Among the earliest approaches, we find the developmental theory of Abraham Maslow, emphasizing a hierarchy of needs and motivations; the existential psychology of Rollo May acknowledging human choice and the tragic aspects of human existence; and the person-centered or client-centered therapy of Carl Rogers, which is centered on the client's capacity for self-direction and understanding of his or her own development. Client-centered therapy is non-directive; the therapist listens to the client without judgment, allowing the client to come to insights by themselves. The therapist should ensure that all of the client's feelings are being considered and that the therapist has a firm grasp on the concerns of the client while ensuring that there is an air of acceptance and warmth. A client-centered therapist engages in active listening during therapy sessions.

Existential psychotherapies, an application of humanistic psychology, apply existential philosophy, which emphasizes the idea that humans have the freedom to make sense of their lives. They are free to define themselves and do whatever it is they want to do. This is a type of humanistic therapy that forces the client to explore the meaning of their life, as well as its purpose. There is a conflict between having freedoms and having limitations. Examples of limitations include genetics, culture, and many other factors. Existential therapy involves trying to resolve this conflict.

The Behavioral Perspective

Behavioral theories of depression emphasize the role maladaptive actions play in the onset and maintenance of depression. These theories stem from work concerning the principles of learning and conditioning from the early to mid-1900s. Ivan Pavlov and B. F. Skinner are often credited with the establishment of behavioral psychology with their research on classical conditioning and operant conditioning, respectively. Collectively, their research established that certain behaviors could be learned or unlearned, and these theories have been applied in a variety of contexts, including abnormal psychology. Theories specifically applied to depression emphasize the reactions individuals have to their environment and how they develop adaptive or maladaptive coping strategies. Beck's cognitive theory of depression (mentioned above) originally began as cognitive-specific therapy and as a response to psychodynamic and behavioral therapies of the time, but has since evolved to include and incorporate behavioral aspects, hence the more current umbrella term of *cognitive behavioral therapy*. CBT focuses on challenging and changing unhelpful cognitive distortions (e.g., thoughts, beliefs, and attitudes) and behaviors, improving emotional regulation, and the development of personal coping strategies that target solving current problems. Originally, it was designed to treat depression, but its uses have been expanded to include treatment of a number of mental health conditions, including anxiety. CBT includes a number of cognitive or behavior psychotherapies that treat defined psychopathologies using evidence-based techniques and strategies. Cognitive-based therapies include, but are not limited to, rational emotive therapy (REBT), cognitive therapy, acceptance and commitment therapy, dialectical behavior therapy, reality therapy/choice theory, cognitive processing therapy, EMDR, and multimodal therapy. All of these therapies are a blending of cognitive- and behavior-based elements, and they are popular choices in treating depression.

Glossary

cognitive behavioral therapy: a therapy focused on challenging and changing unhelpful cognitive distortions and behaviors, improving emotional regulation, and developing personal coping strategies that target solving current problems

diathesis-stress model: theory in which certain predispositions or vulnerability factors influence one's reaction to stress

hopelessness theory: cognitive theory of depression proposing that a style of thinking that perceives negative life events as having stable and global causes leads to a sense of hopelessness and then to depression

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7.12: Treatments for Mood Disorders

Learning Objectives

- Compare treatment methods for major depressive disorder
- Examine treatments for bipolar disorder

What Are Some of the Well-Supported Treatments for Mood Disorders?

Depressive Disorders



Figure **7.12.1**: Those taking MAOIs should avoid eating foods high in tyramine.

There are many treatment options available for people with MDD. First, a number of antidepressant medications are available, all of which target one or more of the neurotransmitters implicated in depression. The earliest antidepressant medications were monoamine oxidase inhibitors (MAOIs). Monoamine oxidase inhibitors (MAOIs) inhibit monoamine oxidase, an enzyme involved in deactivating dopamine, norepinephrine, and serotonin. Although effective in treating depression, monoamine oxidase inhibitors (MAOIs) can have serious side effects. Patients taking MAOIs may develop dangerously high blood pressure if they take certain drugs (e.g., antihistamines) or eat foods containing tyramine, an amino acid commonly found in foods such as aged cheeses, wine, and soy sauce. Tricyclics, the second-oldest class of antidepressant medications, block the reabsorption of norepinephrine, serotonin, or dopamine at synapses, resulting in their increased availability. Tricyclics are most effective for treating vegetative and somatic symptoms of depression. Like MAOIs, they have serious side effects, the most concerning of which is being cardiotoxic.

Selective serotonin reuptake inhibitors (SSRIs; e.g., Fluoxetine) and serotonin and norepinephrine reuptake inhibitors (SNRIs; e.g., Duloxetine) are the most recently introduced antidepressant medications. SSRIs, the most commonly prescribed antidepressant medication, block the reabsorption of serotonin, whereas SNRIs block the reabsorption of serotonin and norepinephrine. SSRIs and SNRIs have fewer serious side effects than do MAOIs and tricyclics. In particular, they are less cardiotoxic, less lethal in overdose, and produce fewer cognitive impairments. They are not, however, without their own side effects, which include but are not limited to difficulty having orgasms, gastrointestinal issues, and insomnia.

It should be noted that anti-depressant medication may not work equally for all people. This approach to treatment often involves experimentation with several medications and dosages, and may tend to be more effective when paired with physical exercise and psychotherapy.

Watch It

This video explains some of the medications used to treat depression



You can view the transcript for "Depression Medication" here (opens in new window).

Other biological treatments for people with depression include electroconvulsive therapy (ECT), transcranial magnetic stimulation (TMS), and deep brain stimulation. Electroconvulsive therapy (ECT) involves inducing a seizure after a patient takes muscle relaxants and is under general anesthesia. Electroconvulsive therapy (ECT) is viable treatment for patients with severe depression or who show resistance to antidepressants although the mechanisms through which it works remain unknown. A common side effect is confusion and memory loss, usually short-term (Schulze-Rauschenbach, Harms, Schlaepfer, Maier, Falkai, & Wagner, 2005).

Repetitive transcranial magnetic stimulation (TMS) is a noninvasive technique administered while a patient is awake. Brief pulsating magnetic fields are delivered to the cortex, inducing electrical activity. Transcranial magnetic stimulation (TMS) has fewer side effects than ECT (Schulze-Rauschenbach et al., 2005), and while outcome studies are mixed, there is evidence that TMS is a promising treatment for patients with MDD who have shown resistance to other treatments (Rosa et al., 2006). Most recently, deep brain stimulation is being examined as a treatment option for patients who did not respond to more traditional treatments like those already described. Deep brain stimulation involves implanting an electrode in the brain. The electrode is connected to an implanted neurostimulator, which electrically stimulates that particular brain region. Although there is some evidence of its effectiveness (Mayberg et al., 2005), additional research is needed.

Several psychosocial treatments have received strong empirical support, meaning that independent investigations have achieved similarly positive results—a high threshold for examining treatment outcomes. Psychosocial treatments include but are not limited to behavior therapy, cognitive therapy, and interpersonal therapy. Behavior therapies focus on increasing the frequency and quality of experiences that are pleasant or help the patient achieve mastery. Cognitive therapies primarily focus on helping patients identify and change distorted automatic thoughts and assumptions (e.g., Beck, 1967). Cognitive-behavioral therapies are based on the rationale that thoughts, behaviors, and emotions affect and are affected by each other. Interpersonal therapy for depression focuses largely on improving interpersonal relationships by targeting problem areas, specifically unresolved grief, interpersonal role disputes, role transitions, and interpersonal deficits. Finally, there is also some support for the effectiveness of short-term psychodynamic therapy for depression (Leichsenring, 2001). The short-term treatment focuses on a limited number of important issues, and the therapist tends to be more actively involved than in more traditional psychodynamic therapy.

Bipolar Disorders



Figure **7.12.2**: Nausea is a common physical side effect of lithium treatment.

Patients with BD are typically treated with pharmacotherapy. Antidepressants such as SSRIs and SNRIs are the primary choice of treatment for depression, whereas for BD, lithium is the first-line treatment choice because SSRIs and SNRIs have the potential to induce mania or hypomania in patients with BD. Mood stabilizers such as lithium act on several neurotransmitter systems in the brain through complex mechanisms, including reduction of excitatory (dopamine and glutamate) neurotransmission, and increasing of inhibitory (GABA) neurotransmission (Lenox & Hahn, 2000). Lithium has strong efficacy for the treatment of BD (Geddes, Burgess, Hawton, Jamison, & Goodwin, 2004). However, a number of side effects can make lithium treatment difficult for patients to tolerate. Side effects include impaired cognitive function (Wingo, Wingo, Harvey, & Baldessarini, 2009), as well as physical symptoms such as nausea, tremor, weight gain, and fatigue (Dunner, 2000). Some of these side effects can improve with continued use; however, medication noncompliance remains an ongoing concern in the treatment of patients with BD. Anticonvulsant medications (e.g., carbamazepine and valproate) are also commonly used to treat patients with BD, either alone or in conjunction with lithium.

There are several adjunctive treatment options for people with BD. Interpersonal and social rhythm therapy (IPSRT; Frank et al., 1994) is a psychosocial intervention focused on addressing the mechanism of action posited in social zeitgeber theory to predispose patients who have BD to relapse, namely sleep disruption. A growing body of literature provides support for the central role of sleep dysregulation in BD (Harvey, 2008). Consistent with this literature, IPSRT aims to increase rhythmicity of patients' lives and encourage vigilance in maintaining a stable rhythm. The therapist and patient work to develop and maintain a healthy balance of activity and stimulation such that the patient does not become overly active (e.g., by taking on too many projects) or inactive (e.g., by avoiding social contact). The efficacy of IPSRT has been demonstrated in that patients who received this treatment show reduced risk of episode recurrence and are more likely to remain well (Frank et al., 2005).

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7.13: Suicide

Learning Objectives

- Discuss the relationship between mood disorders and suicidal ideation as well as factors associated with suicide



Figure **7.13.1**: Having protective factors like social support is crucial to coping with emotional pain.

For some people with mood disorders, the extreme emotional pain they experience becomes unendurable. Overwhelmed by hopelessness, devastated by incapacitating feelings of worthlessness, and burdened with the inability to adequately cope with such feelings, they may consider suicide to be a reasonable way out. **Suicide**, defined by the CDC as "death caused by self-directed injurious behavior with any intent to die as the result of the behavior" (CDC, 2013a), in a sense represents an outcome of several things going wrong all at the same time (Crosby, Ortega, & Melanson, 2011). Not only must the person be biologically or psychologically vulnerable, but he or she must also have the means to perform the suicidal act, and must lack the necessary protective factors (e.g., social support from friends and family, religion, coping skills, and problem-solving skills) that provide comfort and enable one to cope during times of crisis or great psychological pain (Berman, 2009).

Suicide is not listed as a disorder in the DSM-5; however, suffering from a mental disorder—especially a mood disorder—poses the greatest risk for suicide. Around 90% of those who complete suicides have a diagnosis of at least one mental disorder, with mood disorders being the most frequent (Fleischman, Bertolote, Belfer, & Beautrais, 2005). In fact, the association between major depressive disorder and suicide is so strong that one of the criteria for the disorder is thoughts of suicide, as discussed above (APA, 2013). Suicide rates can be difficult to interpret because some deaths that appear to be accidental may in fact be acts of suicide (e.g., automobile crash).

Antidepressant medications may also have an effect on suicidal thoughts as a troubling side effect.[1] Studies have shown that the use of antidepressants is correlated with an increased risk of suicidal behavior and thinking (suicidality) in those aged under 25. This problem has been serious enough to warrant government intervention by the FDA to warn of the increased risk of suicidality during antidepressant treatment. According to the FDA, the heightened risk of suicidality occurs within the first one to two months of treatment.

The National Institute for Health and Care Excellence (NICE) places the excess risk in the "early stages of treatment." A meta-analysis suggests that the relationship between antidepressant use and suicidal behavior or thoughts is age-dependent. Compared with a placebo, the use of antidepressants is associated with an increase in suicidal behavior or thoughts among those 25 or younger (OR=1.62). There is no effect or possibly a mild protective effect among those aged 25 to 64 (OR=0.79). Antidepressant treatment has a protective effect against suicidality among those aged 65 and over (OR=0.37). Nevertheless, investigations into U.S. suicide rates have uncovered these facts:

- Suicide was the 10th leading cause of death for all ages in 2010 (Centers for Disease Control and Prevention [CDC], 2012).
- There were 38,364 suicides in 2010 in the United States—an average of 105 each day (CDC, 2012).
- Suicide among males is four times higher than among females and accounts for 79% of all suicides; firearms are the most commonly used method of suicide for males, whereas poisoning is the most commonly used method for females (CDC, 2012).
- From 1991 to 2003, suicide rates were consistently higher among those 65 years and older. Since 2001, however, suicide rates among those ages 25–64 have risen consistently, and, since 2006, suicide rates have been greater for those ages 65 and older (CDC, 2013b). This increase in suicide rates among middle-aged Americans has prompted concern in some quarters that baby boomers (individuals born between 1946–1964) who face economic worry and easy access to prescription medication may be particularly vulnerable to suicide (Parker-Pope, 2013).

- The highest rates of suicide within the United States are among American Indians/Alaskan natives and non-Hispanic Whites (CDC, 2013b).
- Suicide rates vary across the United States, with the highest rates consistently found in the mountain states of the west (Alaska, Montana, Nevada, Wyoming, Colorado, and Idaho) (Berman, 2009).

Contrary to popular belief, suicide rates peak during the springtime (April and May), not during the holiday season or winter. In fact, suicide rates are generally lowest during the winter months (Postolache et al., 2010).

Celebrity Suicides



Figure **7.13.2**: Celebrity chef Anthony Bourdain.

In early June 2018, celebrity chef Anthony Bourdain was working on an episode of *Parts Unknown* in Strasbourg, with his frequent collaborator and friend Éric Ripert. On June 8, Ripert became worried when Bourdain had missed dinner and breakfast. He subsequently found Bourdain dead of an apparent suicide by hanging in his room at Le Chambard hotel in Kaysersberg near Colmar. Bourdain was 61 years old.

Christian de Rocquigny du Fayel, the public prosecutor for Colmar, said Bourdain's body bore no signs of violence and the suicide appeared to be an impulsive act. Rocquigny du Fayel disclosed that Bourdain's toxicology results were negative for narcotics, showing only a trace of a therapeutic non-narcotic medication. Bourdain's body was cremated in France on June 13, 2018, and his ashes were returned to the United States two days later.
Bourdain's mother, Gladys Bourdain, told The New York Times, "He is absolutely the last person in the world I would have ever dreamed would do something like this."

Following the news of Bourdain's death, various celebrity chefs and other public figures expressed sentiments of condolence. Among them were fellow chefs Andrew Zimmern and Gordon Ramsay and former astronaut Scott Kelly. CNN issued a statement, saying that Bourdain's "talents never ceased to amaze us and we will miss him very much." Former U.S. President Barack Obama, who dined with Bourdain in Vietnam on an episode of *Parts Unknown*, wrote on Twitter, "He taught us about food—but more importantly, about its ability to bring us together. To make us a little less afraid of the unknown." On June 8, 2018, CNN aired *Remembering Anthony Bourdain*, a tribute program.

In the days following Bourdain's death, fans paid tribute to him outside his now-closed former place of employment, Brasserie Les Halles. Cooks and restaurant owners gathered together and held tribute dinners and memorials and donated net sales to the National Suicide Prevention Lifeline.

Bourdain's death came just days after the suicide of designer Kate Spade.

Watch this video from CBS in the aftermath of Bourdain's death as it discusses some of the risk factors for suicide.



You can view the transcript for "Anthony Bourdain's death: Dr. LaPook on suicide risk factors" here (opens in new window).

Risk Factors for Suicide



Figure 7.13.3: Alcoholism is a significant risk factor for suicide.

Suicidal risk is especially high among people with substance abuse problems. Individuals with alcohol dependence are at 10 times greater risk for suicide than the general population (Wilcox, Conner, & Caine, 2004). The risk of suicidal behavior is especially high among those who have made a prior suicide attempt. Among those who attempt suicide, 16% make another attempt within a year and over 21% make another attempt within four years (Owens, Horrocks, & House, 2002). Suicidal individuals may be at high risk for terminating their life if they have a lethal means in which to act, such as a firearm in the home (Brent & Bridge, 2003). Withdrawal from social relationships, feeling as though one is a burden to others, and engaging in reckless and risk-taking behaviors may be precursors to suicidal behavior (Berman, 2009). A sense of entrapment or feeling unable to escape one's miserable feelings or external circumstances (e.g., an abusive relationship with no perceived way out) predicts suicidal behavior (O'Connor, Smyth, Ferguson, Ryan, & Williams, 2013). Tragically, reports of suicides among adolescents following instances of cyberbullying have emerged in recent years. In one widely publicized case, Phoebe Prince, a 15-year-old high school student in Massachusetts, committed suicide following incessant harassment and taunting from her classmates via texting and Facebook (McCabe, 2010).

Suicides can have a contagious effect on people. For example, another's suicide, especially that of a family member, heightens one's risk of suicide (Agerbo, Nordentoft, & Mortensen, 2002). Additionally, widely publicized suicides tend to trigger copycat suicides in some individuals. One study examining suicide statistics in the United States from 1947 to 1967 found that the rates of suicide skyrocketed for the first month after a suicide story was printed on the front page of the *New York Times* (Phillips, 1974). Austrian researchers found a significant increase in the number of suicides by firearms in the three weeks following extensive reports in Austria's largest newspaper of a celebrity suicide by gun (Etzersdorfer, Voracek, & Sonneck, 2004). A review of 42 studies concluded that media coverage of celebrity suicides is more than 14 times more likely to trigger copycat suicides than is coverage of non-celebrity suicides (Stack, 2000). This review also demonstrated that the medium of coverage is important: televised stories are considerably less likely to prompt a surge in suicides than are newspaper stories. Research suggests that a trend appears to be emerging whereby people use online social media to leave suicide notes, although it is not clear to what extent suicide notes on such media might induce copycat suicides (Ruder, Hatch, Ampanozi, Thali, & Fischer, 2011). Nevertheless, it is reasonable to conjecture that suicide notes left by individuals on social media may influence the decisions of other vulnerable people who encounter them (Luxton, June, & Fairall, 2012).

One possible contributing factor in suicide is brain chemistry. Contemporary neurological research shows that disturbances in the functioning of serotonin are linked to suicidal behavior (Pompili et al., 2010). Low levels of serotonin predict future suicide attempts and suicide completions, and low levels have been observed post-mortem among suicide victims (Mann, 2003). Serotonin dysfunction, as noted earlier, is also known to play an important role in depression; low levels of serotonin have also been linked to aggression and impulsivity (Stanley et al., 2000). The combination of these three characteristics constitutes a potential formula for suicide—especially violent suicide. A classic study conducted during the 1970s found that patients with major depressive disorder who had very low levels of serotonin attempted suicide more frequently and more violently than did patients with higher levels (Asberg, Thorén, Träskman, Bertilsson, & Ringberger, 1976; Mann, 2003).

Suicidal thoughts, plans, and even off-hand remarks ("I might kill myself this afternoon") should always be taken extremely seriously. People who contemplate terminating their life need immediate help. Below are links to two excellent websites that contain resources (including hotlines) for people who are struggling with suicidal ideation, have loved ones who may be suicidal, or who have lost loved ones to suicide: American Foundation for Suicide Prevention and American Association of Suicidology.

Watch It: Treatments for Suicide Prevention

Effective treatments for suicide are **dialectical behavioral therapy (DBT)**, **cognitive therapy for suicide prevention (CT-SP),** and **brief cognitive behavioral therapy (BCBT)**. These plans generally begin with a safety plan, then learning methods to manage emotions (such as mindfulness), changing unhealthy coping mechanisms, and managing thoughts.



You can view the transcript for "Suicide Prevention Treatment" here (opens in new window).

Dialectical Behavior Therapy

The most notable and heavily researched treatment that has been shown to reduce suicidal behaviors regardless of the intent to die, is dialectical behavior therapy (DBT). Dialectical behavior therapy (DBT) has four main components: individual therapy, skills training, phone coaching, and a consultation team. DBT's main goal is to teach the patient skills to regulate emotions and improve relationships with others (suicidality is always targeted at the forefront of care). Skills are taught through validation and acceptance with a genuine focus on behavioral change. DBT was one of the first evidence-based treatments shown to be effective in decreasing repetitive self-harm behaviors and suicide attempts. More recent results have demonstrated DBT's continued impact on decreasing suicidal behaviors among high risk individuals such as those with borderline personality disorder, and decreasing suicide ideation and self-harm among adolescents. However, while DBT has shown impressive results in managing suicidal behaviors, it is not solely devoted to treating suicidality, and replicated results for reliably decreasing suicidal ideation are not consistent across all DBT randomized controlled trials (RCTs).

Cognitive Therapy for Suicide Prevention

Another effective treatment that targets the "suicidal mode" is cognitive therapy for suicide prevention (CT-SP). Cognitive therapy for suicide prevention (CT-SP) treats the clinical characteristics of suicidal behaviors by using various cognitive therapy techniques, which have proven successful for treating an extensive array of psychiatric disorders. In a well-powered randomized controlled trial (RCT) (with a deliberately longer follow-up period than previous RCTs—18 months), Brown and

colleagues[2] found that patients in CT-SP treatment were 50% less likely to attempt suicide compared to those in the usual care treatment group. The researchers also found significant reductions in levels of depression and hopelessness in the CT-SP treatment group compared to the control. This study showed high internal validity; replication of the data in a real world setting (e.g., a community-based outpatient setting) with varied samples (e.g., those who have not attempted suicide, but with severe ideation) is a pending next step for the researchers of CT-SP.

Brief Cognitive Behavior Therapy

Brief cognitive behavior therapy (BCBT) was used in one well-powered randomized controlled trial (RCT) with suicidal, active duty U.S. Army soldiers and was shown to be effective for reducing suicide attempts.[3] As its name indicates, this modality is brief (i.e., 12 sessions) to accommodate short-term treatment environments. This variation of CBT suicide-focused care emphasizes common effective treatment elements, developing skills (e.g., emotion regulation, mindfulness), a focus on the suicidal mode, and the development of self-management. Rudd and colleagues followed soldiers for 24 months and found that compared to treatment as usual, those in the brief cognitive behavior therapy (BCBT) group were 60% less likely to attempt suicide.[4]

Glossary

brief cognitive behavior therapy: a modified CBT program with 12 session designed to help patients after a suicide attempt; they develop skills and self-management techniques

cognitive therapy for suicide prevention: a type of CBT for patients following a suicide attempt in which they are taught new ways of thinking during suicidal episodes

dialectical behavior therapy: a therapy focused on teaching a patient skills to regulate emotions and improve relationships with others

suicidal ideation: thoughts of death by suicide, thinking about or planning suicide, or making a suicide attempt

suicide: death caused by intentional, self-directed injurious behavior

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7.14: Case Studies- Mood Disorders

Learning Objectives

- Identify mood disorders in case studies

Let's use the information we've learned in this module to examine a few fictional characters.

Case Study: A.J. from *The Sopranos*

We'll start with the case study of Anthony Soprano, Jr. (referred to as A.J.) from *The Sopranos* (a HBO television series, 1999–2005). A.J. started a new job working construction and was getting more stable in his life following dropping out of community college. He met a girl named Blanca at the construction site and they started dating.

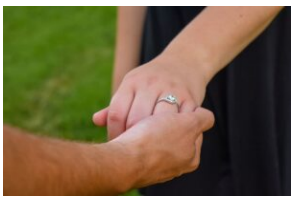


Figure **7.14.1**: A.J. became sad and irritable following his failed engagement.

The two became really close, and A. J. eventually proposed to Blanca. After some reconsideration, she decided that A.J. was not right for her and broke up with him. Following the breakup, A.J. is sad and lethargic. A.J. continued to work at the construction site for some time, but the sight of Blanca talking to other men becomes too much for him, so he eventually quits. After the breakup with Blanca, A.J. started sleeping all the time and would not come out of his room. He had a decreased appetite and anhedonia. He seemed to lack energy for quite some time. There were no suicidal ideations initially. Though his symptoms seemed to be improving slightly, after an incident with another student, he again confined himself to his room. He attempted to kill himself by jumping into a pool with a cinderblock around his leg while his parents were out of the house. Luckily, his father came home and saved him prior to there being any significant damage. A.J. was admitted to an inpatient psychiatric facility and received the therapy he needed.

Treatment

Proper treatment of A.J.'s diagnosis would, given his severe symptom levels, include beginning with antidepressant medication. Psychotherapy, specifically CBT (which has shown great success with a combination of antidepressant medication), DBT, CT-SP, or BCBT, may also prove helpful.

Case Study: Eeyore from *Winnie the Pooh*

Background Information



Figure **7.14.2**: Eeyore.

Eeyore is an older gray donkey. There are no documents indicating the exact age. Eeyore does not have an occupation. One main difficulty Eeyore has elaborated on is his detachable tail, which seems to cause him several problems. He has indicated that his goals are to remain strong for his friends despite his lack of self-confidence, and as a result, he often feels lonely without support from others that he is close to. Some forms of coping mechanisms include trying to feel useful in the presence of others and also trying his best to find pleasure in life.

Description of the Problem

Eeyore constantly insists that his tail falls off rather frequently. Eeyore's posture typically involves a slumped head and droopy eyes, and he commonly says, "thanks for noticing me." Sluggish movement is also apparent, without any physical cause for movement delay. He seems to step on his tail often and fall down. Eeyore indicates that sometimes it seems that even his close friends do not need him. Around friends, he typically makes comments about his relative unimportance and travels near the back of the pack. He also stated that although he tries to force a smile, a real smile has not existed in a long time, even though others try to cheer him up. He often feels empty even when accompanied by friends. Eeyore also seems to experience a loss of energy throughout the day, although sleeping habits are not explicitly expressed.

Diagnosis

Eeyore met criteria including depressed mood most of the day, markedly diminished interest or pleasure in activities, fatigue or loss of energy nearly every day, feelings of worthlessness, and diminished ability to think or concentrate.

Case Study: Ashlynn

Ashlynn is a 21-year-old college student and political science major who has struggled with depression on and off since beginning college. She was picked up by campus police after being caught vandalizing a man's apartment and was recommended to a psychiatrist after exhibiting erratic behavior. She explained that she was only at the man's apartment because he was a classmate and she wanted to prove to him how much she liked him. She spoke about how that, even though he didn't know it yet, they were destined to be together and that they would someday run for office together and be the first-ever married president and vice president. She was irritable and argumentative, and defensive about her behavior. She indicated that she didn't care about her finals anymore (though she had consistently good grades in the past), she had been pulling all-nighters to research politicians, and had been experimenting with illicit drugs.

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7.15: Putting It Together- Mood Disorders



In this chapter, we learned about mood disorders—disorders in which a person experiences severe disturbances in mood and emotion. They include depressive disorders and bipolar and related disorders. Depressive disorders include major depressive disorder, which is characterized by episodes of profound sadness and loss of interest or pleasure in usual activities and other associated features, and persistent depressive disorder, which is marked by a chronic state of sadness.

Bipolar disorder is characterized by mood states that vacillate between sadness and euphoria; a diagnosis of bipolar disorder requires experiencing at least one manic episode, which is defined as a period of extreme euphoria, irritability, and increased activity.

Mood disorders appear to have a genetic component, with genetic factors playing a more prominent role in bipolar disorder than in depression. Both biological and psychological factors are important in the development of depression. People who suffer from mental health problems, especially mood disorders, are at heightened risk for suicide.

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CHAPTER OVERVIEW

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8.1: Why It Matters- Eating and Sleep Disorders

Why learn about eating and sleep disorders?



Figure **8.1.1**: Good sleep is an essential part of a healthy lifestyle.

Eating and sleeping are essential to human survival. Humans need food to survive to promote healthy development. Eating food provides nutrients to build bones and grow muscle, regulate and make hormones, breathe, and produce energy. In order for humans to continue developing, sleep is fundamental. In this module, you will learn about the fundamentals of hunger and sleep and how if there is disruption, there can be significant health consequences.

Sleep is a basic necessity of life, as important to our health and well-being as air, food, and water. When we sleep well, we wake up feeling refreshed, alert, and ready to face daily challenges. When we don't, every part of our lives can suffer. Our jobs, relationships, productivity, health, and safety (and that of those around us) are all put at risk. There are many common myths about sleep. We hear them frequently and may even experience them far too often. Sometimes the myths can be characterized as "old wives tales," but there are other times the incorrect information can be serious and even dangerous. The National Sleep Foundation has compiled this list of common myths about sleep and the facts that dispel them.

There are quite a few misconceptions regarding eating and sleep disorders, so let's begin by dispelling some common myths about sleep.

Myth #1. Daytime sleepiness always means a person isn't getting enough sleep.

Excessive daytime sleepiness is a condition in which an individual feels very drowsy during the day and has an urge to fall asleep when he/she should be fully alert and awake. The condition, which can occur even after getting enough night-time sleep, can be a sign of an underlying medical condition or sleep disorder such as narcolepsy or sleep apnea. These problems can often be treated, and symptoms should be discussed with a physician. Daytime sleepiness can be dangerous and puts a person at risk for drowsy driving, injury, and illness and can impair mental abilities, emotions, and performance.

Myth #2: You can "cheat" on the amount of sleep you need.

Sleep experts say most adults need between seven and nine hours of sleep each night for optimum performance, health, and safety. When we don't get adequate sleep, we accumulate a sleep debt that can be difficult to "pay back" if it becomes too big. Sleep deprivation has been linked to health problems such as obesity and high blood pressure, negative mood and behavior, decreased productivity, and safety issues in the home, on the job, and on the road.

Myth #3: During sleep, your brain rests.

The body rests during sleep; however, the brain remains active, gets "recharged," and still controls many body functions including breathing. When we sleep, we typically drift between two sleep states, rapid eye movement (REM) and non-REM, in 90-minute cycles. Non-REM sleep has three stages with distinct features, ranging from stage one drowsiness, when one can be easily awakened, to "deep sleep" stage three, when awakenings are more difficult and where the most positive and restorative effects of sleep occur; however, even in the deepest non-REM sleep, our minds can still process information. REM sleep is an active sleep where dreams occur, breathing and heart rate increase and become irregular, muscles relax, and eyes move back and forth under the eyelids.

Myth #4: If you wake up in the middle of the night, it is best to lie in bed, count sheep, or toss and turn until you eventually fall back asleep.

Waking up in the middle of the night and not being able to go back to sleep is a symptom of insomnia. Relaxing imagery or thoughts may help to induce sleep more than counting sheep, which some research suggests may be more distracting than relaxing.

Whichever technique is used, most experts agree that if you do not fall back asleep within 15–20 minutes, you should get out of bed, go to another room and engage in a relaxing activity such as listening to music or reading. Return to bed when you feel sleepy. Avoid watching the clock.

Myth #5: The older you get, the fewer hours of sleep you need.

Sleep experts recommend a range of seven to nine hours of sleep for the average adult. While sleep patterns change as we age, the amount of sleep we need generally does not. Older people may wake more frequently through the night and may actually get less night-time sleep, but their sleep need is no less than younger adults. Because they may sleep less during the night, older people tend to sleep more during the day. Naps planned as part of a regular daily routine can be useful in promoting wakefulness after the person awakens. According to sleep experts, teens need at least 8.5–9.25 hours of sleep each night, compared to an average of seven to nine hours each night for most adults. Adult internal biological clocks also keep them awake later in the evening and keep them sleeping later in the morning; however, many schools begin classes early in the morning, when a teenager's body wants to be asleep. As a result, many teens come to school too sleepy to learn, through no fault of their own.

Myth #6: Health problems such as obesity, diabetes, hypertension, and depression are unrelated to the amount and quality of a person's sleep.

Studies have found a relationship between the quantity and quality of one's sleep and many health problems. For example, insufficient sleep affects growth hormone secretion that is linked to obesity; as the amount of hormone secretion decreases, the chance for weight gain increases. Blood pressure usually falls during the sleep cycle; however, interrupted sleep can adversely affect this normal decline, leading to hypertension and cardiovascular problems. Research has also shown that insufficient sleep impairs the body's ability to use insulin, which can lead to the onset of diabetes. More and more scientific studies are showing correlations between poor and insufficient sleep and disease.

Myth #7: Snoring is a common problem, especially among men, but it isn't harmful.

Although snoring may be harmless for most people, it can be a symptom of a life-threatening sleep disorder called sleep apnea, especially if it is accompanied by severe daytime sleepiness. Sleep apnea is characterized by pauses in breathing that prevent air from flowing into or out of a sleeping person's airways. People with sleep apnea awaken frequently during the night gasping for breath. The breathing pauses reduce blood oxygen levels, can strain the heart and cardiovascular system, and increase the risk of cardiovascular disease. Snoring on a frequent or regular basis has been directly associated with hypertension. Obesity and a large neck can contribute to sleep apnea. Sleep apnea can be treated. Men and women who snore loudly, especially if pauses in the snoring are noted, should consult a physician.



Figure **8.1.2**: Eating disorders are dangerous disorders with a high mortality rate; they can affect people of all shapes and sizes.

Now let's examine a few myths about eating disorders.

Myth #1: Only women and girls can get an eating disorder.

False. According to the National Eating Disorders Association, 10 million men and boys in the United States will suffer from an eating disorder. Eating disorders affect a diverse array of people of various ethnicities, ages, genders, body weight, and socioeconomic groups.

Myth #2: You can tell someone is suffering from an eating disorder by the way they look.

False. Anorexia nervosa is an eating disorder in which a person unreasonably limits food intake to prevent weight gain. Individuals who suffer from this disorder may appear extremely thin, but that may be the result of struggling with the disorder for a long time. The other most common eating disorder, bulimia nervosa, uses bingeing and purging to control weight. These people may appear healthy, despite the internal damage being done to their bodies.

Myth #3: Only external influences, such as peer pressure or distorted physical images, can cause the onset of an eating disorder.

False. While it is difficult to pinpoint the cause of an eating disorder, research conducted by the National Institute of Health suggests that genetic, psychological, behavioral, biological, and social factors can heighten the risk.

Myth #4: Eating disorders are a choice.

False. Eating disorders are not lifestyle choices. People don't choose to have an eating disorder like they might choose to eat only vegetables or fish. Eating disorders are caused by a complex interaction of genetic, biological, behavioral, psychological, and social factors.

Myth #5: Eating disorders are not really serious.

False. Some research has shown a direct correlation between eating disorders and suicide attempts. If untreated, eating disorders can cause an imbalance in electrolytes that can result in a stroke or heart attack, intestinal distress, brain damage, and multi-organ failure.

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8.2: Introduction to Feeding and Eating Disorders

What you'll learn to do: describe feeding and eating disorders



Eating is essential for survival, and it is no surprise that a drive like hunger exists to ensure that we seek out sustenance. In this section, we will review the fundamentals of hunger and eating as a primer to differentiate healthy and normal hunger and eating from disordered eating. You will learn about hunger, metabolism, body weight, and how disordered eating can have significant health consequences.

Watch It

This Mental Health Minute video about eating disorders from the National Institute of Mental Health (NIMH) gives a general summary of eating disorders, who it affects, and how treatment can be effective.



You can view the transcript for "Mental Health Minute: Eating Disorders" here (opens in new window).

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8.3: Hunger and Eating

Learning Objectives

- Describe how hunger and eating are regulated in the human body

Understanding Hunger

There are a number of physiological mechanisms that serve as the basis for hunger. When our stomachs are empty, they contract, causing both hunger pangs and the secretion of chemical messages that travel to the brain to serve as a signal to initiate feeding behavior. When our blood glucose levels drop, the pancreas and liver generate a number of chemical signals that induce hunger (Konturek et al., 2003; Novin, Robinson, Culbreth, & Tordoff, 1985) and thus initiate feeding behavior.

For most people, once they have eaten, they feel **satiation**, or fullness and satisfaction, and their eating behavior stops. Like the initiation of eating, satiation is also regulated by several physiological mechanisms. As blood glucose levels increase, the pancreas and liver send signals to shut off hunger and eating (Drazen & Woods, 2003; Druce, Small, & Bloom, 2004; Greary, 1990). The food's passage through the gastrointestinal tract also provides important satiety signals to the brain (Woods, 2004), and fat cells release **leptin**, a satiety hormone.

The various hunger and satiety signals that are involved in the regulation of eating are integrated in the brain. Research suggests that several areas of the hypothalamus and hindbrain are especially important sites where this integration occurs (Ahima & Antwi, 2008; Woods & D'Alessio, 2008). Ultimately, activity in the brain determines whether or not we engage in feeding behavior (Figure **8.3.1**).

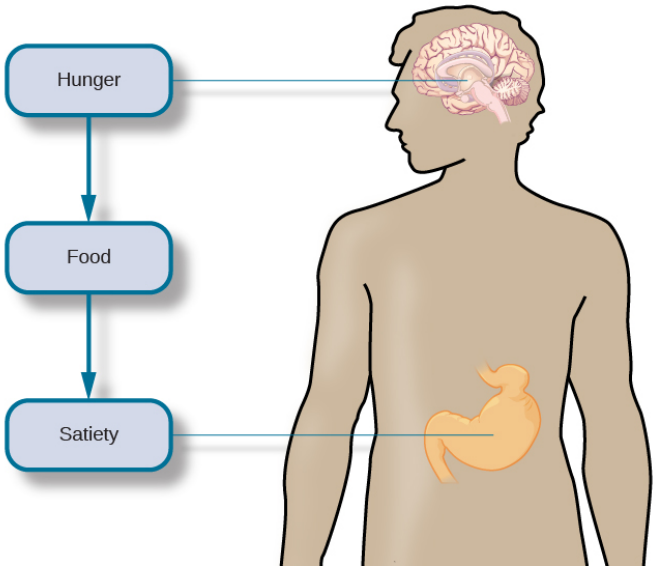


Figure **8.3.1**: Hunger and eating are regulated by a complex interplay of hunger and satiety signals that are integrated in the brain.

Metabolism and Body Weight

Our body weight is affected by a number of factors, including gene-environment interactions, and the number of calories we consume versus the number of calories we burn in daily activity. If our caloric intake exceeds our caloric use, our bodies store excess energy in the form of fat. If we consume fewer calories than we burn off, then stored fat will be converted to energy. Our energy expenditure is obviously affected by our levels of activity, but our body's metabolic rate also comes into play. A person's **metabolic rate** is the amount of energy that is expended in a given period of time, and there is tremendous individual variability in our metabolic rates. People with high rates of metabolism are able to burn off calories more easily than those with lower rates of metabolism.

We all experience fluctuations in our weight from time to time, but generally, most people's weights fluctuate within a narrow margin, in the absence of extreme changes in diet and/or physical activity. This observation led some to propose a set-point theory

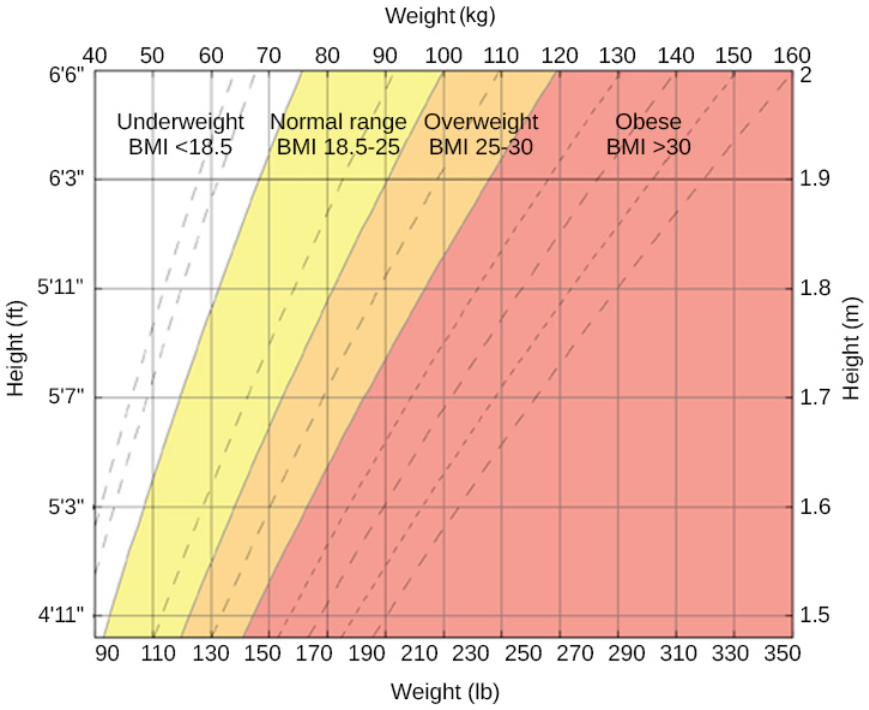
of body weight regulation. The **set-point theory** asserts that each individual has an ideal body weight, or set point, which is resistant to change. This set-point is genetically predetermined; efforts to move our weight significantly from the set-point are resisted by compensatory changes in energy intake and/or expenditure (Speakman et al., 2011).

Obesity

One common measure of health and body weight, and to differentiate the status of someone's weight from underweight, normal, overweight, and obese is the **Body Mass Index (BMI)**.

When someone weighs more than what is generally accepted as healthy for a given height, they are considered overweight or obese. According to the Centers for Disease Control and Prevention (CDC), an adult with a **body mass index** (BMI) between 25 and 29.9 is considered **overweight** (Figure 2). An adult with a Body Mass Index (BMI) of 30 or higher is considered **obese** (Centers for Disease Control and Prevention [CDC], 2012). People who are so overweight that they are at risk for death are classified as morbidly obese. **Morbid obesity** is defined as having a BMI over 40.

However, it should be noted that recently the utility of the BMI has been questioned.[1] Specifically, the BMI has been used as a healthy weight indicator by the World Health Organization (WHO), the CDC, and other groups, its value as an assessment tool has been questioned. The BMI is most useful for studying populations, which is the work of these organizations. It is less useful in assessing an individual since height and weight measurements fail to account for important factors like fitness level. An athlete, for example, may have a high BMI because the tool doesn't distinguish between the body's percentage of fat and muscle in a person's weight.



30" area covers approximately 140–350 pounds at height 4'11" and extends to approximately 265–350 pounds at height 6'6.""
width="614">

Figure **8.3.2**: This chart shows how adult BMI is calculated. Individuals find their height on the *y*-axis and their weight on the *x*-axis to determine their BMI.

Being extremely overweight or obese is a risk factor for several negative health consequences. Negative health consequences include, but are not limited to, an increased risk for cardiovascular disease, stroke, Type 2 diabetes, liver disease, sleep apnea, colon cancer, breast cancer, infertility, and arthritis. Given that it is estimated that around one-third of the adult U.S. population is obese and that nearly two-thirds of adults and one in six children qualify as overweight (CDC, 2012), there is substantial interest in trying to understand how to combat this important public health concern.

Watch It

Watch this video to learn about abnormal eating behaviors, including the most common eating disorders (which we'll examine in detail soon), and a summary of basic treatment strategies.

You can view the transcript for "Eating Disorder: Presentation & Treatment" here (opens in new window).

Dig Deeper: Prader-Willi Syndrome



Figure 8.3.3: Eugenia Martínez Vallejo, depicted in this 1680 painting, may have had Prader-Willi syndrome. At just eight years old, she weighed approximately 120 pounds, and she was nicknamed "La Monstrua" (the monster).

Prader-Willi Syndrome (PWS) is a genetic disorder that results in persistent feelings of intense hunger and reduced rates of metabolism. Typically, affected children have to be supervised around the clock to ensure that they do not engage in excessive eating. Currently, PWS is the leading genetic cause of morbid obesity in children, and it is associated with a number of cognitive deficits and emotional problems.

While genetic testing can be used to make a diagnosis, there is a number of behavioral diagnostic criteria associated with PWS. From birth to two years of age, lack of muscle tone and poor sucking behavior may serve as early signs of PWS. Developmental delays are seen between the ages of six and 12, and excessive eating and cognitive deficits associated with PWS usually onset a little later.

While the exact mechanisms of PWS are not fully understood, there is evidence that affected individuals have hypothalamic abnormalities. This is not surprising, given the hypothalamus's role in regulating hunger and eating. However, as you will learn in the next section of this module, the hypothalamus is also involved in the regulation of sexual behavior. Consequently, many individuals suffering from PWS fail to reach sexual maturity during adolescence.

There is no current treatment or cure for PWS. However, if weight can be controlled in these individuals, then their life expectancies are significantly increased (historically, sufferers of PWS often died in adolescence or early adulthood). Advances in the use of various psychoactive medications and growth hormones continue to enhance the quality of life for individuals with PWS (Cassidy & Driscoll, 2009; Prader-Willi Syndrome Association, 2012).

Glossary

leptin: satiety hormone

metabolic rate: amount of energy that is expended in a given period of time

morbid obesity: adult with a BMI over 40

obese: adult with a BMI of 30 or higher

overweight: adult with a BMI between 25 and 29.9

satiation: fullness; satisfaction

set point theory: assertion that each individual has an ideal body weight, or set point, that is resistant to change

1. Robert H. Shmerling, M. D. (2020, June 23). How useful is the body mass index (BMI)? https://www.health.harvard.edu/blog/how-useful-is-the-body-mass-index-bmi-201603309339. ↵

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8.4: Anorexia Nervosa

Learning Objectives

- Describe anorexia nervosa, including the etiology, symptoms, and psychological health complications
- Describe treatment options for anorexia nervosa

While nearly two out of three U.S. adults struggle with issues related to being overweight, a smaller but significant portion of the population has eating disorders that typically result in being normal weight or underweight. Often, individuals are fearful of gaining weight. Individuals who suffer from bulimia nervosa and anorexia nervosa face many adverse health consequences (Mayo Clinic, 2012a, 2012b).

Anorexia nervosa (AN) is an eating disorder characterized by the maintenance of a bodyweight well below average through starvation and/or excessive exercise. Individuals suffering from anorexia nervosa often have a **distorted body image**, referenced in literature as a type of body dysmorphia, meaning that they view themselves as overweight even though they are not.

Signs and Symptoms



Figure **8.4.1**: While being physically active is healthy, excessive exercise may be indicative of a serious issue.

People with anorexia nervosa may see themselves as overweight, even when they are dangerously underweight. People with anorexia nervosa typically weigh themselves repeatedly, severely restrict the amount of food they eat, often exercise excessively, and/or may force themselves to vomit or use laxatives to lose weight. Anorexia nervosa has the highest mortality rate of any mental disorder. While many people with this disorder die from complications associated with starvation, others die of suicide.

Symptoms include the following:

- extremely restricted eating
- extreme thinness (emaciation)
- a relentless pursuit of thinness and unwillingness to maintain a normal or healthy weight
- intense fear of gaining weight
- distorted body image, a self-esteem that is heavily influenced by perceptions of body weight and shape, or a denial of the seriousness of low body weight

The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-4) criteria for anorexia describes the ways people attempt to lose weight into two subtypes: restrictive and binge-eating/purging. In addition, both subtypes will sometimes engage in excessive exercise in an effort to lose weight as well. A patient can be diagnosed with anorexia when they have a bodyweight that is less than 85% of normal BMI in conjunction with fear and preoccupation with gaining weight and a distorted body image and perception despite being underweight.[1]

Other symptoms may develop over time:

- thinning of the bones (osteopenia or osteoporosis)
- mild anemia and muscle wasting and weakness
- brittle hair and nails
- dry and yellowish skin
- growth of fine hair all over the body (lanugo)
- severe constipation
- low blood pressure slowed breathing and pulse
- damage to the structure and function of the heart

- difficulty concentrating
- brain damage
- multiorgan failure
- drop in internal body temperature, causing a person to feel cold all the time
- lethargy, sluggishness, or feeling tired all the time
- amenorrhea
- infertility

Link to Learning

Content warning: This video clip may contain potentially disturbing or difficult images of a person with anorexia nervosa.

Watch this news story about an Italian advertising campaign to raise public awareness of anorexia nervosa.

Watch It

Watch this video to learn more about anorexia nervosa. Pay close attention to the dangerous effects the illness can have on the body, including osteoporosis, brain complications, and heart symptoms.



You can view the transcript for "Anorexia nervosa – causes, symptoms, diagnosis, treatment & pathology" here (opens in new window).

Etiology

Estimations are between 0.3%-2% of young women and 0.1%-0.3 % of males will develop anorexia. Anorexia is a serious disease, especially given that people with anorexia aged 15 to 24 have 10 times the risk of dying compared to their similar age peers. Furthermore, the male individuals with anorexia are at even higher risk of dying due to late diagnosis as a result of sociocultural influences that males do not have eating disorders.[2]

Genetic

Anorexia nervosa is highly heritable. Twin studies have shown a heritability rate of between 28%-58%.[3] First-degree relatives of those with anorexia have roughly 12 times the risk of developing anorexia. Association studies have been performed, studying 128 different polymorphisms related to 43 genes including genes involved in the regulation of eating behavior, motivation and reward mechanics, personality traits, and emotion. Consistent associations have been identified for polymorphisms associated with agouti-related peptide, brain-derived neurotrophic factor, catechol-o-methyl transferase, SK3, and opioid receptor delta-1. Epigenetic modifications, such as DNA methylation, may contribute to the development or maintenance of anorexia nervosa, though clinical research in this area is in its infancy.

A 2019 study found a genetic relationship with mental disorders, such as schizophrenia, obsessive-compulsive disorder, anxiety disorder and depression; and metabolic functioning with a negative correlation with fat mass, type 2 diabetes and leptin.[4]

Environmental

Obstetric complications: prenatal and perinatal complications may factor into the development of anorexia nervosa, such as preterm birth, maternal anemia, diabetes mellitus, preeclampsia, placental infarction, and neonatal heart abnormalities. Neonatal complications may also have an influence on harm avoidance, one of the personality traits associated with the development of anorexia nervosa .

Neuroendocrine dysregulation: altered signaling of peptides that facilitate communication between the gut, brain, and adipose tissue, such as ghrelin, leptin, neuropeptide Y, and orexin, may contribute to the pathogenesis of anorexia nervosa by disrupting regulation of hunger and satiety.



Figure **8.4.2**: Those with celiac disease can't consume foods like bread because they contain gluten.

People with gastrointestinal disorders may be more at risk of developing disorders of eating practices than the general population, principally restrictive eating disturbances. An association of anorexia nervosa with celiac disease has been found. The role that gastrointestinal symptoms play in the development of eating disorders seems rather complex. Some authors report that unresolved symptoms prior to gastrointestinal disease diagnosis may create a food aversion in these persons, causing alterations to their eating patterns. Other authors report that greater symptoms throughout their diagnosis led to greater risk. Some people with celiac disease, irritable bowel syndrome, or inflammatory bowel disease who are not conscious about the importance of strictly following their diet choose to consume their trigger foods to promote weight loss. On the other hand, individuals with good dietary management may develop anxiety, food aversion, and eating disorders because of concerns around cross-contamination of their foods. Some authors suggest that medical professionals should evaluate the presence of unrecognized celiac disease in all people with an eating disorder, especially if they present any gastrointestinal symptom (such as decreased appetite, abdominal pain, bloating, distension, vomiting, diarrhea, or constipation), weight loss, or growth failure; and also routinely ask celiac patients about weight or body shape concerns, dieting or vomiting for weight control, to evaluate the possible presence of eating disorders, especially in women.

Studies have hypothesized the continuance of disordered eating patterns may be epiphenomena of starvation. The results of the Minnesota Starvation Experiment showed normal controls exhibit many of the behavioral patterns of anorexia nervosa when subjected to starvation. This may be due to the numerous changes in the neuroendocrine system, which results in a self-perpetuating cycle.

Anorexia nervosa is more likely to occur in a person's pubertal years. Some explanatory hypotheses for the rising prevalence of eating disorders in adolescence are "increase of adipose tissue in girls, hormonal changes of puberty, societal expectations of increased independence and autonomy that are particularly difficult for anorexic adolescents to meet; [and] increased influence of the peer group and its values."

Psychological

Early theories of the cause of anorexia linked it to childhood sexual abuse or dysfunctional families; the evidence is conflicting, and well-designed research is needed. The fear of food is known as sitiophobia, cibophobia, and is part of the differential diagnosis. Other psychological causes of anorexia include low self-esteem, feeling like there is a lack of control, depression, anxiety, and loneliness.

Sociological

Anorexia nervosa has been increasingly diagnosed since 1950; the increase has been linked to vulnerability and internalization of body ideals. People in professions where there is a particular social pressure to be thin (such as models and dancers) were more likely to develop anorexia, and those with anorexia have much higher contact with cultural sources that promote weight loss. Anorexia can also be observed for people who partake in certain sports, such as jockeys and wrestlers. There is a higher incidence and prevalence of anorexia nervosa in sports with an emphasis on aesthetics, where low body fat is advantageous, and sports in

which one has to make weight for competition. Family group dynamics can play a role in the cause of anorexia. When there is constant pressure from people to be thin, teasing, and bullying can cause low self-esteem and other psychological symptoms.

Media effects



Figure 8.4.3: Young women in our society are inundated with images of extremely thin models (sometimes accurately depicted and sometimes digitally altered to make them look even thinner). These images may contribute to eating disorders. (credit: Peter Duhon)

Persistent exposure to media that presents body ideals may constitute a risk factor for body dissatisfaction and anorexia nervosa. The cultural ideal for body shape for men versus women continues to favor slender women and athletic, V-shaped muscular men. A 2002 review found that, of the magazines most popular among people aged 18 to 24 years, those read by men, unlike those read by women, were more likely to feature ads and articles on shape than on diet. Body dissatisfaction and internalization of body ideals are risk factors for anorexia nervosa that threaten the health of both male and female populations.

Websites that stress the importance of attainment of body ideals extol and promote anorexia nervosa through the use of religious metaphors, lifestyle descriptions, and “thinspiration” or “fitspiration” (inspirational photo galleries and quotes that aim to serve as motivators for the attainment of body ideals). Pro-anorexia websites reinforce the internalization of body ideals and the importance of their attainment.

The media portray a false view of what people truly look like. In magazines and movies and even on billboards, most of the actors/models are digitally altered in multiple ways. People then strive to look like these “perfect” role models when in reality they are not near perfection themselves

While both anorexia and bulimia nervosa occur in men and women of many different cultures, Caucasian females from Western societies tend to be the most at-risk population. Recent research indicates that females between the ages of 15 and 19 are most at risk, and it has long been suspected that these eating disorders are culturally bound phenomena that are related to messages of a thin ideal often portrayed in popular media and the fashion world (Smink et al., 2012). While social factors play an important role in the development of eating disorders, there is also evidence that genetic factors may predispose people to these disorders (Collier & Treasure, 2004).

Treatment

Patients with anorexia nervosa require and benefit from a multidisciplinary approach to treatment, involving nutritional support, psychological counseling, and behavioral modification. The aggressiveness of treatment can be determined by their health stability including a patient’s weight and age. If an adult patient has lost 15% of their set point bodyweight it is recommended that they participate in inpatient treatment or a highly structured outpatient program. However, it is recommended children and adolescents to do inpatient in combination with family therapy before the 15% weight-loss threshold to avoid developmental risks and damage. [5]

The Maudsley method

Effective family-oriented therapy for people suffering from anorexia or other eating disorders is called the **Maudsley method**, which was developed at the Maudsley Hospital in London. The Maudsley method has three phases to help the person with anorexia recover and also guide the family through their recovery: weight restoration, return of control to the adolescent, and then establishing a healthy identity.

In phase 1, clinicians work with parents and siblings so that they learn strategies to coach and encourage patients to eat more—although the exact nutritional strategy is left up to the families. In phase 2, the therapy focus shifts from eating to identify and addressing changes in the family dynamics to promote and facilitate recovery. During phase 3, when the child has gained an appropriate amount of weight, clinicians work together with the patients and families to help the patient become more independent and improve relationships within the family.[6]

Nutritional therapy to promote weight gain

Patients with anorexia often suffer from severe malnutrition, which causes them to have a more negative, obsessive, and manipulative mindset. When a person with anorexia enters treatment, it is important to keep their mentality in perspective and coordinate a treatment plan that meets the patient at their current health status and be sympathetic, rather than punitive, in manner. A first step is to promote weight gain—often an estimated amount of two to three pounds per week during inpatient care and one pound per week in outpatient settings.

Medication options

Although medications are often prescribed for patients with anorexia nervosa, there is limited evidence to support their effectiveness—either at promoting weight gain or at alleviating psychological distress. There are two general types of medications prescribed: antidepressants and antipsychotics.

Psychotherapy to foster recovery and prevent relapse

Once patients gain enough weight to benefit from psychotherapy and behavioral interventions, the goal of treatment shifts from medical stabilization and gaining weight to learning how to recognize their distorted thinking about food and develop coping strategies to deal with their emotions and stress to avoid relapse. Estimations of relapse within one year of being discharged is approximately 50% of patients who recover in inpatient programs.[7]

Case Study

"Sarah's" parents, "Mr. and Mrs. Rachlin," were worried about their 15-year-old's health; over the past year, she had been on a diet that didn't seem to stop. Sarah had no history of being overweight and was always within a normal weight range with a healthy BMI. Her parents struggled to understand why she was on a diet because she had never been overweight, and Sarah did not seem happier on her diet. Sarah's parents also noticed changes in her social behaviors and Sarah began to express concerns about her body and her presentation. She showed signs of peer pressure and began to adapt her wardrobe to fit in with her new friends. Also, Sarah was taking a heavy schedule of honors classes and was on the lacrosse team. In addition to her eating habits changing, her parents also noticed an increase in her stress level and time spent exercising.

Sarah's dieting behaviors made her more withdrawn, depressed, and anxious. Every time her parents expressed their concerns, she burst into tears or shouted at them. She had stopped eating with the family and spent most of her time in her bedroom. She was still going to school and doing extremely well in her exams, but even her friends were worried about her. Sarah's parents had repeatedly suggested that she see the doctor but she insisted that there was nothing wrong with her, asking them why they couldn't just leave her alone. Mr. and Mrs. Rachlin had started to disagree about how to handle Sarah's behavior and argued frequently. As a result of this, Sarah's younger brother and sister were very angry with her and wouldn't talk to her.

Finally, Mr. and Mrs. Rachlin received a phone call from the school to say that Sarah had fainted and was in the hospital.

She was discharged a short while later but with the recommendation that they see an eating disorder specialist. Emma's parents took her to the GP who referred her to Priory's eating disorder service. She was immediately admitted to a Priory hospital as an

inpatient because her weight was dangerously low. Emma also had a very low heart rate (bradycardia) and a low temperature (hypothermia).

After a thorough assessment of not only her physical condition but also her mental health, Sarah had repeated blood tests and an electrocardiogram (ECG). Her blood work revealed anemia and other tests found bradycardia and hypothermia.[8]

Glossary

anorexia nervosa: an eating disorder characterized by an individual maintaining bodyweight that is well below average through starvation and/or excessive exercise

distorted body image: individuals view themselves as overweight even though they are not

Maudsley method: family therapy that occurs in three phases to help patients and their families in recovery from an eating disorder

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8.5: Bulimia Nervosa and Binge-Eating Disorder

Learning Objectives

- Describe the characteristics, complications, and health outcomes of bulimia nervosa
- Explain binge eating disorder

People suffering from **bulimia nervosa** engage in binge eating behavior that is followed by an attempt to compensate for a large amount of consumed food. Purging the food by inducing vomiting or through the use of laxatives are two common compensatory behaviors. Some affected individuals engage in excessive amounts of exercise to compensate for their binges.

People with bulimia nervosa may be slightly underweight, normal weight, or overweight.

Symptoms include the following:

- chronically inflamed and sore throat
- swollen salivary glands in the neck and jaw area
- worn tooth enamel and increasingly sensitive and decaying teeth as a result of exposure to stomach acid
- acid reflux disorder and other gastrointestinal problems
- intestinal distress and irritation from laxative abuse
- severe dehydration from purging of fluids
- electrolyte imbalance (too low or too high levels of sodium, calcium, potassium, and other minerals), which can lead to stroke or heart attack

Bulimia is associated with many adverse health consequences that can include kidney failure, heart failure, and tooth decay. In addition, these individuals often suffer from anxiety and depression, and they are at an increased risk for substance abuse (Mayo Clinic, 2012b).

The lifetime prevalence rate for bulimia nervosa is estimated at around 1% for women and less than 0.5% for men (Smink, van Hoeken, & Hoek, 2012). The rates of bulimia increased during the 1980s and early 1990s and have remained stable since then. Researchers are finding that eating disorders are caused by a complex interaction of genetic, biological, behavioral, psychological, and social factors.

Dig Deeper: Bulimia



Figure **8.5.1**: Princess Diana spoke openly about her struggles with bulimia.

An iconic fashion figure and celebrity, Princess Diana was a prominent public figure that was open about how bulimia affected her life. Read this article about how she struggled and how she attempted to use bulimia as a coping mechanism to deal with some of her life stressors.

Watch It

Watch this video to review the basics about anorexia and bulimia, and contrast those with the obsessive-compulsive related body dysmorphic disorder.

You can view the transcript for "Eating and Body Dysmorphic Disorders: Crash Course Psychology #33" here (opens in new window).

Binge-Eating Disorder

Binge-eating disorder (BED) is the most prevalent eating disorder, affecting approximately 2.8% of females and 1% of males.[1] Binge-eating disorder (BED) is characterized by recurrent episodes of binge eating that are not combined with compensatory methods to avoid weight gain. Thus, the majority of binge-eating disorder (BED) cases are overweight or obese. Unlike bulimia, BED binges are not followed by purging.[2]. Of those patients affected, research estimates that 28.4% of people with current BED are receiving treatment and 43.6% of people with BED will receive treatment at some point in their lives.[3]

Binge eating is the core symptom of BED; however, not everyone who binge eats has BED. An individual may occasionally binge eat without experiencing many of the negative physical, psychological, or social effects of BED. This example may be considered an eating problem (or not), rather than a disorder. Precisely defining binge eating can be problematic; however, binge eating episodes in BED are generally described as having the following potential features:

- eating much faster than normal, perhaps in a short space of time
- eating until feeling uncomfortably full
- eating a large amount when not hungry
- subjective loss of control over how much or what is eaten
- binges may be planned in advance, involving the purchase of special binge foods, and the allocation of a specific time for binging, sometimes at night
- eating alone or secretly due to embarrassment over the amount of food consumed
- there may be a dazed mental state during the binge
- not being able to remember what was eaten after the binge
- feelings of guilt, shame, or disgust following a food binge



Figure **8.5.2**: Binge-eating is characterized by over-consuming large amounts of food in a short amount of time. Unlike bulimia, it is not followed by purging, but it does often result in feelings of shame or frustration.

In contrast to bulimia nervosa, binge-eating episodes are not regularly followed by activities intended to prevent weight gain, such as self-induced vomiting, laxative or enema misuse, or strenuous exercise. BED is characterized more by overeating than dietary restriction and over concern about body shape. Obesity is common in persons with BED, as are depressive features, low self-esteem, stress, and boredom.

Treatments for Bulimia and Binge-Eating Disorder

There are several supported psychosocial treatments for bulimia. CBT, which involves teaching a person to challenge automatic thoughts and engage in behavioral experiments (for example, in session eating of "forbidden foods"), has a small amount of evidence supporting its use.

Seeking treatment early for eating disorders is essential as people with eating disorders are at higher risk for suicide and medical complications. People with eating disorders can often have other mental disorders (such as depression or anxiety) or problems with

substance use. Complete recovery is possible. Treatment plans are tailored to individual needs and may include one or more of the following:

- individual, group, and/or family psychotherapy
- medical care and monitoring
- nutritional counseling
- medications

Psychotherapies



Figure **8.5.3**: Keeping track of what you eat and how you feel throughout the day can be helpful and insightful.

Psychotherapies such as a family-based therapy called the Maudsley approach, where parents of adolescents with anorexia nervosa assume responsibility for feeding their child, appear to be very effective in helping people gain weight and improve eating habits and moods. This and other types of family-based treatments (FBT) are particularly helpful for younger adolescents to gradually take more control of their eating habits after learning more about the consequences of their eating choices.

To reduce or eliminate binge-eating and purging behaviors, people may undergo CBT, which is another type of psychotherapy that helps a person learn how to identify distorted or unhelpful thinking patterns and recognize and change inaccurate beliefs. By using CBT, people record how much food they eat and periods of vomiting with the purpose of identifying and avoiding emotional fluctuations that bring on episodes of bulimia on a regular basis. People undergoing CBT who exhibit early behavioral changes are most likely to achieve the best treatment outcomes in the long run. Researchers have also reported some positive outcomes for interpersonal psychotherapy and dialectical behavior therapy.

Medications

Antidepressants of the selective serotonin reuptake inhibitors (SSRI) class may have a modest benefit in treating bulimia. This includes fluoxetine, which is FDA approved, for the treatment of bulimia. Other antidepressants such as sertraline may also be effective against bulimia. Topiramate may also be useful, but has greater side effects. Compared to placebo, the use of a single antidepressant has been shown to be effective.

Combining medication with counseling can improve outcomes in some circumstances. Some positive outcomes of treatments can include abstinence from binge eating, a decrease in obsessive behaviors to lose weight and in-shape preoccupation, less severe psychiatric symptoms, and a desire to counter the effects of binge eating as well as an improvement in social functioning and reduced relapse rates.

? Case Study: Reeya

Reeya is a young Indian woman in her early 20s born in Britain. She was diagnosed with bulimia nervosa in her late teens and later came into contact with an urban eating disorder specialist in the hospital as an outpatient.

Reeya's mother passed away when she was a toddler. Thereafter, she moved in with her grandparents. Her father remarried a short time later and she moved back in with her father and step-mother. She did not get along with her step-mother. She described her relationship with her parents (father and step-mother) as "hostile" and recounted pervasive family pressure in terms of high expectations around education, marriage, and career.

Reeya attended an all-girls grammar school and described it as being quite competitive. She could not recall having had any difficulties with body image or eating prior to the age of 16. When she was in year 12, she recalled feeling quite large and began to weigh herself. At that time, Reeya and her friend began a Weightwatchers diet plan to lose weight for a big ball at the

school. Reeya went from being 55 kg (121 lbs) to 47 kg (103 lbs) and felt a sense of happiness, pride, and achievement, even though her BMI measured slightly underweight. She regained the weight when going to college.

Reeya went to university at age 18 and described her living circumstances there as stressful and lonely. She pursued a course that was dominated by young women and she did not get along with her flatmates. She recounted her first year of university as being the hardest year of her life. Reeya discussed feeling quite self-conscious about her appearance when she started university. She described developing an increasing pre-occupation with weight, concerns over body image, daily intake restriction, and then overeating/binges in the evenings, followed by occasional purging.

Reeya sought help through her supportive boyfriend who accompanied her to the doctor as a first point of access.[4]

Watch It

This video provides an overview of binge-eating disorder.



You can view the transcript for "Understanding Binge Eating Disorder" here (opens in new window).

Glossary

binge eating disorder: type of eating disorder characterized by binge eating and associated distress

bulimia nervosa: type of eating disorder characterized by binge eating followed by purging

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8.6: Avoidant/Restrictive Food Intake Disorder and Rumination Disorder

Learning Objectives

- Describe the characteristics, complications, and health outcomes of avoidant/restrictive food intake disorder (ARFID)
- Describe rumination disorder

Avoidant/Restrictive Food Intake Disorder (ARFID)

Avoidant/restrictive food intake disorder (ARFID) is an eating or feeding disturbance associated with an apparent lack of interest in eating or food. Previously defined as a disorder exclusive to children and adolescents, the DSM-5 broadened the disorder to include adults who limit their eating and are affected by related physiological or psychological problems, but who do not fall under the definition of another eating disorder.



Figure **8.6.1**: Someone with ARFID may avoid eggplant altogether because of its color.

People with avoidant/restrictive food intake disorder (ARFID) have an inability to eat certain foods. “Safe” foods may be limited to certain food types and even specific brands. In some cases, individuals with the condition will exclude whole food groups, such as fruits or vegetables. Sometimes excluded foods can be refused based on color. Some may only like very hot or very cold foods, very crunchy or hard-to-chew foods or very soft foods, or avoid sauces.

Most people with avoidant/restrictive food intake disorder (ARFID) will still maintain a healthy or typical body weight. There are no specific outward appearances associated with ARFID. Sufferers can experience physical gastrointestinal reactions to adverse foods such as retching, vomiting, or gagging. Some studies have identified symptoms of social avoidance due to their eating habits. Most, however, would change their eating habits if they could.

In a research study of children and adolescents who were receiving treatments for an eating disorder at a clinic, 14% of the patients met the criteria for AFRID and were more likely to be younger and male.[1]

Diagnosis

The DSM-5 defines the following diagnostic criteria for avoidant/restrictive food intake disorder (ARFID):

- disturbance in eating or feeding, as evidenced by one or more of the following:
  - substantial weight loss (or, in children, absence of expected weight gain)
  - nutritional deficiency
  - dependence on a feeding tube or dietary supplements
  - significant psychosocial interference
- disturbance not due to unavailability of food or to observation of cultural norms
- disturbance not due to anorexia nervosa or bulimia nervosa, and no evidence of disturbance in body shape or weight
- disturbance not better explained by another medical condition or mental disorder, or when occurring concurrently with another condition, the disturbance exceeds what is normally caused by that condition

Associated Conditions

The determination of the cause of ARFID has been difficult due to the lack of diagnostic criteria and concrete definition. However, many have proposed other conditions that co-occur with ARFID.

There are different kinds of sub-categories identified for ARFID:

- sensory-based avoidance, where the individual refuses food intake based on smell, texture, color, brand, presentation
- a lack of interest in consuming the food, or tolerating it nearby
- food associated with fear-evoking stimuli that have developed through a learned history
- anorexia and bulimia co-occurring in individuals suffering from ARFID

Autism

Symptoms of ARFID are usually found with symptoms of other disorders or with neurodivergence. Some form of feeding disorder is found in 80% of children that also have a developmental disability. Children often exhibit symptoms of obsessive-compulsive disorder and autism. Although many people with ARFID have symptoms of these disorders, they usually do not qualify for a full diagnosis. Strict behavior patterns and difficulty adjusting to new things are common symptoms in patients that are on the autistic spectrum.

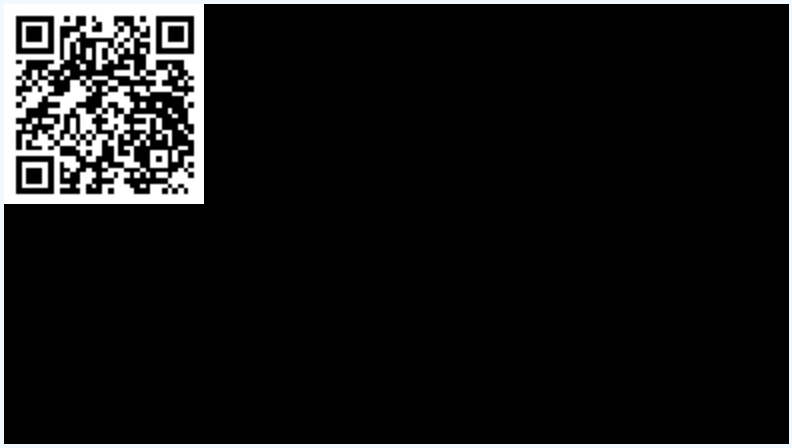
A study done by Schreck at Pennsylvania State University compared the eating habits of children with autism spectrum disorder (ASD) and typically developing children. After analyzing their eating patterns, they suggested that the children with some degree of ASD have a higher degree of selective eating. These children were found to have similar patterns of selective eating and favored more energy-dense foods, such as nuts and whole grains. Eating a diet of energy-dense foods could put these children at a greater risk for health problems such as obesity and other chronic diseases due to the high fat and low fiber content of energy-dense foods. Due to the tie to ASD, children are less likely to outgrow their selective eating behaviors and most likely should meet with a clinician to address their eating issues.

Anxiety Disorder

Specific food avoidances could be caused by food phobias that cause great anxiety when a person is presented with new or feared foods. Most eating disorders are related to a fear of gaining weight. Those who have ARFID do not have this fear, but the psychological symptoms and anxiety created are similar. Nearly half of the children with an AFRID diagnosis report emetophobia (i.e., fear of vomiting),[2] and others may report a fear of choking.

Watch It

Watch this video to better understand how the experience of ARFID is more than just picky eating.



You can view the transcript for “Avoidant/restrictive food intake disorder (ARFID) Signs & Symptoms” here (opens in new window).



Figure **8.6.2**: ARFID often involves certain foods due to sensory concerns, and a common treatment method includes systematic desensitization and gradual exposure to the types of foods that have been avoided. It is also helpful to identify concerns and review the progress.

For Adults

With time, the symptoms of ARFID can lessen and can eventually disappear without treatment. However, in some cases treatment will be needed as the symptoms persist into adulthood. The most common type of treatment for ARFID is some form of CBT. Working with a clinician can help to change behaviors more quickly than symptoms may typically disappear without treatment.

For Children

Children can benefit from a four-stage, in-home treatment program based on the principles of systematic desensitization. The four stages of the treatment are record, reward, relax, and review.[3]

- In the **record stage**, children are encouraged to keep a log of their typical eating behaviors without attempting to change their habits as well as their cognitive feelings.
- The **reward stage** involves systematic desensitization. Children create a list of foods that they might like to try eating someday. These foods may not be drastically different from their normal diet, but perhaps a familiar food prepared in a different way. Because the goal is for the children to try new foods, children are rewarded when they sample new foods.
- The **relaxation stage** is most important for those children that suffer severe anxiety when presented with unfavorable foods. Children learn to relax to reduce the anxiety that they feel. Children work through a list of anxiety-producing stimuli and can create a storyline with relaxing imagery and scenarios. Often these stories can also include the introduction of new foods with the help of a real person or fantasy person. Children then listen to this story before eating new foods as a way to imagine themselves participating in an expanded variety of foods while relaxed.
- The final stage, **review**, is important to keep track of the child's progress. It is important to include both one-on-one sessions with the child, as well as with the parent in order to get a clear picture of how the child is progressing and if the relaxation techniques are working.

? Rumination Disorder

Rumination is an eating disorder characterized by having the contents of the stomach regurgitated (drawn back up into the mouth), and either re-chewed, re-swallowed, or spit out. RD must take place repeatedly for at least a month and not be attributed to other gastrointestinal or medical issues.

Signs and symptoms of rumination disorder include the backward flow of recently eaten food from the stomach to the mouth. RD typically occurs immediately to 15 to 30 minutes after eating. Rumination often occurs without retching or gagging. Rumination may be proceeded by a feeling of pressure, the need to belch, nausea, or discomfort. Some people with rumination disorder experience bloating, heartburn, diarrhea, constipation, abdominal pain, headaches, dizziness, or sleeping difficulties. Complications of the disorder include weight loss, malnutrition, and electrolyte imbalance.

Causes

Rumination disorder may occur following a viral illness, emotional stress, or physical injury. RD is theorized that while the initial stressor improves, an altered sensation in the abdomen persists. RD ultimately results in the relaxation of the muscle at the bottom of the esophagus. To relieve this discomfort, people with rumination disorder use abdominal wall muscles to expel and regurgitate foods. As a result of the relief of symptoms, the person repeats the same response when the discomfort returns. Overtime, the person unconsciously adopts this learned behavior.

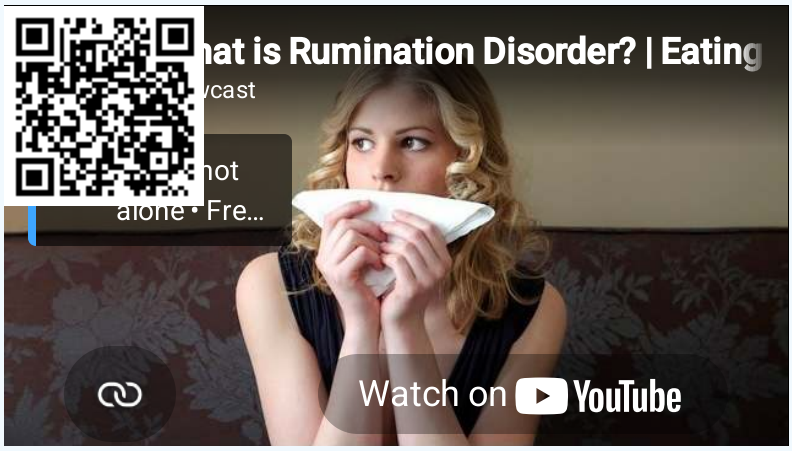
Some cases of rumination disorder occur without a precipitating event or illness. Other people with the disorder describe also having ingestion, which may serve as a trigger. Studies have shown that some people with rumination disorder also have depression, anxiety, or an eating disorder. These conditions may likewise play a role in rumination disorder. Conditions like depression and anxiety are known to be more common in people with other functional gastrointestinal conditions as well, for example, irritable bowel syndrome.

Treatment

The main treatment of rumination disorder is behavioral therapy. Treatment may involve habitat reversal strategies, relaxation, diaphragmatic breathing, and biofeedback. These types of therapies can often be administered by a gastroenterologist. Other professionals, such as nurse practitioners, psychologists, massage therapists, and recreational therapists may also be involved in care. Ensuring adequate nutrition is essential and treatment will also involve managing other symptoms, such as anxiety, nausea, and stomach discomfort (which may involve anti-depressive agents or SSRIs).

Watch It

Watch this video to learn about the rumination eating disorder and how it is related to bulimia and other binge-related eating disorders.



You can view the transcript for “What is Rumination Disorder? | Eating Disorders” here (opens in new window).

Glossary

avoidant/restrictive food intake disorder: apparent lack of interest in eating or food

rumination disorder: individuals with rumination disorder regurgitate the contents of their stomach and then either re-chew, re-swallow, or spit out the vomit

emetophobia: fear of vomiting

1. Statistics & Research on Eating Disorders. National Eating Disorders Association. (2020, May 8). https://www.nationaleatingdisorders.org/statistics-research-eating-disorders. ↵
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8.7: Pica

Learning Objectives

- Describe the characteristics, health outcomes, and treatment options for pica

Pica

Pica is a psychological disorder characterized by an appetite for substances that are largely non-nutritive, such as ice, soap, hair, paper, metal, soil, stones, glass, or chalk. The term pica originates in the Latin word for magpie (picave), a bird that is famed for its unusual eating behaviors, where it is known to eat almost anything. Here are types of cravings someone with pica may have:

- acuphagia (sharp objects)
- amylophagia (starch)
- cautopyreiophagia (burnt matches)
- coniophagia (dust)
- coprophagia (feces)
- emetophagia (vomit)
- geomelophagia (raw potatoes)
- geophagia (earth, soil, or clay)
- hyalophagia (glass)
- lithophagia (stones)
- mucophagia (mucus)
- pagophagia (ice)
- plumbophagia (lead)
- trichophagia (hair, wool, and other fibers)
- urophagia (urine)
- hematophagia (vampirism) (blood)
- xylophagia (wood or derivates of wood such as paper)



Figure **8.7.1**: Consuming non-nutritional substances like rocks can lead to surgical emergencies.

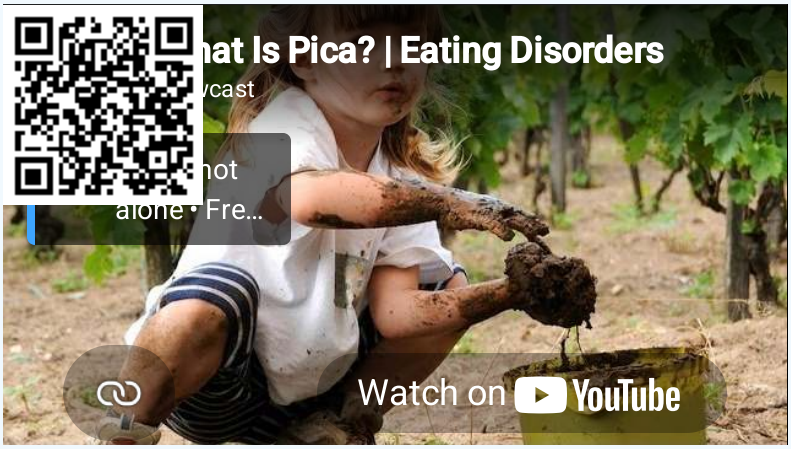
According to the DSM-5 criteria, for these actions to be considered pica, they must persist for more than one month at an age when eating such objects is considered developmentally inappropriate, not part of a culturally sanctioned practice, and sufficiently severe to warrant clinical attention. Pica may lead to intoxication in children, which can result in an impairment of both physical and mental development. In addition, it may cause surgical emergencies to address intestinal obstructions, as well as more subtle symptoms such as nutritional deficiencies and parasitosis. Pica has been linked to other mental and emotional disorders. Stressors such as emotional trauma, maternal deprivation, family issues, parental neglect, pregnancy, and a disorganized family structure[failed verification] are strongly linked to pica as a form of comfort.

Pica is most commonly seen in pregnant women, small children, iron- and zinc-deficient children, malnourished children, and people with intellectual disabilities.[1]

Children eating painted plaster containing lead may suffer brain damage from lead poisoning. A similar risk exists from eating soil near roads that existed before the phase-out of tetraethyllead or that were sprayed with oil (to settle dust) contaminated by toxic PCBs or dioxin. In addition to poisoning, a much greater risk exists of gastrointestinal obstruction or tearing in the stomach. Another risk of eating soil is the ingestion of animal feces and accompanying parasites. Pica can also be found in animals such as dogs and cats.

Watch It

Watch this video to review the basic symptoms and characteristics of pica, potential causes and risk factors, and treatment basics in children and adults.



You can view the transcript for "What Is Pica? | Eating Disorders" here (opens in new window).

Causes

According to the DSM-5, mineral deficiencies are occasionally associated with pica, but biological abnormalities are rarely found. People practicing forms of pica, such as geophagy, pagophagy, and amylophagy, (soil, ice, or starches) are more likely anemic, have low hemoglobin concentration in their blood, lower levels of red blood cells (hematocrit), or have lower plasma zinc levels. Specifically, practicing geophagy is more likely to be associated with anemia or low hemoglobin. Practicing pagophagy and amylophagy is more highly associated with anemia. Additionally, children and pregnant women may be more likely to have anemia or low hemoglobin relative to the general population. More recently, cases of pica have been tied to the obsessive-compulsive spectrum, and a move has arisen to consider OCD as the cause of pica.

If nutritional deficits and lead exposure are ruled out, then pica is likely brought on by emotional distress. In these instances, the child may also display abnormal behaviors such as using strange language, engaging in strange play, or having relationship issues with other peers.[2]

Diagnosis

No single test confirms pica, but because pica can occur in people who have lower than normal nutrient levels and poor nutrition (malnutrition), the health care provider should test blood levels of iron and zinc. Hemoglobin can also be checked to test for anemia. Lead levels should always be checked in children who may have eaten paint or objects covered in lead-paint dust. The healthcare provider should test for infection if the patient has been eating contaminated soil or animal waste.

The DSM-5 posits four criteria that must be met for a person to be diagnosed with pica:[3]

- A person must have been eating non-nutritive nonfoods for at least one month.
- This eating must be considered abnormal for the person's stage of development.
- Eating these substances cannot be associated with a cultural practice that is considered normal in the social context of the individual.
- For people who currently have a medical condition (e.g., pregnancy) or a mental disorder (e.g., autism spectrum disorder), the action of eating non-nutritive nonfoods should only be considered pica if it is dangerous and requires extra medical

investigation or treatment on top of what they are already receiving for their pre-existing condition.

Epidemiology

The prevalence of pica is difficult to establish because of differences in definition and the reluctance of patients to admit to abnormal cravings and ingestion, thus leading to the prevalence recordings of pica among at-risk groups being in the range of 8%-65% depending on the study.

Based on compiled self-report and interview data of pregnant and postpartum women, pica is most prevalent geographically in Africa, with an estimated prevalence of 44.8%, followed by North and South America (23.0%) and Eurasia (17.5%).[4] Factors associated with pica in this population were determined to be anemia and low levels of education, both of which are associated with low socioeconomic backgrounds. Two studies of adults with intellectual disability living in institutions found that 21.8% and 25.8% of these groups suffered from pica.

Prevalence rates for children are unknown. Young children commonly place non-nutritious material into their mouths. This activity occurs in 75% of 12-month-old infants, and 15% of two- to three-year-old children. In institutionalized children with mental retardation, pica occurs in ten to 33% of the population.

Treatment

Treatment for pica may vary by patient and suspected cause (e.g., child, developmentally disabled, pregnant, or psychogenic) and may emphasize psychosocial, environmental, and family-guidance approaches; iron deficiency may be treatable through iron supplement by dietary changes. An initial approach often involves screening for, and if necessary, treating any mineral deficiencies or other comorbid conditions. For pica that appears to be of psychogenic cause, therapy and medication such as SSRIs have been used successfully. However, previous reports have cautioned against the use of medication until all non-psychogenic causes have been ruled out.

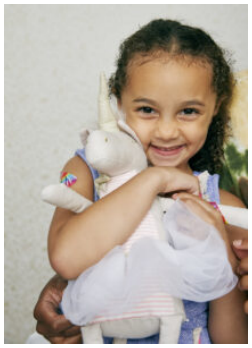


Figure **8.7.2**: A favorite stuffed animal or toy can be used as a reward to reinforce healthy eating behaviors.

A clinician will look at likely causes of pica to help determine the best treatment method. For example, if pica is a result of social inattention, a treatment strategy could involve ignoring the pica and giving praise and attention for other desirable behaviors. If pica is a way for a person to escape an activity or situation, the reason why the person wants to escape the activity should be examined and the person should be moved to a new situation. If pica is motivated by sensory feedback, an alternative method of feeling that sensation should be provided. Other nonmedication techniques might include other ways for oral stimulation such as chewing gum or eating popcorn.

Behavior-based treatment options can be useful for developmentally disabled and mentally ill individuals with pica. Behavioral treatments for pica have been shown to reduce pica severity by 80% in people with intellectual disabilities.[5] These treatments may involve using positive reinforcement for healthy eating behavior. Some methods use aversion therapy where the patient learns through positive reinforcement which foods are good and which ones they should not eat. Often, treatment is similar to the treatment of obsessive-compulsive or addictive disorders (such as exposure therapy). Success with treatment is high and generally fades with age, but it varies depending on the cause of the disorder. Developmental causes tend to have a lower success rate.

Treatment techniques include the following:

- presentation of attention, food, or toys, not contingent on pica being attempted
- differential reinforcement with positive reinforcement if pica is not attempted and consequences if pica is attempted

- discrimination training between edible and inedible items with negative consequences if pica is attempted
- visual screening with eyes covered for a short time after pica is attempted
- aversive presentation contingent on pica being attempted:
  - oral taste (e.g., lemon)
  - smell sensation (e.g., ammonia)
  - physical sensation (e.g., water mist in face)
- physical restraint
  - self-protection devices that prohibit placement of objects in the mouth
  - brief restraint contingent on pica being attempted
  - time-out contingent on pica being attempted
  - overcorrection with attempted pica resulting in required washing of self, disposal of nonedible objects, and chore-based punishment
  - negative practice (inedible object held against patient's mouth without allowing ingestion)

Case Study: Skyler

Skyler has had a history of chronic abdominal pain since the age of three. Now age seven, she was checked into the ER for pain. In the past, she has eaten non-food items like brick pieces, dirt, and paint, despite her parent's persistent attempts to get her to stop. At the ER, her physical examination was normal; however, on her oral examination, several teeth showed damage and severe attrition.

The patient was investigated for serum iron, serum zinc, and hemoglobin and went through a stool and urine test. There was a significant reduction in serum ferritin, calcium, and zinc levels, indicating iron and zinc deficiency.

Treatment

The dental treatment planned was a restoration of several teeth and the extraction of one. Skyler was put on iron, zinc, and calcium supplements alongside a deworming treatment.

Skyler was called every month for a follow-up. During the second month, the mother reported a reduction in consumption of brick pieces and the habit finally stopped after six months. The patient is currently on medications and the eating behaviors have not resurfaced.[6]

Glossary

pica: eating disorder that describes the consumption of non-nutritive substances

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6. Advani, S., Kochhar, G., Chachra, S., & Dhawan, P. (2014). Eating everything except food (PICA): A rare case report and review. Journal of International Society of Preventive & Community Dentistry, 4(1), 1–4. https://doi.org/10.4103/2231-0762.127851↵

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8.8: Introduction to Sleep-Wake Disorders

What you'll learn to do: distinguish between normal and abnormal sleep and examine the characteristics, epidemiology, and etiology of sleep-wake disorders



Many people experience disturbances in their sleep at some point in their lives. Depending on the population and sleep disorder being studied, between 30% and 50% of the population suffers from a sleep disorder at some point in their lives (Bixler, Kales, Soldatos, Kaels, & Healey, 1979; Hossain & Shapiro, 2002; Ohayon, 1997, 2002; Ohayon & Roth, 2002). This section will describe sleep, why we sleep, sleep disorders, and sleep disorder treatment.

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8.9: Understanding Sleep

Learning Objectives

- Describe sleep, the sleep-wake cycle, and the stages of sleep

We spend approximately one-third of our lives sleeping. Given that the average life expectancy for U.S. citizens falls between 73 and 79 years old (Singh & Siahpush, 2006), we can expect to spend approximately 25 years of our lives sleeping. Some animals never sleep (e.g., several fish and amphibian species); other animals can go extended periods of time without sleep and without apparent negative consequences (e.g., dolphins); yet some animals (e.g., rats) die after two weeks of sleep deprivation (Siegel, 2008). Why do we devote so much time to sleeping? Is it absolutely essential that we sleep? This section will consider these questions and explore various explanations for why we sleep.

What Is Sleep?

Sleep is distinguished by low levels of physical activity and reduced sensory awareness. As discussed by Siegel (2008), a definition of sleep must also include mention of the interplay of the circadian and homeostatic mechanisms that regulate sleep. Homeostatic regulation of sleep is evidenced by sleep rebound following sleep deprivation. Sleep rebound refers to the fact that a sleep-deprived individual will tend to take longer falling asleep during subsequent opportunities for sleep. Sleep is characterized by certain patterns of activity of the brain that can be visualized using electroencephalography (EEG), and different phases of sleep can be differentiated using EEG as well (Figure 1).

Sleep comprises several different stages that can be differentiated from one another by the patterns of brain wave activity that occur during each stage. These changes in brain wave activity can be visualized using electroencephalography (EEG) and are distinguished from one another by both the frequency and amplitude of brain waves. Sleep can be divided into two different general phases: rapid eye movement (REM) sleep and non-REM (NREM) sleep. Rapid eye movement (REM) sleep is characterized by darting movements of the eyes under closed eyelids. Brain waves during rapid eye movement (REM) sleep appear very similar to brain waves during wakefulness. In contrast, non-REM (NREM) sleep is subdivided into three stages distinguished from each other and from wakefulness by characteristic patterns of brain waves. The first three stages of sleep are non-REM (NREM) sleep, while the fourth and final stage of sleep is REM sleep. In this section, we will discuss each of these stages of sleep and their associated patterns of brain wave activity.

NREM Stages of Sleep

The first stage of NREM sleep is known as stage one sleep. Stage one sleep is a transitional phase that occurs between wakefulness and sleep, the period during which we drift off to sleep. During this time, there is a slowdown in both the rates of respiration and heartbeat. In addition, stage one sleep involves a marked decrease in both overall muscle tension and core body temperature.

In terms of brain wave activity, stage one sleep is associated with both alpha and theta waves. The early portion of stage one sleep produces alpha waves, which are relatively low frequency (8–13Hz), and high amplitude patterns of electrical activity (waves) that become synchronized. As we move into stage two sleep, the body goes into a state of deep relaxation. Theta waves still dominate the activity of the brain, but they are interrupted by brief bursts of activity known as sleep spindles. Stage three of sleep is often referred to as deep sleep or slow-wave sleep because these stages are characterized by low frequency (up to 4 Hz), high amplitude delta waves. During this time, an individual's heart rate and respiration slow dramatically.

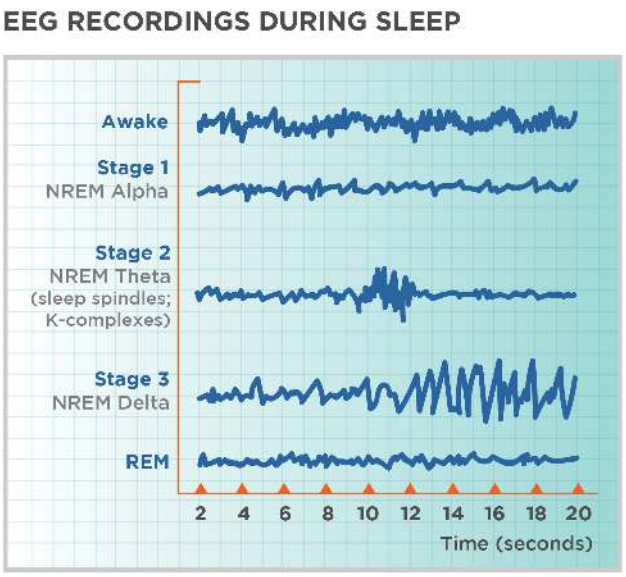


Figure 8.9.1: Brainwave activity changes dramatically across the different stages of sleep.

REM Sleep

As mentioned earlier, REM sleep is marked by rapid movements of the eyes. The brain waves associated with this stage of sleep are very similar to those observed when a person is awake, as shown in Figure 5, and this is the period of sleep in which dreaming occurs. It is also associated with paralysis of muscle systems in the body with the exception of those that make circulation and respiration possible. Therefore, no movement of voluntary muscles occurs during REM sleep in a normal individual; REM sleep is often referred to as paradoxical sleep because of this combination of high brain activity and lack of muscle tone. Like NREM sleep, REM has been implicated in various aspects of learning and memory (Wagner, Gais, & Born, 2001), although there is disagreement within the scientific community about how important both NREM and REM sleep are for normal learning and memory (Siegel, 2001).

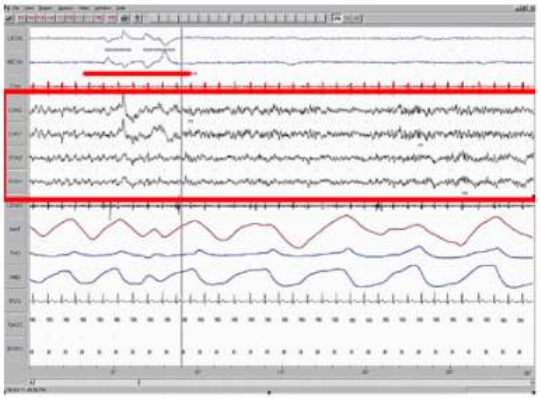


Figure 8.9.2: A period of REM is marked by the short red line segment. The brain waves associated with REM sleep, outlined in the red box, look very similar to those seen during wakefulness.

If people are deprived of REM sleep and then allowed to sleep without disturbance, they will spend more time in REM sleep in what would appear to be an effort to recoup the lost time in REM. This is known as the REM rebound, and it suggests that REM sleep is also homeostatically regulated. Aside from the role that REM sleep may play in processes related to learning and memory, REM sleep may also be involved in emotional processing and regulation. In such instances, REM rebound may actually represent an adaptive response to stress in non-depressed individuals by suppressing the emotional salience of aversive events that occurred in wakefulness (Suchecki, Tiba, & Machado, 2012).

While sleep deprivation in general is associated with a number of negative consequences (Brown, 2012), the consequences of REM deprivation appear to be less profound (as discussed in Siegel, 2001). In fact, some have suggested that REM deprivation can actually be beneficial in some circumstances. For instance, REM sleep deprivation has been demonstrated to improve symptoms of people suffering from major depression, and many effective antidepressant medications suppress REM sleep (Riemann, Berger, & Volderholzer, 2001; Vogel, 1975). The hypnogram below shows a person's passage through the stages of sleep.

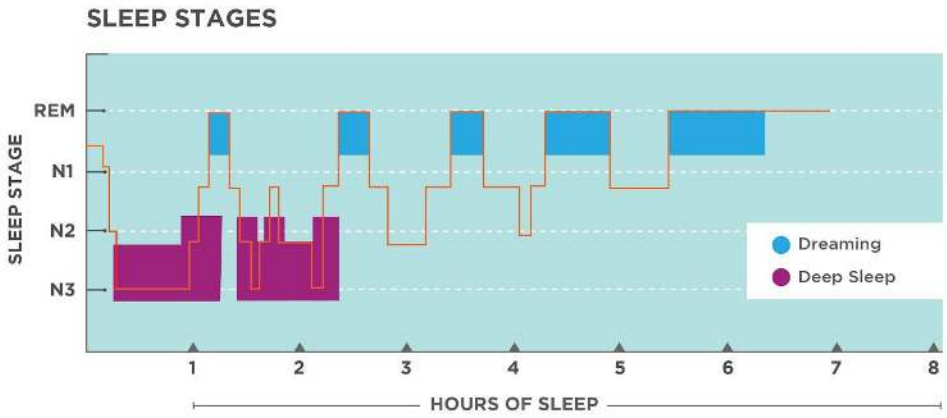


Figure **8.9.3**: This hypnogram illustrates how an individual moves through the various stages of sleep. Deeper NREM sleep occurs early on in the night, while the duration of REM sleep increases as the night progresses.

Sleep and Hormones

Sleep-wake cycles seem to be controlled by multiple brain areas acting in conjunction with one another. Some of these areas include the thalamus, the hypothalamus, and the pons. As already mentioned, the hypothalamus contains the SCN—the biological clock of the body—in addition to other nuclei that, in conjunction with the thalamus, regulate slow-wave sleep. The pons is important for regulating REM sleep (National Institutes of Health, n.d.).

Sleep is also associated with the secretion and regulation of a number of hormones from several endocrine glands, including melatonin, follicle stimulating hormone (FSH), luteinizing hormone (LH), and growth hormone (National Institutes of Health, n.d.). You have read that the pineal gland releases melatonin during sleep (Figure 2). Melatonin is thought to be involved in the regulation of various biological rhythms and the immune system (Hardeland et al., 2006). During sleep, the pituitary gland secretes both FSH and LH, which are important in regulating the reproductive system (Christensen et al., 2012; Sofikitis et al., 2008). The pituitary gland also secretes growth hormone during sleep, which plays a role in physical growth and maturation as well as other metabolic processes (Bartke, Sun, & Longo, 2013).

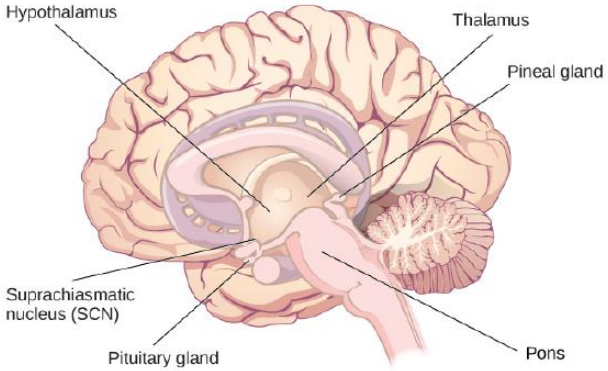


Figure **8.9.4**: The pineal and pituitary glands secrete a number of hormones during sleep.

Why Do We Sleep?

Given the central role that sleep plays in our lives and the number of adverse consequences that have been associated with sleep deprivation, one would think that we would have a clear understanding of why it is that we sleep. Unfortunately, this is not the

case; however, several hypotheses have been proposed to explain the function of sleep. It is quite possible that sleep serves no single, universally adaptive function, and different species have evolved different patterns of sleep in response to their unique evolutionary pressures. The benefits of sleep listed include maintaining a healthy weight, lowering stress levels, improving mood, and increasing motor coordination as well as a number of benefits related to cognition and memory formation (the National Sleep Foundation (n.d.).

Adaptive Function of Sleep

One popular hypothesis of sleep incorporates the perspective of evolutionary psychology. Evolutionary psychology is a discipline that studies how universal patterns of behavior and cognitive processes have evolved over time as a result of natural selection. Variations and adaptations in cognition and behavior make individuals more or less successful in reproducing and passing their genes to their offspring. One hypothesis from this perspective might argue that sleep is essential to restore resources that are expended during the day. While this is an intuitive explanation of sleep, there is little research that supports this explanation.

Another evolutionary hypothesis of sleep holds that our sleep patterns evolved as an adaptive response to predatory risks, which increase in darkness. Thus we sleep in safe areas to reduce the chance of harm. Again, this is an intuitive and appealing explanation for why we sleep. Comparative research indicates, however, that the relationship that exists between predatory risk and sleep is very complex and equivocal.

Cognitive Function of Sleep

Another theory regarding why we sleep involves sleep's importance for cognitive function and memory formation (Rattenborg, Lesku, Martinez-Gonzalez, & Lima, 2007). Indeed, we know sleep deprivation results in disruptions in cognition and memory deficits (Brown, 2012), leading to impairments in our abilities to maintain attention, make decisions, and recall long-term memories. Moreover, these impairments become more severe as the amount of sleep deprivation increases (Alhola & Polo-Kantola, 2007). Furthermore, slow-wave sleep after learning a new task can improve resultant performance on that task (Huber, Ghilardi, Massimini, & Tononi, 2004) and seems essential for effective memory formation (Stickgold, 2005).

Sleep has also been associated with other cognitive benefits. Research indicates that included among these possible benefits are increased capacities for creative thinking (Cai, Mednick, Harrison, Kanady, & Mednick, 2009; Wagner, Gais, Haider, Verleger, & Born, 2004), language learning (Fenn, Nusbaum, & Margoliash, 2003; Gómez, Bootzin, & Nadel, 2006), and inferential judgments (Ellenbogen, Hu, Payne, Titone, & Walker, 2007).

Glossary

alpha wave: type of relatively low frequency, relatively high amplitude brain wave that becomes synchronized; characteristic of the beginning of stage one sleep

delta wave: type of low frequency, high amplitude brain wave characteristic of stage three sleep

non-REM (NREM): period of sleep outside periods of REM sleep

rapid eye movement (REM) sleep: period of sleep characterized by brain waves very similar to those during wakefulness and by darting movements of the eyes under closed eyelids

stage one sleep: first stage of sleep; transitional phase that occurs between wakefulness and sleep; the period during which a person drifts off to sleep

stage two sleep: second stage of sleep; the body goes into deep relaxation; characterized by the appearance of sleep spindles

stage three sleep: third stage of sleep; deep sleep characterized by low frequency, high amplitude delta waves

sleep rebound: sleep-deprived individuals will experience longer sleep latencies during subsequent opportunities for sleep

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8.10: Insomnia Disorder

Learning Objectives

- Describe symptoms and health consequences of insomnia



Figure **8.10.1**: Insomnia is often accompanied by feelings of anxiety and frustration.

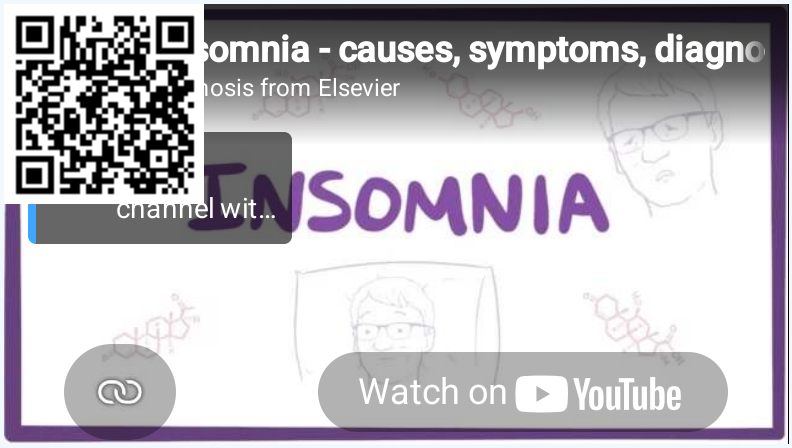
Insomnia

A person with **insomnia** has a consistent difficulty in falling or staying asleep and is the most common sleep disorders. Individuals with insomnia often experience long delays between the times that they go to bed and actually fall asleep. In addition, those with insomnia may wake up several times during the night only to find that they have difficulty getting back to sleep. As mentioned earlier, one of the criteria for insomnia involves experiencing these symptoms for at least three nights a week for at least one month's time (Roth, 2007).

It is not uncommon for people suffering from insomnia to experience increased levels of anxiety about their inability to fall asleep. Insomnia becomes a self-perpetuating cycle because increased anxiety leads to increased arousal, and higher levels of arousal make the prospect of falling asleep even more unlikely. Chronic insomnia is almost always associated with feeling overtired and may be associated with symptoms of depression.

Watch It

Watch this video to learn more about the causes, symptoms, diagnosis, and treatment of insomnia.



You can view the transcript for "Insomnia – causes, symptoms, diagnosis, treatment & pathology" here (opens in new window).

Etiology

There may be many factors that contribute to insomnia, including age, drug use, exercise, mental status, and bedtime routines. Not surprisingly, insomnia treatment may take one of several different approaches. People who suffer from insomnia might limit their use of stimulant drugs (such as caffeine) or increase their amount of physical exercise during the day. Some people might turn to

over-the-counter (OTC) or prescribed sleep medications to help them sleep, but this should be done sparingly because many sleep medications result in dependence and alter the nature of the sleep cycle, and they can increase insomnia over time. Individuals who continue to have insomnia, particularly if it affects their quality of life, should seek professional treatment.

Treatment

Treatment for insomnia includes both pharmacological and non-pharmacological treatment approaches. Common pharmacological treatment for insomnia includes Benzodiazepines; however, there also may be a withdrawal syndrome with rapid eye movement (REM) rebound. In addition, there are two newer benzodiazepine-like agents, zolpidem and zaleplon, which have fewer side effects, but good efficacy. Other agents for insomnia include sedating antidepressants and over-the-counter sleep products (sedating antihistamines).[1]

Dig Deeper: Use of Sleep-aid medication

While sleep-aid medication may be effective in treating insomnia, it is essential to be cautious and careful about your treatment plan involving this medication. It is important to consider all forms of treatment and consult with your doctor for treatment of sleep disorders. Read this article about "How to use sleep aids safely."

An understanding of the electrophysiologic and neurochemical correlates of the stages of sleep is useful in defining and understanding sleep disorders. Excessive daytime sleepiness is often associated with obstructive sleep apnea or depression. Medications, including amphetamines, may be used to induce daytime alertness. Parasomnias include disorders of arousal and of REM sleep. Chronic medical illnesses can become symptomatic during specific sleep stages. Many medications affect sleep stages and can thus cause sleep disorders or exacerbate the effect of chronic illnesses on sleep.[2]

Some forms of psychotherapy, such as CBT, can help sufferers of insomnia. CBT focuses on cognitive processes and problem behaviors. The treatment of insomnia likely would include stress management techniques and changes in problematic behaviors that could contribute to insomnia (e.g., spending more waking time in bed). CBT has been demonstrated to be quite effective in treating insomnia (Savard, Simard, Ivers, & Morin, 2005; Williams, Roth, Vatthauer, & McCrae, 2013). Cognitive therapies for insomnia can help a patient to identify disruptive thought patterns, examine their validity, then replace harmful thoughts using strategies such as shifting attention, decatastrophizing, or reappraisal.[3]

Dig Deeper: Sleep Disorder Medication Dosage and Women

Read this article to learn about how FDA approval may not mean that the proper dosages are recommended for all groups of people, especially in the treatment of sleep disorders.

Solutions to support healthy sleep

Many college students struggle to get the recommended seven to nine hours of sleep each night. However, for some, it's not because of all-night partying or late-night study sessions. It's simply that they feel so overwhelmed and stressed that they cannot fall asleep or stay asleep. One or two nights of sleep difficulty is not unusual, but if you experience anything more than that, you should seek a doctor's advice.



Figure **8.10.2**: Compared to caffeinated drinks, herbal teas may be more affective in helping you achieve a good night's sleep.

Here are some tips to maintain healthy sleep:

- Stick to a sleep schedule, even on the weekends. Try going to bed and waking up at the same time every day to keep your biological clock in sync so your body gets in the habit of sleeping every night.
- Avoid anything stimulating for an hour before bed, including exercise and bright light from devices.
- Exercise daily.
- Avoid naps.
- Keep your bedroom temperature between 60 and 67 degrees. People sleep better in cooler temperatures.
- Avoid alcohol, cigarettes, caffeine, and heavy meals before bed. It may feel like alcohol helps you sleep, but it actually disrupts REM sleep and leads to frequent awakenings.
- Heavy meals may make you sleepy, but they can also lead to frequent awakenings due to gastric distress.
- If you cannot fall asleep, leave your bed and do something else until you feel tired again.
- Train your body to associate the bed with sleeping rather than other activities like studying, eating, or watching television shows.

Glossary

insomnia: a consistent difficulty in falling or staying asleep

sleep aids: prescribed and over-the-counter medication that helps people fall asleep

1. Pagel, J. F., & Parnes, B. L. (2001). Medications for the Treatment of Sleep Disorders: An Overview. *Primary care companion to the Journal of clinical psychiatry*, 3(3), 118–125. https://doi.org/10.4088/pcc.v03n0303↵
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8.11: Narcolepsy and Hypersomnolence

Learning Objectives

- Describe symptoms and factors associated with narcolepsy
- Describe symptoms and factors associated with hypersomnolence disorder

Narcolepsy



Figure **8.11.1**: Those with narcolepsy may have a difficult time staying awake at work.

A person with **narcolepsy** cannot resist falling asleep at inopportune times. These sleep episodes are often associated with **cataplexy**, which is a lack of muscle tone or muscle weakness, and in some cases involves complete paralysis of the voluntary muscles similar to the kind of paralysis experienced by healthy individuals during REM sleep (Burgess & Scammell, 2012; Hishikawa & Shimizu, 1995; Luppi et al., 2011). Narcoleptic episodes take on other features of REM sleep. For example, around one-third of individuals diagnosed with narcolepsy experience vivid, dream-like hallucinations during narcoleptic attacks (Chokroverty, 2010).

Surprisingly, narcoleptic episodes are often triggered by states of heightened arousal or stress. The typical episode can last from a minute or two to half an hour. Once awakened from a narcoleptic attack, people report that they feel refreshed (Chokroverty, 2010). Obviously, regular narcoleptic episodes could interfere with the ability to perform one's job or complete schoolwork, and in some situations, narcolepsy can result in significant harm and injury (e.g., driving a car or operating machinery or other potentially dangerous equipment).

In order to make a diagnosis of narcolepsy, an individual must have symptoms occurring at least three times a week over the past three months. In addition, one of the following must be present:

- hypocretin deficiency
- episodes of cataplexy occurring at least several times a month
- REM sleep latency of fewer than 15 minutes or two or more sleep-onset REM periods (SOREMPs) and a mean sleep latency of fewer than eight minutes.

Etiology

Narcolepsy affects both males and females equally. Symptoms often start in childhood, adolescence, or young adulthood (ages seven to 25), but can occur at any time in life. It is estimated that anywhere from 135,000 to 200,000 people in the United States have narcolepsy. However, since this condition often goes undiagnosed, the number may be higher. Since people with narcolepsy are often misdiagnosed with other conditions, such as psychiatric disorders or emotional problems, it can take years for someone to get the proper diagnosis. There are two types: narcolepsy type 1 (formerly narcolepsy with cataplexy) and narcolepsy type 2 (formerly narcolepsy without cataplexy).

Narcolepsy may have several causes. Nearly all people with narcolepsy who have cataplexy have extremely low levels of the naturally occurring chemical hypocretin, which promotes wakefulness and regulates REM sleep. Hypocretin levels are usually normal in people who have narcolepsy without cataplexy. Although the cause of narcolepsy is not completely understood, current research suggests that narcolepsy may be the result of a combination of factors working together to cause a lack of hypocretin. These factors include

- **autoimmune disorders**. When cataplexy is present, the cause is most often the loss of brain cells that produce hypocretin. Although the reason for this cell loss is unknown, it appears to be linked to abnormalities in the immune system. Autoimmune disorders occur when the body's immune system turns against itself and mistakenly attacks healthy cells or tissue. Researchers believe that in individuals with narcolepsy, the body's immune system selectively attacks the hypocretin-containing brain cells because of a combination of genetic and environmental factors.
- **family history**. Most cases of narcolepsy are sporadic, meaning the disorder occurs in individuals with no known family history. However, clusters in families sometimes occur—up to 10% of individuals diagnosed with narcolepsy with cataplexy report having a close relative with similar symptoms.
- **brain injuries**. Rarely, narcolepsy results from traumatic injury to parts of the brain that regulate wakefulness and REM sleep or from tumors and other diseases in the same regions.

Treatment

Although there is no cure for narcolepsy, some of the symptoms can be treated with medicines and lifestyle changes. When cataplexy is present, the loss of hypocretin is believed to be irreversible and lifelong. Excessive daytime sleepiness (EDS) and cataplexy can be controlled in most individuals with medications.

Medications

- **Modafinil**. The initial line of treatment is usually a central nervous system stimulant such as modafinil. Modafinil is usually prescribed first because it is less addictive and has fewer side effects than older stimulants. For most people, these drugs are generally effective at reducing daytime drowsiness and improving alertness.
- **Amphetamines**. Generally, narcolepsy is treated using psychomotor stimulant drugs, such as amphetamines (Mignot, 2012). These drugs promote increased levels of neural activity. Narcolepsy is associated with reduced levels of the signaling molecule hypocretin in some areas of the brain (De la Herrán-Arita & Drucker-Colín, 2012; Han, 2012), and traditional stimulant drugs do not have direct effects on this system. Therefore, it is quite likely that new medications that are developed to treat narcolepsy will be designed to target the hypocretin system.
- **Amphetamine-like stimulants**. In cases where modafinil is not effective, doctors may prescribe amphetamine-like stimulants such as methylphenidate to alleviate EDS. However, these medications must be carefully monitored because they can have side effects like irritability and nervousness, shakiness, disturbances in heart rhythm, and night-time sleep disruption. In addition, health care professionals should be careful when prescribing these drugs and people should be careful using them because the potential for abuse is high with any amphetamine.
- **Antidepressants**. Two classes of antidepressant drugs have proven effective in controlling cataplexy in many individuals: tricyclics (including imipramine, desipramine, clomipramine, and protriptyline) and selective serotonin and noradrenergic reuptake inhibitors (including venlafaxine, fluoxetine, and atomoxetine). In general, antidepressants produce fewer adverse effects than amphetamines. However, troublesome side effects still occur in some individuals, including impotence, high blood pressure, and heart rhythm irregularities.
- **Sodium oxybate**. Sodium oxybate (also known as gamma hydroxybutyrate or GHB) has been approved by the FDA to treat cataplexy and excessive daytime sleepiness in individuals with narcolepsy. It is a strong sedative that must be taken twice a night. Due to safety concerns associated with the use of this drug, the distribution of sodium oxybate is tightly restricted

Lifestyle changes

Not everyone with narcolepsy can consistently maintain a fully normal state of alertness using currently available medications. Drug therapy should accompany various lifestyle changes. The following strategies may be helpful:

- **Take short naps**. Many individuals take short, regularly scheduled naps at times when they tend to feel sleepiest.
- **Maintain a regular sleep schedule**. Going to bed and waking up at the same time every day, even on the weekends, can help people sleep better.
- **Avoid caffeine or alcohol before bed**. Individuals should avoid alcohol and caffeine for several hours before bedtime.
- **Avoid smoking.** Individuals should avoid smoking, especially at night.
- **Exercise daily**. Exercising for at least 20 minutes per day at least four or five hours before bedtime also improves sleep quality and can help people with narcolepsy avoid gaining excess weight.
- **Avoid large, heavy meals right before bedtime**. Eating very close to bedtime can make it harder to sleep.

- **Relax before bed**. Relaxing activities such as a warm bath before bedtime can help promote sleepiness. Also, make sure the sleep space is cool and comfortable.
- **Take safety precautions.** Safety precautions, particularly when driving, are important for everyone with narcolepsy. People with untreated symptoms are more likely to be involved in automobile accidents, although the risk is lower among individuals who are taking appropriate medication. EDS and cataplexy can lead to serious injury or death if left uncontrolled. Suddenly falling asleep or losing muscle control can transform actions that are ordinarily safe, such as walking down a long flight of stairs, into hazards.
- **Modify work/school schedules.** The Americans with Disabilities Act requires employers to provide reasonable accommodations for all employees with disabilities. Adults with narcolepsy can often negotiate with employers to modify their work schedules so they can take naps when necessary and perform their most demanding tasks when they are most alert. Similarly, children and adolescents with narcolepsy may be able to work with school administrators to accommodate special needs, like taking medications during the school day, modifying class schedules to fit in a nap, and other strategies.
- **Participate in support groups.** Additionally, support groups can be extremely beneficial for people with narcolepsy who want to develop better coping strategies or feel socially isolated due to embarrassment about their symptoms. Support groups also provide individuals with a network of social contacts who can offer practical help and emotional support.

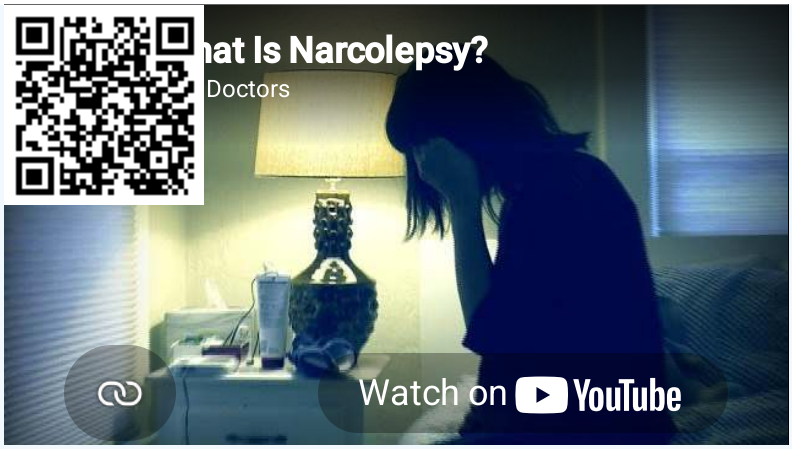


Figure 8.11.2: A relaxing bath before bed may contribute to a better night's sleep and sleep schedule, which can help in treating narcolepsy.

There is a tremendous amount of variability among sufferers, both in terms of how symptoms of narcolepsy manifest and the effectiveness of currently available treatment options. This variation is illustrated by McCarty's (2010) case study of a 50-year-old woman who sought help for the excessive sleepiness during normal waking hours that she had experienced for several years. She indicated that she had fallen asleep at inappropriate or dangerous times, including while eating, while socializing with friends, and while driving her car. During periods of emotional arousal, the woman complained that she felt some weakness in the right side of her body. Although she did not experience any dream-like hallucinations, she was diagnosed with narcolepsy as a result of sleep testing. In her case, the fact that her cataplexy was confined to the right side of her body was quite unusual. Early attempts to treat her condition with a stimulant drug alone were unsuccessful. However, when a stimulant drug was used in conjunction with a popular antidepressant, her condition improved dramatically.

Watch It

Watch this video about Michelle, who suffered from narcolepsy when she experienced emotional distress. She describes her symptoms with sleep disturbances, her diagnosis, and how she was empowered by her diagnosis.



You can view the transcript for "What Is Narcolepsy?" here (opens in new window).

Hypersomnolence Disorder

Hypersomnia is a pathological state characterized by a lack of alertness during the waking episodes of the day. Hypersomnia is not to be confused with fatigue, which is a normal physiological state. Daytime sleepiness appears most commonly during situations where little interaction is needed. Hypersomnolence disorder affects approximately 5% of the population and is more common in men than females.[1]

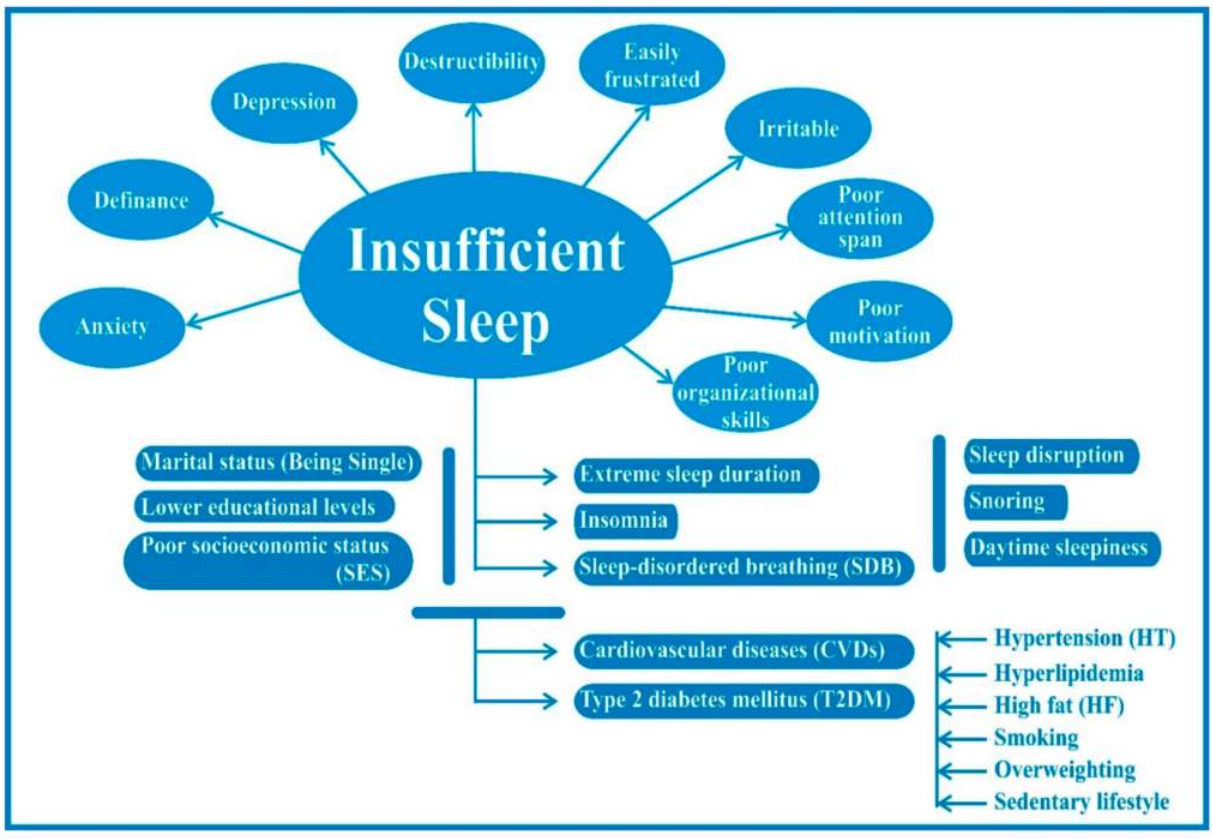


Figure **8.11.1**: Insufficient sleep poses major health risks throughout a person's lifetime.

In 2008, the CDC examined data from over 400,000 subjects throughout the United States and found that 11.1% reported that they had had insufficient rest or sleep every day during the preceding 30 days. Females (12.4%) were more likely than males (9.9%), and non-Hispanic blacks (13.3%) were more likely than other racial/ethnic groups to report insufficient rest or sleep. A recent study conducted among Korean adults (19 years and older) found that the prevalence of excessive day sleepiness was 11.9%. A recent

cross-sectional Japanese study, which used a web-based questionnaire to ask about health-related quality of life issues, found that respondents aged 20 to 25 years and who were either students or full-time employees (11% of the sample), reported that they suffered from insufficient sleep syndrome.[2]

Etiology

Hypersonia may be caused by another sleep disorder (such as narcolepsy or sleep apnea), dysfunction of the autonomic nervous system, or drug or alcohol abuse. In some cases, it results from a physical problem, such as a tumor, head trauma, or injury to the central nervous system. Medical conditions including multiple sclerosis, depression, encephalitis, epilepsy, or obesity may contribute to hypersomnolence sleep disorder. Some people appear to have a genetic predisposition to hypersomnia; in others, there is no known cause. Typically, hypersomnia is first recognized in adolescence or young adulthood.

Treatment

Treatment is symptomatic in nature. Stimulants, such as amphetamine, methylphenidate, and modafinil, may be prescribed. Other drugs used to treat hypersomnia include clonidine, levodopa, bromocriptine, antidepressants, and monoamine oxidase inhibitors. Changes in behavior (for example, avoiding night work and social activities that delay bedtime) and diet may offer some relief. Patients should avoid alcohol and caffeine.

Watch It

This short clip explains hypersomnia.



You can view the transcript for "What is hypersomnia?" here (opens in new window).

Insufficient Sleep Syndrome

Although not yet classified as a disorder in the DSM-5, insufficient sleep syndrome is recognized by the International Classification of Sleep Disorders (ICSD-3) as a curtailed sleep pattern that has persisted for at least three months for most days of the week, along with complaints of sleepiness during the day. Further, a resolution of sleepiness complaints is shown to follow an extension of total sleep time. Frequently occurring episodes of insufficient sleep are associated with the experience of unfavorable mental and physical well-being. Sleep insufficiency is sometimes confused with insomnia, but the opportunity to sleep differs in the two disorders (with insomnia sufferers typically being unable to sleep despite having opportunities to do so). Insufficient sleep is associated with a range of negative health and social outcomes, including an adverse performance at school and in the labor market. Reduced sleep duration has been linked to seven of the 15 leading causes of death in the United States, including cardiovascular disease, malignant neoplasm, cerebrovascular disease, accidents, diabetes, septicemia, and hypertension. The evidence suggests that the link between inadequate sleep and negative outcomes is more direct, whereas the link between excessive sleep and negative outcomes seems to be more indirect (i.e., excessive sleep is driven by underlying chronic health conditions and not vice versa). Hence, the impact of insufficient sleep appears to be the more salient issue in our society and, because of its broad-ranging effects, represents a major public health concern. In this review, we note some of the more serious consequences of insufficient sleep, and additionally consider how these might be best addressed by changes in

individual behavior, actions by employers, and by public policy measures.[footnote]Chattu, V.K.; Manzar, M.D.; Kumary, S.; Burman, D.; Spence, D.W.; Pandi-Perumal, S.R. The Global Problem of Insufficient Sleep and Its Serious Public Health Implications. Healthcare 2019, 7, 1. ↵

Glossary

cataplexy: lack of muscle tone or muscle weakness, and in some cases complete paralysis of the voluntary muscles

narcolepsy: sleep disorder in which the sufferer cannot resist falling to sleep at inopportune times

sleep drunkenness: difficulty transitioning from wake to sleep; a symptom found in hypersomniac patients

1. Since the patients' attention levels are impaired, their quality of life may be impacted as well. This is especially true for people whose jobs request high levels of attention, for example, in the healthcare field. Indeed, the lack of attention can cause injuries to self or others, which makes this disorder a real public health issue. Hypersomnolence disorder is characterized by excessive daytime somnolence (EDS). However, the patient in question must have no other symptoms or signs of narcolepsy (cataplexy, sleep paralysis, hypnagogic hallucinations), and no breathing-related sleep disorder like sleep apnea. In addition, their excessive daytime somnolence (EDS) is not a result of insufficient sleep or due to the effects of a substance or another general medical condition. In other words, this is a diagnosis of exclusion once all other causes of excessive daytime somnolence (EDS) have been ruled out. According to ICD-10, the EDS should occur nearly every day for a month or recurrently for shorter periods of time. According to the DSM-5, the main symptom of hypersomnia is EDS, or prolonged night-time sleep, which has occurred for at least three months prior to diagnosis. **Sleep drunkenness**, a difficulty to transition from wake to sleep is also a symptom found in hypersomniac patients. Patients suffering from sleep drunkenness report waking with confusion, disorientation, slowness and repeated returns to sleep. According to the American Academy of Sleep Medicine, hypersomniac patients often take long naps during the day that are mostly unrefreshing. Researchers found that naps are usually more frequent and longer in those with hypersomnolence than for those in control groups. Furthermore, 75% of those with hypersomnolence reported that short naps are not refreshing, compared to controls.
2. Chattu, V. K., Sakhamuri, S. M., Kumar, R., Spence, D. W., BaHammam, A. S., & Pandi-Perumal, S. R. (2018). Insufficient Sleep Syndrome: Is it time to classify it as a major noncommunicable disease?. Sleep science (Sao Paulo, Brazil), 11(2), 56–64. https://doi.org/10.5935/1984-0063.20180013↵

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8.12: Parasomnias

Learning Objectives

- Explain NREM sleep arousal disorders (sleepwalking and night terrors)
- Explain REM sleep behavior disorder (RBD)

Parasomnias

A **parasomnia** is one of a group of sleep disorders in which unwanted, disruptive motor activity and/or experiences *during* sleep play a role. Parasomnias can occur in either REM or NREM phases of sleep. Parasomnias include NREM sleep arousal disorders of sleepwalking and sleep terrors, nightmare disorder, REM sleep behavior disorder, and restless leg syndrome.

Link to Learning

Listen to the following clip from NPR's "This American Life" episode "Fear of Sleep" to hear about some experiences of people with parasomnia sleep disorders and how it has affected their lives. Listen specifically to the 1:40–6:25 timeframe.

NREM Sleep Arousal Disorders

Sleepwalking



Figure **8.12.1**: Sleepwalking and night terrors take place during non-REM sleep.

In **sleepwalking** or somnambulism, the sleeper engages in relatively complex behaviors ranging from wandering about to driving an automobile. During periods of sleepwalking, sleepers often have their eyes open, but they are not responsive to attempts to communicate with them. Sleepwalking most often occurs during slow-wave sleep, but it can occur at any time during a sleep period in some affected individuals (Mahowald & Schenck, 2000).

Historically, somnambulism has been treated with a variety of pharmacotherapies ranging from benzodiazepines to antidepressants. However, the success rate of such treatments is questionable. Guilleminault et al. (2005) found that sleepwalking was not alleviated with the use of benzodiazepines. However, all their somnambulistic patients who also suffered from sleep-related breathing problems showed a marked decrease in sleepwalking when their breathing problems were effectively treated.

Night Terrors

Night terrors result in a sense of panic in the sufferer and are often accompanied by screams and attempts to escape from the immediate environment (Mahowald & Schenck, 2000). Although individuals suffering from night terrors appear to be awake, they generally have no memories of the events that occurred and attempts to console them are ineffective. Typically, individuals suffering from night terrors will fall back asleep again within a short time. Night terrors apparently occur during the NREM phase of sleep (Provini, Tinuper, Bisulli, & Lagaresi, 2011). Generally, treatment for night terrors is unnecessary unless there is some underlying medical or psychological condition that is contributing to the night terrors (Mayo Clinic, n.d.).



Figure **8.12.2**: Nightmares occur during REM sleep.

Nightmare disorder is a sleep disorder characterized by frequent nightmares. The nightmares, which often portray the individual in a situation that jeopardizes his or her life or personal safety, usually occur during the REM stages of sleep (unlike night terrors, which occur during NREM sleep and do not follow a storyline in the way that nightmares do). Though most people have experienced at least one nightmare during their life, subjects with nightmare disorder experience them with a greater frequency.

Nightmares also have to be distinguished from bad dreams, which are less emotionally intense. Furthermore, nightmares contain more stories of aggression than bad dreams and more unhappy endings. Finally, people experiencing nightmares feel more fear than for bad dreams.

Comorbidity

Studies have reported that nightmare disorders were present in 50–70% of the cases for PTSD, in 17.5% for depression, in 18.3% for insomnia, in 16.7% for schizophrenia, and in 49% for borderline personality disorder.[1] For all psychiatric disorders taken together, nightmare disorders are present in 29.9% of the cases, a much bigger rate than for the general population, which is 2%-5%. Nightmare disorders can also be associated with sleep disorders such as night terrors, chronic insomnia, and sleep-disordered breathing. The presence of nightmares before a trauma would influence the severity of PTSD symptoms. Furthermore, having nightmares is linked to a significantly higher risk of attempting suicide and of death by suicide.

Nightmares also seem to be correlated with some personality factors. Studies found an association between anxiety, depression, and nightmares in insomnia, while only a small relationship was found in other populations. Neuroticism would be also link to nightmares. Nevertheless, people with a higher score of neuroticism could be better at recalling their nightmares during the self-report assessment, which could have an influence on this association.

REM Sleep Behavior Disorder (RBD)

REM sleep behavior disorder (RBD) occurs when the muscle paralysis associated with the REM sleep phase does not occur. Individuals who suffer from RBD have high levels of physical activity during REM sleep, especially during disturbing dreams. These behaviors vary widely, but they can include kicking, punching, scratching, yelling, and behaving like an animal that has been frightened or attacked.



Figure **8.12.3**: Restless leg syndrome commonly kicks in during the evening hours or during sleep, frustrating a person's ability to relax or get a good night's sleep.

The prevalence of REM sleep behavior disorder (RBD) is approximately 0.5%-1% in the general population and 2% in older adults. A large number of people are undiagnosed. The male to female diagnosis ratio is as high as 9:1. People with REM sleep behavior disorder (RBD) often have a comorbidity with mental health illness like a mood disorder and take anti-depressants or a history of traumatic brain injury.[2] This disorder is associated with a number of neurodegenerative diseases such as Parkinson's disease. In fact, this relationship is so robust that some view the presence of RBD as a potential aid in the diagnosis and treatment of a number of neurodegenerative diseases (Ferini-Strambi, 2011).

Clonazepam, an anti-anxiety medication with sedative properties, is most often used to treat RBD. It is administered alone or in conjunction with doses of melatonin (the hormone secreted by the pineal gland). As part of treatment, the sleeping environment is often modified to make it a safer place for those suffering from RBD (Zangini, Calandra-Buonaura, Grimaldi, & Cortelli, 2011).

Restless Leg Syndrome

A person with **restless leg syndrome** has uncomfortable sensations in the legs during periods of inactivity or when trying to fall asleep. This discomfort is relieved by deliberately moving the legs, which, not surprisingly, contributes to difficulty in falling or staying asleep. Restless leg syndrome is quite common and has been associated with a number of other medical diagnoses, such as chronic kidney disease and diabetes (Mahowald & Schenck, 2000). There are a variety of drugs that treat restless leg syndrome: benzodiazepines, opiates, and anticonvulsants.

Dig Deeper: A Sleepwalking Defense?

On January 16, 1997, Scott Falater sat down to dinner with his wife and children and told them about the difficulties he was experiencing on a project at work. After dinner, he prepared some materials to use in leading a church youth group the following morning, and then he attempted to repair the family's swimming pool pump before retiring to bed. The following morning, he awoke to barking dogs and unfamiliar voices from downstairs. As he went to investigate what was going on, he was met by a group of police officers who arrested him for the murder of his wife (Cartwright, 2004; CNN, 1999).

Yarmila Falater's body was found in the family's pool with 44 stab wounds. A neighbor called the police after witnessing Falater standing over his wife's body before dragging her into the pool. Upon a search of the premises, police found blood-stained clothes and a bloody knife in the trunk of Falater's car, and he had blood stains on his neck.

Remarkably, Falater insisted that he had no recollection of hurting his wife in any way. His children and his wife's parents all agreed that Falater had an excellent relationship with his wife and they couldn't think of a reason that would provide any sort of motive to murder her (Cartwright, 2004).

Scott Falater had a history of regular episodes of sleepwalking as a child, and he had even behaved violently toward his sister once when she tried to prevent him from leaving their home in his pajamas during a sleepwalking episode. He suffered from no apparent anatomical brain anomalies or psychological disorders. It appeared that Scott Falater had killed his wife in his sleep, or at least, that is the defense he used when he was tried for his wife's murder (Cartwright, 2004; CNN, 1999). In Falater's case, a jury found him guilty of first degree murder in June of 1999 (CNN, 1999); however, there are other murder cases where the sleepwalking defense has been used successfully. As scary as it sounds, many sleep researchers believe that homicidal sleepwalking is possible in individuals suffering from the types of sleep disorders described below (Broughton et al., 1994; Cartwright, 2004; Mahowald, Schenck, & Cramer Bornemann, 2005; Pressman, 2007).

Glossary

parasomnia: a group of sleep disorders where unwanted, disruptive motor activity, or other experiences during sleep play a role in their life

nightmare disorder: the sleep disorder that is characterized by frequent nightmares

night terrors: when a person experiences a sense of panic that is often accompanied by screams and attempts to escape from the immediate environment

REM sleep behavior disorder (RBD): when the muscle paralysis associated with the REM sleep phase does not occur

restless leg syndrome: the sleep disorder where a person has uncomfortable sensations in the legs during periods of inactivity or when trying to fall asleep.

1. Swart, Marijke L.; van Schagen, Annette M.; Lancee, Jaap; van den Bout, Jan (2013). "Prevalence of Nightmare Disorder in Psychiatric Outpatients". *Psychotherapy and Psychosomatics*. 82 (4): 267–268. doi:10.1159/000343590. ISSN 1423-0348. PMID 23735876. ↵
2. UpToDate. https://www.uptodate.com/contents/rapid-eye-movement-sleep-behavior-disorder. ↵

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8.13: Breathing-Related Sleep Disorders

Learning Objectives

- Describe sleep apnea and other breathing-related sleep disorders

Sleep Apnea

Sleep apnea is defined by episodes during which a sleeper's breathing stops. Sleep apnea episodes can last 10–20 seconds or longer and often are associated with brief periods of arousal. While individuals suffering from sleep apnea may not be aware of these repeated disruptions in sleep, they do experience increased levels of fatigue. Many individuals diagnosed with sleep apnea first seek treatment because their sleeping partners indicate that they snore loudly and/or stop breathing for extended periods of time while sleeping (Henry & Rosenthal, 2013).

Sleep apnea is much more common in people who are overweight and it may exacerbate cardiovascular disease (Sánchez-de-la-Torre, Campos-Rodriguez, & Barbé, 2012). While sleep apnea is less common in people who are thin, anyone, regardless of their weight, who snores loudly or gasps for air while sleeping, should be checked for sleep apnea.

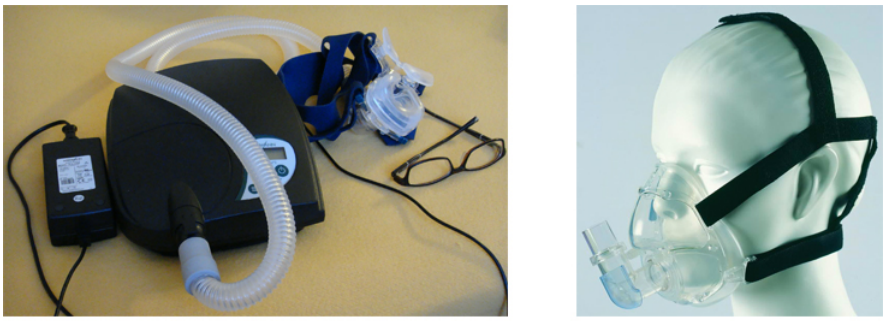
While people are often unaware of their sleep apnea, they are keenly aware of some of the adverse consequences of insufficient sleep. One person explained that the sleep apnea affected his daily life saying, "I'm in a job where there's a premium on being mentally alert. I was really sleepy . . . and having trouble concentrating. . . . It was getting to the point where it was kind of scary" (Henry & Rosenthal, 2013, p. 52).

There are two main types of sleep apnea: obstructive sleep apnea**,** listed as obstructive sleep apnea hypopnea (OSAH) in the DSM-5, and central sleep apnea. **Obstructive sleep apnea** occurs when an individual's airway becomes blocked during sleep and air is prevented from entering the lungs. It is diagnosed if a person has at least five obstructive apneas per hour of sleep combined with breathing disturbances such as snoring and daytime sleepiness or at least fifteen obstructive apneas per hour of sleep without other symptoms. The apnea is characterized by at least ten-second disturbances in breathing. In **central sleep apnea (CSA)**, disruption in signals sent from the brain that regulate breathing cause periods of interrupted breathing (White, 2005). Central sleep apnea is less common than obstructive sleep apnea hypopnea (OSAH) and is more commonly associated with illnesses.

Approximately 2%-9% of adults in the United States have obstructive sleep apnea and about 0.9% of adults over the age of 40 are diagnosed with central sleep apnea, but many cases are estimated to be undiagnosed. Both are more common in men than women. There are multiple factors that contribute to being at risk for sleep apnea, including obesity, hormonal imbalances, smoking cigarettes, family history, and the size and positioning of the jaw, throat, and tongue.[1]

Treatment

One of the most common treatments for sleep apnea involves the use of a special device during sleep. A **continuous positive airway pressure (CPAP)** device includes a mask that fits over the sleeper's nose and mouth, which is connected to a pump that pumps air into the person's airways, forcing them to remain open, as shown in Figure 1. Some newer continuous positive airway pressure (CPAP) masks are smaller and cover only the nose. This treatment option has proven to be effective for people suffering from mild to severe cases of sleep apnea (McDaid et al., 2009). However, alternative treatment options are being explored because consistent compliance by users of continuous positive airway pressure (CPAP) devices is a problem. EPAP (excitatory positive air pressure) device has shown promise in double-blind trials as one such alternative (Berry, Kryger, & Massie, 2011). Treatment of sleep apnea also involves treading comorbid medical conditions, sometimes through surgery or medications.



(a) (b)

Figure **8.13.1**: (a) A typical CPAP device used in the treatment of sleep apnea is (b) affixed to the head with straps and a mask that covers the nose and mouth.

- Tongue Surgery
  - This procedure involves advancing one of the main tonguemuscles, the genioglossus muscle, forward, thereby limiting the backward fall of the tongue during sleep. This procedure addresses the same sites of potential obstruction as the hyoid advancement, and numerous studies have shown a high success rate.
- Nasal Surgery
  - Both daytime nasal obstruction and nocturnal nasal congestionhave been shown as risk factors for sleep-disordered breathing. Therefore, the treatmentof nasal obstruction plays an important role in sleep apneasurgery. Three anatomic areas of the nose that may contribute to obstruction are the septum, the turbinates, and the nasal valve. The most common nasal surgical procedure consists of straightening out the septumand reducing the size of the turbinates, which creates more room in the nose and allows air to pass smoothly and without effort.
- Tongue Surgery
  - The tongue has an established role in obstruction in sleep apnea. One treatment involves moving one of the main tonguemuscles, the genioglossus muscle, forward; thereby limiting the backward fall of the tongue during sleep. This procedure addresses the same sites of potential obstruction as the hyoid advancement, and numerous studies have shown a high success rate. Another effective surgical treatment to reduce is to reduce the amount of tissue at the tonguebase.
- UPPP
  - UPPP, or in full, uvulopalatopharyngoplasty, has been the most common sleep apnea surgical procedure performed during the past 25 years. This procedure was developed to remove excess tissue from the soft palateand pharynx to remove the obstruction from the upper airway. Patientswho suffer from snoringgain a great deal of improvement from this procedure as snoringis often due to the reverberation of the soft palatewith the back wall of the pharynx.
- Hyoid Advancement
  - The hyoidbone is a small bone in the neck where the muscles of the tonguebase and pharynxattach. Patientswith sleep apnea often have a large tonguebase. During the deep stages of sleep, normal muscle tone is relaxed, and the base of the tonguefalls back and can make contact with the back wall of the pharynxresulting in obstruction. Through a very minimally invasive procedure, the hyoidbone is surgically repositioned anteriorly by placing a suture around it and suspending it to the front of the jaw bone. This procedure results in an expansion of the airwayand prevents collapse.
- Lower Jaw Advancement
  - Abnormality of the maxillofacial skeletonis a well-recognized risk factor of obstructivesleep apnea. Sleep apnea patients usually have small, narrow jaws that result in diminished airway dimension, which leads to nocturnal obstruction.

Maxillomandibular advancement achieves enlargement of the entire upper airway through the expansion of the skeletal framework that encircles the airway.

- Tracheostomy
  - Tracheostomy is a technique that creates a passageway for air to get to the lungs directly from the trachea in the neck. This technique bypasses any potential sites of obstruction from the upper airway. Permanent tracheostomy as a long-term treatment of obstructive sleep apnea remains an option when all other forms of non-surgical and surgical treatments have failed. Though it may seem excessive, it is an extremely effective surgical option reserved for the very sick patient. [2]

SIDS



Figure 8.13.2: The Safe to Sleep campaign educates the public about how to minimize risk factors associated with SIDS. This campaign is sponsored in part by the National Institute of Child Health and Human Development.

In **sudden infant death syndrome (SIDS)**, an infant stops breathing during sleep and dies. Infants younger than 12 months appear to be at the highest risk for SIDS, and boys have a greater risk than girls. A number of risk factors have been associated with SIDS, including premature birth, smoking within the home, and hyperthermia. There may also be differences in both brain structure and function in infants that die from SIDS (Berkowitz, 2012; Mage & Donner, 2006; Thach, 2005).

The substantial amount of research on SIDS has led to a number of recommendations to parents to protect their children (Figure 2). For one, research suggests that infants should be placed on their backs when put down to sleep, and their cribs should not contain any items that pose suffocation threats, such as blankets, pillows, or padded crib bumpers (cushions that cover the bars of a crib). Infants should not have caps placed on their heads when put down to sleep in order to prevent overheating, and people in the child's household should abstain from smoking in the home. Recommendations like these have helped to decrease the number of infant deaths from SIDS in recent years (Mitchell, 2009; Task Force on Sudden Infant Death Syndrome, 2011).

Glossary

central sleep apnea: sleep disorder with periods of interrupted breathing due to a disruption in signals sent from the brain that regulate breathing

continuous positive airway pressure (CPAP): device used to treat sleep apnea; includes a mask that fits over the sleeper's nose and mouth, which is connected to a pump that pumps air into the person's airways, forcing them to remain open

obstructive sleep apnea: sleep disorder defined by episodes when breathing stops during sleep as a result of blockage of the airway

sleep apnea: sleep disorder defined by episodes during which breathing stops during sleep

sudden infant death syndrome (SIDS): infant (one-year-old or younger) with no apparent medical condition suddenly dies during sleep

1. Sleep Apnea - Causes & Symptoms. Sleep Foundation. (2020, September 25). https://www.sleepfoundation.org/sleep-apnea. ↵
2. Sleep Apnea - Treatment Options. Sleep Foundation. (2020, September 25). https://www.sleepfoundation.org/sleep-apnea. ↵

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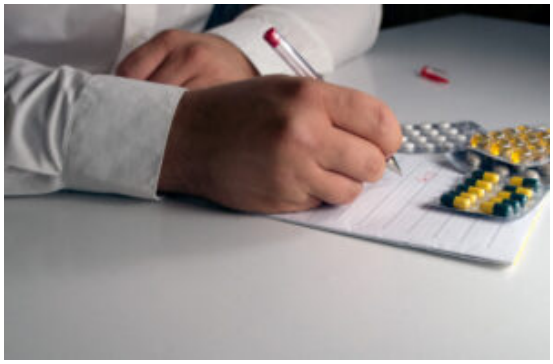
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8.14: Introduction to Perspectives and Treatments Related to Eating and Sleep Disorders

What you'll learn to do: examine various psychological perspectives and treatment methods related to eating and sleep disorders



In this section, we will review psychological perspectives and treatment for eating and sleep disorders. In addition, you will learn about common psychological approaches treating eating and sleep disorders, know important factors to consider when seeking treatment, and describe treatments and research for different types of eating and sleep disorders.

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8.15: Understanding and Treating Eating Disorders

Learning Objectives

- Describe biological, cognitive, and behavioral approaches to treating eating disorders

Eating disorders can affect people of all ages, racial/ethnic backgrounds, body weights, and genders. Eating disorders frequently appear during the teen years or young adulthood, but may also develop during childhood or later in life. These disorders affect both genders, although rates among women are higher than among men. Like women who have eating disorders, men can also have a distorted sense of body image. It is important to seek treatment early for eating disorders. People with eating disorders are at higher risk for suicide and medical complications. Some people with eating disorders may also have other mental disorders (such as depression or anxiety) or problems with substance use.

Researchers are finding that eating disorders are caused by a complex interaction of genetic, biological, behavioral, psychological, and social factors. This research has lead to many different and integrative approaches and techniques for treating eating disorders. It is important to also note that eating disorder treatment is highly influenced by the individual patient and the effectiveness of treatment plans varies, depending on a wide variety of factors. Some treatment plans include medications, psychotherapy, nutritional counseling, medical care, or some combination of these approaches. Treatment location also varies, as the levels of care provided for the patient depend on their medical and psychiatric stability; there are inpatient, residential, partial hospitalization, intensive outpatient, and outpatient options.

Let's examine a few of the psychological perspectives related to eating disorders and examine treatment options from several of these perspectives.

Biological Approaches

Eating behavior is a complex process controlled by the neuroendocrine system, of which the hypothalamus-pituitary-adrenal-axis (HPA axis) is a major component. Dysregulation of the hypothalamus-pituitary-adrenal-axis (HPA axis) has been associated with eating disorders, such as irregularities in the manufacture, amount, or transmission of certain neurotransmitters, hormones, or neuropeptides and amino acids such as homocysteine, elevated levels of which are found in anorexia and bulimia, as well as depression. Some of the neurotransmitters and hormones linked to eating disorders include

- serotonin: a neurotransmitter, known for being involved in depression, that has an inhibitory effect on eating behavior.
- norepinephrine: both a neurotransmitter and a hormone; abnormalities in either capacity may affect eating behavior.
- Dopamine: in addition to being a precursor of norepinephrine and epinephrine also regulates the rewarding property of food.
- neuropeptide Y (also known as NPY): a hormone that encourages eating and decreases metabolic rate. Blood levels of NPY are elevated in patients with anorexia nervosa, and studies have shown that injection of this hormone into the brain of rats with restricted food intake increases their time spent running on a wheel. Normally the hormone stimulates eating in healthy patients, but under conditions of starvation it increases their activity rate, probably to increase the chance of finding food. The increased levels of NPY in the blood of patients with eating disorders can in some ways explain the instances of extreme over-exercising found in most anorexia nervosa patients.
- leptin and ghrelin: leptin is a hormone produced primarily by the fat cells in the body; it has an inhibitory effect on appetite by inducing a feeling of satiety. Ghrelin is an appetite-inducing hormone produced in the stomach and the upper portion of the small intestine. Circulating levels of both hormones are an important factor in weight control. While often associated with obesity, both hormones and their respective effects have been implicated in the pathophysiology of anorexia nervosa and bulimia nervosa. Leptin can also be used to distinguish between constitutional thinness found in a healthy person with a low BMI and an individual with anorexia nervosa.
- Gut bacteria and immune system: studies have shown that a majority of patients with anorexia and bulimia nervosa have elevated levels of autoantibodies that affect hormones and neuropeptides that regulate appetite control and the stress response. There may be a direct correlation between autoantibody levels and associated psychological traits.

Genetic influences

While past findings have described eating disorders as primarily psychological, environmental, and sociocultural, further studies have uncovered evidence that there is a genetic component related to a predisposition toward eating disorders.[1] Twin studies have found slight instances of genetic variance when considering the different criterion of both anorexia nervosa and bulimia nervosa as endophenotypes contributing to the disorders as a whole. A genetic link has been found on chromosome 1 in multiple family members of an individual with anorexia nervosa. An individual who is a first-degree relative of someone who has had or currently has an eating disorder is seven to 12 times more likely to have an eating disorder themselves. Twin studies also show that at least a portion of the vulnerability to develop eating disorders can be inherited, and there is evidence to show that there is a genetic locus that shows susceptibility for developing anorexia nervosa.

People with gastrointestinal disorders may be at greater risk of developing disordered eating practices than the general population, principally restrictive eating disturbances; there is an association of anorexia nervosa with celiac disease.

There are also various childhood personality traits associated with the development of eating disorders. During adolescence, these traits may become intensified due to a variety of physiological and cultural influences such as the hormonal changes associated with puberty, stress related to the approaching demands of maturity, and socio-cultural influences and perceived expectations, especially in areas that concern body image. Eating disorders have been associated with a fragile sense of self and with disordered mentalization. Many personality traits have a genetic component and are highly heritable. Maladaptive levels of certain traits may be acquired as a result of anoxic or traumatic brain injury, neurodegenerative diseases such as Parkinson's disease, neurotoxicity such as lead exposure, bacterial infection such as Lyme disease or parasitic infection such as Toxoplasma gondii as well as hormonal influences. While studies are still continuing via the use of various imaging techniques such as fMRI, these traits have been shown to originate in various regions of the brain such as the amygdala and the prefrontal cortex. Disorders in the prefrontal cortex and the executive functioning system have been shown to affect eating behavior.

Biological Treatments

Depending on the medical stability of the patient, in general, medical treatment plans for eating disorders include medical care and monitoring, nutritional counseling and interventions, medications, or a combination of these approaches. Typical treatment goals include restoring adequate nutrition, bringing weight to a healthy level, reducing excessive exercise, and stopping binge-purge and binge-eating behaviors.

Two pharmaceuticals, prozac and vyvanse, have been approved by the FDA to treat bulimia nervosa and binge-eating disorder, respectively. Olanzapine has also been used off-label to treat anorexia nervosa. Studies are also underway to explore psychedelic and psychedelic-adjacent medicines such as MDMA, psilocybin, and ketamine for anorexia nervosa and binge-eating disorder.

Watch It

Watch this video to learn about eating disorder treatment, especially the components of care and how the medical stability of the patient determines both the level and types of treatments prescribed.



You can view the transcript for "What Does Treatment Look Like?" here (opens in new window).

Cognitive Approaches

Cognitive attentional bias may have an effect on eating disorders. Attentional bias is the preferential attention toward certain types of information in the environment while simultaneously ignoring others. Individuals with eating disorders can be thought to have dysfunctional schemas and knowledge structures that may bias judgment, thought, and behavior in a manner that is self-destructive or maladaptive. They may have developed a disordered schema that focuses on body size and eating. Thus, this information is given the highest level of importance and overvalued among other cognitive structures. Researchers have found that people who have eating disorders tend to pay more attention to stimuli related to food. For people struggling to recover from an eating disorder or addiction, this tendency to pay attention to certain signals while discounting others can make recovery that much more difficult.

Studies have utilized the Stroop task (which measures reaction time to different stimuli) to assess the probable effect of attentional bias on eating disorders. This may involve separating food and eating words from body shape and weight words. Such studies have found that anorexic subjects were slower to color name food-related words than control subjects. Other studies have noted that individuals with eating disorders have significant attentional biases associated with eating and weight stimuli.[2]

Cognitive treatments

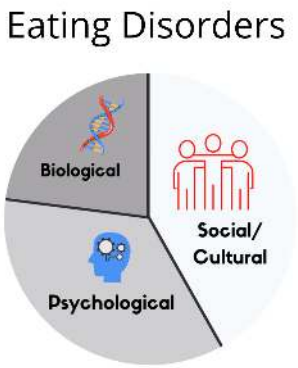


Figure 8.15.1: The biopsychosocial model emphasizes the interplay between biological, psychological, and sociocultural forces. Research on eating disorders indicates that there is a strong sociocultural influence.

Cognitive treatment approaches focus on helping patients with eating disorders shift the way that they think about eating and their eating behaviors. The primary form of cognitive treatment comes in the form of psychotherapy, the most common of which for treating eating disorders include acceptance and commitment therapy (ACT), CBT, enhanced CBT, and cognitive remediation therapy.

Acceptance and Commitment Therapy

The goal of ACT is to focus on changing your actions rather than your thoughts and feelings. Patients are taught to identify core values and commit to creating goals that fulfill these values. A popular metaphor for ACT has us compare the journey through life as driving a bus full of noisy passengers (or thoughts), but ignoring unwanted or negative thoughts by still purposefully driving to the destination. For someone with an eating disorder, negative thoughts may be related to their body, appearance, or self-worth. Through ACT, they can learn to be mindful of these thoughts but not act on them.[3]

Cognitive Behavioral Therapy

CBT is a relatively short-term, symptom-oriented therapy focusing on the beliefs, values, and cognitive processes that maintain the eating disorder behavior. It aims to modify distorted beliefs and attitudes about the meaning of weight, shape, and appearance, which are correlated to the development and maintenance of the eating disorder.

CBT-Enhanced

A common form of CBT that is used to treat eating disorders is called CBT-enhanced (CBT-E) and was developed by Christopher G. Fairburn throughout the 1970s and 1980s. Originally intended for bulimia nervosa specifically, it was eventually extended to all eating disorders. Within Fairburn's enhanced CBT is CBT-Ef, designed to deal particularly with eating habits, and CBT-Eb for other issues that do not directly involve eating. It is described as "enhanced" because it uses a variety of new strategies and

procedures to improve outcomes and because it includes modules to address certain obstacles to change that are “external” to the core eating disorder, namely clinical perfectionism, low self-esteem, and interpersonal difficulties.

Although CBT-E uses a variety of generic cognitive and behavioral interventions (such as addressing cognitive biases), unlike some forms of CBT, it favors the use of strategic changes in behavior to modify thinking rather than direct cognitive restructuring. The eating disorder psychopathology may be likened to a house of cards with the strategy being to identify and remove the key cards that are supporting the eating disorder, thereby bringing down the entire house.[4]

Cognitive Remediation Therapy

For individuals with anorexia nervosa, CRT is an interactive treatment that combines practical exercises with discussions about their relevance to the patient’s everyday life. Cognitive remediation therapy was adapted for anorexia nervosa by Professor Kate Tchanturia and colleagues at the Institute of Psychiatry, Psychology, and Neuroscience to address the process rather than the content of thinking, thus helping patients to develop a metacognitive awareness of their own thinking style. The treatment is hypothesized to work by strengthening and refining neural circuits, and by learning and transferring new cognitive strategies to appropriate situations. The aim is to identify and target the cognitive impairments specific to each patient, and to motivate the patient to engage in meta-cognitive processes, i.e., to consider their cognitive/thinking styles and to explore alternative strategies, which in turn might lead to behavioral changes. By becoming aware of problematic cognitive styles, the patient can reflect on how these affect everyday life and learn to develop new strategies. The intervention was originally developed for adults with chronic anorexia nervosa, but it has been explored for younger patients as well.

✔ Watch It

This video provides an overview of some of the main treatments for eating disorders, including CBT-E and interpersonal therapy (IPT).

You can view the transcript for “Treatment for Eating Disorders” here (opens in new window).

Behavioral Approaches

Behavioral theories about eating disorders related to ways that behavior is learned and reinforced. For example, if someone who loses weight is praised or rewarded for their appearance but took extreme measures to look that way, this praise could contribute to the development or maintenance of an eating disorder. What types of behaviors are rewarded and encouraged are strongly influenced by sociocultural factors, which we’ll examine soon. Another behavioral connection to eating disorders is the way in which anxiety or stress become associated with eating or overeating; efforts to avoid that anxiety may result in starvation or binging, and the reduction of anxiety acts as a reinforcer for the behavior.

Treatments: Dialectical Behavior Therapy (DBT)

Dialectical behavior therapy assumes that the most effective place to begin treatment is with changing behaviors. Treatment focuses on developing skills to replace maladaptive eating disorder behaviors. Skills focus on building mindfulness skills and becoming more effective in interpersonal relationships, emotion regulation, and distress tolerance.

? Link to Learning

An obvious part of the recovery process in overcoming eating disorders involves changing eating behavior. Those with the disorder must overcome unhealthy relationships with food and behavioral techniques, and using behavioral tools such as exposure therapy and systematic desensitization can gradually help a person to become less anxious about certain foods or situations. For example, a person with ARFID may avoid certain textures of food, but can be gradually introduced to them. A person with anorexia may become anxious about eating in public or around others.

In November 2020, an 18-year old TikTok user Sara Sadok gained popularity by inviting others to create a TikTok duet in which they “eat with her” and enjoy a meal together. This video and subsequent duets raised awareness of eating disorders and the importance of taking small steps to reduce anxiety surrounding food.



Figure 8.15.2: Diet culture is prevalent on social media and can have a negative influence on an individual's body image.

There is a strong sociocultural influence on the development of eating disorders.
Western society especially places a cultural emphasis on thinness that can influence how people view their bodies. A child's perception of external pressure to achieve the ideal body that is represented by the media predicts the child's body image dissatisfaction, body dysmorphic disorder, and an eating disorder.[5] "The cultural pressure on men and women to be 'perfect' is an important predisposing factor for the development of eating disorders".[6] Further, when women of all races base their evaluation of themselves upon what is considered the culturally ideal body, the incidence of eating disorders increases.

While there are many influences to how an individual processes their body image, the media does play a major role. Along with the media, parental influence, peer influence, and self-efficacy beliefs also play a large role in an individual's view of themselves. The way the media presents images can have a lasting effect on an individual's perception of their body image. Countless magazine ads and commercials depict thin celebrities who appear to gain nothing but attention from their looks. Unfortunately, this has led to the belief that in order to fit in, one must look a certain way. Televised beauty competitions such as the Miss America Competition contribute to the idea of what it means to be beautiful because competitors are evaluated on the basis of opinions about idealized beauty standards.

To try to address unhealthy body image in the fashion world, in 2015, France passed a law requiring models to be declared healthy by a doctor to participate in fashion shows. The law also requires re-touched images to be marked as such in magazines.

There is a relationship between "thin ideal" social media content and body dissatisfaction and eating disorders among young adult women, especially in the Western hemisphere. New research points to an "internalization" of distorted images online, as well as negative comparisons among young adult women. Most studies have been based in the United States, the United Kingdom, and Australia; these are places where the thin ideal is strong among women as well as the strive for the "perfect" body.

In addition to mere media exposure, there is an online "pro-eating disorder" community. Through personal blogs and Twitter, this community promotes eating disorders as a "lifestyle," and continuously posts pictures of emaciated bodies and tips on how to stay thin. The hashtag "#proana" (pro-anorexia) is a product of this community as well as images promoting weight loss, tagged with the term "thinspiration." According to social comparison theory, young women have a tendency to compare their appearance to others, which can result in a negative view of their own bodies and altering of eating behaviors, which in turn can develop disordered eating behaviors.

Socioeconomic status (SES) has been viewed as a risk factor for eating disorders, presuming that possessing more resources allows for an individual to actively choose to diet and reduce body weight. Some studies have also shown a relationship between increasing body dissatisfaction with increasing SES. However, once high SES has been achieved, this relationship weakens and, in some cases, no longer exists.



Figure 8.15.3: A myth about ballet is that you need to be thin to be a successful ballerina. This notion, paired with the competitive nature of ballet has resulted in some dancers developing eating disorders.

Another risk factor for developing eating disorders involve participating in some sports. Athletes and eating disorders tend to go hand in hand, especially in sports where weight is a competitive factor. Gymnastics, horseback riding, wrestling, bodybuilding, and dancing are just a few that fall into this category of weight-dependent sports. Eating disorders among individuals that participate in competitive activities, especially women, often lead to having physical and biological changes related to their weight that often mimic prepubescent stages. Oftentimes as women's bodies change, they lose their competitive edge, which leads them to take extreme measures to maintain their younger body shape. Men often struggle with binge eating followed by excessive exercise while focusing on building muscle rather than losing fat, but this goal of gaining muscle is just as much an eating disorder as obsessing over thinness. The following statistics taken from Susan Nolen-Hoeksema's book, (ab)normal psychology, show the estimated percentage of athletes that struggle with eating disorders based on the category of sport:

- aesthetic sports (dance, figure skating, and gymnastics)—35%
- weight dependent sports (judo and wrestling)—29%
- endurance sports (cycling, swimming, running)—20%
- technical sports (golf and high jumping)—14%
- ball game sports (volleyball and soccer)—12%

Although most of these athletes develop eating disorders to keep their competitive edge, others use exercise as a way to maintain their weight and figure. This is just as serious as regulating food intake for competition. Even though there is mixed evidence showing at what point athletes are challenged with eating disorders, studies show that regardless of competition level, all athletes are at higher risk for developing eating disorders than non-athletes, especially those that participate in sports where thinness is a factor.

Pressure from society is also seen within the homosexual community. Homosexual men are at greater risk of eating disorder symptoms than heterosexual men.

CROSS-CULTURAL STUDIES

Most of the cross-cultural studies use definitions from the DSM, which has been criticized as reflecting a Western cultural bias. Thus, assessments and questionnaires may not be constructed to detect some of the cultural differences associated with different disorders. Also, when looking at individuals in areas potentially influenced by Western culture, few studies have attempted to measure how much an individual has adopted the mainstream culture or retained the traditional cultural values of the area. Lastly, the majority of the cross-cultural studies on eating disorders and body image disturbances occurred in Western nations and not in the countries or regions being examined.

African Perspective

In the majority of many African communities, thinness is generally not seen as an ideal body type and most pressure to attain a slim figure may stem from influence or exposure to Western culture and ideology. Traditional African cultural ideals are reflected in the practice of some health professionals; in Ghana, pharmacists sell appetite stimulants to women who desire to, as Ghanaians stated, "grow fat." Girls are told that if they wish to find a partner and birth children they must gain weight. On the contrary, there are certain taboos surrounding a slim body image, specifically in West Africa. Lack of body fat is linked to poverty and HIV/AIDS.

However, the emergence of Western and European influence, specifically with the introduction of fashion and modeling shows and competitions, is changing certain views among body acceptance, and the prevalence of eating disorders has consequently increased. This acculturation is also related to how South Africa is concurrently undergoing rapid, intense urbanization. Such modern development is leading to cultural changes, and professionals cite rates of eating disorders in this region will increase with urbanization, specifically with changes in identity, body image, and cultural issues.

Other factors that are cited to be related to the increasing prevalence of eating disorders in African communities can be related to sexual conflicts, such as psychosexual guilt, first sexual intercourse, and pregnancy. Traumatic events that are related to both family (i.e., parental separation) and eating-related issues are also cited as possible effectors. Religious fasting, particularly around times of stress, and feelings of self-control are also cited as determinants in the onset of eating disorders.

China

In China as well as other Asian countries, Westernization, migration from rural to urban areas, after-effects of sociocultural events, and disruptions of social and emotional support are implicated in the emergence of eating disorders. In particular, risk factors for eating disorders include higher SES, preference for a thin body ideal, history of child abuse, high anxiety levels, hostile parental relationships, jealousy towards media idols, and above-average scores on the body dissatisfaction and interoceptive awareness sections of the Eating Disorder Inventory. Similarly to the West, researchers have identified the media as a primary source of pressures relating to physical appearance, which may even predict body change behaviors in males and females.

Hong Kong

From the early to mid-1990s, a variant form of anorexia nervosa was identified in Hong Kong. This variant form did not share features of anorexia in the West, notably “fat-phobia” and distorted body image. Patients attributed their restrictive food intake to somatic complaints, such as epigastric bloating, abdominal or stomach pain, or a lack of hunger or appetite. Compared to Western patients, individuals with this variant anorexia demonstrated bulimic symptoms less frequently and tended to have lower pre-morbid body mass index. This form disapproves the assumption that a “fear of fatness or weight gain” is the defining characteristic of individuals with anorexia nervosa.

India

In the past, the available evidence did not suggest that unhealthy weight loss methods and eating disordered behaviors are common in India as proven by stagnant rates of clinically diagnosed eating disorders. However, it appears that rates of eating disorders in urban areas of India are increasing based on surveys from psychiatrists who were asked whether they perceived eating disorders to be a “serious clinical issue” in India. In a 2015 survey, 23.5% of respondents believed that rates of eating disorders were rising in Bangalore, 26.5% claimed that rates were stagnant, and 42%, the largest percentage, expressed uncertainty.[7] It has been suggested that urbanization and SES are associated with increased risk for body weight dissatisfaction. However, due to the physical size of and diversity within India, trends may vary throughout the country.

Other Approaches

The psychodynamic view on eating disorders focuses on understanding the unconscious forces and motives that influence the disorder. Humanistic approaches take a positive approach to help the individual see themselves as more than their disorder. The humanistic approach attempts to take a positive lens during therapy. A therapist views their patient as an individual rather than a person who a member of a group that represents a category of an eating disorder. The shift in the individual focus allows the therapist and the client to take a holistic viewpoint to observe their behavior which allows for acknowledgment of their positive traits and behaviors, their ability to use their personal instincts to find healing, agency, and fulfillment within themselves. Humanistic therapy is beneficial to people with eating disorders because it is an effective therapeutic technique for people with depression, anxiety, addiction, and relationship issues including themselves. Given the positive focus and individualistic nature of the humanistic approach, people with eating disorders may be able to address their low self-esteem, lack of comfort with themselves or their lives, and a feeling of “wholeness,” and those who struggle with finding personal meaning in their lives.[8]

Interpersonal psychotherapy (IPT) is humanistic therapy, originally created to treat depression, and is also effective in treating eating disorders. It is similar to CBT, but the therapist does not attempt to uncover distorted thoughts systematically by giving homework or other assignments. However, as evidence arises during the course of therapy, the therapist calls attention to distorted thinking in relation to significant others. The therapist is warm, empathetic, and positive, working to help the patient resolve interpersonal issues that can also reduce eating disorder behaviors.

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8.16: Treating Sleep Disorders

Learning Objectives

- Examine sleep disorders and sleep disorder treatment from various psychological approaches

Biological Approaches

The biological approach to sleep disorders consists of medical interventions that include surgical, non-surgical, pharmacological, and nonpharmacological treatments. For example, treatment for sleep apnea consists of non-surgical as well as surgical treatments. Nonsurgical biological treatments include general behavioral changes such as weight loss, adjusting body positioning during sleep (avoiding lying on the back), or using mechanical devices to alleviate pressure, such as a CPAP or BIPAP.[1] While breathing devices can help, patients often find the devices frustrating and do not wear them often enough. Several surgical procedures (sleep surgery) are used to treat sleep apnea, although they are normally a third line of treatment for those who reject or are not helped by CPAP treatment or dental appliances. Surgical treatment for obstructive sleep apnea needs to be individualized to address all anatomical areas of obstruction; it may involve nasal, throat, tongue, or other surgeries.

Research suggests that REM sleep behavior disorder (RBD), may have a hereditary component to it. A total of 632 participants, half with RBD and half without, completed self-report questionnaires. The results of the study suggest that people with RBD are more likely to report having a first-degree relative with the same sleep disorder than people of the same age and sex that do not have the disorder.[2] More research needs to be conducted to gain further information about the hereditary nature of sleep disorders.

Medications and somatic treatments may provide the most rapid symptomatic relief from some sleep disturbances. Certain disorders like narcolepsy are best treated with prescription drugs such as modafinil. Others, such as chronic and primary insomnia, may be more amenable to behavioral interventions with more durable results. Major classes of prescription insomnia medications include benzodiazepine hypnotics, non-benzodiazepine hypnotics, and melatonin receptor agonists. All these medications have potential side effects, like drowsiness, dizziness, impaired sleep quality, or cognitive deficits, that may outweigh the benefits for some users.

Non-pharmacological treatments for sleep disorders such as insomnia include changing sleep habits and patterns, relaxation therapy, hypnosis, or alternative forms of medicine such as the usage of essential oils, acupuncture, or supplements.

In addressing sleep disorders and possible solutions, there is often a lot of buzz surrounding melatonin. Research suggests that melatonin is useful in helping people to fall asleep faster (decreased sleep latency), to stay asleep longer, and to experience improved sleep quality. In order to test this, a study was conducted that compared subjects that had taken melatonin to subjects that had taken a placebo pill in subjects with primary sleep disorders. Researchers assessed sleep onset latency, total minutes slept, and overall sleep quality in the melatonin and placebo groups to note the differences. In the end, researchers found that melatonin decreased sleep onset latency and increased total sleep time, but had an insignificant and inconclusive impact on the quality of sleep compared to a placebo group.[3]



Figure **8.16.1**: Acupuncture or other forms of alternative medicine may be used to treat sleep disorders.

Cognitive Approaches

The cognitive approach in treating sleep disorders focuses on reframing how people think about sleep and the act of sleeping to provoke change in their thoughts and sleeping habits. For example, cognitive therapy may be used to help some with insomnia to challenge their thoughts around sleep and reappraise their thoughts. Paradoxical intention is one technique that has a patient participate in their most feared behavior, such as staying awake, because one possible reason for their lack of sleep is something

akin to “performance anxiety” that they will be unable to sleep. By tackling their fear head-on, they can reduce this anxiety and get to sleep more easily.[4]

Mindfulness, meditation, relaxation training, guided imagery, and breathing exercises are all cognitive related techniques that aid in treating sleep disorders.

Behavioral Approaches

Stimulus control helps to build an association between the bedroom and sleep by limiting the type of activities allowed in the bedroom. An example of stimulus control is going to bed only when you are sleepy and getting out of bed if you’ve been awake for 20 minutes or more. This helps to break an unhealthy association between the bedroom and wakefulness. Sleep restriction involves a strict schedule of bedtimes and wake times and limits time in bed to only when a person is sleeping.[5]

Psychodynamic Approaches

The psychodynamic approach originated from the work of Sigmund Freud and emphasizes unconscious psychological processes. A systematic review found that traumatic childhood experiences (such as family conflict or sexual trauma) significantly increases the risk for a number of sleep disorders in adulthood, including sleep apnea, narcolepsy, and insomnia.

Freud not only emphasized childhood experiences in contributing to disorders, but believed that dreams represented an opportunity to gain access to the unconscious. By analyzing dreams, Freud thought people could increase self-awareness and gain valuable insight to help them deal with the problems they faced in their lives. Freud made distinctions between the manifest content and the latent content of dreams.

Manifest content is the actual content, or storyline, of a dream. **Latent content**, on the other hand, refers to the hidden meaning of a dream. For instance, if a woman dreams about being chased by a snake, Freud might have argued that this represents the woman’s fear of sexual intimacy, with the snake serving as a symbol of a man’s penis.

Freud was not the only theorist to focus on the content of dreams. The 20th century Swiss psychiatrist Carl Jung believed that dreams allowed us to tap into the **collective unconscious**. The collective unconscious, as described by Jung, is a theoretical repository of information he believed to be shared by everyone. According to Jung, certain symbols in dreams reflected universal archetypes with meanings that are similar for all people regardless of culture or location.

While Freud’s and Jung’s psychodynamic contributions lacked empirical support, their theories serve as an important foundation for the current and ongoing understanding of sleep through their connection between sleep stages and dreaming, which began the scientific process of examining consciousness of sleep and dreams, mental health, and the biology of sleep disorders.

Watch It

This video highlights how one sleep center studies and treats sleep disorders.



You can view the transcript for “Treatment and care of sleep disorders” here (opens in new window).

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8.17: Case Studies- Eating and Sleep Disorders

Learning Objectives

- Identify eating and sleep disorders in case studies

Case study: The Hollywood star

A female patient describes her childhood as being fairly normal and happy until the unexpected death of her father that she had difficulty coping with. Later in life, she became an actress and worked hard in her various roles, but due to the immense pressure of her public-facing career, she maintained a strict diet of only 300 calories a day while taking laxatives. She mentioned she was afraid of being "outed" by the press and tried to keep her eating habits under wraps. At one point she was diagnosed with lupus (but this was found to be incorrect). No other medical conditions were apparent, though it is noted that she does have comorbid depression. When she presented with symptoms, she was a very low weight for her age and height.

The case study above is based on the true story of Portia de Rossi who, in her book *Unbearable Lightness: A Story of Loss and Gain*, describes her struggle with anorexia and bulimia. Watch more of her story here.

Case Study: Taisha

Taisha is in her first semester of sophomore year at college and went to student counseling for help with increased depression and anxiety. She is a high-achieving student who's made the Dean's list every term but has been struggling with concentration and fatigue. She didn't do as well on her mid-terms as she'd hoped and has complaints that she can't sleep and she is constantly exhausted. She's resorted to OTC sleeping pills for sleep and energy drinks to keep her awake during the day. She knows this isn't a healthy routine, but doesn't know what else to do; if she doesn't take something to sleep, she barely gets two or three hours of rest each night.

Treatment

CBT would be a good first-line treatment for Taisha. Though medication may help in the short-term, a focus on cognitive processes and problem behaviors, as well as include stress management techniques could help improve the patient's symptoms. CBT has been demonstrated to be very effective in treating insomnia.

Case Study: Carson

Carson is a 50-year-old man who went to the doctor due to his wife's frustration with his snoring. He had a consistent history of loud and frequent snoring after he turned 40. After his 40th birthday, he changed jobs and therefore had to change his routine. Over a period of five years, he gained 40 pounds, reduced his exercise, and had increased stress. When Carson slept, his wife was frightened by the snoring noise, but also noticed the occasional pauses in his breathing during sleep, and at times he awakened to his snoring. During the day, he also reported frequent morning headaches and acid reflux. He had seasonal allergies, which resulted in occasional nasal congestion, yet he always woke up with a dry mouth and sore throat. Also, in his sleep, he had frequent bouts of movement and he tossed and turned. Carson constantly felt sleepy during the day, especially while reading and driving.[1]

Treatment

In this case, the best course of treatment, following a sleep study, would likely be to try a continuous positive airway pressure (CPAP) device, which involves the use of a mask that fits over the sleeper's nose and mouth, which mask is connected to a pump that forces air into the person's airways, helping them remain open. Other possible treatments (especially for those who are unable or unwilling to use a CPAP machine) include surgery of the nose, tongue, jaw, trachea, or uvula. These types of surgeries help expand airways and stop blockages so the patient can breathe better at night, and thus, sleep better.

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8.18: Putting It Together- Eating and Sleep Disorders



In this chapter, we learned about eating and sleep disorders and their treatments. It is important to note that neither eating and sleep disorders are choices. Rather, eating disorders are a mental illness, not a lifestyle choice. Eating disorders affect all human beings regardless of gender and age, despite socio-cultural myths that only women are affected.

There are many health contributors to sleep disorders, including physical traits (i.e., weight and nose and throat anatomy) and environmental influences (i.e., stress, trauma, caffeine, and shift work). Sleep disorders have a significant negative effect on a person's mental health and physical well being.

The treatment of eating and sleep disorders is complex, and effective treatment is often a combination of approaches including biological, cognitive, humanistic, behavioral. In summary, both eating and sleep disorders can be disruptive to a person's life and cause life-threatening complications threatening his or her medical stability. Treatment for both eating and sleep disorders is possible and people should seek help with expedience and with support from family and their communities.

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CHAPTER OVERVIEW

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9.3: Psychoactive Drugs and Substance Use Disorder
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9.1: Why It Matters- Substance-Related and Addictive Disorders

Why learn about substance-related and addictive disorders?



Substance-related disorders, including both substance dependence and substance abuse, can lead to large societal problems. Substance related-disorders are found to be greatest in individuals ages 18–25, with a higher likelihood occurring in men compared to women, and urban residents compared to rural residents. On average, general medical facilities hold 20% of patients with substance-related disorders that possibly lead to psychiatric disorders later on. Over 50% of individuals with substance-related disorders will often have a “dual diagnosis” where they are diagnosed with substance abuse as well as a psychiatric diagnosis, the most common being major depression, personality disorder, anxiety disorders, and dysthymia.

Substance abuse, also known as drug abuse, is a patterned use of a substance (drug) in which the user consumes the substance in amounts or with methods that are harmful to themselves or others. The substances used are often associated with levels of intoxication that alter judgment, perception, attention, and physical control, all of which are unrelated to medical effects. Much of the drug discussion is that the main abused substances are illegal drugs and alcohol; however, it is becoming more common that prescription drugs and tobacco are a prevalent problem.

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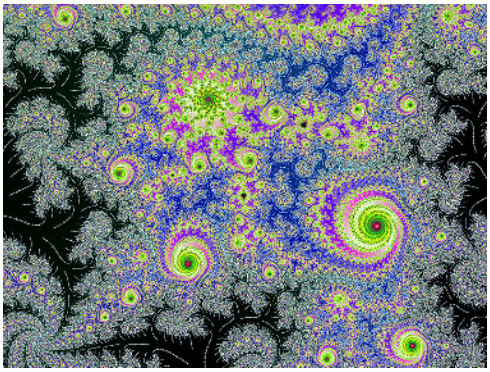
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9.2: Introduction to Substance-Related Disorders

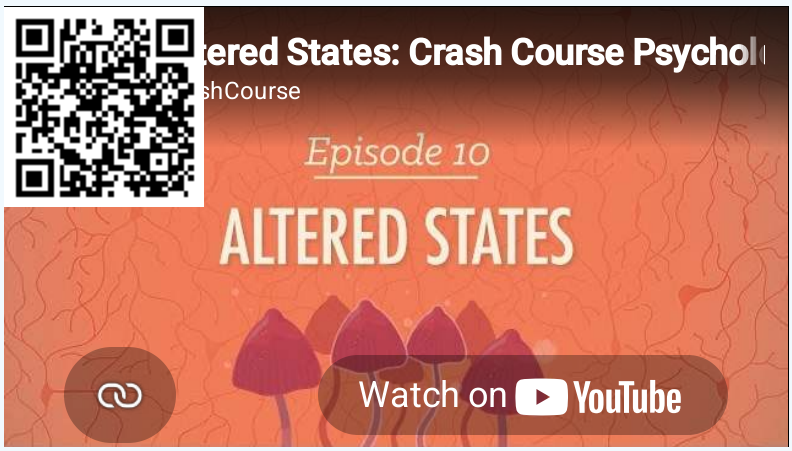
What you'll learn to do: examine the characteristics and complications of depressants, alcohol, and stimulants



In this section, you will learn about some of the general characteristics of commonly abused drugs, such as depressants, stimulants, and hallucinogens. Then you'll learn more about alcohol use and abuse and complications caused by stimulant use disorder.

Watch It

This CrashCourse video gives an excellent overview of altered states of consciousness caused by psychoactive drugs.



You can view the transcript "Altered States: Crash Course Psychology #10" here (opens in new window).

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9.3: Psychoactive Drugs and Substance Use Disorder

Learning Objectives

- Describe how substance abuse disorders are diagnosed

Substance Abuse Disorders

The *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5) is used by clinicians to diagnose individuals suffering from various psychological disorders. Drug use disorders are addictive disorders, and the criteria for specific substance (drug) use disorders are described in DSM-5. Drug categories listed in the DSM-5 include those for alcohol, opioids, depressants, stimulants, hallucinogens, inhalants, and other or unknown substances. A person who has a **substance use disorder** often uses more of the substance than they originally intended to and continues to use that substance despite experiencing significant adverse consequences. For individuals diagnosed with a substance use disorder, there is a compulsive pattern of drug use that is often associated with both physical and psychological dependence.

Physical dependence involves changes in normal bodily functions—the user will experience withdrawal from the drug upon cessation of use. In contrast, a person who has **psychological dependence** has an emotional, rather than physical, need for the drug and may use the drug to relieve psychological distress. **Tolerance** is linked to physiological dependence, and it occurs when a person requires more and more drug to achieve effects previously experienced at lower doses. Tolerance can cause the user to increase the amount of drug used to a dangerous level—even to the point of overdose and death.

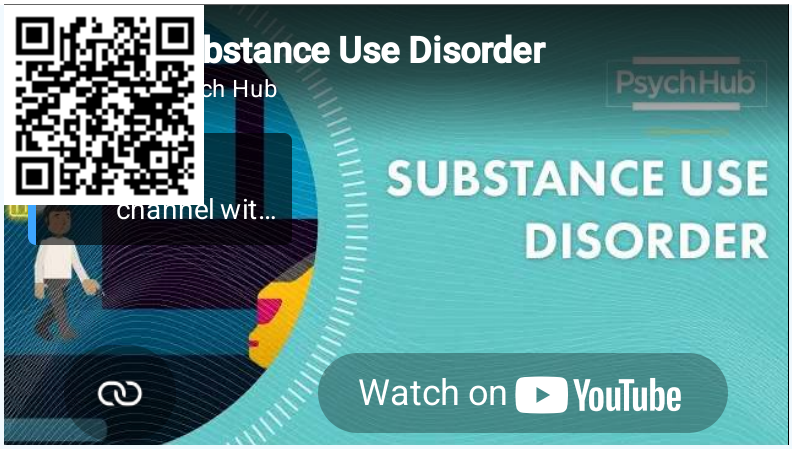
Drug **withdrawal** includes a variety of negative symptoms experienced when drug use is discontinued. Negative symptoms usually are opposite of the effects of the drug. For example, withdrawal from sedative drugs often produces unpleasant arousal and agitation. In addition to withdrawal, many individuals who are diagnosed with substance use disorders will also develop tolerance to these substances. Psychological dependence, or drug craving, is a recent addition to the diagnostic criteria for substance use disorder in DSM-5. Dependence levels are an important factor because we can develop tolerance and experience withdrawal from any number of drugs that we do not abuse. In other words, physical dependence in and of itself is of limited utility in determining whether or not someone has a substance use disorder.

In the DSM-5, each category of drugs has a use disorder listed, such as alcohol use disorder or opioid use disorder (further categorized as mild, moderate, or severe). Each drug category also lists an intoxication disorder, such as alcohol intoxication or cannabis intoxication, and a withdrawal disorder, such as alcohol withdrawal or opioid withdrawal. Intoxication disorders are attributed to recent drug use that causes significant and problematic psychological or behavioral changes. Withdrawal disorders are caused by clinically significant behavioral or psychological changes that occur after the cessation of drug abuse.

Watch It

All the substance use disorders we'll learn about in this module share similar characteristics, but vary depending on the drug that causes the addiction. In general terms, those who suffer from a substance use disorder share these common characteristics. They do the following:

- develop a tolerance for the drug
- experience withdrawal symptoms when the substance leaves their body
- take risks because of the substance
- spend a lot of time thinking about the drug
- stop doing meaningful things and withdraw from activities and others
- take more of the drug than they intended to
- crave the drug
- struggle to quit



You can view the transcript for "Substance Use Disorder" here (opens in new window).

Link to Learning

Read through this fascinating comic created by Stuart McMillen about psychologist Bruce Alexander's Rat Park study on addiction.

For more information on Bruce Alexander's study and a better understanding of addiction, listen to Johann Hari's TED Talk, "Everything you think you know about addiction is wrong."

Drug Categories

The effects of all **psychoactive drugs** occur through their interactions with our endogenous neurotransmitter systems. There are many psychoactive drugs, and their relationships are shown in Figure 1. Drugs can act as agonists or antagonists of a given neurotransmitter system. An agonist facilitates the activity of a neurotransmitter system, and antagonists impede neurotransmitter activity.

The main categories of drugs are **depressants**, **stimulants**, and **hallucinogens**. You will learn more about these types of drugs in the coming pages.

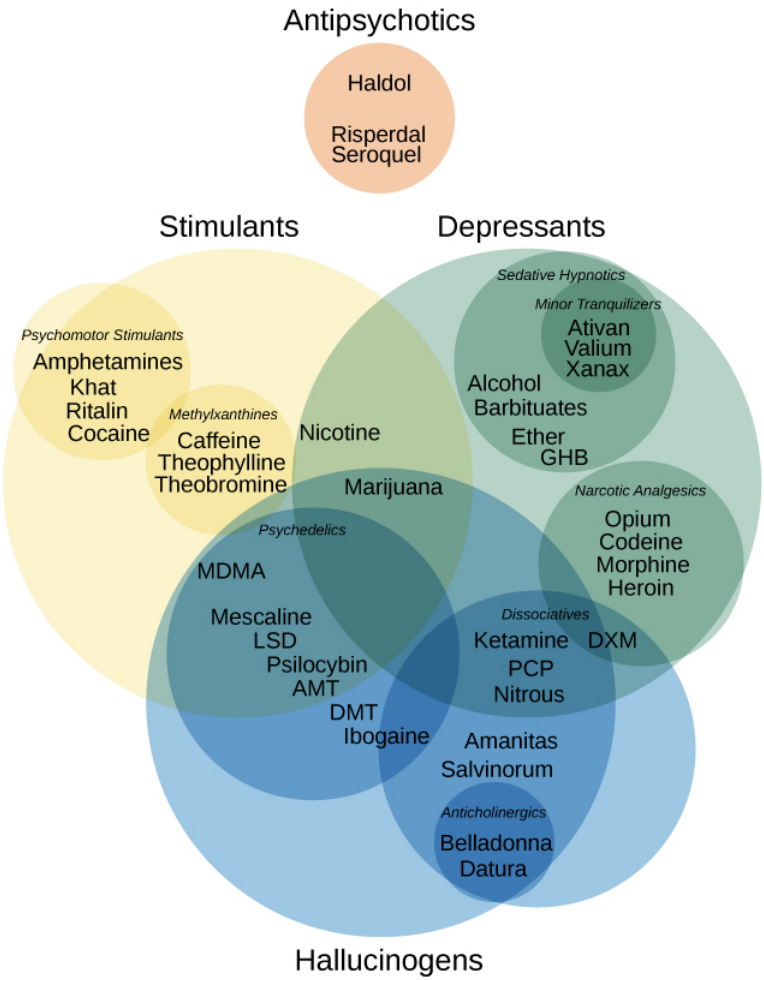


Figure **9.3.1**: This figure illustrates various drug categories and overlap among them. (credit: modification of work by Derrick Snider)

Glossary

depressant: drug that tends to suppress central nervous system activity

hallucinogen: one of a class of drugs that results in profound alterations in sensory and perceptual experiences, often with vivid hallucinations

physical dependence: changes in normal bodily functions that cause a drug user to experience withdrawal symptoms upon cessation of use

psychoactive drugs: chemical substances that alters brain functioning, causing changes to consciousness, perception, mood, and thoughts

psychological dependence: emotional, rather than a physical, need for a drug that may be used to relieve psychological distress

stimulant: drug that tends to increase overall levels of neural activity, including caffeine, nicotine, amphetamines, and cocaine

substance use disorder: DSM-5 diagnosis for those who show a compulsive pattern of drug use associated with both physical and psychological dependence

tolerance: state of requiring increasing quantities of the drug to gain the desired effect

withdrawal: variety of negative symptoms experienced when drug use is discontinued

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9.4: Alcohol-Related Disorders

Learning Objectives

- Explain how depressants impact nervous system activity, including the short- and long-term physiological and psychological effects of alcohol
- Describe the diagnostic features, development, and risk factors of alcohol use disorder, alcohol intoxication, and alcohol withdrawal

Understanding Alcohol

Ethanol, which we commonly refer to as alcohol, is in a class of psychoactive drugs known as depressants (Figure 1). A **depressant** is a drug that tends to suppress central nervous system activity. Other depressants include barbiturates and benzodiazepines. Barbiturates and benzodiazepines share in common their ability to serve as agonists of the gamma-aminobutyric acid (GABA) neurotransmitter system. Because gamma-aminobutyric acid (GABA) has a quieting effect on the brain, gamma-aminobutyric acid (GABA) agonists also have a quieting effect; these types of drugs are often prescribed to treat both anxiety and insomnia.

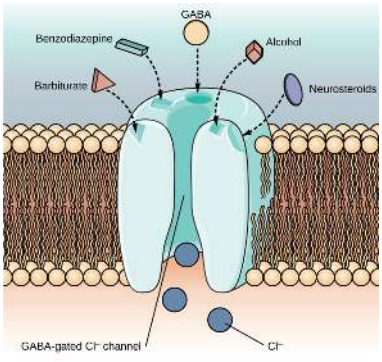


Figure **9.4.1**: The GABA-gated chloride (Cl-) channel is embedded in the cell membrane of certain neurons. The channel has multiple receptor sites where alcohol, barbiturates, and benzodiazepines bind to exert their effects. The binding of these molecules opens the chloride channel, allowing negatively-charged chloride ions (Cl-) into the neuron's cell body. Changing its charge in a negative direction pushes the neuron away from firing; thus, activating a GABA neuron has a quieting effect on the brain.

Acute alcohol administration results in a variety of changes to consciousness. Alcohol intoxication is measured for legal and medical uses in terms of blood alcohol content (BAC). A blood alcohol content (BAC) of 0.10 (0.10% or one-tenth of 1%) means that there are 0.10 grams of alcohol for every 100 milliliter of blood. While a blood alcohol content (BAC) of 0.0 is sober, in the United States, a BAC of 0.08 is legally intoxicated, and any level above that is considered very impaired. BAC levels above 0.40 are potentially fatal. At rather low doses, alcohol use is associated with feelings of euphoria. As the dose increases, people report feeling sedated. Generally, alcohol is associated with decreases in reaction time and visual acuity, lowered levels of alertness, and reduction in behavioral control. With excessive alcohol use, a person might experience a complete loss of consciousness and/or difficulty remembering events that occurred during a period of intoxication (McKim & Hancock, 2013). In addition, if a pregnant woman consumes alcohol, her infant may be born with a cluster of birth defects and symptoms collectively called fetal alcohol spectrum disorder (FASD) or fetal alcohol syndrome (FAS).

With repeated use of many central nervous system depressants, such as alcohol, a person becomes physically dependent upon the substance and will exhibit signs of both tolerance and withdrawal. Psychological dependence on these drugs is also possible. Therefore, the abuse potential of central nervous system depressants is relatively high.

Drug withdrawal is usually an aversive experience, and it can be a life-threatening process in individuals who have a long history of very high doses of alcohol and/or barbiturates. A history of high doses of substances is of such concern that people who are trying to overcome addiction to these substances should only do so under medical supervision.

Alcohol-Related Disorders

Alcoholism, more appropriately referred to as **alcohol use disorder** (**AUD**), is, broadly, any drinking of alcohol that results in mental and/or physical health problems ranging anywhere from mild and imperceptible without special testing to severe and grossly overt. The disorder was previously divided into two types: alcohol abuse and alcohol dependence. The DSM–5 integrates the two DSM–4 disorders, alcohol abuse and alcohol dependence, into a single disorder called alcohol use disorder (AUD) with mild, moderate, and severe sub-classifications.

In a medical context, alcoholism is said to exist when two or more of the following conditions are present: a person drinks large amounts of alcohol over a long time period, a person has difficulty cutting down, acquiring and drinking alcohol takes up a great deal of his or her time, alcohol is strongly desired, usage results in not fulfilling responsibilities, usage results in social problems, usage results in health problems, usage results in risky situations, withdrawal occurs when stopping, and alcohol tolerance has occurred with use. Under DSM–5, anyone meeting any two of the 11 criteria (shown in Table 1) during the same 12-month period would receive a diagnosis of AUD. The severity of AUD—mild, moderate, or severe—is based on the number of criteria met.

The DSM-5 classifies alcoholism as alcohol-use disorder, alcohol intoxication, and alcohol withdrawal. **Alcohol intoxication** is diagnosed by the recent ingestion of alcohol and clinically significant problematic behavioral or psychological changes (e.g., inappropriate sexual or aggressive behavior, mood lability, impaired judgment) that developed during, or shortly after, alcohol ingestion. One (or more) of the following signs or symptoms develop during, or shortly after, alcohol use:

- slurred speech
- incoordination
- unsteady guilt
- nystagmus
- impairment in attention or memory
- stupor or coma

Alcohol intoxication is a risk factor in some cases of catastrophic injury, in particular for unsupervised recreational activity. A study in the province of Ontario, Canada, based on epidemiological data from 1986, 1989, 1992, and 1995 revealed that 79.2% of the 2,154 catastrophic injuries recorded for the study were preventable, of which 346 (17%) involved alcohol consumption. Alcohol intoxication is the negative health effects due to the recent drinking of ethanol (alcohol). When severe, it may become a medical emergency. Some effects of alcohol intoxication, such as euphoria and lowered social inhibition, are central to alcohol's desirability.

Alcohol withdrawal describes a set of symptoms that can occur following a reduction in alcohol use after a period of excessive use. Symptoms typically include anxiety, shakiness, sweating, vomiting, fast heart rate, and a mild fever. More severe symptoms may include seizures, seeing or hearing things that others do not, and delirium tremens (DTs). Delirium tremens (DTs) may include alcohol hallucinosis in which patients have transient visual, auditory, or tactile hallucinations, but are otherwise clear. Withdrawal seizures are seizures that occur within 48 hours of alcohol cessations and occur either as a single generalized tonic-clonic seizure or as a brief episode of multiple seizures. Symptoms typically begin around six hours following the last drink, are worst at 24 to 72 hours, and improve by seven days. Delirium tremens describe a hyperadrenergic state, disorientation, tremors, diaphoresis, and impaired attention/consciousness. Alcohol withdrawal may occur in those who are alcohol dependent. This may occur following a planned or unplanned decrease in alcohol intake.

In the Western world, about 15% of people have problems with alcoholism at some point in time. About half of people with alcoholism will develop withdrawal symptoms upon reducing their use, with 4% developing severe symptoms. Among those with severe symptoms, up to 15% die. Benzodiazepines are effective for the management of symptoms as well as the prevention of seizures. Certain vitamins are also an important part of the management of alcohol withdrawal syndrome. In those with lesser symptoms, treatment at home may be possible with daily visits with a health care provider.

Diagnostic Criteria for Alcohol Use Disorder

Table 1. DSM–5 Criteria for Alcohol Use Disorder.

In the past year, have you:	
The presence of at least 2 of these symptoms indicates **Alcohol Use Disorder (AUD).** The severity of the AUD is defined as: **Mild**: The presence of 2 to 3 symptoms **Moderate**: The presence of 4 to 5 symptoms **Severe**: The presence of 6 or more symptoms	Had times when you ended up drinking more, or longer, than you intended?
	More than once wanted to cut down or stop drinking, or tried to, but couldn't?
	Spent a lot of time drinking? Or being sick or getting over other aftereffects?
	Wanted a drink so badly you couldn't think of anything else
	Found that drinking—or being sick from drinking—often interfered with taking care of your home or family? Or caused job troubles? Or school problems?
	Continued to drink even though it was causing trouble with your family or friends?
	Given up or cut back on activities that were important or interesting to you, or gave you pleasure, in order to drink?
	More than once gotten into situations while or after drinking that increased your chances of getting hurt (such as driving, swimming, using machinery, walking in a dangerous area, or having unsafe sex)?
	Continued to drink, even though it was making you feel depressed or anxious or adding to another health problem? Or after having had a memory blackout?
	Had to drink much more than you once did to get the effect you want? Or found that your usual number of drinks had much less effect than before?
	Found that when the effects of alcohol were wearing off, you had withdrawal symptoms, such as trouble sleeping, shakiness, restlessness, nausea, sweating, a racing heart, or a seizure? Or sensed things that were not there?

Alcohol use can affect all parts of the body, but it particularly affects the brain, heart, liver, pancreas, and immune system. Alcoholism can result in mental illness, delirium tremens, Wernicke-Korsakoff syndrome (memory loss), irregular heartbeat, an impaired immune response, liver cirrhosis, and increased cancer risk. Drinking during pregnancy can result in fetal alcohol syndrome disorders. Women are generally more sensitive than men to the harmful effects of alcohol, primarily due to their smaller body weight, lower capacity to metabolize alcohol, and higher proportion of body fat. In a small number of individuals, prolonged, severe alcohol abuse ultimately leads to frank dementia.

Etiology

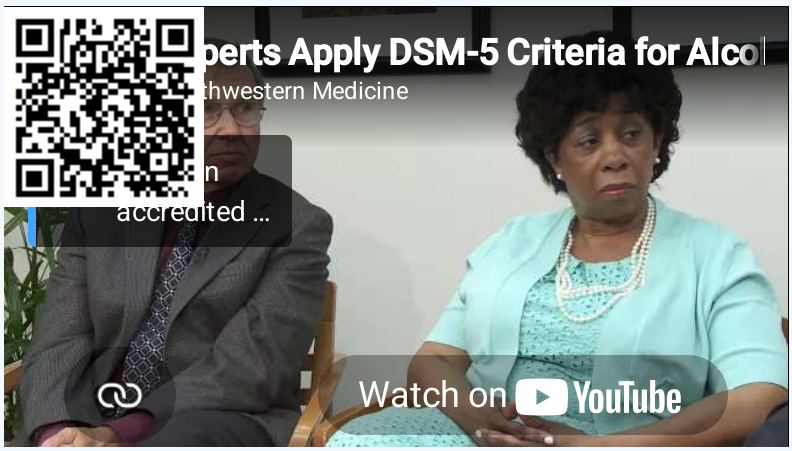
Environmental factors and genetics are two factors affecting risk for alcoholism, with about half the risk attributed to each. Someone with a parent or sibling with alcoholism is three to four times more likely to become an alcoholic themselves, but only a minority of them do. Environmental factors include social, cultural, and behavioral influences. High stress levels and anxiety, as well as alcohol's inexpensive cost and easy accessibility, increase the risk. People may continue to drink partly to prevent or improve symptoms of withdrawal. After a person stops drinking alcohol, they may experience a low level of withdrawal lasting for

months. Medically, alcoholism is considered both a physical and mental illness. Questionnaires are usually used to detect possible alcoholism. Further information is then collected to confirm the diagnosis.

Prevention of alcoholism may be attempted by regulating and limiting the sale of alcohol (particularly to minors), taxing alcohol to increase its cost, and providing education and inexpensive treatment. Prohibition did not work. Treatment of alcoholism may take several forms. Due to medical problems that can occur during withdrawal, alcohol detoxification should be carefully controlled. One common method involves the use of benzodiazepine medications, such as diazepam. These can be either given while admitted to a health care institution or occasionally while a person remains in the community with close supervision. Mental illness or other addictions may complicate treatment. After detoxification, various forms of individual or group therapy or support groups can help keep a person from returning to drinking. One commonly used form of support is the group Alcoholics Anonymous. The medications acamprosate, disulfiram, or naltrexone may also be used to help prevent further drinking.

Watch It

In this video, a medical team discusses alcohol use disorder and we are introduced to a man who explains his own experiences and struggles with alcohol.



You can view the transcript for “Experts Apply DSM-5 Criteria for Alcohol Use Disorder” here (opens in new window).

Epidemiology

The World Health Organization has estimated that as of 2016, there were 380 million people with alcoholism worldwide (5.1% of the population over 15 years of age). As of 2015 in the United States, about 17 million adults (7%) and 0.7 million (2.8%) of those from 12 to 17 years of age are affected. Alcoholism is most common among males and young adults. Geographically, it is least common in Africa (1.1% of the population) and has the highest rates in Eastern Europe (11%). Alcoholism directly resulted in 139,000 deaths in 2013, up from 112,000 deaths in 1990. A total of 3.3 million deaths (5.9% of all deaths) are believed to be due to alcohol. Alcoholism reduces a person’s life expectancy by approximately ten years.

Glossary

depressant: drug that tends to suppress central nervous system activity

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9.5: Stimulant-Related Disorders

Learning Objectives

- Identify stimulants and describe how they affect the brain and body
- Describe the diagnostic features and development of stimulant use disorder, stimulant intoxication, and stimulant withdrawal

Stimulants are drugs that tend to increase overall levels of neural activity. Many of these drugs act as agonists of the dopamine neurotransmitter system. Dopamine activity is often associated with reward and craving; therefore, drugs that affect dopamine neurotransmission often have abuse liability. Drugs in this category include cocaine, amphetamines (including methamphetamine), cathinones (i.e., bath salts), MDMA (ecstasy), nicotine, and caffeine.

The use of stimulants in humans causes rapid weight loss; cardiovascular effects such as an increase in heart rate, respirations, and blood pressure; emotional or mental side effects such as paranoia, anxiety, and aggression; and a change in the survival pathway known as the reward/reinforcement pathway in our brain. An increase in energy, a reduced appetite, increased alertness and a boost in confidence are all additional side effects of stimulant use when introduced to the body.

Cocaine can be taken in multiple ways. While many users snort cocaine, intravenous injection and ingestion are also common. The freebase version of cocaine, known as crack, is a potent, smokable version of the drug. Like many other stimulants, cocaine agonizes the dopamine neurotransmitter system by blocking the reuptake of dopamine in the neuronal synapse. Cocaine's most dangerous side effects include the following:

- increased body temperature (high fever)
- irregular heart rate or rapid heart rate
- high Blood Pressure
- increased risk of heart attack
- strokes
- sudden death from cardiac arrest
- sudden aggression
- sudden extreme paranoia
- feeling, seeing, or hearing things that are not really there (hallucinations), including feeling like there are insects under the skin (this is called formication or coke bugs)

Dig Deeper: Crack Cocaine

Crack (Figure 9.5.1) is often considered to be more addictive than cocaine itself because it is smokable and reaches the brain very quickly. Crack is often less expensive than other forms of cocaine; therefore, it tends to be a more accessible drug for individuals from impoverished segments of society. During the 1980s, many drug laws were rewritten to punish crack users more severely than cocaine users. This led to discriminatory sentencing with low-income, inner-city minority populations receiving the harshest punishments. The wisdom of these laws has recently been called into question, especially given research that suggests crack may not be more addictive than other forms of cocaine, as previously thought (Haasen & Krausz, 2001; Reinerman, 2007).



Figure 9.5.1: Crack rocks like these are smoked to achieve a high. Compared with other routes of administration, smoking a drug allows it to enter the brain more rapidly, which can often enhance the user's experience. (credit: modification of work by U.S. Department of Justice)

Amphetamines have a mechanism of action quite similar to cocaine in that they block the reuptake of dopamine in addition to stimulating its release (Figure 9.5.2). While amphetamines are often abused, they are also commonly prescribed to children diagnosed with attention deficit hyperactivity disorder (ADHD). It may seem counterintuitive that stimulant medications are prescribed to treat a disorder that involves hyperactivity, but the therapeutic effect comes from increases in neurotransmitter activity within certain areas of the brain associated with impulse control.

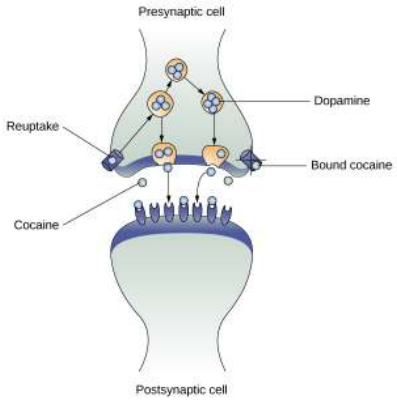


Figure 9.5.2: As one of their mechanisms of action, cocaine and amphetamines block the reuptake of dopamine from the synapse into the presynaptic cell.

In recent years, methamphetamine (meth) use has become increasingly widespread. **Methamphetamine** is a type of amphetamine that can be made from ingredients that are readily available (e.g., medications containing pseudoephedrine, a compound found in many over-the-counter cold and flu remedies). Despite recent changes in laws designed to make obtaining pseudoephedrine more difficult, methamphetamine continues to be an easily accessible and relatively inexpensive drug option (Shukla, Crump, & Chrisco, 2012).

The cocaine, amphetamine, cathinones, and MDMA users seek a **euphoric high**, feelings of intense elation and pleasure, especially in those users who take the drug via intravenous injection or smoking. Repeated use of these stimulants can have significant adverse consequences. Users can experience physical symptoms that include nausea, elevated blood pressure, and increased heart rate. In addition, these drugs can cause feelings of anxiety, hallucinations, and paranoia (Fiorentini et al., 2011). Normal brain functioning is altered after repeated use of these drugs. For example, repeated use can lead to overall depletion among the monoamine neurotransmitters (dopamine, norepinephrine, and serotonin). People may engage in compulsive use of these stimulant substances in part to try to re-establish normal levels of these neurotransmitters (Jayanthi & Ramamoorthy, 2005; Rothman, Blough, & Baumann, 2007).

Caffeine is another stimulant drug. While it is probably the most commonly used drug in the world, the potency of this particular drug pales in comparison to the other stimulant drugs described in this section. Generally, people use caffeine to maintain increased levels of alertness and arousal. Caffeine is found in many common medicines (such as weight loss drugs), beverages, foods, and even cosmetics (Herman & Herman, 2013). While caffeine may have some indirect effects on dopamine neurotransmission, its primary mechanism of action involves antagonizing adenosine activity (Porkka-Heiskanen, 2011).

While caffeine is generally considered a relatively safe drug, high blood levels of caffeine can result in insomnia, agitation, muscle twitching, nausea, irregular heartbeat, and even death (Reissig, Strain, & Griffiths, 2009; Wolt, Ganetsky, & Babu, 2012). In 2012, Kromann and Nielson reported on a case study of a 40-year-old woman who suffered significant ill effects from her use of caffeine. The woman used caffeine in the past to boost her mood and to provide energy, but over the course of several years, she increased her caffeine consumption to the point that she was consuming three liters of soda each day. Although she had been taking a prescription antidepressant, her symptoms of depression continued to worsen and she began to suffer physically, displaying significant warning signs of cardiovascular disease and diabetes. Upon admission to an outpatient clinic for treatment of mood disorders, she met all the diagnostic criteria for substance dependence and was advised to dramatically limit her caffeine intake. Once she was able to limit her use to less than 12 ounces of soda a day, both her mental and physical health gradually improved. Despite the prevalence of caffeine use and the large number of people who confess to suffering from caffeine addiction, this was the first published description of soda dependence appearing in scientific literature.

Nicotine is highly addictive, and the use of tobacco products is associated with increased risks of heart disease, stroke, and a variety of cancers. Nicotine exerts its effects through its interaction with acetylcholine receptors. Acetylcholine functions as a neurotransmitter in motor neurons. In the central nervous system, it plays a role in arousal and reward mechanisms. Nicotine is most commonly used in the form of tobacco products like cigarettes or chewing tobacco; therefore, there is a tremendous interest in developing effective smoking cessation techniques. To date, people have used a variety of nicotine replacement therapies in addition to various psychotherapeutic options in an attempt to discontinue their use of tobacco products. In general, smoking cessation programs may be effective in the short term, but it is unclear whether these effects persist (Cropley, Theadom, Pravettoni, & Webb, 2008; Levitt, Shaw, Wong, & Kaczorowski, 2007; Smedslund, Fisher, Boles, & Lichtenstein, 2004).

Link to Learning

To learn more about some of the most commonly abused prescription and street drugs, check out the Commonly Abused Drugs Chart and the Commonly Abused Prescription Drugs Chart from the National Institute on Drug Abuse.

Stimulant-Related Disorders

Stimulant use disorder is a type of substance use disorder that involves the abuse of stimulants. Stimulants are the most widely used drugs in the world today; approximately 200 million Americans have used some type of stimulant in the past year alone. Caffeine and nicotine are the most popular stimulants used today, with roughly 400 million cups of coffee consumed daily and 36.5 million current cigarette smokers, according to a 2015 study conducted by the Centers for Disease Control and Prevention.

Stimulant disorders described in the DSM-5 include cocaine use disorder, amphetamine-use disorder, and tobacco-use disorder. While not listed as its own disorder, certain isolated conditions related to caffeine are recognized in the DSM-5's "substance-related" section: caffeine intoxication, caffeine withdrawal, and other caffeine-induced disorders.

Diagnostic Criteria

A stimulant use disorder is diagnosed as a pattern of amphetamine-type substance, cocaine, or other stimulant use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. The stimulant is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control stimulant use.
3. A great deal of time is spent in activities necessary to obtain the stimulant, use the stimulant, or recover from its effects.
4. The craving, or a strong desire or urge to use the stimulant is present.
5. Recurrent stimulant use results in a failure to fulfill major role obligations at work, school, or home.
6. Stimulant use is continued despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the stimulant.
7. Important social, occupational, or recreational activities are given up or reduced because of stimulant use.
8. Stimulant use is recurrent in situations in which it is physically hazardous.
9. Stimulant use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the stimulant.
10. Tolerance is present, as defined by either of the following:

- **a**. a need for markedly increased amounts of the stimulant to achieve intoxication or desired effect
- **b**. a markedly diminished effect with continued use of the same amount of the stimulant
- **Note**: This criterion is not considered to be met for those taking stimulant medications solely under appropriate medical supervision, such as medications for attention-deficit/hyperactivity disorder or narcolepsy.

11. Withdrawal is manifested by either of the following:
    - **a**. the characteristic withdrawal syndrome for the stimulant
    - **b**. stimulant (or a closely related substance) used to relieve or avoid withdrawal symptoms
    - **Note**: This criterion is not considered to be met for those taking stimulant medications solely under appropriate medical supervision, such as medications for attention-deficit/hyperactivity disorder or narcolepsy.

If a substance is used over a long period of time and the user becomes dependent upon it, a substance abuse issue begins to appear. Substance abuse may lead to substance dependence and with time, addiction. Both mental and physiological dependence requires the development of tolerance leading to withdrawal symptoms.

Stimulant intoxication is marked by clinically significant problematic behavioral or psychological changes (e.g., euphoria or affective blunting; changes in sociability; hypervigilance; interpersonal sensitivity; anxiety, tension, or anger; stereotyped behaviors; or impaired judgement) that developed during, or shortly after, use of a stimulant. **Cocaine intoxication** refers to the deleterious effects of cocaine on the body. These effects can develop over time with regular use or can be the result of acute intoxication due to a single intake of the substance.

Stimulant withdrawal is marked by the following diagnostic criteria:

A. The cessation of (or reduction in) prolonged amphetamine-type substance, cocaine, or other stimulant use occurs.

B. Dysphoric mood and two (or more) of the following physiological changes develop within a few hours to several days after Criterion A:

- Fatigue.
- Vivid, unpleasant dreams.
- Insomnia or hypersomnia.
- Increased appetite.
- Psychomotor retardation or agitation.

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication or withdrawal from another substance.

Note: Amphetamine, cocaine, or other stimulant withdrawal can only occur in the presence of a moderate or severe amphetamine, cocaine, or other stimulant use disorder. For example, cocaine withdrawal happens when a person who uses a lot of cocaine stops using cocaine. It can also happen when a person who uses a lot of cocaine starts using less cocaine than they did before. When a person who is used to using cocaine stops right away, they will go through what is called a “crash” along with many of other cocaine withdrawal symptoms, like

- paranoia
- depression
- exhaustion (feeling very tired)
- anxiety
- itching
- mood swings (quick changes in emotions
- irritability (feeling easily annoyed)
- fatigue (feeling tired), sleeping many hours
- insomnia (trouble sleeping)
- a very strong craving for more cocaine
- nausea and vomiting

Withdrawal symptoms can last for days, weeks, months, and on rare occasions, years, depending on the frequency and dosages used by the individual. Unless intensive medical and psychological treatment is sought after, there is a very high likelihood of relapse among the user.

Tobacco-Related Disorders



Figure 9.5.3: First-time nicotine users develop a dependence about 32% of the time.

Tobacco use disorder, as described in the DSM-5, is a problematic pattern of tobacco use leading to clinically significant impairment or distress, as manifested by at least two of the 11 symptoms listed in the diagnostic criteria above.

Nicotine dependence is a state of dependence upon nicotine. Nicotine dependence is a chronic, relapsing disease defined as a compulsive craving to use the drug, despite harmful social consequences. Tolerance is another component of drug dependence. Nicotine dependence develops over time as a person continues to use nicotine. Nicotine dependence is a serious public health concern due to it being one of the leading causes of avoidable deaths worldwide.

First-time nicotine users develop a dependence about 32% of the time. There are approximately 976 million smokers in the world. There is an increased frequency of nicotine dependence in people with anxiety disorders. Nicotine is a parasympathomimetic stimulant that attaches to nicotinic acetylcholine receptors in the brain. Neuroplasticity within the brain's reward system occurs as a result of long-term nicotine use, leading to nicotine dependence. There are genetic risk factors for developing dependence. For instance, genetic markers for a specific type of nicotinic receptor (the α5-α3-β4 nicotine receptors) have been linked to increased risk for dependence. Evidence-based medicine can double or triple a smoker's chances of quitting successfully.

Caffeine-Related Disorders

Consumption of one to one-and-a-half grams (1,000–1,500 mg) per day is associated with a condition known as *caffeinism*. Caffeinism usually combines caffeine dependency with a wide range of unpleasant symptoms including nervousness, irritability, restlessness, insomnia, headaches, and palpitations after caffeine use.

Caffeine overdose can result in a state of central nervous system over-stimulation known as caffeine intoxication, a clinically significant temporary condition that develops during, or shortly after, the consumption of caffeine. This syndrome typically occurs only after ingestion of large amounts of caffeine, well over the amounts found in typical caffeinated beverages and caffeine tablets (e.g., more than 400–500 mg at a time).

Caffeine intoxication may be diagnosed if five (or more) of the following symptoms develop after recent consumption of caffeine: restlessness, nervousness, excitement, insomnia, flushed face, diuresis (increased production of urine), gastrointestinal disturbance, muscle twitching, rambling flow of thought and speech, tachycardia (increased heart rate) or cardiac arrhythmia, periods of inexhaustibility, and psychomotor agitation.

Treatment for Stimulant Use Disorders

Treatments for stimulant use disorders first involve addressing any emergency issues, such as treating cocaine-associated high body temperature with a benzodiazepine and physical cooling.

Twelve-step programs like Cocaine Anonymous (modeled on Alcoholics Anonymous and Narcotics Anonymous) have been widely used to help people addicted to cocaine, though some other psychotherapies have proven more helpful. Common therapies include motivational interviewing, contingency management, a community reinforcement approach, and cognitive behavioral therapy. Medications that have been studied to treat cocaine withdrawal include acetylcysteine, baclofen, bupropian, vanoxerine, and vigabatrin. Since there is no antidote nor reversal agent for caffeine intoxication, treatment of mild caffeine intoxication is directed toward symptom relief; severe intoxication may require peritoneal dialysis, hemodialysis, or hemofiltration.

Motivational interviewing (MI) is a treatment approach that helps individuals overcome ambivalent feelings and insecurities. In the process, individuals become motivated to change their behavior and reduce or stop their stimulant use. Five underlying principles guide how providers should interact with clients while using this practice:

- Express empathy through reflective listening.
- Identify discrepancies between a client's goals or values and their current behavior.
- Avoid arguments and direct confrontations with a client.
- Adjust to a client's resistance rather than opposing it directly.
- Support self-efficacy and optimism.

Studies indicate that use of MI for people with stimulant use disorders was associated with reductions in the number of days of stimulant use and the amount of stimulant used per day.

Contingency management (CM) is a type of behavioral therapy grounded in the principles of operant conditioning. Operant conditioning is a method of learning in which desired behaviors are reinforced with prizes, privileges, or cash. For treatment of stimulant use disorders, incentivized behaviors might include attendance at treatment sessions, adherence to prescribed medications for other health conditions, or provision of stimulant-negative urine specimens.

In a clinical setting, reinforcement is often provided in the form of vouchers that can be exchanged for retail goods and services. It may also include access to certain privileges, the opportunity to win a prize, or even direct cash payments. While contingency management (CM) may be structured in several different ways, two widely used approaches include

- the Fishbowl Method (popularized by Dr. Nancy Petry) where clients who have earned an incentive draw a token from a "fishbowl" for a chance to win a prize of varying value.
- voucher-based reinforcement therapy (popularized by Dr. Stephen Higgins) where clients earn vouchers for completion of desired behaviors with the level of vouchers increasing according to an escalating schedule of reinforcement.

Studies included in this evidence review demonstrated that the use of contingency management (CM) for people with stimulant use disorders was associated with reductions in the number of days of stimulant use, stimulant cravings, new stimulant use, and HIV-risk behaviors.

One behavioral therapy approach that is commonly used in combination with CM is the **community reinforcement approach** (CRA). This treatment was originally developed by Nathan Azrin and his colleagues for alcohol use disorder, and was later adapted for stimulant use disorder, particularly cocaine use. The goal of community reinforcement approach (CRA) is to identify behaviors reinforcing stimulant use and make a substance-free lifestyle more rewarding than one that includes drugs and alcohol. Community reinforcement approach (CRA) includes multiple elements such as analyzing clients' substance use, relationship counseling, vocational guidance, and job skills training. CRA therapy also focuses on building social and drug refusal skills. Ultimately, clients are encouraged to make substantial behavioral changes, engage in new recreational activities, and develop new social networks. When used in combination with CM, clients who demonstrate positive behaviors such as drug abstinence, are typically provided material incentives to encourage skills development to build a rewarding substance-free life.

Cognitive behavioral therapy (CBT) is a short-term, goal-oriented psychotherapy treatment that enables individuals to understand their current problems, challenges, and experiences in order to change their behaviors and patterns of thinking. CBT helps clients develop accurate assessments of circumstances and their feelings so they can develop realistic strategies. CBT is used also to address depressive cognitions and other cognitive distortions associated with depression, generalized anxiety disorders, and substance use disorders. Through CBT, people with stimulant use disorders are trained to evaluate faulty patterns of thinking, actions, and negative feelings associated with their drug use. CBT is tailored to the needs of the individual, with the goals of each therapy session uniquely based on the client's experiences with stimulant use and personal circumstances.

Glossary

euphoric high: feelings of intense elation and pleasure from drug use

methamphetamine: type of amphetamine that can be made from pseudoephedrine, an over-the-counter drug; widely manufactured and abused

stimulant: drug that tends to increase overall levels of neural activity; includes caffeine, nicotine, amphetamines, and cocaine

stimulant intoxication: clinically significant problematic behavioral or psychological changes that develop during, or shortly after, the use of a stimulant

stimulant withdrawal: marked by significant behavioral and psychological changes following the cessation of (or reduction in) prolonged amphetamine-type substance, cocaine, or other stimulant use

stimulant use disorder: a type of substance use disorder that involves the abuse of stimulants

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9.6: Introduction to Opioid and Hallucinogen-Related Disorders

What you'll learn to do: examine the characteristics and complications of opioids, hallucinogens, cannabis, phencyclidine, inhalants, and similar disorders



In this section, you will continue to learn about substance use disorders, specifically opioids, such as commonly abused pain relievers, heroin, and hallucinogens.

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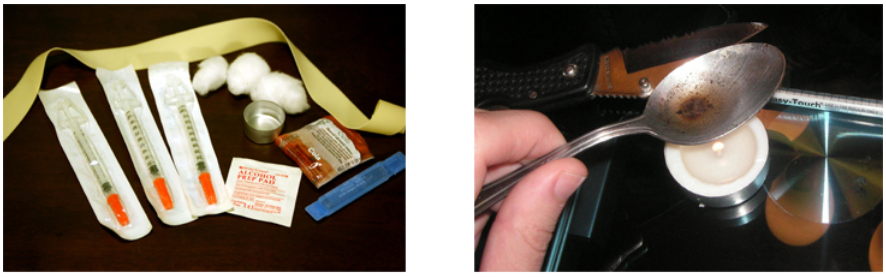
9.7: Opioid-Related Disorders

Learning Objectives

- Identify opioids and describe how they impact the brain and behavior
- Describe the diagnostic features, development, and risk factors of opioid use disorder, opioid intoxication, and opioid withdrawal
- Describe sedative, hypnotic, or anxiolytic-related disorders

An **opioid** is one of a category of drugs that includes heroin, morphine, methadone, and codeine. Opioids have analgesic properties: that is, they decrease pain. Humans have an endogenous opioid neurotransmitter system—the body makes small quantities of opioid compounds that bind to opioid receptors reducing pain and producing euphoria. Thus, opioid drugs, which mimic this endogenous painkilling mechanism, have an extremely high potential for abuse. Natural opioids, called opiates, are derivatives of opium, which is a naturally occurring compound found in the poppy plant. There are now several synthetic versions of opiate drugs (correctly called opioids) that have very potent painkilling effects, and they are often abused. For example, the National Institute of Drug Abuse has sponsored research that suggests the misuse and abuse of the prescription pain killers hydrocodone and oxycodone are significant public health concerns (Maxwell, 2006). In 2013, the U.S. Food and Drug Administration (FDA) recommended tighter controls on their medical use.

Opioids include substances such as heroin, morphine, fentanyl, codeine, oxycodone, and hydrocodone. In the United States, a majority of heroin users begin by using prescription opioids that may also be bought illegally. Historically, heroin has been a major opioid drug of abuse (Figure 1). Heroin can be snorted, smoked, or injected intravenously. Like the stimulants described earlier, the use of heroin is associated with an initial feeling of euphoria followed by periods of agitation. Because heroin is often administered via intravenous injection, users often bear needle track marks on their arms and, like all abusers of intravenous drugs, have an increased risk for contraction of both tuberculosis and HIV.



(a) (b)

Figure 9.7.1: (a) Common paraphernalia for heroin preparation and use are shown here in a needle exchange kit. (b) Heroin is cooked on a spoon over a candle. (credit a: modification of work by Todd Huffman)

Aside from their utility as analgesic drugs, opioid-like compounds are often found in cough suppressants, anti-nausea, and anti-diarrhea medications. Given that withdrawal from a drug often involves an experience opposite to the effect of the drug, it should be no surprise that opioid withdrawal resembles a severe case of the flu. While opioid withdrawal can be extremely unpleasant, it is not life-threatening (Julien, 2005). Still, people experiencing opioid withdrawal may be given **methadone** to make withdrawal from the drug less difficult. Methadone is a synthetic opioid that is less euphorigenic than heroin and similar drugs. **Methadone clinics** help people who previously struggled with opioid addiction manage withdrawal symptoms through the use of methadone. Other drugs, including the opioid buprenorphine, have also been used to alleviate symptoms of opiate withdrawal.

Codeine is an opioid with relatively low potency. Codeine is often prescribed for minor pain, and it is available over-the-counter in some other countries. Like all opioids, codeine does have abuse potential. In fact, abuse of prescription opioid medications is becoming a major concern worldwide (Aquina, Marques-Baptista, Bridgeman, & Merlin, 2009; Casati, Sedefov, & Pfeiffer-Gerschel, 2012).

The Opioid Epidemic

The opioid epidemic, or the opioid crisis, refers to the extensive overuse of opioid drugs, both from medical prescriptions and from illegal sources. The epidemic began slowly in the United States, beginning in the late 1990s, and led to a massive increase in opioid use in recent years, contributing to over 70,000 drug overdose deaths in the United States in 2018. Fentanyl alone, being 50 times stronger than heroin and 100 times stronger than morphine, was causing about 200 overdose deaths per day in 2017.[1]

Opioids are a diverse class of moderately strong, addictive, inexpensive painkillers prescribed by doctors. In the late 1990s, pharmaceutical companies reassured the medical community that patients would not become addicted to opioid pain relievers and healthcare providers began to prescribe them at greater rates. This claim led to widespread misuse of both prescription and non-prescription opioids before it became clear that these medications could indeed be highly addictive.

Though aggressive opioid prescription practices played the biggest role in creating the epidemic, the popularity of illegal substances such as potent heroin and illicit fentanyl have become an increasingly large factor. It has been suggested that decreased supply of prescription opioids caused by opioid prescribing reforms turned people who were already addicted to opioids towards illegal substances.[2]

In 2015, approximately 50% of drug overdoses were not the result of an opioid product from a prescription, though most abusers' first exposure had still been by lawful prescription.[3] By 2018, another study suggested that 75% of opioid abusers started their opioid use by taking drugs which had been obtained in a way other than by legitimate prescription.[4]

Those addicted to opioids, both legal and illegal, are increasingly young, white, and female, with 1.2 million women addicted compared to 0.9 million men in 2015. While men have higher rates of opioid abuse on the whole and are more likely to visit the emergency room or die from an overdose, heroin deaths among women increased at more than twice the rate than among men from 1995 to 2015.[5]

The populations of rural areas of the country have been the hardest hit. Teen abuse of opioids has been noticeably increasing since 2006, using prescription drugs more than any illicit drug except marijuana and more than cocaine, heroin, and methamphetamine combined. The crisis has also changed moral, social, and cultural resistance to street drug alternatives such as heroin.

Opioid-Related Disorders

Opioid use disorder (**OUD**) is a substance use disorder relating to the use of an opioid. Any such disorder causes significant impairment or distress. Signs of the disorder include a strong desire to use opioids, increased tolerance to opioids, difficulty fulfilling obligations, trouble reducing use, and withdrawal symptoms with discontinuation. **Opioid withdrawal** symptoms may include nausea, muscle aches, diarrhea, trouble sleeping, agitation, and a low mood. Addiction and dependence are components of a substance use disorder. Complications may include opioid overdose, suicide, HIV/AIDS, hepatitis C, and problems at school, work, or home.

If a person is appropriately taking opioids for a medical condition, issues of tolerance and withdrawal do not apply.

Epidemiology

Opioid use disorders affect over 16 million people worldwide, over 2.1 million in the United States, and there are over 120,000 deaths worldwide annually attributed to opioids.

Over 16 million people worldwide are opioid-dependent and would meet the criteria for opioid use disorder, three million in the United States alone. Opioid use disorder results in over 120,000 and 47,000 deaths per year worldwide and in the United States, respectively. Opioid-related death is the most lethal drug epidemic in American history. According to the CDC, the age-adjusted drug poisoning death rate involving opioid analgesics increased to 7.0 per 100,000 in 2015. Substance abuse is widespread, with over 20 million suffering from substance use disorder, including alcohol, methamphetamines, and opioids. Nearly 10% of the United States population over the age of 12 has used an illicit drug in the prior month. Of the 20 million Americans with substance abuse, two million are using prescription opioid pain medications, and 500 thousand use heroin. Recreational use of opioids was at

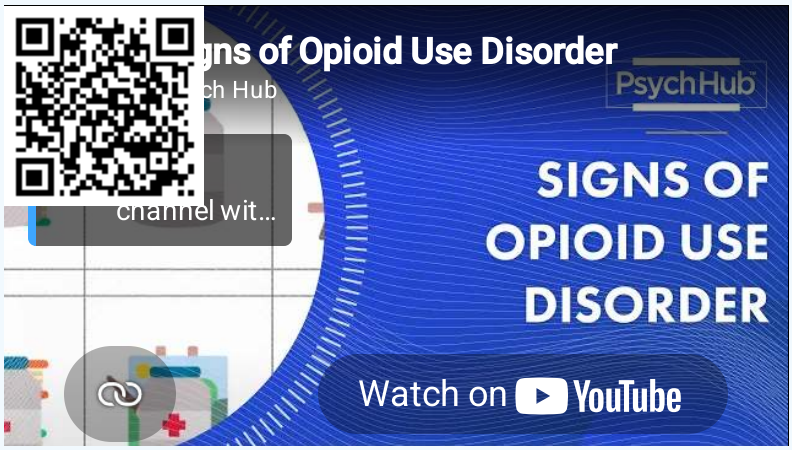
its highest in 2010 and has gradually decreased as the opioid epidemic has gained attention in the United States. Up to 50% of patients on chronic opioid therapy meet the criteria for opioid use disorder.

The prevalence of opioid use and dependency varies by age and gender. Men are more likely to use opioids, become dependent on various opioids, and account for the majority of opioid-related overdoses, although women are prescribed opioids more often than men, and in recent years, women are abusing opioids in record numbers. Studies show that overdose deaths from synthetic opioids in women between ages 30 and 64 have increased 1643%. In 2017, the age-adjusted death rate from drug overdoses involving opioids (per 100,000) was 6.1 for men and 4.2 for women.[6]

Deaths due to opioid use tend to skew at older ages with overdoses from opioids highest among individuals between the ages of 40 and 50. Yet, heroin overdoses peak between the ages of 20 and 30. The peak age of treatment for opioid use disorder is between 20- and 35-year-olds. Patients diagnosed with an opioid-use disorder are more likely to encounter legal problems related to their drug use if they have previous criminal records and high impulsivity.

Watch It

This video explains the struggles of opioid use and how it may lead to a disorder.



You can view the transcript for “Signs of Opioid Use Disorder” here (opens in new window).

Etiology

The etiology of opiate use disorder is multifaceted. Dependence and substance abuse is a product of biological, environmental, genetic, and psychosocial factors. Opioids, including prescription analgesics, derive from the poppy plant. Clinicians prescribe various opioids to control pain, decrease cough, or relieve diarrhea. Opioid-use disorders occur in individuals from all educational and socioeconomic backgrounds. There is a biological base of addiction. Patients can be deficient in neurotransmitters, such as dopamine, making them more likely to seek external sources of endorphins. In an attempt to self-correct this deficit, some individuals may turn to opioids. Separately, a patient with first-degree relatives who have a substance abuse disorder is more likely to develop an opiate use disorder. There is an estimated 50% heritability for opioid use disorder.

When patients diagnosed with opioid use disorder are exposed to an environment that includes opioid use, they may be more likely to develop substance abuse disorder. Environmental influence on opioid use may be secondary to peer relationships or be from a physician’s prescription for a previous injury. Patients with a history of depression, post-traumatic stress disorder (PTSD), or anxiety are more likely to suffer from substance abuse, as well as patients with histories of childhood trauma and abuse. Opioid dependence includes physical or psychological dependence or both. The majority of opioids in use are prescribed, but many are obtained illegally. According to the CDC, there were more than 191 million opioid prescriptions prescribed from 2012 to 2017.

Genetics may also play a role in the development of opioid use disorder. Mu, delta, and kappa are the three different principal receptors for opioids. Mu acts in the brain by decreasing the release of neurotransmitters. Research has demonstrated a genetic basis in the treatment of pain for opioid use disorder. There are no specific pharmacogenomic dosing recommendations, as there is no clear evidence connecting genotype to drug effect, toxicity, or dependence.

Treatment

Individuals with an opioid use disorder are often treated with opioid replacement therapy using methadone or buprenorphine. Being on such treatment reduces the risk of death. Additionally, individuals may benefit from cognitive behavioral therapy, other forms of support from mental health professionals such as individual or group therapy, twelve-step programs, and other peer-support programs. The goal of therapy is to minimize drug-use relapse. The combination of education, motivational enhancement, and self-help groups helps patients change how they think about the ways that opioids affect their lives. Group therapy helps maintain self-control and restraint for patients with substance abuse disorder. Group therapy is cost-effective in comparison to individualized therapy in the treatment of substance abuse.

CBT is most effective if combined with medications; however, there are mixed results on its effectiveness. Education about dealing with pain syndromes and minimizing opioid use can help build rapport and create realistic treatment goals. There is also the need to warn patients to avoid misuse of other drugs, which enhance the effects of opioids such as benzodiazepines, to help prevent overdose. Opioid replacement, maintenance, or substitution therapy involves replacing an opioid with a longer-acting but less euphoric and addicting opioid. The commonly used drugs are buprenorphine and methadone prescribed and given under medical supervision.

Methadone, an oral mu agonist, has been widely used and studied worldwide, and methadone maintenance is a well-established approach in treating opioid use disorder. The advantages of methadone treatment include blocking euphoric effects, decreasing narcotic craving, and reducing the transmission of infectious diseases. Methadone maintenance is non-sedating and is medically safe, provided there is no concomitant use of other prescription or illicit drugs. The maintenance phase begins approximately six weeks after the initiation of therapy. The length of the maintenance phase can last years to an entire lifetime. Tapering off methadone can take weeks or months, depending on the patient's opioid dependence.

An alternative oral, long-acting opioid is buprenorphine for maintenance therapy. Buprenorphine treatment reduces morbidity and mortality. The recommended for buprenorphine is the minimum treatment of 12 months, although, as with methadone, risks of relapse and overdose increase following discontinuation of buprenorphine.

Sedative-Hypnotic- or Anxiolytic-Related Disorders

Just as with the opioid use disorders and others we have learned about thus far, another category of drug use that may lead to mental illness or addiction include sedative, hypnotic, or anxiolytic-related drugs. The DSM-5 categorizes these as either **sedative, hypnotic, or anxiolytic use disorder**; **sedative, hypnotic, or anxiolytic intoxication**; **sedative, hypnotic, or anxiolytic withdrawal**; or another sedative-, hypnotic-, or anxiolytic-induced mental disorder. **Hypnotic** (from Greek *Hypnos*, sleep), or soporific drugs, commonly known as sleeping pills, are a class of psychoactive drugs whose primary function is to induce sleep and for the treatment of insomnia (sleeplessness), or for surgical anesthesia. This group is related to **sedatives**. Whereas the term *sedative* describes drugs that serve to calm or relieve anxiety, the term *hypnotic* generally describes drugs whose main purpose is to initiate, sustain, or lengthen sleep. Because these two functions frequently overlap, and because drugs in this class generally produce dose-dependent effects (ranging from anxiolysis to loss of consciousness), they are often referred to collectively as sedative-hypnotic drugs. Anxiolytic drugs are those used to reduce and treat anxiety, such as benzodiazepines like Xanax or Valium.

It is also important to understand the paradoxical effects of some sedative drugs. Serious complications can occur in conjunction with the use of sedatives, creating the opposite effect as to that intended. Malcolm Lader at the Institute of Psychiatry in London estimates the incidence of these adverse reactions at about 5%, even in short-term use of the drugs.[7] The paradoxical reactions may consist of depression, with or without suicidal tendencies, phobias, aggressiveness, violent behavior, and symptoms sometimes misdiagnosed as psychosis. However, psychosis is more commonly related to benzodiazepine withdrawal syndrome.

Hypnotic drugs are regularly prescribed for insomnia and other sleep disorders, with over 95% of insomnia patients being prescribed hypnotics in some countries. Many hypnotic drugs are habit-forming and, due to many factors known to disturb the human sleep pattern, a physician may instead recommend changes in the environment before and during sleep, better sleep hygiene, the avoidance of caffeine or other stimulating substances, or behavioral interventions such as CBT for insomnia (CBT-I) before prescribing medication for sleep. When prescribed, hypnotic medication should be used for the shortest period of time necessary.

Among individuals with sleep disorders, 13.7% are taking or prescribed nonbenzodiazepines, while 10.8% are taking benzodiazepines, as of 2010, in the USA. Early classes of drugs, such as barbituates, have fallen out of use in most practices but are still prescribed for some patients. In children, prescribing hypnotics is not yet acceptable unless used to treat night terrors or sleepwalking.

Watch It

In this video, psychiatrist Anna Lembke talks about the difficulties some of her patients have in stopping their dependence on benzodiazepines.



You can view the transcript for "Benzodiazepine Withdrawal Difficulties: Stanford Psychiatrist Anna Lembke, M.D." here (opens in new window).

Glossary

anxiolytic: drugs used to reduce and treat anxiety

codeine: opiate with relatively low potency often prescribed for minor pain

hypnotic: describes drugs whose main purpose is to initiate, sustain, or lengthen sleep

methadone: synthetic opioid that is less euphorogenic than heroin and similar drugs; used to manage withdrawal symptoms in opiate users

methadone clinic: uses methadone to treat withdrawal symptoms in opiate users

methamphetamine: type of amphetamine that can be made from pseudoephedrine, an over-the-counter drug; widely manufactured and abused

opiate/opioid: one of a category of drugs that has strong analgesic properties; opiates are produced from the resin of the opium poppy; includes heroin, morphine, methadone, and codeine

sedative: drugs that serve to calm or relieve anxiety

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9.8: Hallucinogen-Related Disorders

Learning Objectives

- Describe hallucinogens and how they affect the brain and behavior
- Describe the diagnostic features, development, and risk factors of hallucinogen-related disorders, including phencyclidine, inhalant use disorder, and other hallucinogens

A **hallucinogen** is one of a class of drugs that results in profound alterations in sensory and perceptual experiences. In some cases, users experience vivid visual hallucinations. Common hallucinogens include psilocybin (shrooms), mescaline (peyote), and LSD. These substances also commonly cause hallucinations of body sensations (e.g., feeling as if you are a giant) and a skewed perception of the passage of time.

Leo Hollister's five criteria for establishing that a drug is hallucinogenic are as follows:

- in proportion to other effects, changes in thought, perception, and mood should predominate;
- intellectual or memory impairment should be minimal;
- stupor, narcosis, or excessive stimulation should not be an integral effect;
- autonomic nervous system side effects should be minimal; and
- addictive craving should be absent.

As a group, hallucinogens are incredibly varied in terms of the neurotransmitter systems they affect. Mescaline and LSD are serotonin agonists, and PCP (angel dust) and ketamine (an animal anesthetic) act as antagonists of the NMDA glutamate receptor. In general, these drugs are not thought to possess the same sort of abuse potential as other classes of drugs discussed in this section.

Some hallucinogens are extracted from plants or mushrooms, and some are synthetic (human-made). Historically, people have used hallucinogens for religious or healing rituals. More recently, people report using these drugs for social or recreational purposes, including to have fun, deal with stress, have spiritual experiences, or just to feel different.

Common classic hallucinogens include the following:

- **LSD** (D-lysergic acid diethylamide) is one of the most powerful mind-altering chemicals. It is a clear or white odorless material made from lysergic acid, which is found in a fungus that grows on rye and other grains. LSD has many other street names, including acid, blotter acid, dots, and mellow yellow.
- **Psilocybin** (4-phosphoryloxy-N, N-dimethyltryptamine) comes from certain types of mushrooms found in tropical and subtropical regions of South America, Mexico, and the United States. Some common names for psilocybin include little smoke, magic mushrooms, and shrooms.
- **Peyote** (mescaline) is a small, spineless cactus with mescaline as its main component. Peyote can also be synthetic. Common names for peyote are buttons, cactus, and mesc.
- **DMT** (N, N-dimethyltryptamine) is a powerful chemical found naturally in some Amazonian plants. Ayahuasca is a tea made from such plants, and when taken in this form, it is also known as hoasca, aya, and yagé. People can also make DMT in a lab. Synthetic DMT usually takes the form of a white crystalline powder that is smoked. A popular name for synthetic DMT is Dimitri.
- **251-NBOMe** is a synthetic hallucinogen with similarities both to LSD and MDMA but that is much more potent. Developed for use in brain research, when sold on the street, it is sometimes called N Bomb or 251.
- **Methylenedioxymethamphetamine** (**MDMA**), commonly known as ecstasy (E) or molly, is a psychoactive drug primarily used for recreational purposes. The desired effects include altered sensations and increased energy, empathy and pleasure. When taken by mouth, effects begin in 30 to 45 minutes and last three to six hours. It is an amphetamine, but often classified with hallucinogens for its hallucinogenic effects. Adverse effects include addiction, memory problems, paranoia, difficulty sleeping, grinding of the teeth, blurred vision, sweating, and a rapid heartbeat. Deaths have been reported due to increased body temperature and dehydration. Following its use, people often feel depressed and tired. MDMA acts primarily by increasing the activity of the neurotransmitters serotonin, dopamine, and noradrenaline in parts of the brain.

Common examples of dissociative drugs include the following:

- **PCP** (Phencyclidine) was developed in the 1950s as a general anesthetic for surgery, but it is no longer used for this purpose due to serious side effects. PCP can be found in a variety of forms, including tablets or capsules; however, liquid and white crystal powder are the most common forms. PCP has various slang names, such as angel dust, hog, love boat, and peace pill.
- **Ketamine** is used as a surgery anesthetic for humans and animals. Much of the ketamine sold on the streets comes from veterinary offices. It mostly sells as a powder or as pills, but it also available as an injectable liquid. Ketamine is snorted or sometimes added to drinks as a date-rape drug. Slang names for ketamine include Special K and Cat Valium.
- **Dextromethorphan** (DXM) is a cough suppressant and mucus-clearing ingredient in some over-the-counter cold and cough medicines (syrups, tablets, and gel capsules). Robo is a common slang name for DXM.
- **Salvia** (Salvia divinorum) is a plant common to southern Mexico and Central and South America. Salvia is typically ingested by chewing fresh leaves or by drinking their extracted juices. The dried leaves of salvia can also be smoked or vaporized and inhaled. Popular names for salvia are diviner's sage, Maria Pastora, Sally-D, and magic mint.

Hallucinogen-Related Disorders

Hallucinogen-related disorders in the DSM-5 include all of the following:

- phencyclidine use disorder
- other hallucinogen use disorder
- phencyclidine intoxication
- other hallucinogen intoxication
- hallucinogen persisting perception disorder
- other phencyclidine-induced disorders
- other hallucinogen-induced disorders
- unspecified phencyclidine-related disorder
- unspecified hallucinogen-related disorder

Similar to the other categories of disorders we have learned about, the hallucinogen-use disorders describe a dependency on the drug, while intoxication refers to changes in behavior and psychology during or immediately following the use of the drug. These categories do not have withdrawal disorders.

Phencyclidine-Use Disorder

Phencyclidine or phenylcyclohexyl piperidine (PCP), also known as angel dust, is a drug used for its mind-altering effects. PCP may cause hallucinations, distorted perceptions of sounds, and violent behavior. As a recreational drug, it is typically smoked, but may be taken by mouth, snorted, or injected. It may also be mixed with cannabis or tobacco.

Adverse effects may include seizures, coma, addiction, and an increased risk of suicide. Flashbacks may occur despite stopping usage. PCP is most commonly used in the United States. While usage peaked in the United States in the 1970s, between 2005 and 2011, an increase in visits to emergency departments as a result of the drug occurred. As of 2017 in the United States, about 1% of people in grade 12 reported using PCP in the prior year while 2.9% of those over the age of 25 reported using it at some point in their lives.

Phencyclidine intoxication occurs when a person's mental capacity to recognize reality, communicate, and relate to others is impaired, thus interfering with the capacity to deal with life demands.

Other Hallucinogen-Use Disorders

Other hallucinogen-use disorders involve a problematic pattern of hallucinogen use (other than PCP/phencyclidine), including LSD (acid), ecstasy (MDMA), ketamine, mushrooms, peyote, etc.

As of 2017, about 10% of people in the United States have used LSD at some point in their lives, while 0.7% have used it in the last year. It was most popular in the 1960s to 1980s. The use of LSD among U.S. adults increased 56.4% from 2015 to 2018. LSD is typically either swallowed or held under the tongue.

Hallucinogen Persisting Perception Disorder

Hallucinogen persisting perception disorder (HPPD) is a chronic disorder in which a person has non-psychotic flashbacks of visual hallucinations or distortions experienced during a previous hallucinogenic drug experience, usually lacking the same feelings of mental intoxication experienced before. Hallucinations are usually not intense or impairing and consist of visual snow, trails and after images (palinopsia), light fractals on flat surfaces, intensified colors, or other psychedelic visuals. To be diagnosed, the disorder must cause distress or impairment in work or everyday life. The flashbacks may be continuous or just occasional. Symptoms often get worse when they're focused on.

The prevalence of HPPD was unknown as of 2018. Estimates in the 1960s and 1970s were around one in 20 for intermittent HPPD among regular users of hallucinogens. It is not clear how common chronic HPPD may be, but one estimate in the 1990s was that one in 50,000 regular users might have chronic hallucinations.

The only certain cause for HPPD is the prior use of hallucinogens. Some evidence points to phenethylamines carrying a slightly greater risk than lysergamides or tryptamines. Some sort of disinhibition of visual processing may be involved. It has been suggested MDMA (ecstasy) use with other drugs is linked to the development of HPPD.

Diagnostic Criteria for Phencyclidine and Other Hallucinogen Use Disorder

A. A problematic pattern of phencyclidine [or other hallucinogen] use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. Phencyclidine [or other hallucinogen] is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control phencyclidine (or other hallucinogen) use.
3. A great deal of time is spent in activities necessary to obtain phencyclidine (or other hallucinogen), use phencyclidine, or recover from its effects.
4. Craving, or a strong desire or urge to use phencyclidine (or other hallucinogen).
5. Recurrent phencyclidine (or other hallucinogen) use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated absences from work or poor performance related to phencyclidine use; phencyclidine-related absences, suspensions, or expulsions from school; or neglect of children or household).
6. Continued phencyclidine (or other hallucinogen) use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of phencyclidine (e.g., arguments with a spouse about consequences of intoxication or physical fights).
7. Important social, occupational, or recreational activities are given up or reduced because of phencyclidine (or other hallucinogen) use.
8. Recurrent phencyclidine (or other hallucinogen) use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by a phencyclidine).
9. Phencyclidine (or other hallucinogen) use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by phencyclidine.
10. Tolerance, as defined by either of the following:
    - a. A need for markedly increased amounts of phencyclidine to achieve intoxication or desired effect.
    - b. A markedly diminished effect with continued use of the same amount of phencyclidine.

PCP was initially made in 1926 and brought to market as an anesthetic medication in the 1950s. Its anesthetic effects were discovered by Victor Maddox, a chemist at Parke-Davis in Michigan, while investigating synthetic analgesic agents. It was approved for use as an investigational drug under the brand names Sernyl and Sernylan in the 1950s as an anesthetic, but because of its long terminal half-life and adverse side-effects, such as hallucinations, mania, delirium, and disorientation, it was removed from the market in 1965 and limited to veterinary use, until that was disallowed in 1978.

Treatment

There are no FDA-approved medications to treat addiction to hallucinogen addiction. While behavioral treatments can be helpful for patients with a variety of addictions, scientists need more research to find out if behavioral therapies are effective for addiction to hallucinogens. For HPDD, some antidepressant and antipsychotic medications can be used to improve mood and treat psychosis. Behavioral therapies can be used to help people cope with fear or confusion associated with visual disturbances. Management of

PCP intoxication mostly consists of supportive care—controlling breathing, circulation, and body temperature—and, in the early stages, treating psychiatric symptoms. Benzodiazepines, such as lorazepam, are the drugs of choice to control agitation and seizures (when present).

Inhalant-Related Disorders

Inhalants are a broad range of household and industrial chemicals whose volatile vapors or pressurized gases can be concentrated and breathed in via the nose or mouth to produce intoxication in a manner not intended by the manufacturer. Inhalants are inhaled at room temperature through volatilization (in the case of gasoline or acetone) or from a pressurized container (e.g., nitrous oxide or butane), and do not include drugs that are sniffed after burning or heating. For example, amyl nitrite (poppers), nitrous oxide, and toluene—a solvent widely used in contact cement, permanent markers, and certain types of glue—are considered inhalants, but smoking tobacco, cannabis, and crack are not, even though these drugs are inhaled as smoke.



Figure **9.8.2**: Even unintentional inhalant use can lead to inhalant intoxication marked by problematic behavioral or psychological changes.

While a small number of inhalants are prescribed by medical professionals and used for medical purposes, as in the case of inhaled anesthetics and nitrous oxide (an anxiolytic and pain relief agent prescribed by dentists), inhalants typically refer to the use of household and industrial propellants, glues, fuels, and other products in a manner not intended by the manufacturer to produce intoxication or other psychoactive effects. These products are used as recreational drugs for their intoxicating effect. According to a 1995 report by the National Institute on Drug Abuse, the most serious inhalant abuse occurs among homeless children and teens who “live on the streets completely without family ties.” Inhalants are the only substances used more by younger teens than by older teens. The practices are known colloquially as “sniffing”, “huffing,” or “bagging.”

Inhalant disorders described in the DSM-5 include inhalant use disorder, inhalant intoxication, other inhalant-induced disorders, and unspecified inhalant-related disorder. **Inhalant use disorder** is defined as a problematic pattern of use of a hydrocarbon-based inhalant substance leading to clinically significant impairment or distress.

Inhalant intoxication occurs frequently during inhalant use disorder but also may occur among individuals whose use does not meet the criteria for inhalant use disorder. Inhalant intoxication is identified as the following:

A. Recent intended or unintended short-term, high-dose exposure to inhalant substances, including volatile hydrocarbons such as toluene or gasoline.

B. Clinically significant problematic behavioral or psychological changes (e.g., belligerence, assaultiveness, apathy, and impaired judgment) that developed during, or shortly after, exposure to inhalants.

C. Two (or more) of the following signs or symptoms developing during, or shortly after, inhalant use or exposure:

- dizziness
- nystagmus
- incoordination
- slurred speech
- unsteady guilt
- lethargy
- depressed reflexes
- psychomotor retardation
- tremor
- generalized muscle weakness

- blurred vision or diplopia
- stupor or coma
- euphoria

The effects of inhalants range from an alcohol-like intoxication and intense euphoria to vivid hallucinations, depending on the substance and the dose. Some inhalant users are injured due to the harmful effects of the solvents or gases or due to other chemicals used in the products that they are inhaling. In some cases, users have died from hypoxia (lack of oxygen), pneumonia, cardiac failure or arrest, or aspiration of vomit. Brain damage is typically seen with chronic long-term use of solvents as opposed to short-term exposure.

Glossary

251-NBOMe: a synthetic hallucinogen with similarities both to LSD and MDMA but that is much more potent

DMT: a powerful chemical found naturally in some Amazonian plants

DXM: a cough suppressant and mucus-clearing ingredient in some over-the-counter cold and cough medicines

hallucinogen: one of a class of drugs that results in profound alterations in sensory and perceptual experiences, often with vivid hallucinations

HPPD: a chronic disorder in which a person has non-psychotic flashbacks of visual hallucinations or distortions experienced during a previous hallucinogenic drug experience, usually lacking the same feelings of mental intoxication experienced before

inhalants: a broad range of household and industrial chemicals whose volatile vapors or pressurized gases can be concentrated and breathed in via the nose or mouth to produce intoxication

inhalant intoxication: occurs frequently during inhalant use disorder but also may occur among individuals whose use does not meet criteria for inhalant use disorder

inhalant use disorder: a problematic pattern of use of a hydrocarbon-based inhalant substance leading to clinically significant impairment or distress

ketamine: used as a surgery anesthetic for humans and animals

LSD: is one of the most powerful mind-altering chemicals and it is a clear or white odorless material made from lysergic acid, which is found in a fungus that grows on rye and other grains

MDMA: commonly known as ecstasy (E) or molly, is a psychoactive drug primarily used for recreational purposes and the desired effects include altered sensations, increased energy, and empathy as well as pleasure

peyote: a small, spineless cactus with mescaline as its main ingredient and can also be synthetic

PCP: also known as angel dust, is a drug used for its mind-altering effects and may cause hallucinations, distorted perceptions of sounds, and violent behavior

salvia: a plant common to southern Mexico and Central and South America and is typically ingested by chewing fresh leaves or by drinking their extracted juices

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9.9: Cannabis-Related Disorders

Learning Objectives

- Describe the diagnostic features, development, and risk factors of cannabis-related disorders

Cannabis



Figure **9.9.1**: Marijuana is the most commonly used illegal drug in the world.

Cannabis, also known as marijuana, is a psychoactive drug from the cannabis plant used primarily for medical or recreational purposes. The main psychoactive component of cannabis is tetrahydrocannabinol (THC), which is one of the 483 known compounds in the plant, including at least 65 other cannabinoids, including cannabidiol (CBD). Cannabis can be used by smoking, vaporizing, or consuming within food or as an extract.

Cannabis has various mental and physical effects, which include euphoria, altered states of mind and sense of time, difficulty concentrating, impaired short-term memory and body movement, relaxation, and an increase in appetite. The onset of effects is felt within minutes if smoked, and about 30 to 60 minutes when cooked and eaten. The effects last for two to six hours, depending on the amount used. At high doses, mental effects can include anxiety, delusions (including ideas of reference), hallucinations, panic, paranoia, and psychosis. There is a strong relation between cannabis use and the risk of psychosis, though the direction of causality is debated.

Physical effects include increased heart rate, difficulty breathing, nausea, and behavioral problems in children whose mothers used cannabis during pregnancy; short-term side effects may also include dry mouth and red eyes. Long-term adverse effects may include addiction, decreased mental ability in those who started regular use as adolescents, chronic coughing, and susceptibility to respiratory infections.

Cannabis is mostly used recreationally or as a medicinal drug, although it may also be used for spiritual purposes. In 2013, between 128 and 232 million people used cannabis (2.7% to 4.9% of the global population between the ages of 15 and 65). It is the most commonly used illegal drug in the world, though it is also legal in some jurisdictions, with the highest use among adults (as of 2018) in Zambia, the United States, Canada, and Nigeria. While cannabis was previously an illicit substance, a majority of states across the United States have legalized the medical use of cannabis with a doctor’s recommendation, and more and more states are also legalizing the recreational use of marijuana.

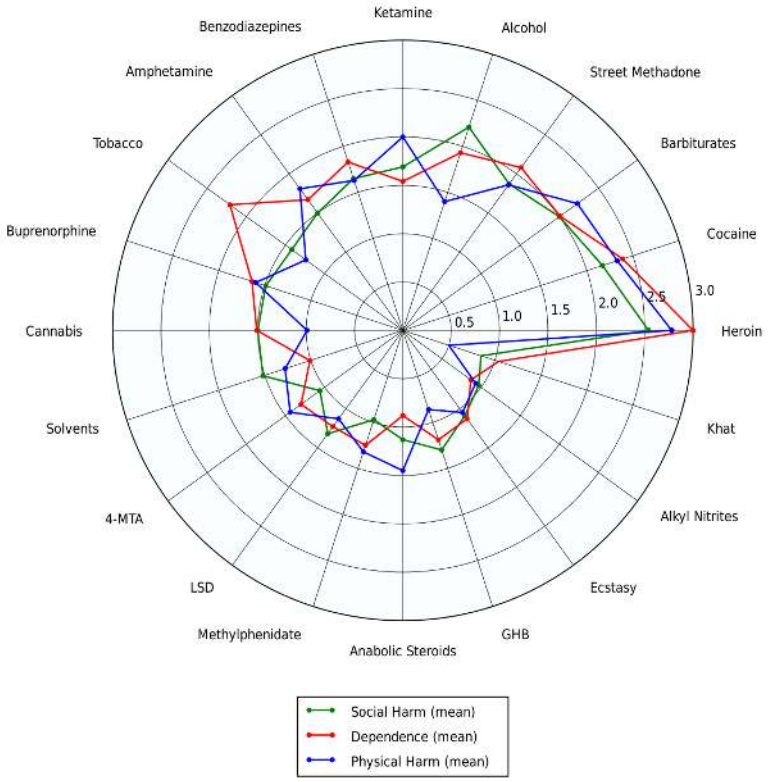


Figure 9.9.2: Addiction experts in psychiatry, chemistry, pharmacology, forensic science, and epidemiology and the police and legal services engaged in delphic analysis regarding 20 popular recreational drugs. Cannabis was ranked 11th in dependence, 17th in physical harm, and 10th in social harm.

To screen for cannabis-related problems, several methods are used. Scales specific to cannabis include the Cannabis Abuse Screening Test (CAST), Cannabis Use Identification Test (CUDIT), and Cannabis Use Problems Identification Test (CUPIT). Scales for general drug use disorders are also used, including the Severity Dependence Scale (SDS), Drug Use Disorder Identification Test (DUDIT), and Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST). However, there is no gold standard and both older and newer scales are still in use.

Watch It

This TED talk examines the effects of marijuana on the brain and body and investigates its dangers.



You can view the transcript for "Is marijuana bad for your brain? – Anees Bahji" here (opens in new window).

Diagnosis of Cannabis-Related Disorders

The diagnostic criteria of the varying effects of cannabis are listed below, as defined by the DSM-5.

Cannabis Intoxication

1. recent use of cannabis

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9.10: Introduction to Perspectives and Treatments Related to Substance-Related Disorders

What you'll learn to do: examine various perspectives and treatment methods related to substance-related and addictive disorders



In this section, we will take a closer look at the various psychological perspectives and see how they explain addiction and substance-related disorders. Addiction is a complicated phenomenon, influenced by genetic, biological, environmental, and social factors, which also makes treatment programs more nuanced. Below are some key points to consider related to treatment:

- Drug addiction can be treated, but it's not simple. Addiction treatment must help the person do the following:
  - stop using drugs
  - stay drug-free
  - be productive in the family, at work, and in society
- Successful treatment has several steps:
  - detoxification
  - behavioral counseling
  - medication (for opioid, tobacco, or alcohol addiction)
  - evaluation and treatment for co-occurring mental health issues such as depression and anxiety
  - long-term follow-up to prevent relapse
- Medications and devices can be used to manage withdrawal symptoms, prevent relapse, and treat co-occurring conditions.
- Behavioral therapies help patients to
  - modify their attitudes and behaviors related to drug use,
  - increase healthy life skills, and
  - persist with other forms of treatment, such as medication.

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9.11: Perspectives on Addiction

Learning Objectives

- Describe and discuss the various psychological explanations for addiction

There are a number of genetic and environmental risk factors for explaining drug or alcohol abuse that vary across the population. Genetic and environmental risk factors each account for roughly half of an individual's risk for developing an addiction; the contribution from epigenetic risk factors to the total risk is unknown. Even in individuals with a relatively low genetic risk, exposure to sufficiently high doses of an addictive drug for a long period of time (e.g., weeks to months) can result in an addiction.

Watch It

This video explains how substance abuse disorders may develop.



You can view the transcript for "The development of substance use – Why do people use legal and illegal substances? | Khan Academy" here (opens in new window).

Genetic and Biological Approaches to Addiction

Epidemiological studies estimate that genetic factors account for 40–60% of the risk factors for alcoholism. Similar rates of heritability for other types of drug addiction have been indicated by other studies. Overall, the data implicating specific genes in the development of drug addiction is mixed for most genes. One reason for this may be that the case is due to a focus of current research on common variants. Many addiction studies focus on common variants with an allele frequency of greater than 5% in the general population; however, when associated with the disease, these only confer a small amount of additional risk with an odds ratio of 1.1%–1.3%.

Genome-wide association studies (GWAS) are used to examine genetic associations with dependence, addiction, and drug use. These studies employ an unbiased approach to finding genetic associations with specific phenotypes and give equal weight to all regions of DNA, including those with no ostensible relationship to drug metabolism or response. These studies rarely identify genes from proteins previously described via animal-knockout models and candidate-gene analysis. Instead, large percentages of genes involved in processes such as cell adhesion are commonly identified.

Cross addiction is when one already has a predisposed addiction and then starts to become addicted to something different. If one family member has a history of addiction, the chances of a relative or close family developing those same habits are much higher than one who has not been introduced to addiction at a young age. In a recent study done by the National Institute on Drug Abuse, from 2002 to 2017, overdose deaths have almost tripled amongst male and females. In 2017, 72,306 overdose deaths happened in the United States that were reported.

Epigenetic Factors

Epigenetic genes and their products (e.g., proteins) are the key components through which environmental influences can affect the genes of an individual; they also serve as the mechanism responsible for transgenerational epigenetic inheritance, a phenomenon in which environmental influences on the genes of a parent can affect the associated traits and behavioral phenotypes of their offspring (e.g., behavioral responses to environmental stimuli). In addiction, epigenetic mechanisms play a central role in the pathophysiology of the disease; it has been noted that some of the alterations to the epigenome that arise through chronic exposure to addictive stimuli during an addiction can be transmitted across generations, in turn affecting the behavior of one's children (e.g., the child's behavioral responses to addictive drugs and natural rewards).

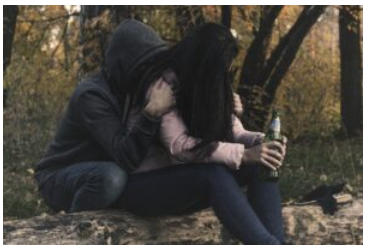


Figure **9.11.1**: Reasons for addiction may be influenced by a myriad of factors, including genetic and biological, social, environmental, and personal reasons.

The general classes of epigenetic alterations that have been implicated in transgenerational epigenetic inheritance include DNA mythylation, histone modifcations, and downregulation or upregulation of microRNAs. With respect to addiction, more research is needed to determine the specific heritable epigenetic alterations that arise from various forms of addiction in humans and the corresponding behavioral phenotypes from these epigenetic alterations that occur in human offspring. More generally, the heritable behavioral phenotypes that are derived from addiction-induced epigenetic alterations and transmitted from parent to offspring may serve to either increase or decrease the offspring's risk of developing an addiction.

Psychodynamic Approaches

The psychodynamic approach emphasizes systematic study of the psychological forces that underlie human behavior, feelings, and emotions and how they might relate to early experience. It is especially interested in the dynamic relations between conscious motivation and unconscious motivation. The psychodynamic approach may explain addiction associated with unconscious factors from the past that could create something similar to a trauma response of negative coping by substances. The negative coping by partaking in drugs and alcohol could turn into addiction easily if the unconscious factors are not resolved based on the approach.

According to Edward Khantzian's **self-medication hypothesis** (SMH), a person may choose to medicate by using a drug as an "ego solvent," which acts on parts of the self that are cut off from consciousness by defense mechanisms.[1] According to Khantzian, drug-dependent individuals generally experience more psychiatric distress than nondrug-dependent individuals, and the development of drug dependence involves the gradual incorporation of the drug effects and the need to sustain these effects into the defensive structure-building activity of the ego itself. The addict's choice of drug is a result of the interaction between the psychopharmacologic properties of the drug and the affective states from which the addict was seeking relief. The drug's effects substitute for defective or non-existent ego mechanisms of defense. The addict's drug of choice, therefore, is not random.

Khantzian revisited the self-medication hypothesis (SMH), suggesting there is more evidence that psychiatric symptoms, rather than personality styles, lie at the heart of drug use disorders. Khantzian specified that the two crucial aspects of the self-medication hypothesis (SMH) were that (1) drugs of abuse produce a relief from psychological suffering and (2) the individual's preference for a particular drug is based on its psychopharmacological properties. The individual's drug of choice is determined through experimentation, whereby the interaction of the main effects of the drug, the individual's inner psychological turmoil, and underlying personality traits identify the drug that produces the desired effects.

While Khantzian takes a psychodynamic approach to self-medication, David Duncan proposed a self-medication model that focuses on behavioral factors. Duncan described the nature of positive reinforcement (e.g., the "high feeling" or approval from peers), negative reinforcement (e.g. reduction of negative affect), and avoidance of withdrawal symptoms, all of which are seen in those who develop problematic drug use, but are not all found in all recreational drug users. While earlier behavioral formulations of drug dependence using operant conditioning maintained that positive and negative reinforcement were necessary for drug

dependence, Duncan maintained that drug dependence was not maintained by positive reinforcement, but rather by negative reinforcement. Duncan applied a public health model to drug dependence, where the agent (the drug of choice) infects the host (the drug user) through a vector (e.g., peers), while the environment supports the disease process through stressors and lack of support. A crucial determinant of whether a drug user develops drug abuse is the presence or absence of negative reinforcement, which is experienced by problematic users, but not by recreational users. According to Duncan, drug dependence is an avoidance behavior, where an individual finds a drug that produces a temporary escape from a problem, and taking the drug is reinforced as an operant behavior.

Personality theories of addiction are psychological models that associate personality traits or modes of thinking (i.e., affective states) with an individual's proclivity for developing an addiction. Models of addiction risk that have been proposed in psychology literature include an affect dysregulation model of positive and negative psychological effects, the reinforcement sensitivity theory model of impulsiveness and behavioral inhibition, and an impulsivity model of reward sensitization and impulsiveness.

Research has consistently shown strong associations between affective disorders and substance use disorders. Specifically, people with mood disorders are at increased risk of substance use disorders. Affect and addiction can be related in a variety of ways as they play a crucial role in influencing motivated behaviors. For instance, affect facilitates action, directs attention, prepares the individual for a physical response, and guides behavior to meet particular needs. Moreover, affect is implicated in a range of concepts relevant to addiction: positive reinforcement, behavior motivation, regulation of cognition and mood, and reasoning and decision making. Emotion-motivated reasoning has been shown to influence addictive behaviors via selecting outcomes that minimize negative affective states while maximizing positive affective states.

Cognitive Approaches

Cognitive control, and particularly inhibitory control over behavior, is impaired in addiction. Executive functions (collectively referred to as executive function and cognitive control) are a set of cognitive processes that are necessary for the cognitive control of behavior: selecting and successfully monitoring behaviors that facilitate the attainment of chosen goals. Executive functions include basic cognitive processes such as attentional control, cognitive inhibition, inhibitory control, working memory, and cognitive flexibility. Higher-order executive functions require the simultaneous use of multiple basic executive functions and include planning and fluid intelligence (e.g., reasoning and problem-solving).

Expectancy theory proposes that an individual will behave or act in a certain way because they are motivated to select a specific behavior over others due to what they expect the result of that selected behavior will be. In essence, the motivation of the behavior selection is determined by the desirability of the outcome. However, at the core of the theory is the cognitive process of how an individual processes the different motivational elements. As related to addiction, expectancy theory explains how there may be a motivation to experience the "high" of the substance and the euphoric state that the drug brings to the body. Also, this euphoric state may motivate individuals in the future to take the substance again and again, and hence exacerbating the addiction process. Expectancy theory may also explain how some view drug use as "cool," or that what they perceive as the benefits of drug use outweigh the consequences.

Behavioral Approaches

Behaviorists explain drug use and addiction by emphasizing the way that these behaviors are learned or habitualized. According to social learning theory, which holds that behaviors are learned through imitating and observing others, someone might be peer pressured into trying drugs for the first time. Operant conditioning, which emphasizes repeating behaviors that result in a reward, may explain why an individual would seek out a "high" or the reduction in stress they may get from using a drug. Eventually, this could lead to an increased desire to obtain or use the drug.

As mentioned above, the self-medication hypothesis argues that specific psychiatric withdrawal symptoms, which increase during abstinence, may be the main driver of persistent drug use because of negative reinforcement. Positive reinforcement theories, by contrast, argue that the excessively rewarding effects of drugs drive persistent drug use independently of the withdrawal syndrome. The challenge for positive reinforcement theories is to explain why drug use persists when addicts claim the drug has lost its value. To solve this contradiction, there are several theories like those that emphasize the classical conditioning of behaviors, where an addicted person has learned to associate certain behaviors and drug use, so that they become dependent on the drug.[2]

Environmental Factors

Environmental risk factors for addiction are the experiences of an individual during their lifetime that interact with the individual's genetic composition to increase or decrease his or her vulnerability to addiction. A number of different environmental factors have been implicated as risk factors for addiction, including various psychosocial stressors. The National Institute on Drug Abuse (NIDA) cites lack of parental supervision, the prevalence of peer substance use, drug availability, and poverty as risk factors for substance use among children and adolescents. The brain disease model of addiction posits that an individual's exposure to an addictive drug is the most significant environmental risk factor for addiction. However, many researchers, including neuroscientists, indicate that the brain disease model presents a misleading, incomplete, and potentially detrimental explanation of addiction.

Adverse childhood experiences (ACEs) are various forms of maltreatment and household dysfunction experienced in childhood. A study of 900 court cases involving children who experienced abuse found that a vast amount of them went on to suffer from some form of addiction in their adolescence or adult life. This pathway towards addiction, which is opened through stressful experiences during childhood, can be avoided by a change in environmental factors throughout an individual's life and opportunities of professional help. If one has friends or peers who engage in drug use favorably, the chances of them developing an addiction increases. Family conflict and home management may also lead to alcohol or other drug use.

Age

Adolescence represents a period of unique vulnerability for developing an addiction. In adolescence, the incentive-rewards systems in the brain mature well before the cognitive control center. Therefore, adolescents are increasingly likely to act on their impulses and engage in risky, potentially addicting behavior before considering the consequences. Not only are adolescents more likely to initiate and maintain drug use, but once addicted they are more resistant to treatment and more liable to relapse.

Statistics have shown that those who start to drink alcohol at a younger age are more likely to become dependent later on. About 33% of the population tasted their first alcohol between the ages of 15 and 17, while 18% experienced it prior to this. As for alcohol abuse or dependence, the numbers start off high with those who first drank before they were 12 and then drop off after that. For example, 16% of alcoholics began drinking prior to turning 12 years old, while only 9% first touched alcohol between 15 and 17. This percentage is even lower, at 2.6%, for those who first started the habit after they were 21.

Most individuals are exposed to and use addictive drugs for the first time during their teenage years. In the United States, there were just over 2.8 million new users of illicit drugs in 2013 (~7,800 new users per day); among them, 54.1% were under 18 years of age. In 2011, there were approximately 20.6 million people in the United States over the age of 12 with an addiction. Over 90% of those with an addiction began drinking, smoking, or using illicit drugs before the age of 18.

Comorbid Disorders

Individuals with comorbid (i.e., co-occurring) mental health disorders such as depression, anxiety, attention-deficit/hyperactivity disorder (ADHD) or post-traumatic stress disorder are more likely to develop substance use disorders. The NIDA cites early aggressive behavior as a risk factor for substance use. A study by the National Bureau of Economic Research found that there is a "definite connection between mental illness and the use of addictive substances" and a majority of mental health patients participate in the use of these substances: 38% alcohol, 44% cocaine, and 40% cigarettes.

Sociocultural Factors

Due to cultural variations, the proportion of individuals who develop a drug or behavioral addiction within a specified time period (i.e., the prevalence) varies over time, by country, and across national population demographics (e.g., by age group, socioeconomic status (SES), etc.)

Internationally, the United States and Eastern Europe contain the countries with the highest substance abuse disorder occurrence (5%-6%). Africa, Asia, and the Middle East contain countries with the lowest worldwide occurrence (1%-2%). Across the globe, those that tended to have a higher prevalence of substance dependence were in their twenties, unemployed, and male. The National Survey on Drug Use and Health (NSDUH) reports on substance dependence/abuse rates in various population demographics across the United States. When surveying populations based on race and ethnicity in those ages 12 and older, it was observed that

American Indian/Alaskan Natives were among the highest rates and Asians were among the lowest rates in comparison to other racial/ethnic groups.

Substance Use in Racial/Ethnic Groups

Race/Ethnicity	Dependence/Abuse Rate
Asian	4.6%
Black	7.4%
White	8.4%
Hispanic	8.6%
Mixed race	10.9%
Native Hawaiian/Pacific Islander	11.3%
American Indian/Alaskan Native	14.9%

When surveying populations based on gender in those ages 12 and older, it was observed that males had a higher substance dependence rate than females. However, the difference in the rates are not apparent until after age 17. *Drug and Alcohol Dependence* reports that older adults abuse drugs including alcohol at a rate of 15–20%. It's estimated that 52 million Americans beyond 12 years old have abused a substance.

Substance Use in Different Genders w/ Respect to Age

Age	Male	Female
12 and older	10.8%	5.8%
12–17	5.3%	5.2%
18 or older	11.4%	5.8%

Alcohol dependence or abuse rates were shown to have no correspondence with any person's education level when populations were surveyed in varying degrees of education from ages 26 and older. However, when it came to illicit drug use, there was a correlation in which those that graduated from college had the lowest rates. Furthermore, dependence rates were greater in unemployed populations ages 18 and older and in metropolitan-residing populations ages 12 and older.

Illicit Drug Dependence Demographics (Education, Employment, and Regional)

Education level	Rates	Employment status	Rates	Region	Rates
high school	2.5%	unemployed	15.2%	large metropolitan	8.6%
no degree, college	2.1%	part-time	9.3%	small metropolitan	8.4%
college graduate	0.9%	full-time	9.5%	non-metropolitan	6.6%

The National Opinion Research Center at the University of Chicago reported an analysis on disparities within admissions for substance abuse treatment in the Appalachian region, which comprises 13 states and 410 counties in the eastern part of the United States. While their findings for most demographic categories were similar to the national findings by NSDUH, they had different results for racial/ethnic groups that varied by sub-regions. Overall, Whites were the demographic with the largest admission rate (83%), while Alaskan Native, American Indian, Pacific Islander, and Asian populations had the lowest admissions (1.8%).

Alcohol and Drug Abuse Around the World

In Asia, the prevalence of alcohol dependence is not as high as is seen in other regions. Many Asians (30% to 50% of people of Chinese, Japanese, and Korean ancestry) have at least one *ALDH2*2* allele, which may cause alcohol flush reaction, or a sensitivity to alcohol that causes flushes or blotches on the body after alcoholic consumption. In Europe in 2015, the estimated prevalence

among the adult population was 18.4% for heavy episodic alcohol use (in the past 30 days); 15.2% for daily tobacco smoking; and 3.8, 0.77, 0.37 and 0.35% in 2017 for cannabis, amphetamine, opioid, and cocaine use, respectively. The mortality rates for alcohol and illicit drugs were highest in Eastern Europe.

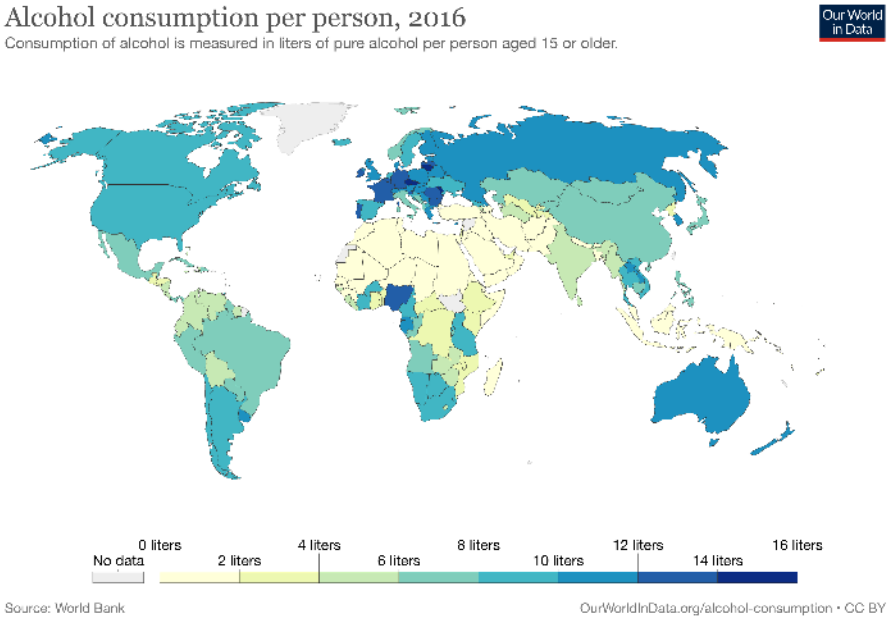


Figure **9.11.2**: Alcohol consumption throughout the world. Hannah Ritchie (2018) – "Alcohol Consumption". Published online at OurWorldInData.org. Retrieved from: 'https://ourworldindata.org/alcohol-consumption' [Online Resource]

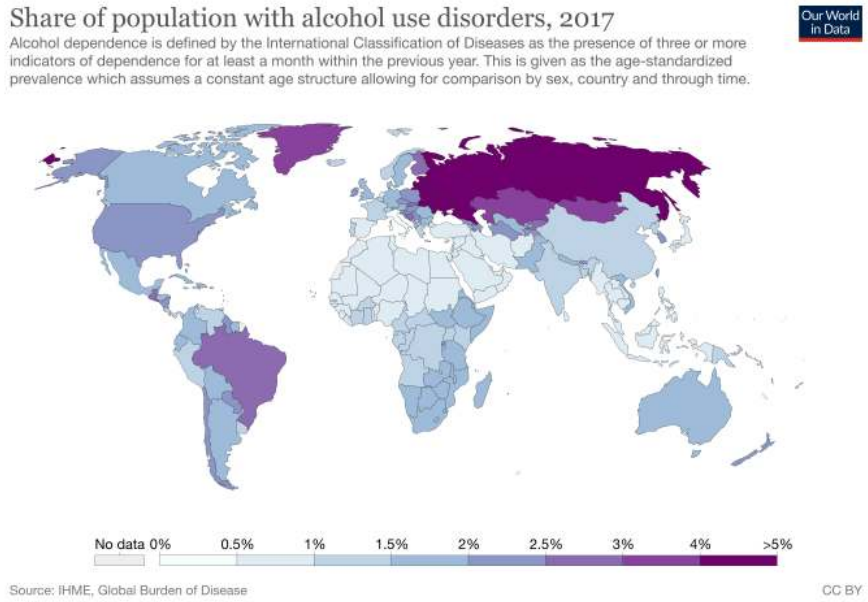


Figure **9.11.3**: Alcohol use disorder around the world is estimated to affect around 1.4% of the population. For most countries, the rate of prevalence is around 1 or 2%, but some countries, such as Russia, Greenland, and Brazil, have higher prevalence rates. Hannah Ritchie (2019) – "Drug Use". *Published online at OurWorldInData.org.* Retrieved from: 'https://ourworldindata.org/drug-use' [Online Resource]

The realities of opioid use and abuse in Latin America may be deceptive if observations are limited to epidemiological findings. In the United Nations Office on Drugs and Crime report, although South America produced 3% of the world's morphine and heroin and 0.01% of its opium, the prevalence of use is uneven. According to the Inter-American Commission on Drug Abuse Control, consumption of heroin is low in most Latin American countries, although Colombia is the area's largest opium producer. Mexico, because of its border with the United States, has the highest incidence of use.

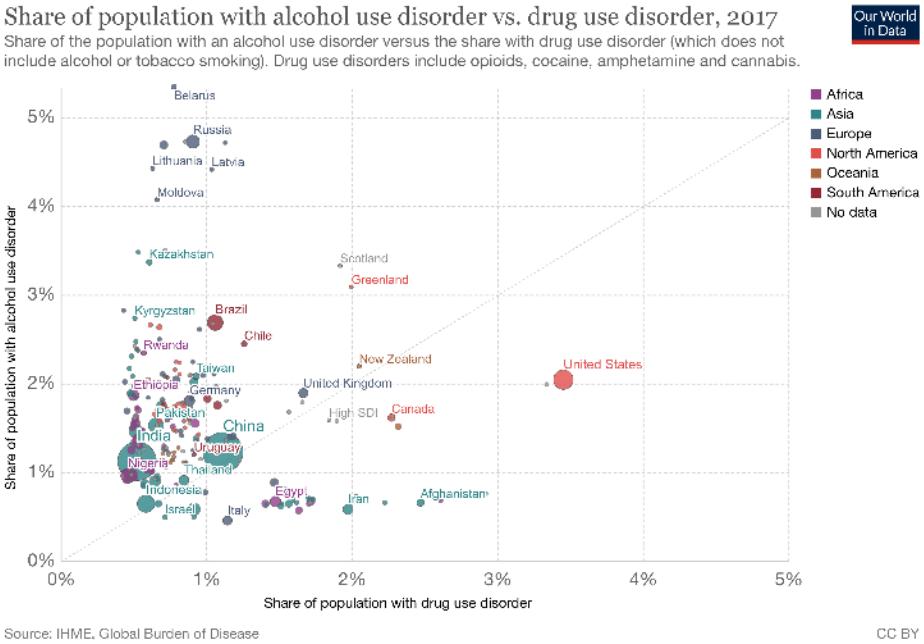


Figure **9.11.4**:While alcohol abuse is more common in Russia and Eastern Europe, the United States has the highest percentage of the population suffering from drug use disorders. Hannah Ritchie (2019) – "Drug Use". *Published online at OurWorldInData.org.* Retrieved from: 'https://ourworldindata.org/drug-use' [Online Resource]

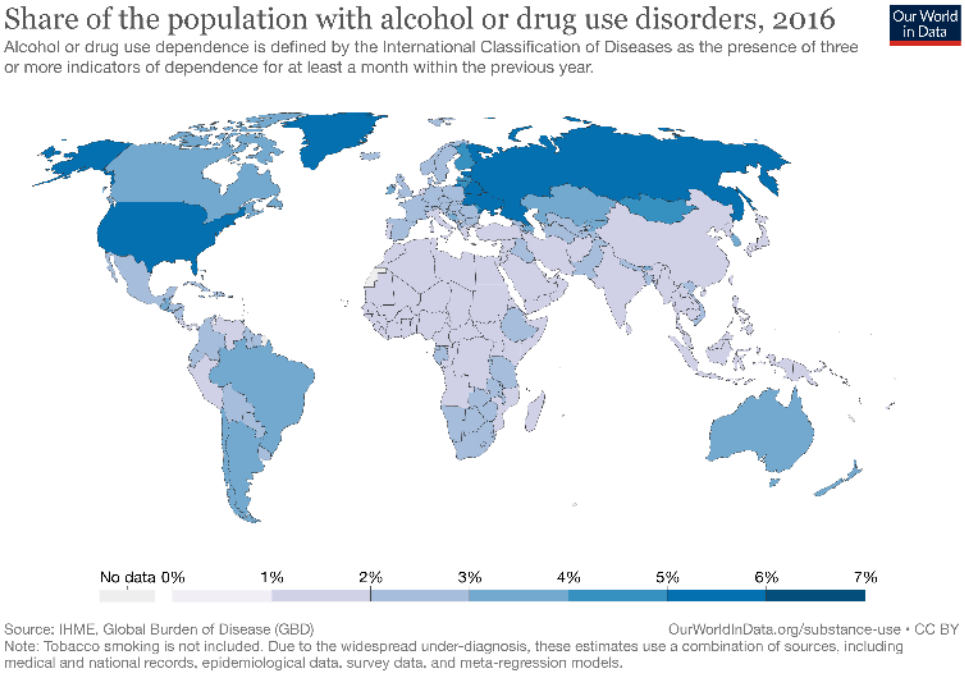


Figure **9.11.5**: Map showing the share of the population with alcohol or drug use disorders. Hannah Ritchie (2019) – "Drug Use". *Published online at OurWorldInData.org.* Retrieved from: 'https://ourworldindata.org/drug-use' [Online Resource]



Figure 9.11.6: One-third of inpatient hospital costs and 20% of all deaths in the United States every year are the result of untreated addictions and risky substance use.

Based upon representative samples of the U.S. youth population in 2011, the lifetime prevalence of addictions to alcohol and illicit drugs has been estimated to be approximately 8% and 2%-3%, respectively. Based upon representative samples of the U.S. adult population in 2011, the 12-month prevalence of alcohol and illicit drug addictions were estimated at roughly 12% and 2%-3% respectively. The lifetime prevalence of prescription drug addictions is currently around 4.7%.

As of 2016, about 22 million people in the United States need treatment for an addiction to alcohol, nicotine, or other drugs. Only about 10%, or a little over two million, receive any form of treatment, and those that do generally do not receive evidence-based care. One-third of inpatient hospital costs and 20% of all deaths in the United States every year are the result of untreated addictions and risky substance use. In spite of the massive overall economic cost to society, which is greater than the cost of diabetes and all forms of cancer combined, most doctors in the United States lack the training to effectively address drug addiction.

In 2019, opioid addiction was acknowledged as a national crisis in the United States. An article in *The Washington Post* stated that "America's largest drug companies flooded the country with pain pills from 2006 through 2012, even when it became apparent that they were fueling addiction and overdoses.

Watch It

In this video, Dr. Femke Buisman-Pijlman explains how biological, psychological, and social factors interact to either increase or reduce the risk that a person may develop a drug use disorder.



You can view the transcript for "Biopsychosocial Model" here (opens in new window).

Other Addictions

There are other types of addictions, unrelated to drugs, that elicit similar behaviors and patterns of abuse in individuals. Recall that a mental disorder is characterized by deviance, dysfunction, distress, and danger. Some behaviors may become so addictive that a person's thinking is dysfunctional, their behaviors are deviant, and that causes obvious distress. One disorder fitting this category in the DSM-5 is **gambling disorder**. This is classified when an individual has at least four of the following symptoms in a 12-month period:

- Needs to gamble with increasing amounts of money in order to achieve the desired excitement

- Is restless or irritable when attempting to cut down or stop gambling
- Has made repeated unsuccessful efforts to control, cut back, or stop gambling
- Is often preoccupied with gambling (e.g., having persistent thoughts of reliving past gambling experiences, handicapping or planning the next venture, thinking of ways to get money with which to gamble)
- Often gambles when feeling distressed (e.g., helpless, guilty, anxious, depressed)
- After losing money gambling, often returns another day to get even ("chasing" one's losses)
- Lies to conceal the extent of involvement with gambling
- Has jeopardized or lost a significant relationship, job, education, or career opportunity because of gambling
- Relies on others to provide money to relieve desperate financial situations caused by gambling

Would you consider yourself addicted to any social media platforms or games? Excessive Internet use has *not* been recognized as a disorder by the World Health Organization, the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), or the *International Classification of Diseases* (ICD-11), though the diagnosis of **gaming disorder** has been included in the *International Classification of Diseases* (ICD-11). Gaming disorder is defined as the problematic, compulsive use of video games that results in significant impairment to an individual's ability to function in various life domains over a prolonged period of time.

Mental health professionals have debated including compulsive shopping, stealing, excessive sexual behavior, and internet use as possible disorders, though there is controversy about whether or not a repetitive behavior in and of itself should constitute an addiction. There is also disagreement about whether these types of addictions should constitute a separate clinical entity, or whether they are a manifestation of underlying psychiatric disorders. Research has approached the question from a variety of viewpoints, with no universally standardized or agreed definitions, leading to difficulties in developing evidence-based recommendations.

Glossary

gambling disorder: mental disorder defined by an addiction to gambling

gaming disorder: controversial disorder described in the ICD-11 related to video game addiction

expectancy theory: idea that an individual will behave or act in a certain way because they are motivated to select a specific behavior over others due to what they expect the result of that selected behavior will be

personality theories of addiction: psychological models that associate personality traits or modes of thinking (i.e., affective states) with an individual's proclivity for developing an addiction

self-medication hypothesis: idea that an individual uses a substance or any exogenous influence to self-administer treatment for physical or psychological ailments

1. Khantzian E.J. (1997). "The self-medication hypothesis of drug use disorders: A reconsideration and recent applications". *Harvard Review of Psychiatry*. 4 (5): 231–244. doi:10.3109/10673229709030550. PMID 9385000. S2CID 39531697. ↵
2. Hogarth, L. Addiction is driven by excessive goal-directed drug choice under negative affect: translational critique of habit and compulsion theory. Neuropsychopharmacol. 45, 720–735 (2020). https://doi.org/10.1038/s41386-020-0600-8↵

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9.12: Treating Addiction

Learning Objectives

- Describe the process and effectiveness of pharmacological approaches to substance-use treatment
- Explain cognitive and behavioral approaches to treating substance-related disorders

Drug addiction can be treated, but it's not simple. Most patients need long-term or repeated care to stop using completely and recover their lives. Addiction treatment must help the person do the following:

- stop using drugs
- stay drug-free
- be productive in the family, at work, and in society

Depending on the severity of substance use disorder, and the given substance, early treatment of acute withdrawal may include medical detoxification. Of note, acute withdrawal from heavy alcohol use should be done under medical supervision to prevent a potentially deadly withdrawal syndrome known as delirium tremens.

Therapists often classify people with chemical dependencies as either interested or not interested in changing. About 11% of Americans with substance use disorder seek treatment, and 40–60% of those people relapse within a year. Treatments usually involve planning for specific ways to avoid the addictive stimulus, and therapeutic interventions intended to help a client learn healthier ways to find satisfaction. Clinical leaders in recent years have attempted to tailor intervention approaches to specific influences that affect addictive behavior, using therapeutic interviews in an effort to discover factors that led a person to embrace unhealthy, addictive sources of pleasure or relief from pain.

Principles of Effective Treatment

Based on scientific research since the mid-1970s, the following key principles should form the basis of any effective treatment program:

- Addiction is a complex but treatable disease that affects brain function and behavior.
- No single treatment is right for everyone.
- People need to have quick access to treatment.
- Effective treatment addresses all of the patient's needs, not just his or her drug use.
- Staying in treatment long enough is critical.
- Counseling and other behavioral therapies are the most commonly used forms of treatment.
- Medications are often an important part of treatment, especially when combined with behavioral therapies.
- Treatment plans must be reviewed often and modified to fit the patient's changing needs.
- Treatment should address other possible mental disorders.
- Medically assisted detoxification is only the first stage of treatment.
- Treatment doesn't need to be voluntary to be effective.
- Drug use during treatment must be monitored continuously.
- Treatment programs should test patients for HIV/AIDS, hepatitis B and C, tuberculosis, and other infectious diseases as well as teach them about steps they can take to reduce their risk of these illnesses.

What Are Treatments for Drug Addiction?

There are many options that have been successful in treating drug addiction, including:

- behavioral counseling
- medication
- medical devices and applications used to treat withdrawal symptoms or deliver skills training
- evaluation and treatment for co-occurring mental health issues such as depression and anxiety
- long-term follow-up to prevent relapse

A range of care with a tailored treatment program and follow-up options can be crucial to success. Treatment should include both medical and mental health services as needed. Follow-up care may include community- or family-based recovery support systems. Various types of programs offer help in drug rehabilitation, including residential treatment (in-patient/out-patient), local support groups, extended care centers, recovery or sober houses, addiction counseling, mental health, and medical care. Some rehab centers offer age- and gender-specific programs.

Drug or Addiction Counseling

Drug rehabilitation is the process of medical or psychotherapeutic treatment for dependency on psychoactive substances such as alcohol, prescription drugs, and street drugs such as cannabis, cocaine, heroin, or amphetamines. The general intent is to enable the patient to confront substance dependence, if present, and cease substance abuse to avoid the psychological, legal, financial, social, and physical consequences that can be caused, especially by extreme abuse.

Psychological dependency is addressed in many drug rehabilitation programs by attempting to teach the person new methods of interacting in a drug-free environment. In particular, patients are generally encouraged, or possibly even required, to not associate with peers who still use the addictive substance. Twelve-step programs encourage addicts not only to stop using alcohol or other drugs, but to examine and change habits related to their addictions. For legal drugs such as alcohol, complete abstention—rather than attempts at moderation, which may lead to relapse—is also emphasized ("One is too many, and a thousand is never enough.") Whether moderation is achievable by those with a history of abuse remains a controversial point.

Medications and devices can be used to manage withdrawal symptoms, prevent relapse, and treat co-occurring conditions.

Withdrawal. Medications and devices can help suppress withdrawal symptoms during detoxification. Detoxification is not in itself "treatment," but only the first step in the process. Patients who do not receive any further treatment after detoxification usually resume their drug use. One study of treatment facilities found that medications were used in almost 80% of detoxifications (SAMHSA, 2014). In November 2017, the Food and Drug Administration (FDA) granted a new indication to an electronic stimulation device, NSS-2 Bridge, for use in helping reduce opioid withdrawal symptoms. This device is placed behind the ear and sends electrical pulses to stimulate certain brain nerves. Also, in May 2018, the FDA approved lofexidine, a non-opioid medicine designed to reduce opioid withdrawal symptoms.

Relapse prevention. Patients can use medications to help re-establish normal brain function and decrease cravings. Medications are available for the treatment of opioid (heroin, prescription pain relievers), tobacco (nicotine), and alcohol addiction. Scientists are developing other medications to treat stimulant (cocaine, methamphetamine) and cannabis (marijuana) addiction. People who use more than one drug, which is very common, need treatment for all the substances they use.

- **Opioids:** Methadone (Dolophine® and Methadose®), buprenorphine (Suboxone®, Subutex®, Probuphine® , and Sublocade™), and naltrexone (Vivitrol®) are used to treat opioid addiction. Acting on the same targets in the brain as heroin and morphine, methadone and buprenorphine suppress withdrawal symptoms and relieve cravings. Naltrexone blocks the effects of opioids at their receptor sites in the brain and should be used only in patients who have already been detoxified. All medications help patients reduce drug seeking and related criminal behavior and help them become more open to behavioral treatments. A NIDA study found that once treatment is initiated, both a buprenorphine/naloxone combination and an extended release naltrexone formulation are similarly effective in treating opioid addiction. Because full detoxification is necessary for treatment with naloxone, initiating treatment among active users was difficult, but once detoxification was complete, both medications had similar effectiveness.



Figure **9.12.1**: Some people are able to quit cold turkey and never pick up their addiction again, but for most people, drug rehabilitation and treatment is a process with several ups and downs utilizing a variety of treatment methods, including medications and therapy.

- **Tobacco:** Nicotine replacement therapies have several forms, including the patch, spray, gum, and lozenges. These products are available over the counter. The FDA has approved two prescription medications for nicotine addiction: bupropion (Zyban®) and varenicline (Chantix®). They work differently in the brain, but both help prevent relapse in people trying to quit. The medications are more effective when combined with behavioral treatments, such as group and individual therapy as well as telephone quit lines. A few antidepressants have been proven to be helpful in the context of smoking cessation/nicotine addiction. These medications include bupropion and nortriptyline. Bupropion inhibits the re-uptake of nor-epinephrine and dopamine and has been FDA approved for smoking cessation, while nortriptyline is a tricyclic antidepressant that has been used to aid in smoking cessation but has not been FDA approved for this indication.
- ***Alcohol:*** Three medications have been FDA approved for treating alcohol addiction and a fourth, topiramate, has shown promise in clinical trials (large-scale studies with people). The three approved medications are as follows:
  - **Naltrexone** blocks opioid receptors that are involved in the rewarding effects of drinking and in the craving for alcohol. It reduces relapse to heavy drinking and is highly effective in some patients. Genetic differences may affect how well the drug works in certain patients.
  - **Acamprosate (Campral®)** may reduce symptoms of long-lasting withdrawal, such as insomnia, anxiety, restlessness, and dysphoria (generally feeling unwell or unhappy). It may be more effective in patients with severe addiction.
  - **Disulfiram (Antabuse®)** interferes with the breakdown of alcohol. Acetaldehyde builds up in the body, leading to unpleasant reactions that include flushing (warmth and redness in the face), nausea, and irregular heartbeat if the patient drinks alcohol. Compliance (taking the drug as prescribed) can be a problem, but it may help patients who are highly motivated to quit drinking.

Below are common treatments for various types of disorders:

- benzodiazepines intoxication: flumazenil

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9.13: Case Studies- Substance-Abuse Disorders

Learning Objectives

- Identify substance abuse disorders in case studies

Case Study: Benny

The following story comes from Benny, a 28-year-old living in the Metro Detroit area, USA. Read through the interview as he recounts his experiences dealing with addiction and recovery.



Q: How long have you been in recovery?

Benny: I have been in recovery for nine years. My sobriety date is April 21, 2010.

Q: What can you tell us about the last months/years of your drinking before you gave up?

Benny: To sum it up, it was a living hell. Every day I would wake up and promise myself I would not drink that day and by the evening I was intoxicated once again. I was a hardcore drug user and excessively taking ADHD medication such as Adderall, Vyvance, and Ritalin. I would abuse pills throughout the day and take sedatives at night, whether it was alcohol or a benzodiazepine. During the last month of my drinking, I was detached from reality, friends, and family, but also myself. I was isolated in my dark, cold, dorm room and suffered from extreme paranoia for weeks. I gave up going to school and the only person I was in contact with was my drug dealer.

Q: What was the final straw that led you to get sober?

Benny: I had been to drug rehab before and always relapsed afterwards. There were many situations that I can consider the final straw that led me to sobriety. However, the most notable was on an overcast, chilly October day. I was on an Adderall bender. I didn't rest or sleep for five days. One morning I took a handful of Adderall in an effort to take the pain of addiction away. I knew it wouldn't, but I was seeking any sort of relief. The damage this dosage caused to my brain led to a drug-induced psychosis. I was having small hallucinations here and there from the chemicals and a lack of sleep, but this time was different. I was in my own reality and my heart was racing. I had an awful reaction. The hallucinations got so real and my heart rate was beyond thumping. That day I ended up in the psych ward with very little recollection of how I ended up there. I had never been so afraid in my life. I could have died and that was enough for me to want to change.

Q: How was it for you in the early days? What was most difficult?

Benny: I had a different experience than most do in early sobriety. I was stuck in a drug-induced psychosis for the first four months of sobriety. My life was consumed by Alcoholics Anonymous meetings every day and sometimes two a day. I found guidance, friendship, and strength through these meetings. To say early sobriety was fun and easy would be a lie. However, I did learn it was possible to live a life without the use of drugs and alcohol. I also learned how to have fun once again. The most difficult part about early sobriety was dealing with my emotions. Since I started using drugs and alcohol that is what I used to deal with my emotions. If I was happy I used, if I was sad I used, if I was anxious I used, and if I couldn't handle a situation I used. Now that the drinking and drugs were out of my life, I had to find new ways to cope with my emotions. It was also very hard leaving my old friends in the past.

A link to an interactive elements can be found at the bottom of this page.

Q: What reaction did you get from family and friends when you started getting sober?

Benny: My family and close friends were very supportive of me while getting sober. Everyone close to me knew I had a problem and were more than grateful when I started recovery. At first they were very skeptical because of my history of relapsing after treatment. But once they realized I was serious this time around, I received nothing but loving support from everyone close to me. My mother was especially helpful as she stopped enabling my behavior and sought help through Alcoholics Anonymous. I have amazing relationships with everyone close to me in my life today.

Q: Have you ever experienced a relapse?

Benny: I experienced many relapses before actually surrendering. I was constantly in trouble as a teenager and tried quitting many times on my own. This always resulted in me going back to the drugs or alcohol. My first experience with trying to become sober, I was 15 years old. I failed and did not get sober until I was 19. Each time I relapsed my addiction got worse and worse. Each time I gave away my sobriety, the alcohol refunded my misery.

Q: How long did it take for things to start to calm down for you emotionally and physically?

Benny: Getting over the physical pain was less of a challenge. It only lasted a few weeks. The emotional pain took a long time to heal from. It wasn't until at least six months into my sobriety that my emotions calmed down. I was so used to being numb all the time that when I was confronted by my emotions, I often freaked out and didn't know how to handle it. However, after working through the 12 steps of AA, I quickly learned how to deal with my emotions without the aid of drugs or alcohol.

Q: How hard was it getting used to socializing sober?

Benny: It was very hard in the beginning. I had very low self-esteem and had an extremely hard time looking anyone in the eyes. But after practice, building up my self-esteem and going to AA meetings, I quickly learned how to socialize. I have always been a social person, so after building some confidence I had no issue at all. I went back to school right after I left drug rehab and got a degree in communications. Upon taking many communication classes, I became very comfortable socializing in any situation.

Q: Was there anything surprising that you learned about yourself when you stopped drinking?

Benny: There are surprises all the time. At first it was simple things, such as the ability to make people smile. Simple gifts in life such as cracking a joke to make someone laugh when they are having a bad day. I was surprised at the fact that people actually liked me when I wasn't intoxicated. I used to think people only liked being around me because I was the life of the party or someone they could go to and score drugs from. But after gaining experience in sobriety, I learned that people actually enjoyed my company and I wasn't the "prick" I thought I was. The most surprising thing I learned about myself is that I can do anything as long as I am sober and I have sufficient reason to do it.

Q: How did your life change?

Benny: I could write a book to fully answer this question. My life is 100 times different than it was nine years ago. I went from being a lonely drug addict with virtually no goals, no aspirations, no friends, and no family to a productive member of society. When I was using drugs, I honestly didn't think I would make it past the age of 21. Now, I am 28, working a dream job sharing my experience to inspire others, and constantly growing. Nine years ago I was a hopeless, miserable human being. Now, I consider myself an inspiration to others who are struggling with addiction.

Q: What are the main benefits that emerged for you from getting sober?

Benny: There are so many benefits of being sober. The most important one is the fact that no matter what happens, I am experiencing everything with a clear mind. I live every day to the fullest and understand that every day I am sober is a miracle. The benefits of sobriety are endless. People respect me today and can count on me today. I grew up in sobriety and learned a level of maturity that I would have never experienced while using. I don't have to rely on anyone or anything to make me happy. One of the greatest benefits from sobriety is that I no longer live in fear.

Case Study: Lorrie

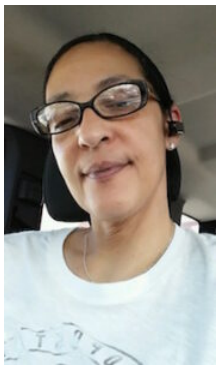


Figure **9.13.1**: Lorrie.

Lorrie Wiley grew up in a neighborhood on the west side of Baltimore, surrounded by family and friends struggling with drug issues. She started using marijuana and “popping pills” at the age of 13, and within the following decade, someone introduced her to cocaine and heroin. She lived with family and occasional boyfriends, and as she puts it, “I had no real home or belongings of my own.”

Before the age of 30, she was trying to survive as a heroin addict. She roamed from job to job, using whatever money she made to buy drugs. She occasionally tried support groups, but they did not work for her. By the time she was in her mid-forties, she was severely depressed and felt trapped and hopeless. “I was really tired.” About that time, she fell in love with a man who also struggled with drugs.

They both knew they needed help, but weren’t sure what to do. Her boyfriend was a military veteran so he courageously sought help with the VA. It was a stroke of luck that then connected Lorrie to friends who showed her an ad in the city paper, highlighting a research study at the National Institute of Drug Abuse (NIDA), part of the National Institutes of Health (NIH.) Lorrie made the call, visited the treatment intake center adjacent to the Johns Hopkins Bayview Medical Center, and qualified for the study.

“On the first day, they gave me some medication. I went home and did what addicts do—I tried to find a bag of heroin. I took it, but felt no effect.” The medication had stopped her from feeling it. “I thought—well that was a waste of money.” Lorrie says she has never taken another drug since. Drug treatment, of course is not quite that simple, but for Lorrie, the medication helped her resist drugs during a nine-month treatment cycle that included weekly counseling as well as small cash incentives for clean urine samples.

To help with heroin cravings, every day Lorrie was given the medication buprenorphine in addition to a new drug. The experimental part of the study was to test if a medication called clonidine, sometimes prescribed to help withdrawal symptoms, would also help prevent stress-induced relapse. Half of the patients received daily buprenorphine plus daily clonidine, and half received daily buprenorphine plus a daily placebo. To this day, Lorrie does not know which one she received, but she is deeply grateful that her involvement in the study worked for her.

The study results? Clonidine worked as the NIDA investigators had hoped.

“Before I was clean, I was so uncertain of myself and I was always depressed about things. Now I am confident in life, I speak my opinion, and I am productive. I cry tears of joy, not tears of sadness,” she says. Lorrie is now eight years drug free. And her boyfriend? His treatment at the VA was also effective, and they are now married. “I now feel joy at little things, like spending time with my husband or my niece, or I look around and see that I have my own apartment, my own car, even my own pots and pans. Sounds silly, but I never thought that would be possible. I feel so happy and so blessed, thanks to the wonderful research team at NIDA.”

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- One patientu2019s story: NIDA clinical trials bring a new life to a woman struggling with opioid addiction. **Provided by**: NIH. **Located at**: https://www.drugabuse.gov/drug-topics/treatment/one-patients-story-nida-clinical-trials-bring-new-life-to-woman-struggling-opioid-addiction. **License**: *Public Domain: No Known Copyright*

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9.14: Putting It Together- Substance-Related and Addictive Disorders



Substance use disorders describe the persistent use of drugs (including alcohol) despite substantial harm and adverse consequences. As you have learned in this module, substance use disorders are characterized by an array of mental/emotional, physical, and behavioral problems, such as chronic guilt, an inability to reduce or stop consuming the substance(s) despite repeated attempts, driving while intoxicated, and physiological withdrawal symptoms. Drug classes that are involved in substance use disorders include alcohol, caffeine, cannabis, phencyclidine and other hallucinogens, inhalants, opioids, sedatives, hypnotics or anxiolytics, stimulants, tobacco, and other or unknown substances. In the DSM-5 diagnosis of a substance use disorder, the severity of an individual's substance use disorder is ranked as mild, moderate, or severe on the basis of how many of the 11 diagnostic criteria are met.

In 2017, 271 million people globally (5.5% of adults) were estimated to have used one or more illicit drugs. In 2017, substance use disorders from illicit substances directly resulted in 585,000 deaths. Direct deaths from drug use, other than alcohol, have increased over 60% from 2000 to 2015; alcohol use resulted in an additional three million deaths in 2016. For all these reasons and more, it is essential to know the criteria, risk factors, and treatment methods for substance abuse disorders.

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CHAPTER OVERVIEW

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10.1: Why It Matters- Sexual Deviations and Dysfunctions

Why learn about sexual deviations and dysfunctions?



Sexual deviations and dysfunctions describe differences in gender, sex, and sexual behavior or preferences. In this module, we will learn about sex, gender dysphoria, sexual dysfunctions (which relate to concerns with sexual performance), and sexual deviations (mostly concerned with "abnormal" sexual desires and paraphilias).

Sexologists such as Henry Havelock Ellis and Alfred Kinsey began conducting research in the area of human sexuality during the first half of the 20th century. This work was groundbreaking and controversial in the scientific arena, but it paved the way for research on sex and sexual behavior. In the 1950s, sex therapy was concerned with "controlling sexual expression" and repressing what was then-considered deviant behaviors, such as homosexuality or having sex too often. Now, of course, the DSM-5 does not consider homosexuality a disorder, and we understand the dangers of conversion therapy.

William Masters and Virginia Johnson are credited with revolutionizing sex therapy in the mid-century and began treating sexual dysfunction by using couple therapy and behavioral interventions that focused on being present in the moment, such as sensate focus exercises. Through the following decades, sex therapy became increasingly common and taboos surrounding sex research decreased.

Despite more acceptance surrounding the discussion of sex and sexuality, sexual dysfunction disorders are not commonly discussed and often go undiagnosed. It is suggested that about 43% of women and 31% of men experience some type of sexual dysfunction, including sexual desire disorders, sexual arousal disorders, orgasm disorder, sexual pain disorders, or other sexual disorders.[1]

Sexual behavior and functioning, considered a biological drive, is influenced by a myriad of things, including psychosocial factors such as personal and family experiences, societal and religious influences, personal beliefs and expectations, etc. For this reason, sexual disorders are psychological in nature and described in the DSM-5, as they may have a somatic or psychogenic cause. A clear example is erectile dysfunction, whose causes may include circulatory problems and performance anxiety. Sex therapy often integrates psychotherapeutic techniques and medical ones, such as Viagra (sildenafil) to increase erectile response and Paxil (paroxetine) to treat premature ejaculation. Sex therapists assist those experiencing problems in overcoming their problems, and in so doing, possibly regain an active sex life. The transformative approach to sex therapy aims to understand the psychological, biological, pharmacological, relational, and contextual aspects of sexual problems.

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1. Avasthi, A., Grover, S., & Sathyanarayana Rao, T. S. (2017). Clinical Practice Guidelines for Management of Sexual Dysfunction. Indian journal of psychiatry, 59(Suppl 1), S91–S115. https://doi.org/10.4103/0019-5545.196977↵

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10.2: Introduction to Sex and Gender

What you'll learn to do: describe sexuality, sex, gender, and gender dysphoria



Human sexuality refers to people's sexual interest in and attraction to others; it is the capacity to have erotic or sexual feelings and experiences. Sexuality differs from biological sex, in that *sexuality* refers to the capacity for sexual feelings and attraction while *biologicalsex* refers to how one's anatomy, physiology, hormones, and genetics are classified (typically as male, female, or intersex). Sexuality is also separate from gender identity, which is a person's sense of their own *gender*, or sociocultural classification (i.e., man, woman, or another gender) based on biological sex (i.e., male or female). It is also distinct from—although it shapes—*sexual orientation*, or one's emotional and sexual attraction to a particular sex or gender.

Sexuality may be experienced and expressed in a variety of ways, including thoughts, fantasies, desires, beliefs, attitudes, values, behaviors, practices, roles, and relationships. These manifest themselves not only in biological, physical, and emotional ways, but also in sociocultural ways, which have to do with the effects of human society and culture on one's sexuality. Some researchers believe that sexual behavior is determined by genetics; however, others assert that it is largely molded by the environment. Human sexuality impacts, and is impacted by, cultural, political, legal, and philosophical aspects of life, and can interact with issues of morality, ethics, theology, spirituality, or religion.

In this section, you will learn about the variations in sexual orientation and gender identity, symptoms and factors associated with gender dysphoria, and the basic biological mechanisms regulating sexual behavior and motivation.

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10.3: Sexual Behavior

Learning Objectives

- Understand basic biological mechanisms regulating sexual behavior and motivation

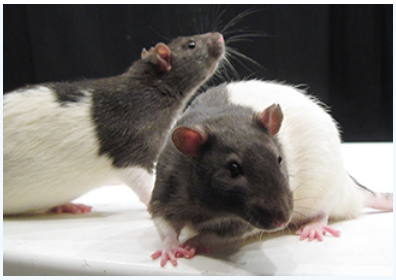


Figure **10.3.1**: A male rat that cannot engage in sexual behavior still seeks receptive females, suggesting that the ability to engage in sexual behavior and the motivation to do so are mediated by different systems in the brain. (credit: Jason Snyder)

Like food, sex is an important part of our lives. From an evolutionary perspective, the reason is obvious—the perpetuation of the species. Sexual behavior in humans, however, involves much more than reproduction. This section provides an overview of research that has been conducted on human sexual behavior and motivation. This section will close with a discussion of issues related to gender and sexual orientation.

Physiological Mechanisms of Sexual Behavior and Motivation

Much of what we know about the physiological mechanisms that underlie sexual behavior and motivation comes from animal research. As you have learned, the hypothalamus plays an important role in motivated behaviors, and sex is no exception. In fact, lesions to an area of the hypothalamus called the medial preoptic area completely disrupt a male rat's ability to engage in sexual behavior. Surprisingly, medial preoptic lesions do not change how hard a male rat is willing to work to gain access to a sexually receptive female (Figure **10.3.1**). This suggests that the ability to engage in sexual behavior and the motivation to do so may be mediated by neural systems distinct from one another.

Animal research suggests that limbic system structures such as the amygdala and nucleus accumbens are especially important for sexual motivation. Damage to these areas results in a decreased motivation to engage in sexual behavior while leaving the ability to do so intact (Figure **10.3.2**) (Everett, 1990). Similar dissociations of sexual motivation and sexual ability have also been observed in the female rat (Becker, Rudick, & Jenkins, 2001; Jenkins & Becker, 2001).

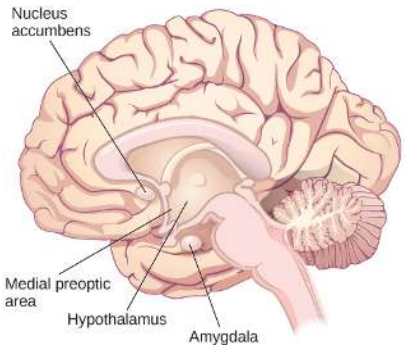


Figure **10.3.2**: The medial preoptic area, an area of the hypothalamus, is involved in the ability to engage in sexual behavior, but it does not affect sexual motivation. In contrast, the amygdala and nucleus accumbens are involved in motivation for sexual behavior, but they do not affect the ability to engage in it.

Although human sexual behavior is much more complex than that seen in rats, some parallels between animals and humans can be drawn from this research. The worldwide popularity of drugs used to treat erectile dysfunction (Conrad, 2005) speaks to the fact that sexual motivation and the ability to engage in sexual behavior can also be dissociated in humans. Moreover, disorders that

involve abnormal hypothalamic function are often associated with hypogonadism (reduced function of the gonads) and reduced sexual function (e.g., the genetic disorder Prader-Willi syndrome is characterized by hypogonadism).

Given the hypothalamus's role in endocrine function, it is not surprising that hormones secreted by the endocrine system also play important roles in sexual motivation and behavior. For example, many animals show no sign of sexual motivation in the absence of the appropriate combination of sex hormones from their gonads. While this is not the case for humans, there is considerable evidence that sexual motivation for both men and women varies as a function of circulating testosterone levels (Bhasin, Enzlin, Coviello, & Basson, 2007; Carter, 1992; Sherwin, 1988).

Kinsey's Research

Before the late 1940s, access to reliable, empirically based information on sex was limited. Physicians were considered authorities on all issues related to sex, despite the fact that they had little to no training in these issues, and it is likely that most of what people knew about sex had been learned either through their own experiences or by talking with their peers. Convinced that people would benefit from a more open dialogue on issues related to human sexuality, Dr. Alfred Kinsey of Indiana University initiated large-scale survey research on the topic (Figure 3). The results of some of these efforts were published in two books—*Sexual Behavior in the Human Male* and *Sexual Behavior in the Human Female*—which were published in 1948 and 1953, respectively (Bullough, 1998).



Figure **10.3.3**: In 1947, Alfred Kinsey established The Kinsey Institute for Research, Sex, Gender and Reproduction at Indiana University, shown here in 2011. The Kinsey Institute has continued as a research site of important psychological studies for decades.

At the time, the Kinsey reports were quite sensational. Never before had the American public seen its private sexual behavior become the focus of scientific scrutiny on such a large scale. The books, which were filled with statistics and scientific lingo, sold remarkably well to the general public, and people began to engage in open conversations about human sexuality. As you might imagine, not everyone was happy that this information was being published. In fact, these books were banned in some countries. Ultimately, the controversy resulted in Kinsey losing funding that he had secured from the Rockefeller Foundation to continue his research efforts (Bancroft, 2004).

Although Kinsey's research has been widely criticized as being riddled with sampling and statistical errors (Jenkins, 2010), there is little doubt that this research was very influential in shaping future research on human sexual behavior and motivation. Kinsey described a remarkably diverse range of sexual behaviors and experiences reported by the volunteers participating in his research. Behaviors that had once been considered exceedingly rare or problematic were demonstrated to be much more common and innocuous than previously imagined (Bancroft, 2004; Bullough, 1998).

Link to Learning

Watch this trailer from the 2004 film *Kinsey* that depicts Alfred Kinsey's life and research.

Among the results of Kinsey's research were the findings that women are as interested and experienced in sex as their male counterparts, that both males and females masturbate without adverse health consequences, and that homosexual acts are fairly common (Bancroft, 2004). Kinsey also developed a continuum known as the Kinsey scale that is still commonly used today to categorize an individual's sexual orientation (Jenkins, 2010).

Masters and Johnson's Research

In 1966, William Masters and Virginia Johnson published a book detailing the results of their observations of nearly 700 people who agreed to participate in their study of physiological responses during sexual behavior. Unlike Kinsey, who used personal interviews and surveys to collect data, Masters and Johnson observed people having intercourse in a variety of positions, and they observed people masturbating, manually or with the aid of a device. While this was occurring, researchers recorded measurements of physiological variables, such as blood pressure and respiration rate, as well as measurements of sexual arousal, such as vaginal lubrication and penile tumescence (swelling associated with an erection). In total, Masters and Johnson observed nearly 10,000 sexual acts as a part of their research (Hock, 2008).

Based on these observations, Masters and Johnson divided the **human sexual response cycle** into four phases that are fairly similar in men and women: excitement, plateau, orgasm, and resolution (Figure 4). The **excitement** phase is the arousal phase of the sexual response cycle, and it is marked by erection of the penis or clitoris and lubrication and expansion of the vaginal canal. During **plateau**, women experience further swelling of the vagina and increased blood flow to the labia minora, and men experience full erection and often exhibit pre-ejaculatory fluid. Both men and women experience increases in muscle tone during this time. **Orgasm** is marked in women by rhythmic contractions of the pelvis and uterus along with increased muscle tension. In men, pelvic contractions are accompanied by a buildup of seminal fluid near the urethra that is ultimately forced out by contractions of genital muscles, (i.e., ejaculation). **Resolution** is the relatively rapid return to an unaroused state accompanied by a decrease in blood pressure and muscular relaxation. While many women can quickly repeat the sexual response cycle, men must pass through a longer refractory period as part of resolution. The **refractory period** is a period of time that follows an orgasm during which an individual is incapable of experiencing another orgasm. In men, the duration of the refractory period can vary dramatically from individual to individual with some refractory periods as short as several minutes and others as long as a day. As men age, their refractory periods tend to span longer periods of time.

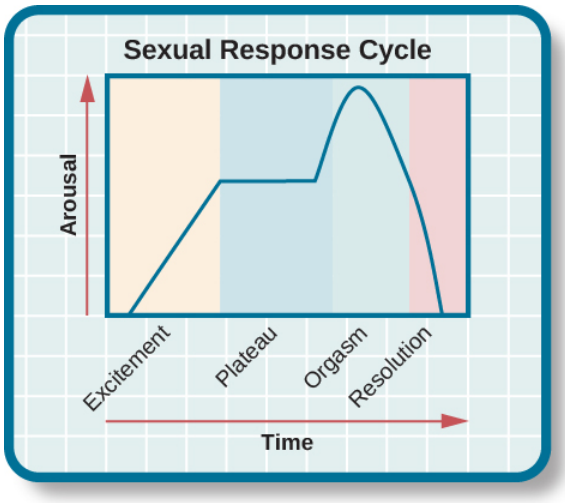


Figure **10.3.4**: This graph illustrates the different phases of the sexual response cycle as described by Masters and Johnson.

These stages are important for understanding the sexual dysfunctions because most of the dysfunctions are associated with the arousal and orgasm phases. In addition to the insights that their research provided with regards to the sexual response cycle and the multi-orgasmic potential of women, Masters and Johnson also collected important information about reproductive anatomy. Their research demonstrated the oft-cited statistic of the average size of a flaccid and an erect penis (3 and 6 inches, respectively) as well as dispelling long-held beliefs about relationships between the size of a man's erect penis and his ability to provide sexual pleasure to his female partner. Furthermore, they determined that the vagina is a very elastic structure that can conform to penises of various sizes (Hock, 2008).

Watch It

Watch the following episode of Crash Course Psychology in which Hank talks about Kinsey, Masters, and Johnson, sexuality, gender identity, hormones, and even looks into the idea of why people have sex.



You can view the transcript for "Let's Talk About Sex: Crash Course Psychology #27" here (opens in new window).

Glossary

asexuality: the lack of sexual attraction to others

biological sex: refers to how one's anatomy, physiology, hormones, and genetics are classified

bisexuality: romantic attraction, sexual attraction, or sexual behavior toward both males and females, or to more than one sex or gender

excitement: phase of the sexual response cycle that involves sexual arousal

human sexual response cycle: a four-stage model of physiological responses to sexual stimulation: excitement, plateau, orgasmic, and resolution phases

orgasm: peak phase of the sexual response cycle associated with rhythmic muscle contractions (and ejaculation)

plateau: phase of the sexual response cycle that falls between excitement and orgasm

refractory period: time immediately following an orgasm during which an individual is incapable of experiencing another orgasm

resolution: phase of the sexual response cycle following orgasm during which the body returns to its unaroused state

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10.4: Sexual Orientation and Gender Identity

Learning Objectives

- Describe variations in sexual orientation and gender identity

Sex and Sexual Orientation

As mentioned in the introduction, **biological sex** refers to how one's anatomy, physiology, hormones, and genetics are classified. **Gender identity** is a person's sense of their own *gender*, or sociocultural classification (i.e., man, woman, or another gender) based on biological sex (i.e., male, female, or intersex). **Sexual orientation** is an enduring pattern of romantic or sexual attraction (or a combination of these) to persons of the opposite sex or gender, the same sex or gender, or to both sexes or more than one gender. These attractions are generally subsumed under heterosexuality, homosexuality, and bisexuality, while **asexuality** (the lack of sexual attraction to others) is sometimes identified as the fourth category.)

According to the American Psychological Association, sexual orientation "also refers to a person's sense of identity based on those attractions, related behaviors, and membership in a community of others who share those attractions."[1] As a sexual orientation, **heterosexuality** is "an enduring pattern of emotional, romantic, and/or sexual attractions" to persons of the opposite sex; someone who is heterosexual is commonly referred to as straight.[2] As a sexual orientation, **homosexuality** is "an enduring pattern of emotional, romantic, and/or sexual attractions" to people of the same sex. The most common terms for homosexual people are lesbian for females and gay for males, but gay also commonly refers to both homosexual females and males. **Bisexuality** is romantic attraction, sexual attraction, or sexual behavior toward both males and females. It may also be defined as romantic or sexual attraction to people of any sex or gender identity, which is also known as **pansexuality**. Varying definitions and strong social norms about sexuality can make sexual orientation difficult to quantify.

While the majority of people identify as heterosexual, there is a sizable population of people within the United States who identify as either homosexual or bisexual. Research suggests that somewhere between three and 10% of the population identifies as homosexual (Kinsey, Pomeroy, & Martin, 1948; LeVay, 1996; Pillard & Bailey, 1995).



Figure **10.4.1**: Between three and 10% of the adult population identifies as homosexual. (credit: Till Krech)

Issues of sexual orientation have long fascinated scientists interested in determining what causes one individual to be heterosexual while another is homosexual. For many years, people believed that these differences arose because of different socialization and familial experiences. However, research has consistently demonstrated that family backgrounds and experiences are very similar among heterosexuals and homosexuals (Bell, Weinberg, & Hammersmith, 1981; Ross & Arrindell, 1988).

Genetic and biological mechanisms have also been proposed, and the balance of research evidence suggests that sexual orientation has an underlying biological component. For instance, over the past 25 years, research has demonstrated gene-level contributions to sexual orientation (Bailey & Pillard, 1991; Hamer, Hu, Magnuson, Hu, & Pattatucci, 1993; Rodriguez-Larralde & Paradisi, 2009), with some researchers estimating that genes account for at least half of the variability seen in human sexual orientation (Pillard &

Bailey, 1998). Other studies report differences in brain structure and function between heterosexuals and homosexuals (Allen & Gorski, 1992; Byne et al., 2001; Hu et al., 2008; LeVay, 1991; Ponseti et al., 2006; Rahman & Wilson, 2003a; Swaab & Hofman, 1990), and even differences in basic body structure and function have been observed (Hall & Kimura, 1994; Lippa, 2003; Loehlin & McFadden, 2003; McFadden & Champlin, 2000; McFadden & Pasanen, 1998; Rahman & Wilson, 2003b). In aggregate, the data suggest that to a significant extent, sexual orientations are something with which we are born.

Although the field once stigmatized non-heterosexual orientations, we now know that homosexuality and bisexuality are in no way related to mental health. Sexual orientation is neither chosen nor changed. Scientists do not know the exact cause of sexual orientation, but they theorize that it is caused by a complex interplay of genetic, hormonal, and environmental influences, and do not view it as a choice. Although no single theory on the cause of sexual orientation has yet gained widespread support, scientists favor biologically based theories. There is considerably more evidence supporting nonsocial, biological causes of sexual orientation than social ones, especially for males. While some people believe that homosexual activity is unnatural, scientific research shows that homosexuality is a normal and natural variation in human sexuality and is not in and of itself a source of negative psychological effects. There is insufficient evidence to support the use of psychological interventions to change sexual orientation.

Misunderstandings about Sexual Orientation

Regardless of how sexual orientation is determined, research has made clear that sexual orientation is not a choice, but rather it is a relatively stable characteristic of a person that cannot be changed. Claims of successful gay conversion therapy have received wide criticism from the research community due to significant concerns with research design, recruitment of experimental participants, and interpretation of data. As such, there is no credible scientific evidence to suggest that individuals can change their sexual orientation (Jenkins, 2010).

Dr. Robert Spitzer, the author of one of the most widely cited examples of successful conversion therapy, apologized to both the scientific community and the gay community for his mistakes, and he publicly recanted his own paper in a public letter addressed to the editor of *Archives of Sexual Behavior* in the spring of 2012 (Carey, 2012). In this letter, Spitzer wrote,

I was considering writing something that would acknowledge that I now judge the major critiques of the study as largely correct. . . . I believe I owe the gay community an apology for my study making unproven claims of the efficacy of reparative therapy. I also apologize to any gay person who wasted time or energy undergoing some form of reparative therapy because they believed that I had proven that reparative therapy works with some “highly motivated” individuals. (Becker, 2012, pars. 2, 5)

Citing research that suggests not only that gay conversion therapy is ineffective, but also potentially harmful, legislative efforts to make such therapy illegal have either been enacted (e.g., it is now illegal in California) or are underway across the United States, and many professional organizations have issued statements against this practice (Human Rights Campaign, n.d.)

Link to Learning

Read this draft of Dr. Spitzer’s letter.

Watch It

Watch this video to learn about variations and differences in biological sex and gender expression.



You can view the transcript for "Sexual orientation and gender identity" here (opens in new window).

Gender Identity

Many people conflate sexual orientation with gender identity because of stereotypical attitudes that exist about homosexuality. In reality, these are two related, but different, issues. **Gender identity** refers to one's sense of being male or female. Generally, our gender identities correspond to our chromosomal and phenotypic sex, but this is not always the case. When individuals do not feel comfortable identifying with the gender associated with their biological sex, then they experience gender dysphoria. **Gender dysphoria,** which we explore in the next section, is a diagnostic category in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) that describes individuals who do not identify as the gender that most people would assume they are. This dysphoria must persist for at least six months and result in significant distress or dysfunction to meet DSM-5 diagnostic criteria. In order for children to be assigned this diagnostic category, they must verbalize their desire to become the other gender.

Cisgender is an umbrella term used to describe people whose sense of personal identity and gender corresponds with their birth sex, while **transgender** is a term used to describe people whose sense of personal identity does not correspond with their birth sex. Approximately 1.4 million U.S. adults or 0.6% of the population are transgender according to a 2016 report.[3]

Many people who are classified as gender dysphoric seek to live their lives in ways that are consistent with their own gender identity. This involves dressing in opposite-sex clothing and assuming an opposite-sex identity. These individuals may also undertake **transgender hormone therapy** in an attempt to make their bodies look more like the opposite sex, and in some cases, they elect to have surgeries to alter the appearance of their external genitalia to resemble that of their gender identity (Figure 10.4.2). Transgender people who attempt to alter their bodies through medical interventions such as surgery and hormonal therapy are called **transsexual** individuals. They may also be known as male-to-female (MTF) or female-to-male (FTM). Not all transgender individuals choose to alter their bodies: many will maintain their original anatomy but may present themselves to society as another gender.



(a)



(b)

Figure **10.4.2**: Chaz Bono, a transgender male, is a well-known person who transitioned from female to male. (a) In the 1970s, the world knew Chaz as Chastity Bono, the daughter of the famous entertaining duo Sonny and Cher; here young Chastity is pictured with Sonny. (b) Later in life, Chaz transitioned to align his physical body with his gender identity. (credit b: modification of work by "dvsross"/Flickr)

Cultural Factors in Sexual Orientation and Gender Identity

Gender is deeply cultural. Like race, it is a social construction with real consequences, particularly for those who do not conform to gender binaries. In order to describe gender as a concept, we need to expand the language we use to describe gender beyond “masculine” or “feminine.” Gender identity, or the way that one thinks about gender and self-identifies, can be woman, man, or genderqueer.

Gender expression, or how one demonstrates gender (based on traditional gender role norms related to clothing, behavior, and interactions) can be feminine, masculine, androgynous, or somewhere along a spectrum. Although gender has traditionally been considered in binary terms (male or female), increasingly gender is being seen as a spectrum; however, our vocabulary is still limited in terms of the ways in which we describe gender identity.

Issues related to sexual orientation and gender identity are very much influenced by sociocultural factors. Even the ways in which we define sexual orientation and gender vary from one culture to the next. While in the United States exclusive heterosexuality is viewed as the norm, there are societies that have different attitudes regarding homosexual behavior. In fact, in some instances, periods of exclusively homosexual behavior are socially prescribed as a part of normal development and maturation. For example, in parts of New Guinea, young boys are expected to engage in sexual behavior with other boys for a given period of time because it is believed that doing so is necessary for these boys to become men (Baldwin & Baldwin, 1989).

There is a two-gendered culture in the United States. We tend to classify an individual as either male or female. However, in some cultures there are additional gender variants resulting in more than two gender categories. For example, in Thailand, you can be male, female, or kathoey. A ***kathoey*** is an individual who would be described as intersexed or transgender in the United States (Tangmunkongvorakul, Banwell, Carmichael, Utomo, & Sleigh, 2010).

Dig Deeper: The Case of David Reimer

In August of 1965, Janet and Ronald Reimer of Winnipeg, Canada, welcomed the birth of their twin sons, Bruce and Brian. Within a few months, the twins were experiencing urinary problems; doctors recommended the problems could be alleviated by having the boys circumcised. A malfunction of the medical equipment used to perform the circumcision resulted in Bruce’s penis being irreparably damaged. Distraught, Janet and Ronald looked to expert advice on what to do with their baby boy. By happenstance, the couple became aware of Dr. John Money at Johns Hopkins University and his theory of psychosexual neutrality (Colapinto, 2000).

Dr. Money had spent a considerable amount of time researching transgender individuals and individuals born with ambiguous genitalia. As a result of this work, he developed a theory of psychosexual neutrality. His theory asserted that we are essentially neutral at birth with regard to our gender identity and that we don’t assume a concrete gender identity until we begin to master

language. Furthermore, Dr. Money believed that the way in which we are socialized in early life is ultimately much more important than our biology in determining our gender identity (Money, 1962).

Dr. Money encouraged Janet and Ronald to bring the twins to Johns Hopkins University, and he convinced them that they should raise Bruce as a girl. Left with few other options at the time, Janet and Ronald agreed to have Bruce's testicles removed and to raise him as a girl. When they returned home to Canada, they brought with them Brian and his "sister," Brenda, along with specific instructions to never reveal to Brenda that she had been born a boy (Colapinto, 2000).

Early on, Dr. Money shared with the scientific community the great success of this natural experiment that seemed to fully support his theory of psychosexual neutrality (Money, 1975). Indeed, in early interviews with the children it appeared that Brenda was a typical little girl who liked to play with "girly" toys and do "girly" things.

However, Dr. Money was less than forthcoming with information that seemed to argue against the success of the case. In reality, Brenda's parents were constantly concerned that their little girl wasn't really behaving as most girls did, and by the time Brenda was nearing adolescence, it was painfully obvious to the family that she was really having a hard time identifying as a female. In addition, Brenda was becoming increasingly reluctant to continue her visits with Dr. Money to the point that she threatened suicide if her parents made her go back to see him again.

At that point, Janet and Ronald disclosed the true nature of Brenda's early childhood to their daughter. While initially shocked, Brenda reported that things made sense to her now, and ultimately, by the time she was an adolescent, Brenda had decided to identify as a male. Thus, she became David Reimer.

David was quite comfortable in his masculine role. He made new friends and began to think about his future. Although his castration had left him infertile, he still wanted to be a father. In 1990, David married a single mother and loved his new role as a husband and father. In 1997, David was made aware that Dr. Money was continuing to publicize his case as a success supporting his theory of psychosexual neutrality. This prompted David and his brother to go public with their experiences in attempt to discredit the doctor's publications. While this revelation created a firestorm in the scientific community for Dr. Money, it also triggered a series of unfortunate events that ultimately led to David committing suicide in 2004 (O'Connell, 2004).

This sad story speaks to the complexities involved in gender identity. While the Reimer case had earlier been paraded as a hallmark of how socialization trumped biology in terms of gender identity, the truth of the story made the scientific and medical communities more cautious in dealing with cases that involve intersex children and how to deal with their unique circumstances. In fact, stories like this one have prompted measures to prevent unnecessary harm and suffering to children who might have issues with gender identity. For example, in 2013, a law took effect in Germany allowing parents of intersex children to classify their children as indeterminate so that children can self-assign the appropriate gender once they have fully developed their own gender identities (Paramaguru, 2013).

Watch this news story to hear more about the experiences of David Reimer and his family.

Hear firsthand about the transgender experience and the disconnect that occurs when one's self-identity is betrayed by one's body. In this brief video interview, Chaz Bono discusses the difficulties of growing up identifying as male, while living in a female body.

Glossary

bisexual: emotional and erotic attractions to both same-sexed individuals and opposite-sexed individuals

cisgender: an umbrella term used to describe people whose sense of personal identity and gender corresponds with their birth sex

gender dysphoria: diagnostic category in DSM-5 for individuals who do not identify as the gender associated with their biological sex

gender expression: how one demonstrates gender based on traditional gender role norms related to clothing, behavior, and interactions

gender identity: a person's sense of their own gender, or sociocultural classification (i.e., man, woman, or another gender) based on biological sex

heterosexuality: an enduring pattern of emotional, romantic, and/or sexual attractions to persons of the opposite sex

homosexuality: an enduring pattern of emotional, romantic, and/or sexual attractions to people of the same sex

pansexuality: romantic or sexual attraction to people of any sex or gender identity

sexual orientation: emotional and erotic attraction to same-sexed individuals, opposite-sexed individuals, or both

transgender: a term used to describe people whose sense of personal identity does not correspond with their birth sex

transgender hormone therapy: use of hormones to make one's body look more like the opposite-sex

transsexual: transgender individuals who attempt to alter their bodies through medical interventions such as surgery and hormonal therapy

1. "Sexual Orientation & Homosexuality". American Psychological Association. 2020. Retrieved February 6, 2020. ↵
2. "Sexual orientation, homosexuality and bisexuality". American Psychological Association. Archived from the original on August 8, 2013. Retrieved August 10, 2013. ↵
3. [footnote]Flores, A., J. Herman, G. Gates, and T. N.T. Brown. "How many adults identify as transgender." The Williams Institute. http://williamsinstitute.law.ucla.edu/wp-content/uploads/How-Many-Adults-Identify-as-Transgender-in-the-United-States.pdf. ↵

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10.5: Gender Dysphoria

Learning Objectives

- Describe symptoms and factors associated with gender dysphoria

In 2013, the diagnosis for gender dysphoria (GD) was renamed from *gender identity disorder* (GID) after criticisms that the latter term was stigmatizing. The DSM-5 also moved this diagnosis out of the sexual disorders category and into a category of its own. In order to be diagnosed with gender dysphoria (GD), a person must experience, for at least six months, a noticeable difference between how he or she experiences/expresses his or her own gender and his or her biological sex. Symptoms related to this difference may include the expressed desire for others to treat or perceive him or her as another gender; discomfort with genitals or sex characteristics; wishing these genitals or sex characteristics were different or aligned with another gender; and/or a strong sense of being another gender despite how others perceive him or her. This discrepancy must cause significant impairment in social, occupational, school, or daily life functioning.

Symptoms



Figure **10.5.1**: A preference for opposite-sex typical toys may be a symptom of gender dysphoria.

The cardinal symptom of gender dysphoria (GD) is distress stemming from an incongruence between a person's felt gender and assigned sex/gender. Symptoms of GD in children include preferences for opposite-sex typical toys, games, or activities; great dislike of their own genitalia; and a strong preference for playmates of the opposite sex. Some children may also experience social isolation from their peers, anxiety, loneliness, and depression. According to the American Psychological Association, transgender children are more likely to experience harassment and violence in school, foster care, residential treatment centers, homeless centers, and juvenile justice programs than other children.[1] The diagnosis for children has been separated from that for adults. The creation of a specific diagnosis for children reflects the lesser ability of children to have insight into what they are experiencing or to express it in the event that they have insight. In order for children to be assigned this diagnostic category, they must verbalize their desire to become the other gender.

In adolescents and adults, symptoms include the desire to be and to be treated as the other gender. Adults with GD are at increased risk for stress, isolation, anxiety, depression, poor self-esteem, and suicide. Studies indicate that transgender people have an extremely high rate of suicide attempts; one study of 6,450 transgender people in the United States found 41% had attempted suicide, compared to a national average of 1.6%. It was also found that suicide attempts were less common among transgender people who said their family ties had remained strong after they came out, but even transgender people at comparatively low risk were still much more likely to have attempted suicide than the general population.[2] Transgender people are also at heightened risk for eating disorders and substance abuse.

Gender Dysphoria and Transgender Individuals

Many people who are diagnosed with gender dysphoria identify as transgender, **genderfluid** (a gender that varies over time), or otherwise **gender non-conforming** (anyone whose appearance and behavior does not reflect the gender roles expected of them) in some way; however, not everyone who identifies as transgender or gender non-conforming experiences gender dysphoria.

Transgender or *transsexual* is an umbrella term for people whose internal experience of gender does not match their biological sex (normally based on first and secondary sex characteristics). Transgender people may experience discomfort or distress due to their gender not aligning with their sex, and therefore wish to transition to being the gender they identify with.



Figure **10.5.2**: Recognizing spaces as gender-neutral is important in helping transgender and non-binary individuals feel included.

Some transgender people feel this way from a very young age, while others go through a period of questioning before realizing they are transgender. Transgender people can be men, women, or **non-binary** (a spectrum of gender identities that are not exclusively masculine or feminine). They can have any sexual orientation, express their gender through their appearance in any way, and may or may not fit into society's views of gender. Every transgender person has different desires for what they want (or do not want) to include in their transition, including surgery and other medical procedures. Transgender people who do not plan to have surgery are sometimes referred to as non-op; **transsexual** is sometimes used to refer to only those who do. It is important not to make assumptions about what is, was, or will be involved in any individual person's transition. **Transition** is any action a transgender person takes in order for the external world to better recognize and reflect their internal gender. This can range from asking people to use different names and pronouns, to a change in dress or appearance, to extensive surgery. The three main forms of transition are social, legal, and medical, although all of these are broad categories that can reflect dozens of different possible actions.

A post-transition specifier was also added for transgender individuals who have transitioned to their chosen gender (i.e., undergone hormonal or surgical procedures to alter their body in a way that matches their experienced gender identity). This specifier helps to ensure post-transition

individuals can continue to receive ongoing hormonal or other treatment as needed.

? Controversy Surrounding the Diagnosis of Gender Dysphoria

The previous diagnosis of gender identity disorder (GID) caused a great deal of controversy. Many transgender people and researchers supported the declassification of gender identity disorder (GID), arguing that the diagnosis pathologizes a natural form of gender variance, reinforces the binary model of gender (i.e., the idea that there are only two genders and that everyone must fit neatly into one of these two genders), and can result in stigmatization of transgender individuals. The official reclassification of gender dysphoria as a disorder in the DSM-5 may help resolve some of these issues, because the term *gender dysphoria* applies only to the discontent experienced by some persons resulting from gender identity issues, rather than suggesting that their identity is disordered.

Advantages and disadvantages exist to classifying gender dysphoria as a disorder, however. Many people argue that the distress associated with gender dysphoria is not caused by any disorder within the individual, but by difficulties encountered from social disapproval of transgender identities and alternative genders. As such, they argue that any form of diagnosis is still stigmatizing and places the "problem" unnecessarily on the individual, rather than on society. However, because gender dysphoria is classified as a disorder in the DSM-5, many insurance companies are willing to cover some of the expenses related to sex-reassignment therapy. Without the classification of gender dysphoria as a medical disorder, sex reassignment therapy may be viewed as cosmetic treatment—rather than medically necessary treatment for many transgender individuals—and thus may not be covered.

Early and Late-Onset Gender Dysphoria

Gender dysphoria in those assigned male at birth tends to follow one of two broad trajectories: early-onset or late-onset. **Early-onset gender dysphoria** is behaviorally visible in childhood. Sometimes gender dysphoria will desist in this group and they will identify as gay or homosexual for a period of time, followed by recurrence of gender dysphoria. This group is usually sexually attracted to members of their natal sex in adulthood. **Late-onset gender dysphoria** does not include visible signs in early childhood, but some report having had wishes to be the opposite sex in childhood that they did not report to others. Trans women who experience late-onset gender dysphoria will usually be sexually attracted to women and may identify as lesbians. It is common for people assigned male at birth who have late-onset gender dysphoria to engage in cross-dressing with sexual excitement. In those individuals who are biologically female, early-onset gender dysphoria is the most common course. This group is usually sexually attracted to women. Trans men who experience late-onset gender dysphoria will usually be sexually attracted to men and may identify as gay.[3]

Epidemiology

Gender dysphoria occurs in one in 30,000 biologically male births and one in 100,000 biologically female births.[4] It is estimated that about 0.005% to 0.014% of biological males and 0.002% to 0.003% of biological females would be diagnosed with gender dysphoria, based on 2013 diagnostic criteria, though this is considered a modest underestimate.[5] Research indicates people who transition in adulthood are up to three times more likely to be biologically male, but that among people transitioning in childhood the sex ratio is close to 1:1.[6]

According to an analysis of national probability samples in 2016, there were 390 per 100,000 adults who were transgender. However, it also suggested that future surveys will probably observe a higher prevalence.[7]

According to a recent national survey, 1.4 million individuals (0.6%) in the United States identify as transgender. It is also believed that these numbers are underrepresented due to the social stigma. Also, a part of this population might not want to engage in studies; hence, the true prevalence remains higher than what is reported. Nevertheless, an increasing shift is observed in this population seeking health care over the last decade.[8]

Substance use disorders are commonly found in men and women with GD, with some studies showing 28% having reported problems with substance use. In a recent study, about 48.3% of a study population had suicidal ideation, and 23.8% had attempted suicide at least once in their lifetime. Although, they were not able to appreciate any clinically significant difference between male-to-female (MTF) or female-to-male (FTM) groups. Anxiety, depression, and personality disorders are also common comorbidities. One study by Madeddu in 2009 found that personality disorder was comorbid in 52% of cases and the most common was Cluster B personality disorders.[9]

Etiology

The etiology of GD remains unclear, but it is thought to originate from a complex biopsychosocial link. Individuals born with congenital adrenal hyperplasia (a condition that involves excessive or deficient production of sex steroids and can alter the development of primary or secondary sex characteristics in some affected infants, children, or adults) or androgen insensitivity syndrome (an intersex condition) are usually brought up and socialized as girls, even though they often cross-dress and have an innate sense of belonging to the opposite sex. These changes are more evident around and during puberty. This is one of the well-established biological links.

Associations have also been found with in-utero exposure to phthalates in plastics and polychlorinated biphenyls. They are known to disrupt the regular endocrinology of sex determination before birth. Phthalates can lead to an increase in total fetal testosterone levels, which in turn increases the risk of autism spectrum disorder as well as GD.

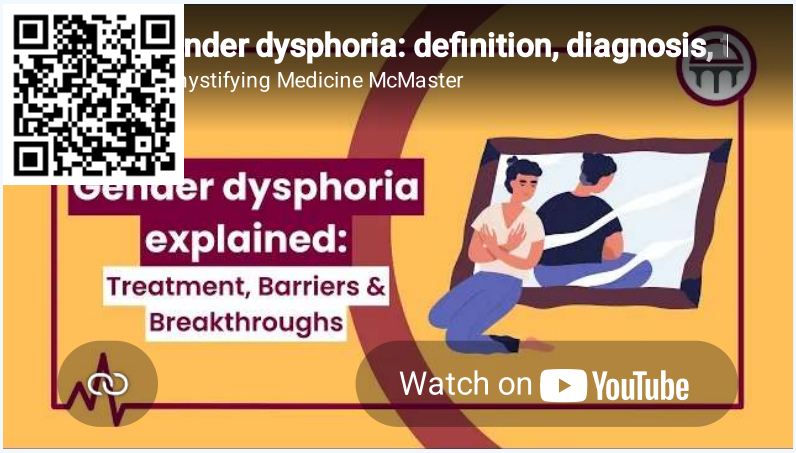
GD has been found to have a higher prevalence in people with psychiatric illnesses such as schizophrenia and autism spectrum disorder. The link seems to be neuroanatomical and needs more research. There was growing evidence those on the autism spectrum have a higher risk of GD; however, certain studies seek to disprove this hypothesis.

There is also growing evidence that childhood abuse, neglect, maltreatment, and physical or sexual abuse may be associated with GD. Individuals reporting higher body dissatisfaction and GD have a worse prognosis in terms of mental health. And as mentioned above in epidemiology, individuals with GD are found to have higher rates of depression, suicidal ideations, and substance use. Neuroanatomical links in those with GD have been found in certain studies, including a faulty neuronal development and differentiation in the hypothalamic links. Functional neuroimaging has shown variations in hemispheric ratios and amygdala connectivity according to gender. A few case reports have reported some association of GD to maternal toxoplasma infection, although additional data is needed for further evidence.

A genetic association is also identified as one of the causes of GD. Heritability and familiarity of GD have been identified: for instance, higher prevalence in monozygotic twins than dizygotic twins. Some alleles (CYP17 and CYP17 T-34C) have also been found to have an association, although it is difficult to say if it is merely association or causation.

Watch It

Watch this video to review the definition, diagnosis, treatment, and challenges of gender dysphoria.



You can view the transcript for "Gender dysphoria: definition, diagnosis, treatment and challenges" here (opens in new window).

This video takes a look at the story of transgender advocate and spokesperson, Jazz Jennings.



You can view the transcript for "Transgender at 11: Listening to Jazz Jennings | 20/20 | ABC News" here (opens in new window).

Learning Objectives

early-onset gender dysphoria: gender dysphoria behaviorally visible in childhood

genderfluid: a gender identity that varies over time

gender non-conforming: describes anyone whose appearance and behavior does not reflect the gender roles expected of them

late-onset gender dysphoria: gender dysphoria that does not include visible signs in early childhood

non-binary: a spectrum of gender identities that are not exclusively masculine or feminine

transsexual: a term sometimes used to refer to individuals with gender dysphoria who choose to undergo sex reassignment surgery

transition: any action a transgender person takes in order for the external world to better recognize and reflect their internal gender

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10.6: Perspectives and Treatments Related to Gender Dysphoria

Learning Objectives

- Examine historical and current perspectives on gender dysphoria
- Describe and evaluate treatments for gender dysphoria

Historical and Modern Perspectives on Gender and Gender Identity

Early Medical Literature

In late-19th-century medical literature, women who chose not to conform to their expected gender roles were called “inverts,” and they were portrayed as having an interest in knowledge and learning and a “dislike and sometimes incapacity for needlework.” During the mid-1900s, doctors pushed for corrective therapy on such women and children, which meant that gender behaviors that were not part of the norm would be punished and changed. The aim of this therapy was to push children back to their “correct” gender roles and thereby limit the number of children who became transgender.

Psychodynamic Perspectives: Freud and Jung’s Views

In 1905, Sigmund Freud presented his theory of psychosexual development in *Three Essays on the Theory of Sexuality*, giving evidence that in the pregenital phase children do not distinguish between sexes, but assume both parents have the same genitalia and reproductive powers. On this basis, he argued that bisexuality was the original sexual orientation and that heterosexuality was resultant of repression during the phallic stage, at which point gender identity became ascertainable. According to Freud, during this stage, children developed an **Oedipus complex** where they had sexual fantasies for the parent ascribed the opposite gender and hatred for the parent ascribed the same gender, and this hatred transformed into (unconscious) transference and (conscious) identification with the hated parent who both exemplified a model to appease sexual impulses and threatened to castrate the child’s power to appease sexual impulses. In 1913, Carl Jung proposed the **Electra complex** as he both believed that bisexuality did not lie at the origin of psychic life and that Freud did not give adequate description to the female child (Freud rejected this suggestion).

1950s and Beyond

During the 1950s and ’60s, psychologists began studying gender development in young children, partially in an effort to understand the origins of homosexuality (which was viewed as a mental disorder at the time). In 1958, the Gender Identity Research Project was established at the UCLA Medical Center for the study of intersex and transsexual individuals. Psychoanalyst Robert Stoller generalized many of the findings of the project in his book *Sex and Gender: On the Development of Masculinity and Femininity* (1968). He is also credited with introducing the term *gender identity* to the International Psychoanalytic Congress in Stockholm, Sweden, in 1963. Behavioral psychologist John Money was also instrumental in the development of early theories of gender identity. His work at Johns Hopkins Medical School’s Gender Identity Clinic (established in 1965) popularized an **interactionist theory of gender identity**, suggesting that, up to a certain age, gender identity is relatively fluid and subject to constant negotiation. His book *Man and Woman, Boy and Girl* (1972) became widely used as a college textbook, although many of Money’s ideas have since been challenged.

Butler’s views



Figure **10.6.1**: Judith Butler is an American philosopher and gender theorist.

In the late 1980s, Judith Butler began lecturing regularly on the topic of gender identity, and in 1990, she published *Gender Trouble: Feminism and the Subversion of Identity*, introducing the concept of **gender performativity,** arguing that both sex and gender are constructed.

Present views

Gender dysphoria (GD) exists when a person suffers discontent due to gender identity, causing them emotional distress. Researchers disagree about the nature of distress and impairment in people with gender dysphoria. Some authors have suggested that people with gender dysphoria suffer because they are stigmatized and victimized by society; if the society was more accepting of transgender identities and non-binary expressions of gender, they would suffer less and/or may not experience dysphoria at all. Other research into genetic variation, hormones, and differences in brain functioning and brain structures suggest evidence for the biological etiology of the symptoms associated with gender dysphoria; however, much of this research is preliminary and still controversial.

As you have read, in the past, gender identity development was mostly viewed through cognitive and behaviorist lens—arguing that struggles with gender identity developed from the environment or cognitive schemas. More recent research has been done to understand biological and influences on gender variances and how early social experiences may create lasting epigenetic changes related to sex differences. Research suggests that, for example, early social experiences may act as such epigenetic influence that they ultimately shape lasting sex differences in brain and behavior, but a lot more research is needed in this field to obtain solid knowledge relevant for understanding GD.

For example, early postmortem studies of transsexual neurological differentiation was focused on the hypothalamic and amygdala regions of the brain. Using magnetic resonance imaging (MRI), some transgender women were found to have female-typical putamina that were larger in size than those of cisgender males.[1] Some trans women have also shown a female-typical central part of the bed nucleus of the stria terminalis (BSTc) and interstitial nucleus of the anterior hypothalamus number 3 (INAH-3), looking at the number of neurons found within each.[2]

? Changing the Stigma

Today, most medical professionals who provide transgender transition services to adults now reject **conversion therapies** (the pseudoscientific practice of trying to change an individual's sexual orientation from homosexual or bisexual to heterosexual using psychological, physical, or spiritual interventions) as abusive and dangerous, believing instead what many transgender people have been convinced of: that when able to live out their daily lives with both a physical embodiment and a social expression that most closely matches their internal sense of self, transgender and transsexual individuals live successful, productive lives virtually indistinguishable from anyone else.

The APA's guidelines for psychotherapy with lesbian, gay, and bisexual clients (American Psychological Association, 2000, 2012) serve as a main reference for clinicians and highlight, among several issues, the need for clinicians to recognize that their own attitudes and knowledge about the experiences of sexual minorities are relevant to the therapeutic process with these clients and that, therefore, mental health care providers must look for appropriate literature, training, and supervision.

Treatment for Gender Dysphoria

The **World Professional Association for Transgender Health** (WPATH) **Standards of Care** (Version 7 from 2011) are considered by some as definitive treatment guidelines for providers. The Standards of Care for the Health of Transsexual, Transgender, and Gender Nonconforming People are international clinical protocols outlining the recommended assessment and treatment for gender non-conforming individuals across the lifespan or transgender or transsexual people who wish to undergo social, hormonal, or surgical transition to the other sex. Clinicians' decisions regarding patients' treatment are often influenced by this standard of care (SOC). They are most widespread standard of care (SOC) used by professionals working with transsexual, transgender, or gender variant people.

Other standards of care (SOC) exist, including the guidelines outlines in Gianna Israel and Donald Tarver's classic 1997 book *Transgender Care*. Several health clinics in the United States (e.g., Tom Waddell Health Center in San Francisco, Callen-Lorde Community Health Center in New York City, and Mazzoni Center in Philadelphia) have developed protocols for transgender

hormone therapy following a **harm reduction model** that is starting to be embraced by increasing numbers of providers. Willingness to provide hormonal therapy based on assessment of individual patients needs, history, and situation with an overriding goal of achieving the best outcome for patients rather than rigidly adhering to arbitrary rules has been successful.

Transgender transition services, the various medical treatments and procedures that alter an individual's primary and/or secondary sexual characteristics, are thus now considered highly successful, medically necessary interventions for many transgender persons, including but not limited to transsexuals, especially those who experience the deep distress of body dysphoria.

Achieving basic human rights for all transgender persons undoubtedly requires increased social acceptance of each individual's own expression of their identity, regardless of their biological gender or social role expectations. However, for those transgender individuals who experience the internal distress of body dysphoria, social acceptance of variation, while vastly important, will not be sufficient. For this segment of the transgender community, some medical services and procedures will also be required in order for these individuals to feel aligned with their bodies and for the distress of body dysphoria to be fully alleviated.

Treatment



Figure **10.6.2**: Some transgender individuals will undergo top or bottom surgeries, while others may choose not to have any surgery at all.

Today, sex reassignment surgery is performed on people who choose to have this change so that their anatomical sex will match their gender identity. Transgender individuals sometimes wish to undergo this type of surgery to refashion their primary sexual characteristics, secondary characteristics, or both, because they feel they will be more comfortable with different genitalia. This may involve removal of penis, testicles or breasts, or the fashioning of a penis, vagina, or breasts. In the past, sex assignment surgery has been performed on infants who are born with ambiguous genitalia. However, current medical opinion is strongly against this procedure on infants since many adults have regretted that these decisions were made for them at birth.

Gender confirmation surgery (or sexual reassignment surgery) refers to any form of surgical procedure performed on a transgender person in order to change their sex characteristics to better reflect their gender identity. Surgical procedures are usually preceded by hormone replacement therapy.

Some forms of gender confirmation surgery include

- **bottom surgery**, or surgery to alter the genitalia.
- **top surgery**, or surgery to alter the chest and breast tissue.
- facial reconstruction surgery, to alter the appearance of the face.

Those who plan to have surgery but not yet done so are often referred to as **pre-op** while those who have already had surgery are referred to as **post-op**. Those who do not wish to include surgery in their transition are referred to as **non-op**.

Sex reassignment surgery performed on nonconsenting minors (babies and children) may result in catastrophic outcomes (including PTSD and suicide—such as in the *David Reimer* case following a botched circumcision) when the individual's sexual identity (determined by neuroanatomical brain wiring) is discrepant with the surgical reassignment previously imposed. Milton Diamond at the John A. Burns School of Medicine of the University of Hawaii recommended that physicians do not perform surgery on children until they are old enough to give informed consent and to assign such infants in the gender to which they will probably best adjust. Diamond believed introducing children to others with differences of sex development could help remove shame and stigma. Diamond considered the intersex condition as a difference of sex development, not as a disorder.

The History of Transition Surgery

The goal of early transition surgeries was the removal of hormone-producing organs (such as the testicles and the ovaries) in order to reduce their masculinizing or feminizing effects. Later, as surgical technique became more complex, the goal became to produce functional sex organs from sex organs that are already present in the patient.

In the United States in 1917, Dr. Alan L. Hart, an American tuberculosis specialist, became one of the first female-to-male transsexuals to undergo hysterectomy and gonadectomy for the relief of gender dysphoria.

In Berlin in 1931, Dora Richter, became the first known transgender woman to undergo the vaginoplasty surgical approach.

This surgery was followed by Lili Elbe in Dresden during 1930–1931. She started with the removal of her original sex organs, the operation supervised by Dr. Magnus Hirschfeld. Lili went on to have four more subsequent operations that included an orchiectomy, an ovary transplant, a penectomy, and ultimately an unsuccessful uterine transplant, the rejection of which resulted in death. An earlier known recipient of this was Magnus Hirschfeld's housekeeper, but their identity is unclear at this time.

In 1951, Dr. Harold Gillies, a plastic surgeon active in World War II, worked to develop the first technique for female-to-male sex reassignment surgery, producing a technique that has become a modern standard, called phalloplasty. Phalloplasty is a cosmetic procedure that produces a visual penis out of grafted tissue from the patient.

Following phalloplasty, in 1999, the procedure for metoidioplasty was developed for female-to-male surgical transition by Drs. Lebovic and Laub. Considered a variant of phalloplasty, metoidioplasty works to create a penis out of the patient's present clitoris. This allows the patient to have a sensation-perceiving penis head. Metoidioplasty may be used in conjunction with phalloplasty to produce a larger, more "cis-appearing" penis in multiple stages.

On 12 June 2003, the European Court of Human Rights ruled in favor of Van Kück, a German trans woman whose insurance company denied her reimbursement for sex reassignment surgery as well as hormone replacement therapy. The legal arguments related to the Article 6 of the European Convention on Human Rights as well as the Article 8. This affair is referred to as *Van Kück vs Germany*.

In 2011, Christiane Völling won the first successful case brought by an intersex person against a surgeon for non-consensual surgical intervention described by the International Commission of Jurists as "an example of an individual who was subjected to sex reassignment surgery without full knowledge or consent."

Psychological Treatments

Treatment for a person diagnosed with GD may include psychotherapy or to support the individual's preferred gender through hormone therapy, gender expression and role, or surgery. Psychotherapy is any therapeutic interaction that aims to treat a psychological problem. This may include psychological counseling, resulting in lifestyle changes or physical changes resulting from medical interventions such as hormonal treatment, genital surgery, electrolysis or laser hair removal, chest/breast surgery, or other reconstructive surgeries. Psychotherapeutic treatment of GD involves helping the patient to adapt. The goal of treatment may simply be to reduce problems resulting from the person's transgender status, for example, counseling the patient in order to reduce guilt associated with cross-dressing or counseling a spouse to help them adjust to the patient's situation.

Until the 1970s, psychotherapy was the primary treatment for gender dysphoria and generally was directed to helping the person adjust to the gender of the physical characteristics present at birth. Though some clinicians still use only psychotherapy to treat gender dysphoria, it may now be used in addition to biological interventions. Attempts to alleviate GD by changing the patient's gender identity to reflect birth characteristics have been ineffective.

Biological Treatments

Biological treatments physically alter primary and secondary sex characteristics to reduce the discrepancy between an individual's physical body and gender identity. Biological treatments for GD without any form of psychotherapy is quite uncommon. Researchers have found that if individuals bypass psychotherapy in their GD treatment, they often feel lost and confused when their biological treatments are complete.[3]

Prepubescent Children

The question of whether to counsel young children to be happy with their biological sex or to encourage them to continue to exhibit behaviors that do not match their biological sex—or to explore a transgender transition—is controversial. The follow-up studies of children with gender dysphoria consistently show that the majority cease to feel transgender during puberty and identify instead as gay or lesbian.[4][5] Other clinicians also report that a significant proportion of young children diagnosed with gender dysphoria later do not exhibit any dysphoria.[6]

Professionals who treat gender dysphoria in children have begun to refer and prescribe hormones, known as puberty blockers, to delay the onset of puberty until a child is believed to be old enough to make an informed decision on whether hormonal gender reassignment leading to surgical gender reassignment will be in that person's best interest.

Psychological and Social Consequences

Overall, psychotherapy, hormone replacement therapy, and sex reassignment surgery together can be effective treating GD when the WPATH standards of care are followed. The overall level of patient satisfaction with both psychological and biological treatments is very high.

After sex reassignment surgery, **transsexual** individuals (people who underwent cross-sex hormone therapy and sex reassignment surgery) tend to be less gender dysphoric. They also normally function well both socially and psychologically. Anxiety, depression, and hostility levels were lower after sex reassignment surgery. They also tend to score well for self-perceived mental health, which is independent from sexual satisfaction. Many studies have been carried out to investigate satisfaction levels of patients after sex reassignment surgery. In these studies, most of the patients have reported being very happy with the results and very few of the patients have expressed regret for undergoing sex reassignment surgery.

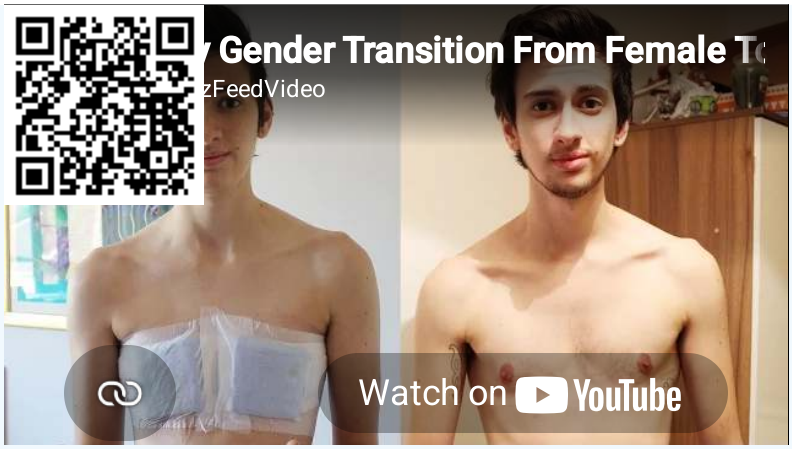
Although studies have suggested that the positive consequences of sex reassignment surgery outweigh the negative consequences, it has been suggested that most studies investigating the outcomes of sex reassignment surgery are flawed as they have only included a small percentage of sex reassignment surgery patients in their studies. These methodological limitations such as lack of double-blind randomized controls, small number of participants due to the rarity of transsexualism, high drop-out rates, and low follow-up rates, which would indicate need for continued study.

Persistent regret can occur after sex reassignment surgery. Regret may be due to unresolved gender dysphoria, or a weak and fluctuating sense of identity, and may even lead to suicide. Risk categories for post-operative regret include being older, having characterized personality disorders with personal and social instability, lacking family support, lacking sexual activity, and expressing dissatisfaction with the results of surgery. During the process of sex reassignment surgery, transsexuals may become victims of different social obstacles such as discrimination, prejudice, and stigmatizing behaviors. The rejection faced by transsexuals is much more severe than what is experienced by LGB individuals. The hostile environment may trigger or worsen internalized transphobia, depression, anxiety, and post-traumatic stress.

Many patients perceive the outcome of the surgery as not only medically but also psychologically important. Social support can help them to relate to their minority identity, ascertain their trans identity, and reduce minority stress. Therefore, it is suggested that psychological support is crucial for patients after sex reassignment surgery, which helps them feel accepted and to have confidence in the outcome of the surgery; also, psychological support will become increasingly important for patients with lengthier sex reassignment surgery process.

Watch It

This video tells the first-person account of Jamie's experience during his hormonal treatment and transition.



You can view the transcript for “My Gender Transition From Female To Male • Dear BuzzFeed” here (opens in new window).

Glossary

bottom surgery: surgery to alter the genitalia

conversion therapies: the pseudoscientific practice of trying to change an individual’s sexual orientation from homosexual or bisexual to heterosexual using psychological, physical, or spiritual interventions

gender confirmation surgery: sexual reassignment surgery or any form of surgical procedure performed on a transgender person in order to change their sex characteristics to better reflect their gender identity

gender performativity: concept arguing that both sex and gender are constructed

harm reduction model: a set of practical strategies and ideas aimed at providing hormonal therapy based on assessment of individual patient’s needs, history, and situation with an overriding goal of achieving the best outcome for patients rather than rigidly adhering to arbitrary rules

identity: the way one understands, describes and expresses oneself and the reflection of those entities to others

interactionist theory of gender identity: a theory that suggests that, up to a certain age, gender identity is relatively fluid and subject to constant negotiation

non-op: those who do not wish to include surgery in their transition

post-op: those who have already had surgery

pre-op: those who plan to have surgery but not yet done so

top surgery: surgery to alter the chest and breast tissue

transgender transition services: the various medical treatments and procedures that alter an individual’s primary and/or secondary sexual characteristics

transsexual: a person who has undergone cross-sex hormone therapy and sex reassignment surgery

World Professional Association for Transgender Health (WPATH) Standards of Care: international clinical protocols outlining the recommended assessment and treatment for gender non-conforming individuals across the lifespan or transgender or transsexual people who wish to undergo social, hormonal, or surgical transition to the other sex

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10.7: Introduction to Sexual Dysfunctions

What you'll learn to do: examine the characteristics, etiology, and treatments for sexual dysfunctions



Sexual health is vital to human society as we have to produce healthy children if society is to survive. Human sexuality is a complicated topic that affects society as a whole. Most societies impose strict rules about who may do what with whom because those rules have helped those societies survive. These rules are not universal, but they are almost always very strongly enforced either by public authorities or informally through social ridicule or castigation.

In this section, you will learn that there are a number of problems that can arise in the act of sex, and these are known collectively as sexual dysfunctions. Sexual dysfunction is difficulty experienced by an individual or a couple during any stage of normal sexual activity, including physical pleasure, desire, preference, arousal, or orgasm.

In this section, we will examine the characteristics, etiology, and treatments of sexual dysfunction disorders, including sexual desire disorders, arousal disorders, orgasm disorders, and pain disorders.

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10.8: Sexual Dysfunction Disorders

Learning Objectives

- Describe sexual dysfunction disorders, including sexual desire disorders, arousal disorders, orgasm disorders, and pain disorders

We first look into the four proposed categories of **sexual dysfunction disorders**: sexual desire disorders, arousal disorders, orgasm disorders and pain disorders as well as some proposed biological and psychological causes of these disorders. Sexual dysfunction among men and women is specifically studied in the fields of andrology and gynecology respectively.

There are several disorders related to sexual dysfunctions outlined in the DSM-5. These include

- delayed ejaculation,
- erectile disorder,
- female orgasmic disorder,
- female sexual interest/arousal disorder,
- genito-pelvic pain/penetration disorder,
- male hypoactive sexual desire disorder,
- premature ejaculation,
- substance or medication-induced sexual dysfunction, or
- other and unspecified sexual dysfunction.

We will not go into exhaustive detail about each disorder, but you should be familiar with the basic descriptions of each. They are summarized in the table below.

Key Takeaways: Sexual Dysfunctions

Table 1. Sexual Dysfunctions as Described in the DSM-5

Disorder	Description	Prevalence
Male hypoactive sexual desire disorder (MHSDD)	Persistently or recurrently deficient (or absent) sexual/erotic thoughts or fantasies and desire for sexual activity, as judged by a clinician with consideration for the patient's age and cultural context, and persisting for at least six months.	Unknown, although 6% of men between ages eight and 24 and 41% of men between 66 and 74 report problems with sexual desire.
Female sexual interest/arousal disorder (FSIAD)	Lack of interest or significantly reduced interest in sexual activity. At least three of the following symptoms occur for a minimum of six months: reduced interest in sex, reduced sexual thought or fantasies, reduced initiation of sexual activity, reduced excitement during sexual activity, reduced excitement to sexual cues, and reduced sensation during sexual encounters.	Unknown
Erectile disorder (ED)	Difficulty in obtaining or maintaining an erection during sexual activity 75–100% of the time.	Prevalence increases with age; 2% in men younger than 40–50 and 40–50% of men older than 60–70 years old.
Delayed ejaculation	Marked delay in ejaculation or infrequency/absence of ejaculation in 75–100% of partnered sexual activity.	Unknown. 75% of men report always ejaculating during sexual activity and 1% complain of problems with ejaculation in the past six months.
Premature ejaculation	Persistent and undesirable premature ejaculation that occurs around one minute or less of vaginal penetration.	Between 1%-3% of men
Female Orgasmic Disorder	Delay in, absence of, or markedly reduced intensity of orgasms during 75–100% of sexual activity.	Large variation; between 10 and 42%. 10% of women report never experiencing an orgasm.[1]
Genito-pelvic pain/penetration disorder (GPPPD)	Formerly called dyspareunia and vaginismus, this is characterized by difficulty or pain during vaginal penetration during intercourse. This may also include fear or anxiety about penetration or tensing and tightening of pelvic floor muscles during intercourse.	Unknown, but 15% of women in North American say they have recurrent pain during intercourse.

Sexual Desire Disorders

Sexual desire disorders or decreased libido are characterized by a lack or absence for some period of time of sexual desire or libido for sexual activity. The condition ranges from a general lack of sexual desire to a lack of sexual desire for the current partner. The condition may have started after a period of normal sexual functioning

or the person may always have had no or low sexual desire. The causes vary considerably, but include a possible decrease in the production of normal estrogen in women or testosterone in both men and women. Other causes may be aging, fatigue, pregnancy, medications (such as SSRIs), or psychiatric conditions, such as depression and anxiety.

Hypoactive Sexual Desire Disorder



Figure **10.8.1**: In some cases, HSDD may put a strain on relationships.

Hypoactive sexual desire disorder (HSDD) is considered a sexual dysfunction and is characterized as a lack or absence of sexual fantasies and desire for sexual activity, as judged by a clinician.

In the DSM-5, hypoactive sexual desire disorder (HSDD) was split into **male hypoactive sexual desire disorder** and **female sexual interest/arousal disorder**. Hypoactive sexual desire disorder (HSDD) was first included in the DSM-3 under the name *inhibited sexual desire disorder*, but the name was changed in the DSM-3-R. Other terms used to describe the phenomenon include sexual aversion and sexual apathy. More informal or colloquial terms are *frigidity* and *frigidness*.

There are various subtypes. HSDD can be general (general lack of sexual desire) or situational (still has sexual desire but lacks sexual desire for current partner), and it can be acquired (HSDD started after a period of normal sexual functioning) or lifelong (the person has always had no/low sexual desire.)

For HSDD to be regarded as a disorder, it must cause marked distress or interpersonal difficulties and not be better accounted for by another mental disorder, a drug (legal or illegal), or some other medical condition. A person with male hypoactive sexual desire disorder or female sexual interest/arousal disorder will not start or respond to their partner's desire for sexual activity.

Male Hypoactive Sexual Desire Disorder (MHSDD)

In the DSM-5, **male hypoactive sexual desire disorder** is characterized by "persistently or recurrently deficient (or absent) sexual/erotic thoughts or fantasies and desire for sexual activity," as judged by a clinician with consideration for the patient's age and cultural context.

Female Sexual Interest/Arousal Disorder (FSIAD)

Female sexual interest/arousal disorder is defined as a "lack of, or significantly reduced, sexual interest/arousal," manifesting through at least three of the following symptoms: no or little interest in sexual activity, no or few sexual thoughts, no or few attempts to initiate sexual activity or respond to partner's initiation, no or little sexual pleasure/excitement in 75–100% of sexual experiences, no or little sexual interest in internal or external erotic stimuli, and no or few genital/nongenital sensations in 75–100% of sexual experiences.

Diagnosing HSDD

For both diagnoses, symptoms must persist for at least six months, cause clinically significant distress, and not be better explained by another condition. Simply having lower desire than one's partner is not sufficient for a diagnosis. Self-identification of a lifelong lack of sexual desire as asexuality precludes diagnosis.

Low sexual desire alone is not equivalent to HSDD because of the requirement in HSDD that the low sexual desire causes marked distress and interpersonal difficulty and because of the requirement that the low desire is not better accounted for by another disorder in the DSM or by a general medical problem. It is therefore difficult to say exactly what causes HSDD. It is easier to describe, instead, some of the causes of low sexual desire.

Etiology of HSDD

The cause of lifelong/generalized HSDD is unknown. In the case of acquired/generalized low sexual desire, possible causes include various medical/health problems, psychiatric problems, low levels of testosterone or high levels of prolactin. One theory suggests that sexual desire is controlled by a balance between inhibitory and excitatory factors. This is thought to be expressed via neurotransmitters in selective brain areas. A decrease in sexual desire may therefore be due to an imbalance between neurotransmitters with excitatory activity like dopamine and norepinephrine and neurotransmitters with inhibitory activity, like serotonin. Low sexual desire can also be a side effect of various medications. In the case of acquired/situational HSDD, possible causes include intimacy difficulty, relationship problems, sexual addiction, and chronic illness of the partner. The evidence for these is somewhat in question. Some claimed causes of low sexual desire are based on empirical evidence. However, some are based merely on clinical observation. In many cases, the cause of HSDD is simply unknown.

Research has not yet examined factors associated with FSIAD and there has been little investigation of the impact of individual factors, notably stress, levels of fatigue, gender identity, health, and other individual attributes and experiences, such as dysfunctional sexual beliefs that may affect sexual desire or response. However, researchers have investigated a number of causes and consequences of low sexual interest in women and low sexual arousal. These elements are broken down into biological factors including medical health, hormones, and medications, and psychological factors including stress, relationships, comorbid mental illness, and history of sexual abuse.[2]

Sexual Arousal Disorders

Sexual arousal disorders were previously known as frigidity in women and impotence in men, though these have now been replaced with less judgmental terms. Impotence is now known as erectile dysfunction, and frigidity has been replaced with a number of terms describing specific problems that can be broken down into four categories. These four categories, as described by the DSM are lack of desire, lack of arousal, pain during intercourse, and lack of orgasm. For both men and women, these conditions can manifest themselves as an aversion to and avoidance of sexual contact with a partner. In men, there may be partial or complete failure to attain or maintain an erection, or a lack of sexual excitement and pleasure in sexual activity. There may be physiological origins to these disorders, such as decreased blood flow or lack of vaginal lubrication. Chronic disease can also contribute as well as the nature of the relationship between the partners.

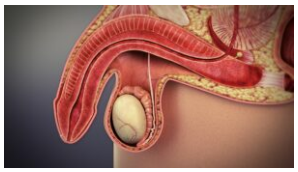


Figure **10.8.2**: Physical damage as a cause of erectile dysfunction is more severe than psychological causes.

Men with **erectile disorder** cannot attain or maintain an erection during sexual activity that is sufficient to allow them to initiate or maintain sexual activity. **Erectile dysfunction** or impotence is a sexual dysfunction characterized by the inability to develop or maintain an erection of the penis. There are various underlying causes, such as damage to the nervi erigentes, which prevents or delays erection, or diabetes as well as cardiovascular disease, which simply decreases blood flow to the tissue in the penis, many of which are medically reversible.

The causes of erectile dysfunction may be psychological or physical. Psychological erectile dysfunction can often be helped by almost anything that the patient believes in; there is a very strong placebo effect. Physical damage is much more severe. One leading physical cause of erectile dysfunction is continual or severe damage taken to the nervi erigentes. These nerves course beside the prostate arising from the sacral plexus and can be damaged in prostatic and colorectal surgeries. Diseases are also common causes of erectile dysfunction; especially in men. Diseases such as cardiovascular disease, multiple sclerosis, kidney failure, vascular disease, and spinal cord injury are the source of erectile dysfunction.

Due to its embarrassing nature and the shame felt by sufferers, the subject was taboo for a long time and is the subject of many urban legends. Folk remedies have long been advocated, with some being advertised widely since the 1930s. The introduction of perhaps the first pharmacologically effective remedy for impotence, sildenafil (trade name Viagra), in the 1990s caused a wave of public attention, propelled in part by the news-worthiness of stories about it and heavy advertising.

It is estimated that around 30 million men in the United States and 152 million men worldwide suffer from erectile dysfunction. However, social stigma, low health literacy, and social taboos lead to underreporting,which makes an accurate prevalence rate hard to determine.

Orgasm Disorders

Ejaculatory and orgasmic disorders are common male sexual dysfunctions. The DSM-5 specifically mentions delayed ejaculation and premature ejaculation (PE), although there are others that may include anejaculation (inability to orgasm), or retrograde ejaculation (where some of the semen travels back to the bladder), or anorgasmia (persistent inability or struggle to orgasm).

Delayed Ejaculation

Delayed ejaculation is a man's inability for or persistent difficulty in achieving orgasm, despite typical sexual desire and sexual stimulation. Generally, a man can reach orgasm within a few minutes of active thrusting during sexual intercourse, whereas a man with delayed ejaculation either does not have orgasms at all or cannot have an orgasm until after prolonged intercourse which might last for 30–45 minutes or more. In most cases, delayed ejaculation presents the condition in which the man can climax and ejaculate only during masturbation, but not during sexual intercourse. It is the least common of the male sexual dysfunctions, and can result as a side effect of some medications. In one survey, 8% of men reported being unable to achieve orgasm over a two-month period or longer in the previous year.

Delayed ejaculation can be *mild* (men who still experience orgasm during intercourse, but only under certain conditions), *moderate* (cannot ejaculate during intercourse but can during fellatio or manual stimulation), *severe* (can ejaculate only when alone), or *most severe* (cannot ejaculate at all). All forms may result in a sense of sexual frustration.

Medical conditions that can cause delayed ejaculation include hypogonadism, thyroid disorders, pituitary disorders such as Cushing's disease, prostate surgery outcome, and drug and alcohol use. Difficulty in achieving orgasm can also result from pelvic surgery that involved trauma to pelvic nerves responsible for orgasm. Some men report a lack of sensation in the nerves of the glans penis, which may or may not be related to external factors, including a history of circumcision. Delayed ejaculation is a possible side effect of certain medications, including selective serotonin reuptake inhibitors (SSRIs); opiates such as morphine, methadone, or oxycodone; many benzodiazepines such as Valium; certain antipsychotics; and antihypertensive.

Psychological and lifestyle factors have been discussed as potential contributors, including insufficient sleep, distraction due to worry, distraction from the environment, anxiety about pleasing their partner, and anxiety about relationship problems.

Premature Ejaculation

Premature ejaculation (PE) is a prevalent sexual dysfunction in men characterized as a persistent and undesirable premature ejaculation that occurs around one minute or less of vaginal penetration and must be experienced in almost all or all (75–100%) occasions during partnered sexual activity.Premature ejaculation (PE) often occurs when a man experiences orgasm and expels semen within a few moments of beginning sexual activity and with minimal penile stimulation. PE has also been called early ejaculation, rapid ejaculation, rapid climax, premature climax, and (historically) ejaculatio praecox.

There is no correct length of time for intercourse to last, but generally, premature ejaculation is thought to occur when ejaculation occurs in around one minute or less from the time of the insertion of the penis. Because of the variability in time required to ejaculate and in partners' desired duration of sex, exact prevalence rates of PE are difficult to determine. For a diagnosis, the patient must have a chronic history of premature ejaculation (at least six months), poor ejaculatory control, and the problem must cause feelings of dissatisfaction as well as distress for the patient, the partner, or both.

Historically attributed to psychological causes, new theories suggest that premature ejaculation may have an underlying neurobiological cause that may lead to rapid ejaculation. The nucleus paragigantocellularis of the brain has been identified as having involvement in ejaculatory control. Scientists have long suspected a genetic link to certain forms of premature ejaculation. However, studies have been inconclusive in isolating the gene responsible for lifelong PE. Other researchers have noted that men who have premature ejaculation have a faster neurological response in the pelvic muscles.

Premature ejaculation can be diagnosed as either lifelong (primary) or acquired (secondary), with acquired PE developing at some point in life after previous normal experiences. Acquired PE may be due to situational factors such as performance anxiety, relationship problems, or some ailments more comment in older men, such as prostatitis, hyperthyroidism, or diabetes.[3]

Female Orgasmic Disorder

In medical and scientific journals, the term **female orgasm disorder** is often referred to as anorgasmia.[4] It represents a type of sexual dysfunction in which a person cannot achieve orgasm despite adequate stimulation. Anorgasmia is also found in men, but not as often as in women.
Anorgasmia is one of the most common sexual problems in women. Symptoms associated with the disorder are most often the marked infrequency/absence of experiencing orgasm or delayed orgasm, heightened frustration and inhibition of sexual desire, fear of failure, prejudices, or avoidance of sexual contact for various reasons.[5]

In a clinical context, orgasm is usually defined strictly by the muscular contractions involved during sexual activity, along with the characteristic patterns of change in heart rate, blood pressure, and often respiration rate and depth. This is categorized as the sudden discharge of accumulated sexual tension during the sexual response cycle, resulting in rhythmic muscular contractions in the pelvic region. However, definitions of orgasm vary and there is a sentiment that consensus on how to consistently classify it is absent. At least twenty-six definitions of orgasm were listed in the journal *Clinical Psychology Review* .

There is some debate whether certain types of sexual sensations should be accurately classified as orgasms, including female orgasms caused by G-spot stimulation alone, and the demonstration of extended or continuous orgasms lasting several minutes or even an hour. The question centers around the clinical definition of orgasm, but this way of viewing orgasm is merely physiological while there are also psychological, endocrinological, and neurological definitions of orgasm. In these and similar cases, the sensations experienced are subjective and do not necessarily involve the involuntary contractions characteristic of orgasm. However, the sensations in both sexes are extremely pleasurable and are often felt throughout the body, causing a mental state that is often described as transcendental, and with vasocongestion and associated pleasure comparable to that of a full-contractionary orgasm.

Primary anorgasmia is a condition where one has never experienced an orgasm. Women with this condition can sometimes achieve a relatively low level of sexual excitement. Frustration, restlessness, and pelvic pain, or a heavy pelvic sensation may occur because of vascular engorgement. On occasion, there may be no obvious reason why orgasm is unobtainable. In such cases, women report that they are unable to orgasm even if they have a caring, skilled partner, adequate time and privacy, and an absence of medical issues that would affect sexual satisfaction.

Secondary anorgasmia is the loss of the ability to have orgasms or loss of the ability to reach orgasm of past intensity. The cause may be multifactorial (alcoholism, depression, grief, pelvic surgery or injuries, certain medications, illness, menopause, or rape).

About 15% of women report difficulties with orgasm, and as many as 10% of women in the United States have never climaxed. It is estimated that only 29% of women always have orgasms with their partner.

Sexual Pain Disorders

Sexual pain disorders affect women almost exclusively and are also known as dyspareunia (painful intercourse) or vaginismus (an involuntary spasm of the muscles of the vaginal wall that interferes with intercourse).

Genito-Pelvic Pain/Penetration Disorder (GPPPD)

Genito-pelvic pain/penetration disorder (GPPPD) is a new diagnosis included in the DSM-5, which merged the revised definitions of both female sexual dysfunctions *dyspareunia* and *vaginismus*.

At least one of the following persistent or recurrent criteria characterizes GPPPD: (1) difficulties with vaginal penetration during intercourse, (2) genito-pelvic pain during vaginal intercourse or penetration attempts, (3) fear or anxiety associated with genito-pelvic pain or vaginal penetration, or (4) tightness of the pelvic floor muscles during attempted vaginal penetration. One or more of these symptoms have to be present for at least six months and must cause clinically significant distress. GPPPD can be classified as either lifelong or acquired and, depending on the level of distress, as mild, moderate, or severe. The fusion of vaginismus and dyspareunia under a new classification and set of criteria was due to the significant overlap in clinical presentation and exceeding difficulties to distinguish between the two reliably.

Etiology of Genito-Pelvic Pain/Penetration Disorder (GPPPD)

Dyspareunia may be caused by insufficient lubrication (vaginal dryness) in women. Poor lubrication may result from insufficient excitement and stimulation or from hormonal changes caused by menopause, pregnancy, or breastfeeding. Irritation from contraceptive creams and foams can also cause dryness as can fear and anxiety about sex.

It is unclear exactly what causes vaginismus, but it is thought that past sexual trauma (such as rape or abuse) may play a role. Another female sexual pain disorder is called vulvodynia or vulvar vestibulitis. In this condition, women experience burning pain during sex which seems to be related to problems with the skin in the vulvar and vaginal areas. The cause is unknown.

The prevalence of GPPPD has not been ascertained due to the novel criteria set. Reported prevalence rates in the general population vary between three and 25% for dyspareunia and 0.4 and 6.6% for vaginismus. Prevalence estimates are heterogeneous due to varying diagnostic criteria, assessment methods, study design, and sample characteristics.

The burden of suffering associated with GPPPD and linked conditions such as *vulvodynia* and provoked *vestibulodynia* is high as symptoms have a detrimental impact on physiological and psychological health, and relational well-being. **Vulvodynia** is chronic pain in the vulva, the area on the outside of a woman's genitals. It is usually described as a sensation of burning, stinging, itching, or rawness. **Vestibulodynia** is chronic pain and discomfort that occurs in the area around the opening of the vagina, inside the inner lips of the vulva. This area is known as the *vestibule*.

GPPPD has been shown to have a negative effect on the woman's overall quality of life, with 60% of women reporting that the disorder compromised their ability to enjoy life. Moreover, it has been linked to depression and anxiety disorder. GPPPD symptoms are often comorbid with a wide range of other sexual dysfunctions and reduced sexual behavior. Many women with GPPPD also experience problems when using tampons or during gynecological examinations. GPPPD has been shown to contribute to declines in self-esteem and feelings of femininity and is associated with a negative body and genital self-image. It can pose a considerable burden on a couple's relationship, especially if they would like to have children.[6]

? Postorgasmic illness syndrome (**POIS**)

Postorgasmic illness syndrome (POIS) is a rare condition in which a person develops flu-like and allergy symptoms after orgasm, whether with a partner, through masturbation, or spontaneously during sleep. Additionally, the condition postorgasm illness syndrome (POIS) may cause symptoms when aroused, including adrenergic-type presentation; rapid breathing, paraesthesia, palpitations, headaches, aphasia, nausea, itchy eyes, fever, muscle pain, and weakness and fatigue. From the onset of arousal, symptoms can persist for up to a week in patients. The etiology of this condition is unknown, however, it is believed to be a pathology of either the immune system or autonomic nervous systems. It is defined as a rare disease by the NIH but the prevalence is unknown. It is not thought to be psychiatric in nature, but it may present as anxiety relating to coital activities and thus may be incorrectly diagnosed as such. There is no known cure or treatment.

Glossary

erectile dysfunction: a sexual dysfunction characterized by the inability to develop or maintain an erection of the penis

hypoactive sexual desire disorder (HSDD): a sexual dysfunction characterized as a lack or absence of sexual fantasies and desire for sexual activity, as judged by a clinician

primary anorgasmia: a condition where one has never experienced an orgasm

postorgasmic illness syndrome (POIS): a rare condition in which a person develops flu-like and allergy symptoms after orgasm, whether with a partner, through masturbation, or spontaneously during sleep

secondary anorgasmia: the loss of the ability to have orgasms or loss of the ability to reach orgasm of past intensity

sexual dysfunction: difficulty experienced by an individual or a couple during any stage of normal sexual activity, including physical pleasure, desire, preference, arousal, or orgasm

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10.9: Treating Sexual Dysfunction Disorders

Learning Objectives

- Examine perspectives and treatments related to sexual dysfunction disorders

Perspectives on Sexual Dysfunction Disorders

There are many factors that may result in a person experiencing sexual dysfunction. These may result from emotional or physical causes. Emotional factors include interpersonal or psychological problems, which can be the result of depression, sexual fears or guilt, past sexual trauma, and sexual disorders, among others.

Sexual dysfunction is especially common among people who have anxiety disorders. Ordinary anxiousness can obviously cause erectile dysfunction in men without psychiatric problems, but clinically diagnosable disorders such as panic disorder commonly cause avoidance of intercourse and premature ejaculation. Pain during intercourse is often a comorbidity of anxiety disorders among women. Therefore, clinicians are careful not to rule out the fact that there may be physiological causes of these dysfunctions as well.

The Biological Model

Physical factors that can lead to sexual dysfunctions include the use of drugs, such as alcohol, nicotine, narcotics, stimulants, antihypertensives, antihistamines, and some psychotherapeutic drugs. Excessive use of alcohol or use of other recreational drugs may cause sexual dysfunction, either by a direct effect on the penile neurovascular system or by causing increased secretion of prolactin, reduction in the production of testosterone, or both. Many medications are commonly associated with sexual dysfunction, including diuretics, antipsychotics, antidepressants, benzodiazepines, buspirone, lithium, disulfiram, oral contraceptives, etc.

For women, almost any physiological change that affects the reproductive system—premenstrual syndrome, pregnancy, the postpartum period, and menopause—can have an adverse effect on libido. A common physiological culprit of anorgasmia is menopause, where one in three women report problems obtaining an orgasm during sexual stimulation following menopause.

For men, diseases such as cardiovascular disease, multiple sclerosis, kidney failure, vascular disease, and spinal cord injury are the source of erectile dysfunction. Historically attributed to psychological causes, new theories suggest that premature ejaculation may have an underlying neurobiological cause that may lead to rapid ejaculation.[1]

The Psychodynamic Model

Psychological factors associated with sexual dysfunction can be divided into three categories: predisposing factors, precipitating factors, and maintaining factors. Predisposing factors include things like trauma, inadequate sexual information, disturbed family relationships, or insecurities. Precipitating factors include unreasonable or negative expectations, lack of harmony in the relationships, infidelity, or depression. Maintaining factors may include any of those same issues, just maintained over a longer time period.[2].

The Sociocultural Model

Cultural and societal myths surrounding sex may also cause sexual dysfunctions. For example, some people erroneously learn that men always think about sex, the man should be the leader, penis size is important, couples must have frequent sex, or sex happens automatically. Other myths perpetuate about orgasms and when they happen, supposed dangers of masturbation, circumcision, and more.[3]

In the context of heterosexual relationships, one of the main reasons for the decline in sexual activity among these couples is the male partner experiencing erectile dysfunction. This can be very distressing for the male partner, causing poor body image, and it can also be a major source of low desire for these men. In aging women, it is natural for the vagina to narrow and become atrophied. If a woman has not been participating in sexual activity regularly (in particular, activities involving vaginal penetration) with her partner, if she does decide to engage in penetrative intercourse, she will not be able to immediately accommodate a penis without risking pain or injury. This can turn into a vicious cycle, often leading to female sexual dysfunction.

According to Emily Wentzell, American culture has anti-aging sentiments that have caused sexual dysfunction to become “an illness that needs treatment” instead of viewing it as the natural part of the aging process it is. Not all cultures seek treatment; for example, a population of men living in Mexico often accept erectile dysfunction as a normal part of their maturing sexuality.[4]

Female Sexual Dysfunction

Several theories have looked at female sexual dysfunction, from medical to psychological perspectives. Three social psychological theories include the self-perception theory, the overjustification hypothesis, and the insufficient justification hypothesis:

- Self-perception theory: people make attributions about their own attitudes, feelings, and behaviors by relying on their observations of external behavior and the circumstances in which those behaviors occur.
- Overjustification hypothesis: when an external reward is given to a person for performing an intrinsically rewarding activity, the person’s intrinsic interest will decrease.
- Insufficient justification: based on the classic cognitive dissonance theory (inconsistency between two cognitions or between a cognition and a behavior will create discomfort), this theory states that people will alter one of the cognitions or behaviors to restore consistency and reduce distress.

The importance of how a woman perceives her behavior should not be underestimated. Many women perceive sex as a chore as opposed to a pleasurable experience, and they tend to consider themselves sexually inadequate, which in turn does not motivate them to engage in sexual activity. Several factors influence a women’s perception of her sexual life. These factors can include race, gender, ethnicity, educational background, socioeconomic status, sexual orientation, financial resources, culture, and religion. Cultural differences are also present in how women view menopause and its impact on health, self-image, and sexuality. A study has found that African-American women are the most optimistic about menopausal life, Caucasian women are the most anxious, Asian women are the most inhibited about their symptoms, and Hispanic women are the most stoic.

About one-third of the women experienced sexual dysfunction, which may lead to women’s loss of confidence in their sexual lives. Since these women had sexual problems, their sexual lives with their partners became a burden without pleasure, and eventually, they may completely lose interest in sexual activity. Some of the women found it hard to be aroused mentally; however, some had physical problems. Several factors can affect female dysfunction, such as situations in which women do not trust their sex partners. The environment where sex occurs is crucial, since being in an extremely public or extremely private place may make some women feel uncomfortable. Inability to concentrate on the sexual activity due to a bad mood or burdens from work may also cause a woman’s sexual dysfunction.[5]

Treating Sexual Dysfunction Disorders

Cognitive sexual therapy (CST) is a cognitive-behavioral integrative psychotherapy aimed specifically to address and treat SDs, articulating evidence-based clinical interventions to scientific understandings of human sexuality. In a cognitive sexual therapy (CST) perspective, distorted sexual cognitions, allied to individuals’ misinterpretations of sexual demands, directly affect emotional, physiological, and behavioral regulation in sexual situations. In this sense, restructuring central and intermediate cognitive processes is essential to foster the acquisition of sexual skills and to promote the development of a more adapted sexual repertoire. Thus, cognitions are understood as mediating factors to be modified, aiming sexual skills implementation and emotional regulation during sexual encounters.

Sexual scripts are ideas of how males and females are supposed to interact with each other, including how each gender should behave in sexual or romantic situations. Being able to flexibilize sexual scripts and behaviors leads to better adaptation to physiological, environmental, and relational changes that negatively impact sexuality over the life cycle, contributing to booster sexual satisfaction even in the presence of sexual function disturbances. The focus of cognitive sexual therapy (CST) interventions is to aid patients and partners towards the development of more flexible and adaptable sexual cognitions and behavioral patterns. CST could permit a regain in sexual function and satisfaction during and after treatment with psychotropic medication, lessen the negative impact of sexual adverse effects in quality of life and therefore, increase adherence and therapeutic effects of pharmacotherapy. Although the theoretical rationale supporting this proposal is evidence-based, feasibility, efficacy, treatment modalities, and procedural aspects of the intervention remain to be empirically tested.



Figure 10.9.1: Sex therapy, at any age, often involves sensate-focused touching.

Sex Therapy

Sex therapy is a strategy for the improvement of sexual function and treatment of sexual dysfunction including premature ejaculation or delayed ejaculation, erectile dysfunction, lack of sexual interest or arousal, and painful sex. It includes dealing with problems imposed by atypical sexual interests (paraphilias); gender dysphoria and being transgender; highly overactive libido or hypersexuality; a lack of sexual confidence; recovering from sexual abuse, such as rape or sexual assault; and sexual issues related to aging, illness, or disability.

It can include sensate focus**,** communication, and fantasy exercises as well as psychodynamic therapy. **Sensate focus** is a sex therapy technique introduced by the Masters and Johnson team. It works by refocusing the participants on their own sensory perceptions and sensuality, instead of goal-oriented behavior focused on the genitals and penetrative sex. Sensate focus has been used to treat problems with body image, erectile dysfunction, orgasm disorders, and lack of sexual arousal.

The exercises are conducted by the couple at home between therapy sessions. Although the couple are nude and touching each other during the exercises, they are instructed to abstain from sexual intercourse during or close to the sessions. Both participants are instructed instead to focus on their own varied sense experience, instead of focusing on performance or orgasm. Initially, the emphasis is on touching in a mindful way for oneself without regard for sexual response or pleasure for oneself or one's partner. In the second stage, they still abstain from intercourse and use touch to learn about their bodies and to identify what is pleasurable. Eventually, further stages lead again to intercourse, with the focus on learning about the partner's body.

Taking about sex can be embarrassing for both the clinician and the patients, so it is important for the clinician to reassure the patient of confidentiality. Treatment plans will vary depending on the condition. For example, sexual desire disorders have no particular treatment plan and do not generally respond well to psychotherapy.[6] Some therapies, like treating vaginismus, include sex education and information to help the patient come to relax or to overcome negative preconceived notions about sex. Relaxation training may be helpful in these instances. Generally, several types of psychoterapies may be used in sex therapy, including psychodynamic, rational emotive therapy, CBT, or behavioral therapies such as systematic desensitization, and Masters and Johnson's behavioral therapy techniques (which focus on talk therapy with a couple making behavioral changes together that they work on as homework assignments following a therapy session).

There are also pharmacological treatment options for some sexual dysfunctions, particularly erectile dysfunction and premature ejaculation. Lifestyle changes such as discontinuing smoking and drug or alcohol abuse can also help in some types of erectile dysfunction. Several oral medications like Viagra, Cialis, and Levitra have become available to help people with erectile dysfunction and have become first-line therapy. These medications provide an easy, safe, and effective treatment solution for approximately 60% of men. In the rest, the medications may not work because of the wrong diagnosis or chronic history. Another type of medication that is effective in roughly 85% of men is called intracavernous pharmacotherapy and involves injecting a vasodilator drug directly into the penis in order to stimulate an erection. This method has an increased risk of priapism if used in conjunction with other treatments and localized pain. Pelvic floor physical therapy has been shown to be a valid treatment for men with sexual problems and pelvic pain.

In 2015, flibanserin was approved in the United States to treat decreased sexual desire in women. While it's effective for some women, it has been criticized for its limited efficacy, and many warnings and contraindications that limit its use. Women experiencing pain with intercourse are often prescribed pain relievers or desensitizing agents; others can be prescribed vaginal lubricants. Many women with sexual dysfunction are also referred to a counselor or sex therapist.[7]

Glossary

cognitive sexual therapy (CST): a cognitive-behavioral integrative psychotherapy aimed specifically to address and treat sexual dysfunctions

sensate focus: a sex therapy technique that works by refocusing the participants on their own sensory perceptions and sensuality, instead of goal-oriented behavior focused on the genitals and penetrative sex

sex therapy: a strategy for the improvement of sexual function and treatment of sexual dysfunction

sexual scripts: ideas of how males and females are supposed to interact with each other, including how each gender should behave in sexual or romantic situations

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10.10: Introduction to Sexual Deviations

What you'll learn to do: examine the characteristics, etiology, and treatments of sexual deviations



What are sexual deviations? As we have discussed, human sexuality spans a range of behaviors, and varies from culture to culture, thus making it hard to define what type of sexual expression is acceptable. From a clinical perspective, there has been some effort to define *sexual deviation* under the umbrella of the sexual paraphilias. The term *paraphilia* comes from the Greek παρά (*para*) "beside" and φιλία (*-philia*) "friendship, love" and was first coined by Friedrich Salomon Krauss, a Croatian Austrian Jewish sexologist in 1903, and used with some regularity by Wilhelm Stekel in the 1920s. The term is used to describe atypical sexual interests, and there remains debate regarding technical accuracy and perceptions of stigma. "Paraphilia" indicates that certain behaviors are somehow deviant when in fact they may be just at different ends of the spectrum[1].

Homosexuality used to be considered a paraphilia, but this is no longer the case (although it some countries it is still considered abnormal behavior). Sexologist John Money popularized the term *paraphilia* as a non-pejorative designation for unusual sexual interests. Money described paraphilia as "a sexuoerotic embellishment of, or alternative to the official, ideological norm." Despite efforts by Money, the term *paraphilia* remains pejorative in most circumstances. For clinical purposes, the term paraphilia is the experience of intense sexual arousal to atypical objects, situations, fantasies, behaviors, or individuals.

In this section, you will become familiar with the sexual paraphilias, previously known as sexual perversions and sexual deviations. Although not innately pathological, a **paraphilic disorder** can evolve if paraphilia invokes harm, distress, or functional impairment on the lives of the affected individual or others. A total of eight paraphilias are listed in the DSM-5 and include pedophilia, exhibitionism, voyeurism, sexual sadism, sexual masochism, frotteurism, fetishism, and transvestic fetishism.

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10.11: Paraphilic Disorders

Learning Objectives

- Describe and differentiate between paraphilias: fetishism, transvestic fetishism, exhibitionism, voyeurism, frotteurism, sexual sadism, sexual masochism, and pedophilia

As mentioned earlier, **paraphilias** are persistent and recurrent sexual interests, urges, fantasies, or behaviors of marked intensity involving objects, activities, or even situations that are atypical in nature. We will discuss the characteristics and etiology of the eight paraphilias listed in the DSM-5 fetishism, transvestic fetishism, exhibitionism, voyeurism, frotteurism, sexual sadism, sexual masochism, and pedophilia. Pedophilia will be discussed in a separate section because of its close relationship to criminal behavior and will be followed by a discussion on sex and violence.

No consensus has been found for any precise border between unusual sexual interests and paraphilic ones. There is debate over which, if any, of the paraphilias should be listed in diagnostic manuals, such as the DSM-5 or ICD-11. The number and taxonomy of paraphilias is under debate; one source lists as many as 549 types of paraphilias. The DSM-5 has specific listings for eight paraphilic disorders, but several sub-classifications of the paraphilias have been proposed and some argue that a fully dimensional, spectrum, or complaint-oriented approach would better reflect the evidence.

The DSM-5 adds a distinction between paraphilias and paraphilic disorders, stating that paraphilias do not require or justify psychiatric treatment in themselves, and defining paraphilic disorder as “a paraphilia that is currently causing distress or impairment to the individual or a paraphilia whose satisfaction has entailed personal harm, or risk of harm, to others.”[1]

Types of Paraphilias



Figure **10.11.1**: Cross-dressing is not always a result of transvestic fetishism.

Fetishism is the use of nonliving objects, most commonly shoes and undergarments, for sexual pleasure. **Transvestic fetishism** is the derivation of sexual arousal from cross-dressing or dressing in clothes of the opposite sex. **Exhibitionism** is the exposure of an individual’s genitalia to unsuspecting strangers for sexual satisfaction. **Voyeurism** is the viewing of an unsuspecting person engaging in disrobing or sexual activity. **Frotteurism** is the touching of or rubbing against a nonconsenting person. **Sexual masochism** is the derivation of sexual arousal from being the recipient of physical or mental abuse and/or humiliation. **Sexual sadism** is when sexual arousal is gained from inflicting mental or physical suffering on a nonconsenting person. **Pedophilia** is any sexual activity with or sexual fantasies about a prepubescent child, where the offender is at least sixteen years of age.

Etiology

The exact etiology of paraphilia and paraphilic disorders is unknown. However, it is thought that a combination of neurobiological, interpersonal, and cognitive processes all play a role. Literature also points towards various genetic factors contributing to the development of pedophilia and pedophilic disorder, with recent evidence displaying a positive correlation of the COMT Val158Met (rs4680) polymorphism in paraphilic child sexual offenders. A recent study focusing on neurotransmission of paraphilic disorders found evidence to suggest that central dopamine plays a key role in the pathogenesis of paraphilic disorders as well as the general disturbance of the conscious regulation of behavior. The results of this study revealed increased levels of serotonin and norepinephrine, with a decreased concentration of DOPAC (3,4-dihydroxyphenylacetic acid) in urine samples of the test population diagnosed with paraphilic disorders. A correlation was made between serotonin and norepinephrine with obsessive disturbances and an association of DOPAC with affective and dissociative disorders.

Epidemiology

Literature is lacking with information pertaining to the epidemiology of paraphilia and paraphilic disorders. Literature is also limited from an epidemiological standpoint, with paraphilia typically studied in a general sense rather than pertaining to specific paraphilia or paraphilic disorders. Paraphilias, in general, are more common in men, with reasons unknown. A recent study conducted looked specifically at the desire for and experience of paraphilic behaviors of a sample population demographically representative of the general population. The sample size contained a total of 1,040 persons classified according to gender, age, education, ethnicity, religion, and location of residency. It was found that almost half of the study population expressed interest in one or more paraphilic categories, with approximately one-third of this population actually acting on this interest at least once. Specifically, fetishism, frotteurism, voyeurism, and masochism held a prevalence of 15.9% (value considered to be statistically unusual), with interest in both males and females. Interest levels in fetishism and masochism revealed no statistically significant difference amongst males and females. It was also found that the most common paraphilic interest amongst men is often voyeurism and fetishism.

Let’s learn a little bit more about the types of paraphillic disorders.



Figure 10.11.2: Foot fetishes are more common than you think.

Sexual fetishism or **erotic fetishism** is a sexual fixation on a nonliving object or nongenital body part. The object of interest is called the **fetish**; the person who has *a fetish* for that object is a **fetishist**. Sexual arousal from a particular body part can be further classified as **partialism**, or sexual interest with an exclusive focus on a specific part of the body other than the genitals. Partialism is categorized as a fetishistic disorder in the DSM-5 only if it causes significant psychosocial distress for the person or has detrimental effects on important areas of their life. Fetishism is diagnosed by recurrent and intense sexual arousal from either the use of nonliving objects or a highly specific focus on non-genital body part(s), as manifested by fantasies, urges, or behaviors that occur for at least six months. It must cause significant stress or impairment and not be related to clothing used in cross-dressing (transvestic disorder) or objects used for genital stimulation, like a vibrator.

Individuals who exhibit partialism sometimes describe the anatomy of interest to them as having an equal or greater erotic attraction for them as do the genitals. Partialism occurs in heterosexual, bisexual, and homosexual individuals. The foot is considered one of the most common partialisms.

? Partialisms

The following are some of the partialisms commonly found among those with fetishes:

Formal name	Common name	Source of arousal
Podophilia	Foot fetish	Foot
Oculophilia	Eye fetish	Eye
Maschalagnia	Armpit fetish	Armpits
Mazophilia	Breast fetish	Breasts
Pygophilia	Buttocks fetish	Buttocks
Nasophilia	Nose fetish	Nose
Trichophilia	Hair fetish	Hair
Alvinophilia	Navel/Belly button fetish	Navel
Alvinolagnia	Belly/Stomach fetish	Belly
Cheirophilia	Hand fetish	Hands
Crurophilia	Leg fetish	Legs

While medical definitions restrict the term *sexual fetishism* to objects or body parts, *fetish* can, in common discourse, also refer to sexual interest in specific activities. This broader usage of *fetish* covers parts or features of the body (including obesity and body modifications), objects, situations and activities (such as **BDSM**—a variety of often erotic practices or roleplaying involving bondage, discipline, dominance and submission, sadomasochism, and other related interpersonal dynamics). Paraphilias such as urophilia, necrophilia, and coprophilia have been described as fetishes.

Eroticasphyxiation is the use of choking to increase the pleasure in sex. The fetish also includes an individualized part that involves choking oneself during the act of masturbation, which is known as auto-erotic asphyxiation. This usually involves a person being connected and strangled by a homemade device that is tight enough to give them pleasure but not tight enough to suffocate them to death. This is dangerous due to the issue of hyperactive pleasure seeking that can result in strangulation when there is no one to help if the device gets too tight and strangles the user.

Transvestic Disorder

Transvestism is the practice of cross-dressing**,** which is the act of wearing items of clothing and other accoutrements commonly associated with the opposite sex within a particular society. The term *cross-dressing* refers to an action or a behavior, without attributing or implying any specific causes or motives for that behavior. Cross-dressing is not synonymous with being transgender. A transvestic disorder is characterized by intense sexual arousal from cross-dressing that has occurred for at least six months, which also causes significant distress or impairment. It should be specified if it occurs with fetishism, meaning arousal by materials related to cross-dressing, or with autogynephilia, which includes arousal by thoughts of oneself as a female.

A transvestic fetishist is a person who cross-dresses as part of a sexual fetish. According to the DSM-4, this fetishism was limited to heterosexual men; however, DSM-5 does not have this restriction, and opens it to women and men, regardless of their sexual orientation.

Transvestic fetishism, *fetishistic transvestism*, and sometimes *transvestism* are also often used to describe *any* sexual behavior or arousal that is in any way triggered by the clothes of the other gender. Especially the latter is problematic because transvestism and cross-dressing are neither a sexual fetish nor do they necessarily have anything to do with sexual behavior or arousal.

Also, not every sexual behavior where clothes of the opposite gender are involved is *transvestic fetishism*; they are also often used in sexual roleplay without being a fetish. Also, many transgendered people, mostly transwomen, also cross-dress before coming out in sexual contexts to relieve their cross-gender feelings. This behavior is likewise not considered *transvestic fetishism*, as it is not cross-dressing for sexual pleasure, rather it is simply their self-gender expression.

Some male transvestic fetishists collect women's clothing, e.g., nightgowns, babydolls, slips, other types of nightwear, lingerie stockings, and pantyhose, items of a distinct feminine look and feel. They may dress in these feminine garments and take photographs of themselves while living out their secret fantasies. Many men love the feeling of wearing silk or nylon and adore the silky fabric of women's nightwear, lingerie, and nylons.

Most transvestic fetishists are said to be heterosexual men, although there are no studies that accurately represent either their sexual orientation or gender, and most information on this is based on anecdotal evidence or informal surveys. A small number of people with transvestic fetishism, as the years pass, want to dress and live permanently as women, and desire surgical or hormonal sex reassignment. In such cases the diagnosis should be changed to transsexualism (or gender dysphoria).

Exhibitionist Disorder



Figure **10.11.3**: Exposing oneself to only an intimate partner may not be considered exhibitionism.

Exhibitionism is the act of exposing in a public or semi-public context those parts of one's body that are not normally exposed—for example, the breasts, genitals, or buttocks. The practice may arise from a desire or compulsion to expose themselves in such a manner to groups of friends or acquaintances, or to strangers for their amusement or sexual satisfaction or to shock the bystander. Exposing oneself only to an intimate partner is normally not regarded as exhibitionism. In law, the act of exhibitionism may be called indecent exposure, "exposing one's person," or other expressions.

When exhibitionistic sexual interest is acted on with a non-consenting person or interferes with a person's quality of life or normal functioning, it can be diagnosed as **exhibitionistic disorder** in the DSM-5. The DSM states that the highest possible prevalence for exhibitionistic disorder in men is 2%-4%. It is thought to be much less common in women.

Exhibitionists in some cases masturbate while exposing themselves (or while fantasizing that he/she is exposing himself/herself) to another person. There is a pattern in which males exhibit themselves and there are three characteristic features of the exhibition: 1) It is performed for unknown women. 2) It takes place where sexual intercourse is impossible (e.g., a crowded shopping center). 3) It seems designed to surprise and shock the woman. The male exhibitionist usually exposes his erect penis, but it is not necessarily essential for the activity. Ejaculation may occur at the moment of exposure or develop later with masturbatory stimulation. Some exhibitionists are aware of a conscious desire to shock or upset their target while others fantasize that the target will become sexually aroused by their display.

Child versus Adult Presentation

Generally, society accepts exhibitionism in children as a natural curiosity, not a disorder; however, if the behaviors continue, a paraphilia is probable. The disorder appears to develop before the age of 18 and rarely is found in people over the age of 50.

Gender and Cultural Differences in Presentation

Most reported cases of exhibitionism involve males. Some scientists argue that women who undress in front of windows (as to invite a person to watch), or who wear low cut outfits are exhibitionists in a sense. Exhibitionism generally appears in Western society and is believed to be almost absent in such countries as Japan, Burma, and India. Additionally, in American society it can be a crime when committed by a male, but when women expose themselves, excluding total nudity, they are often seen as victims of male voyeurism.

Epidemiology

Prevalence and incidence are not easily defined because people with this disorder usually do not seek treatment voluntarily. Exhibitionism is one of the three most common sexual offenses, the other two being voyeurism and pedophilia. It is rarely diagnosed in general mental health clinics, but most professionals believe that it is probably underdiagnosed and under-reported. Risk factors appear to be antisocial history, antisocial personality disorder, alcohol misuse, and pedophilic sexual preference.

Voyeuristic Disorder

Voyeurism is the sexual interest in or practice of spying on people engaged in intimate behaviors, such as undressing, sexual activity, or other actions usually considered to be of a private nature. The term comes from the French *voir* which means "to see." A male voyeur is commonly labelled as "Peeping Tom" or a "Jags," a term which originates from the Lady Godiva legend. However, that term is usually applied to a male who observes somebody secretly and, generally, not in a public space.

It is diagnosed by recurrent and intense sexual arousal stemming from observing an unsuspecting person who is naked, undressing, or engaging in sexual activity. It should cause significant distress and the person must be at least 18 years old.

Research found voyeurism to be the most common sexual law-breaking behavior in both clinical and general populations.[2] In the same study, it was found that 42% of college males who had never been convicted of a crime had watched others in sexual situations. An earlier study indicates that 54% of men have

voyeuristic fantasies, and that 42% have tried voyeurism. In a national study of Sweden, it was found that 7.7% of the population (both men and women) had engaged in voyeurism at some point.[3] It is also believed that voyeurism occurs up to 150 times more frequently than police reports indicate. This same study also indicates that there are high levels of co-occurrence between voyeurism and exhibitionism, finding that 63% of voyeurs also report exhibitionist behavior.

Frotteuristic Disorder

Frotteurism is a paraphilic interest in rubbing, usually one's pelvic area or erect penis, against a non-consenting person for sexual pleasure. It may involve touching any part of the body, including the genital area. **Frotteuristic disorder** is a sexual dysfunction disorder characterized by sexual arousal from rubbing against or touching a non-consenting person.

The DSM-5 lists the following diagnostic criteria for frotteuristic disorder:

- Over a period of at least six months, recurrent and intense sexual arousal from touching or rubbing against a nonconsenting person, as manifested by fantasies, urges, or behaviors.
- The individual has acted on these sexual urges with a nonconsenting person, or the sexual urges or fantasies cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

If the individual has not acted on their interest and experiences no distress or impairment, they are considered to have a frotteuristic sexual interest, but not frotteuristic disorder. Some sexologists distinguish between frotteurism (as pelvic rubbing) and toucherism (as groping with hands), but the DSM does not.

The prevalence of frotteurism is unknown. The DSM estimates that 10–14% of men seen in clinical settings for paraphilias or hypersexuality have frotteuristic disorder, indicating that the population prevalence is lower. However, frotteuristic acts, as opposed to frotteuristic disorder, may occur in up to 30% of men in the general population. The majority of frotteurs are male and the majority of victims are female, although female-on-male, female-on-female, and male-on-male frotteurs exist. This activity is often done in circumstances where the victim cannot easily respond, in a public place such as a crowded train or concert.

Usually, such nonconsensual sexual contact is viewed as a criminal offense: a form of sexual assault albeit often classified as a misdemeanor with minor legal penalties. Conviction may result in a sentence or psychiatric treatment.

Sexual Masochism and Sexual Sadism Disorders

Sexual masochism disorder (SMD) is the condition of experiencing recurring and intense sexual arousal in response to enduring moderate or extreme pain, suffering, or humiliation. Conversely, **sexual sadism disorder** is the condition of experiencing sexual arousal in response to the extreme pain, suffering, or humiliation of others. The words *sadism* and *sadist* are derived from Marquis de Sade. Many of Marquis de Sade's books, including *Justine* (1791), *Juliette* (1797) and *The 120 Days of Sodom* (published posthumously in 1905), are written from a cruelly sadistic viewpoint.

BDSM (bondage, domination, sadomasochism) is a colloquial term relating to individuals who willingly engage in consenting forms of pain or humiliation, typically for sexual purposes. The term BDSM describes the activities between consenting partners that contain sadistic and masochistic elements. Many behaviors such as erotic spanking, tickling, and love-bites that many people think of only as "rough" sex also contain elements of sado-masochism. BDSM is not currently a diagnosable condition in either the DSM or ICD system.

Sexual Masochism

The formal diagnosis of sexual masochism disorder according to the DSM-5 involves "recurrent and intense sexual arousal from the act of being humiliated, beaten, bound, or otherwise made to suffer, as manifested by fantasies, urges, or behaviors." It applies only if the individual experiences clinically significant distress or impairment in social, occupational, or other important areas of functioning.

The prevalence of sexual masochism disorder in the population is unknown, but the DSM-5 suggests that 2.2% of males and 1.3% of females may be involved in BDSM, whether they have sexual masochism disorder or not. Extensive use of pornography depicting humiliation is sometimes associated with sexual masochism disorder.

Behaviors associated with sexual masochism disorder can be acted out alone (e.g., binding, self-sticking pins, self-administration of electric shock, or self-mutilation) or with a partner (e.g., physical restraint, blindfolding, paddling, spanking, whipping, beating, electric shock, cutting, pinning and piercing, and humiliation such as by being urinated or defecated upon, being forced to crawl and bark like a dog, or being subjected to verbal abuse). Behaviors sometimes include being forced to cross-dress or being treated like an infant.

Following a phenomenological study of individuals involved in sexual masochistic sessions, sexual masochism was described as an addiction-like tendency, with several features resembling that of drug addiction: craving, intoxication, tolerance, and withdrawal. It was also demonstrated how the first masochistic experience is placed on a pedestal, with subsequent use aiming at retrieving this lost sensation, much as described in the descriptive literature on addiction. The addictive pattern presented in this study suggests an association with behavioral spin as found in problem gamblers. A behavioral spin is described as a process one goes through that is characterized by a behavior growing in frequency and magnitude. As it develops, it gains its own momentum which the individual finds almost impossible to terminate even when faced with known unwanted outcomes. For the participants in sexual masochistic sessions, the behavioral spin is manifested as a continuously reinforced process leading the masochist to engage in masochistic activity, perpetuating itself, and binding the addict to his/her behavior.

Sexual Sadism Disorder

Sexual sadism disorder refers to the "recurrent and intense sexual arousal from the physical or psychological suffering of another person, as manifested by fantasies, urges, or behaviors." The formal diagnosis of sexual sadism disorder would apply if the individual has acted on these urges with a nonconsenting person or if the urges cause significant distress to the individual.

Paraphilic coercive disorder refers to the preference for non-consenting over consenting sexual partners. It differs from sexual sadism disorder in that although the individual with this disorder may inflict pain or threats of pain in order to gain the compliance of the victim, the infliction of pain is not the actual goal of the

individual. The condition is typically described as a paraphilia and continues to undergo research, but does not appear in the current DSM or ICD. Alternate terms for the condition have included Biastophilia, Coercive Paraphilic Disorder, and Preferential Rape.

With paraphilic coercive disorder, the individual employs enough force to subdue a victim, but with sexual sadism disorder, the individual often continues to inflict harm regardless of the compliance of the victim, which sometimes escalates not only to the death of the victim, but also to the mutilation of the body. What is experienced by the sadist as sexual does not always appear obviously sexual to non-sadists: sadistic rapes do not necessarily include penile penetration of the victim. In a survey of offenses, 77% of cases included sexual bondage, 73% included anal rape, 60% included blunt force trauma, 57% included vaginal rape, and 40% included penetration of the victim by a foreign object. In 40% of cases, the offender kept a personal item of the victim as a souvenir.

On personality testing, sadistic rapists apprehended by law enforcement have shown elevated traits of impulsivity, hypersexuality, callousness, and psychopathy. Although there appears to be a continuum of severity from mild (*hyperdominance* or *BDSM*) to moderate (*paraphilic coercive disorder*) to severe (*sexual sadism disorder*), it is not clear if they are genuinely related or only appear related superficially.

Very little is known about how sexual sadism disorder develops. Most of the people diagnosed with sexual sadism disorder come to the attention of authorities by committing sexually motivated crimes. Surveys have also been conducted to include people who are interested in only mild and consensual forms of sexual pain/humiliation (BDSM).

Most of the people with full-blown sexual sadism disorder are male, whereas the sex ratio of people interested in BDSM is closer to 2:1 male-to-female. People with sexual sadism disorder are at an elevated likelihood of having other paraphilic sexual interests.

Watch It

Watch this video to review the main components of each of the paraphilic disorders in the DSM-5.



You can view the transcript for "What are Paraphilic Disorders?" here (opens in new window).

Key Takeaways: Paraphilic Disorders

Table 1. Paraphilic Disorders as Described in the DSM-5

Disorder	Description	Prevalence
Voyeuristic Disorder	Recurrent and intense sexual arousal from watching another person who is naked, undressing, or engaged in sexual activities.	Lifetime prevalence is estimated at the high end to be up to 12% in males and 4% in females.
Exhibitionistic Disorder	Recurrent and intense sexual arousal from showing genitals to an unsuspecting person.	Highest prevalence predicted to be 2%-4% of males, and unknown for females.
Frotteuristic Disorder	Recurrent and intense sexual arousal from touching or rubbing against a nonconsenting person.	Unknown
Sexual Masochism Disorder	Recurrent and intense sexual arousal from being bound, beaten, or made to suffer in some way.	Unknown; Australia estimates show that 2.2% of males and 1.3% of females were involved in sadomasochistic behaviors in the past 12 months.
Sexual Sadism Disorder	Recurrent and intense sexual arousal from the physical or psychological suffering of another person.	Unknown, estimates vary between 2% and 20%. Of sex offenders in the United States, 10% have sexual sadism.
Pedophilic Disorder	Recurrent and intense sexual arousal and fantasies related to sexual activity with a prepubescent child or children under age 13.	Unknown; highest prevalence approximately 3%-5%.
Fetishistic Disorder	Recurrent and intense sexual arousal from either nonliving objects, or on nongenital body parts.	Unknown
Transvestic Disorder	Recurrent and intense sexual arousal from cross-dressing.	Unknown; fewer than 3% of men report ever being aroused by cross-dressing.

Glossary

BDSM (bondage, domination, sadomasochism): a colloquial term relating to individuals who willingly engage in consenting forms of pain or humiliation, typically for sexual purposes

exhibitionism: the exposure of an individual's genitalia to unsuspecting strangers for sexual satisfaction

frotteurism: the touching of or rubbing against a nonconsenting person

paraphilias: persistent and recurrent sexual interests, urges, fantasies, or behaviors of marked intensity involving objects, activities, or even situations that are atypical in nature

paraphilic coercive disorder: the preference for non-consenting over consenting sexual partners

Partialism: sexual interest with an exclusive focus on a specific part of the body other than the genitals

pedophilia: any sexual activity with a prepubescent child, where the offender/patient is at least sixteen years of age, and the victim is at least five years younger

sexual fetishism or **erotic fetishism:** sexual fixation on a nonliving object or nongenital body part

sexual masochism: the derivation of sexual arousal from being the recipient of physical or mental abuse and/or humiliation

sexual sadism: when sexual arousal is gained from inflicting mental or physical suffering on a nonconsenting person

toucherism: sexual arousal based on grabbing or rubbing one's hands against an unexpecting (and non-consenting) person

transvestic fetishism: the derivation of sexual arousal from cross-dressing or dressing in clothes of the opposite sex

voyeurism: the viewing of an unsuspecting person engaging in disrobing or sexual activity

1. "Paraphilic Disorders". *Diagnostic and Statistical Manual of Mental Disorders (Fifth ed.)*. Philadelphia, Pennsylvania: American Psychiatric Publishing. 2013. pp. 685–686. ↵
2. "The DSM Diagnostic Criteria for Exhibitionism, Voyeurism, and Frotteurism" (PDF). Niklas Langstrom. Retrieved 2013-04-04. ↵
3. Långström, Niklas; Seto, Michael C. (2006). "Exhibitionistic and Voyeuristic Behavior in a Swedish National Population Survey". Archives of Sexual Behavior. 35 (4): 427–35. doi:10.1007/s10508-006-9042-6. PMID 16900414 ↵

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10.12: Pedophilic Disorder

Learning Objectives

- Examine the characteristics, etiology, and treatments for pedophilia
- Explain controversies and complications surrounding the diagnosis of paraphilic disorder

Pedophilia is termed **pedophilic disorder** in the DSM-5, and the manual defines it as a paraphilia involving intense and recurrent sexual urges towards and fantasies about prepubescent children that have either been acted upon or that cause the person with the attraction distress or interpersonal difficulty.

The word *pedophilia* is derived from the Greek words “paidos” (child) and “philia” (love). An estimated 20% of American children have been sexually molested, making pedophilia a common paraphilia. Offenders are usually family friends or relatives. Types of activities vary and may include just looking at a child or undressing and touching a child. However, acts often involve oral sex or touching of genitals of the child or the offender. Studies suggest that children who feel uncared for or lonely may be at higher risk for sexual abuse.

The key feature of this disorder is that the individual experiences sexual arousal when with children that may be equal to, if not greater than, that which they experience with individuals who are physically mature. In other words, an adult or older adolescent experiences a primary or exclusive sexual attraction to prepubescent children. Although girls typically begin the process of puberty at age 10 or 11 and boys at age 11 or 12, criteria for pedophilia extend the cut-off point for prepubescence to age 13. A person must be at least 16 years old, and at least five years older than the prepubescent child, for the attraction to be diagnosed as pedophilia. If an individual is 16 years old and exhibits these behaviors with someone that is at least 5 years younger, he or she would be considered for this disorder.

Another feature involves recurring sexual dreams, behaviors, or urges concerning children that are 13 years old or younger. These urges may be directed toward children of the same sex as the pedophiles, or the other sex. Some pedophiles are attracted to both boys and girls. Some are attracted to only children while others are attracted to children as well as adults. These issues must be persistent for at least six months and must cause impairment to everyday functioning to be considered symptoms.

Pedophilia emerges before or during puberty and is stable over time, although some individuals with pedophilia report that they did not become sexually aroused by children until middle adulthood. It is said to be self-discovered, not chosen.[1] For these reasons, pedophilia has been described as a disorder of sexual preference, phenomenologically similar to a heterosexual or homosexual orientation. This description led to some misunderstandings about wording in the DSM-5, prompting the APA to comment that “APA considers pedophilic disorder a ‘paraphilia,’ not a ‘sexual orientation.’” They said they strongly support efforts to criminally prosecute those who sexually abuse and exploit children and adolescents, and “also support continued efforts to develop treatments for those with pedophilic disorder with the goal of preventing future acts of abuse.”[2]

Pedophiles may limit their activity to exposing themselves to a child (sometimes known as flashing), touching and fondling the child gently, undressing the child and looking at them, or masturbating in front of the child. Consumption of child pornography appears to be a more reliable indicator of pedophilia than molesting a child, although some non-pedophiles also view child pornography. Child pornography may be used for a variety of purposes, ranging from private sexual gratification or trading with other collectors, to preparing children for sexual abuse as part of the child grooming process.

Pedophilic viewers of child pornography are often obsessive about collecting, organizing, categorizing, and labeling their child pornography collection according to age, gender, sex act, and fantasy. When it comes to child sexual exploitation material (CSEM), those whose usage appears to be confined to engagement with the images themselves (e.g., as a facilitator of sexual fantasy or collecting behavior) are labeled as *contact-driven*, and those whose CSEM usage is functionally and directly related to contact sex offending (e.g., as a behavioral substitute, facilitatory factor, or product of a contact offense) are labeled as *fantasy-driven***,** utilizing terminology introduced by Merdian, Curtis, Thakker, Wilson, and Boer (2013)[3].



Figure **10.12.1**: The internet has made it easier for predators to gain access to child pornography or solicit contact with minors.

Researchers Taylor and Quayle reported that pedophilic collectors of child pornography are often involved in anonymous internet communities dedicated to extending their collections. Children are particularly vulnerable to sexual predators, such as pedophiles, on the internet. Forensic science research reveals two models of pedophilia on the internet: a *trust-based seductive model*, in which a pedophile will use the internet to build a relationship of trust with a child, with the end goal of an in-person meeting, and a *direct sexual model*, which is generally a shorter relationship in which the pedophile will bring up sexual activities and solicit images with the minor [4]

Neither the DSM nor the ICD-11 diagnostic criteria require actual sexual activity with a prepubescent youth. The diagnosis can therefore be made based on the presence of fantasies or sexual urges even if they have never been acted upon. On the other hand, a person who acts upon these urges yet experiences no distress about their fantasies or urges can also qualify for the diagnosis. *Acting* on sexual urges is not limited to overt sex acts for purposes of this diagnosis.

Gender and Cultural Differences in Presentation

Until recently, pedophilic individuals had found it relatively easy to gain access to unattended children. Awareness of pedophilia has been raised in the past two decades, and it has become more difficult for these individuals to find children with whom to act out their fantasies. In response to the scarcity of vulnerable children, many pedophiles have turned to chatrooms and child pornography.

Males are more often diagnosed with this disorder than women. Pedophilia is more prevalent among Caucasians than among other ethnicities. It is also known that if a male prefers males, it is more likely that he will repeat his pedophilic actions. This has led certain religious or otherwise radical activists to suggest that pedophilia and homosexuality are “one and the same,” resulting in further media attention to an already well-covered topic.

? Cultural Practices and Pedophilia

One of the biggest issues in assessing behavior as pedophilic or normal is the criteria for pedophilia. E. E. Evans- Pritchard recorded that male Azande warriors (in the northern Congo) routinely took on boy-wives between the ages of twelve and twenty, who helped with household tasks and participated in intercrural (non-penetrative) sex with their older husbands. The practice had died out by the early 20th century, after Europeans had gained control of African countries, but was recounted to Evans-Pritchard by the elders he spoke to.



Figure **10.12.2**: Ethiopia is one of the countries where child marriage is more prevalent.

By Western standards, certain cultures would have a higher prevalence of pedophilia because these cultures allow child weddings, or unions between mature males and prepubescent females. Clearly, it is important to note any religious or cultural

backgrounds in individuals being examined as having pedophilia. This is a very difficult situation, as some groups have voiced the concern that any pedophile can simply convert to a belief system that accommodates and excuses his behaviors. UNICEF has stated that child marriage "represents perhaps the most prevalent form of sexual abuse and exploitation of girls." The effects of child sexual abuse can include depression, post-traumatic stress disorder, anxiety, a propensity to further victimization in adulthood, and physical injury to the child, among other problems. Sexual abuse by a family member is a form of incest and can result in more serious and long-term psychological trauma, especially in the case of parental incest.

Pedophilia and the Law

As we discuss further in this section, under the law, *child sexual abuse* is often used as an umbrella term describing criminal and civil offenses in which an adult engages in sexual activity with a minor or exploits a minor for the purpose of sexual gratification. The American Psychological Association states that "children cannot consent to sexual activity with adults," and condemns any such action by an adult: "an adult who engages in sexual activity with a child is performing a criminal and immoral act which never can be considered normal or socially acceptable behavior." [5]

Several terms have been used to distinguish "true pedophiles" from non-pedophilic and non-exclusive offenders, or to distinguish among types of offenders on a continuum according to strength and exclusivity of pedophilic interest, and motivation for the offense. Exclusive pedophiles are sometimes referred to as **true pedophiles**. They are sexually attracted to prepubescent children, and only prepubescent children. Showing no erotic interest in adults, they can only become sexually aroused while fantasizing about or being in the presence of prepubescent children, or both. Non-exclusive offenders—or "non-exclusive pedophiles"—may at times be referred to as **non-pedophilic offenders**, but the two terms are not always synonymous. Non-exclusive offenders are sexually attracted to both children and adults, and can be sexually aroused by both, though a sexual preference for one over the other in this case may also exist. If the attraction is a sexual preference for prepubescent children, such offenders are considered pedophiles in the same vein as exclusive offenders.

Epidemiology

The most commonly asked question about pedophilia is how frequently it occurs. Obtaining reliable incidence numbers of pedophilia as a preference disorder is difficult as individuals are typically unwilling to admit pedophilic preferences, particularly when offenses have been committed. The prevalence of pedophilia in the general population is not known. The prevalence of a true pedophilic sexual preference is approximately 1%, but when general fantasies are investigated, that prevalence can reach up to 5% among men in the general population.

Some studies suggest that the prevalence of pedophilia may be between 3%-5% in the general population (as reviewed by Seto, 2009). In penile plethysmography, studies of men with sexual offense histories against children, these prevalences can jump from 30% for men with one offense to 61% for men with three or more sexual offenses against children (Blanchard, 2010; Seto, 2009). The most well-known objective method of measuring pedophilic interest is **penile plethysmography (PPG)** or **phallometry**. This method measures genital sexual arousal through sexual stimuli and is based on the relative change in penile response. Sexual preference can be determined as the relative change in penile response to various classes of sexual stimuli (according to Tanner scales), such as prepubescent, pubescent, or adult female or male targets. [6]

Less is known about the prevalence of pedophilia in women, but there are case reports of women with strong sexual fantasies and urges towards children. Frequently, the abuse against a child is carried out in collaboration with a male partner or victims are seen as surrogates to replace less than desirable relationships. The **2009 Plymouth child abuse case** was a child abuse and pedophile ring involving at least five adults from different parts of England who all met on Facebook. The case centered on photographs taken of up to 64 children aged two to five years old by Vanessa George, a nursery (pre-school) worker in Plymouth. It highlighted the issue of child molestation by women, as all but one of the members of the ring were female. The case prompted an increased recognition of the problem of female pedophiles, sex offenders, and the scale of their offending, with one estimate suggesting that at least 10% of sex offenders are female.

Currently, there is no reliable estimate of pedophilia in women and the question remains whether pedophilia, as currently defined, even exists in women[7].The true number of female child molesters may be underrepresented by available estimates, for reasons including a "societal tendency to dismiss the negative impact of sexual relationships between young boys and adult women, as well

as women's greater access to very young children who cannot report their abuse," among other explanations (e.g., Vanessa George who worked with children under the age of 5).

The reason that there is very little known about the prevalence of pedophilia at this time may be because of the severely negative stigma associated with having pedophilia, and therefore, many people with pedophilia only rarely seek help from a mental health professional. Also, abnormal psychology has studied egosyntonic and egodystonic concepts in some detail. **Egosyntonic** refers to the behaviors, values, and feelings that are in harmony with or acceptable to the needs and goals of the ego, or consistent with one's ideal self-image. **Egodystonic** (or *ego alien*) is the opposite, referring to thoughts and behaviors (dreams, compulsions, desires, etc.) that are in conflict, or dissonant, with the needs and goals of the ego, or, further, in conflict with a person's ideal self-image. Many mental disorders, including pedophilic disorder, are egosyntonic, which makes their treatment difficult as the patients may not perceive anything wrong and view their perceptions and behavior as reasonable and appropriate. This corresponds to the general concept in psychiatry of poor insight.

Ego-dystonic sexual orientation includes people who acknowledge that they have a sexual preference for prepubertal children, but wish to change it due to the associated psychological or behavioral problems (or both).

Etiology

Some researchers feel that it is due to biological factors, that one of the male sex hormones predisposes men to be more sexually deviant; however, according to a 2002 study there is no evidence of any link between genetics and pedophilia.

Although what causes pedophilia is not yet known, researchers began reporting a series of findings linking pedophilia with brain structure and function, beginning in 2002. Testing individuals from a variety of referral sources inside and outside the criminal justice system as well as controls, these studies found associations between pedophilia and lower IQs, poorer scores on memory tests, greater rates of non-right-handedness, greater rates of school grade failure over and above the IQ differences, lesser physical height, greater probability of having suffered childhood head injuries resulting in unconsciousness, and several differences in MRI-detected brain structures.

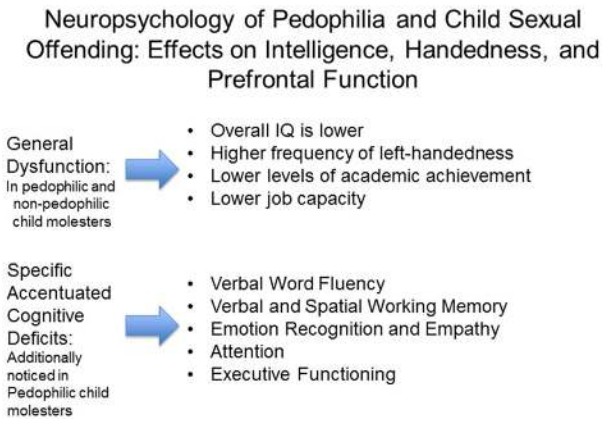


Figure 10.12.3: Overview of neuropsychological findings in pedophilia and child sexual offending.

Such studies suggest that there are one or more neurological characteristics present at birth that cause or increase the likelihood of being pedophilic. Some studies have found that pedophiles are less cognitively impaired than non-pedophilic child molesters. A 2011 study reported that pedophilic child molesters had deficits in response inhibition, but no deficits in memory or cognitive flexibility. Evidence of familial transmittability "suggests, but does not prove that genetic factors are responsible" for the development of pedophilia. A 2015 study indicated that pedophilic offenders have a normal IQ.

Another study, using structural MRI, indicated that male pedophiles have a lower volume of white matter than a control group. Functional magnetic resonance imaging (fMRI) has indicated that child molesters diagnosed with pedophilia have reduced activation of the hypothalamus as compared with non-pedophilic persons when viewing sexually arousing pictures of adults. A 2008 functional neuroimaging study notes that central processing of sexual stimuli in heterosexual "pedophile forensic inpatients" may be altered by a disturbance in the prefrontal networks, which "may be associated with stimulus-controlled behaviors, such as sexual compulsive behaviors." The findings may also suggest "a dysfunction at the cognitive stage of sexual arousal processing."

Blanchard, Cantor, and Robichaud (2006) reviewed the research that attempted to identify hormonal aspects of pedophiles. They concluded that there is some evidence that pedophilic men have less testosterone than controls, but that the research is of poor quality and that it is difficult to draw any firm conclusion from it.

While not causes of pedophilia themselves, childhood abuse by adults or comorbid psychiatric illnesses—such as personality disorders and substance abuse—are risk factors for acting on pedophilic urges. Blanchard, Cantor, and Robichaud addressed comorbid psychiatric illnesses; "The theoretical implications are not so clear. Do particular genes or noxious factors in the prenatal environment predispose a male to develop both affective disorders and pedophilia, or do the frustration, danger, and isolation engendered by unacceptable sexual desires—or their occasional furtive satisfaction—lead to anxiety and despair?" They indicated that, because they previously found mothers of pedophiles to be more likely to have undergone psychiatric treatment, the genetic possibility is more likely.

A study analyzing the sexual fantasies of 200 heterosexual men by using the Wilson Sex Fantasy Questionnaire exam determined that males with a pronounced degree of paraphilic interest (including pedophilia) had a greater number of older brothers, a high 2D:4D digit ratio (which would indicate low prenatal androgen exposure), and an elevated probability of being left-handed, suggesting that disturbed hemispheric brain lateralization may play a role in deviant attractions.

Other research suggests that pedophilia results from certain psychosocial factors (e.g., being sexually abused as a child, or the nature of one's familial interactions). Studies of pedophilia in child sex offenders often report that it co-occurs with other psychopathologies, such as low self-esteem, depression, anxiety, and personality problems. Still others invoke factors such as the following: anomalies in psychological development, the desire to overpower sexual partners, and the belief that sex is a necessary requisite for affection.[8]

Treatment

Current therapeutic approaches to treat pedophilia include psychotherapy as well as pharmacological treatment, among which includes androgen-lowering therapy. CBT aims to reduce attitudes, beliefs, and behaviors that may increase the likelihood of sexual offenses against children. Its content varies widely between therapists, but a typical program might involve training in self-control, social competence and empathy, and use cognitive restructuring to change views on sex with children. The most common form of this therapy is **relapse prevention**, where the patient is taught to identify and respond to potentially risky situations based on principles used for treating addictions.

Antiandrogen drugs (ADT) are used to drastically decrease the testosterone concentration in patients. Antiandrogen drugs (ADT) are also known as **chemical castration** due to the fact that serum testosterone concentration can drop rapidly and even reach castration levels after several weeks. The aim of the treatment is a reduction in sexual drive and, in consequence, a reduced risk of recidivism in paraphilic patients and sexual offenders. Despite a large number of studies, the efficiency of ADT aiming at a reduction of the sex drive and a reduced risk of recidivism is still under debate[9]

However, chemical castration therapy reduces an individual's libido, which then makes some offenders more responsive to the introduction of psychotherapy. This combination therapy is most often utilized in those who are at a high risk of offending. Unlike **surgical castration**, where the gonads are removed through an incision in the body, chemical castration is not as radical and does not remove organs, nor is it a form of sterilization.

As of 2008, most doctors believe that pedophilia cannot be cured. The current approach to deal with pedophiles who have been convicted of sexual abuse is to try to change their behavior. It does not change their sexual attraction. Using this treatment, it is believed that some sex offenders can be prevented from offending again.

? Pedophilia and Child Sexual Abuse or Molestation

The ratio of sex offenders against female children and sex offenders against male children is about 2:1. The term *pedophile* is commonly used by the public to describe all child sexual abuse offenders. This usage is considered problematic by researchers because many child molesters do not have a strong sexual interest in prepubescent children and are consequently not pedophiles. *Pedophilia* is not a legal term and having a sexual attraction to children is not illegal. **Child sexual abuse**, also called **child molestation**, is a form of child abuse in which an adult or older adolescent uses a child for sexual stimulation. Forms of child sexual abuse include engaging in sexual activities with a child (whether by asking or pressuring, force, or by

other means), indecent exposure (of the genitals, female nipples, etc.), child grooming, and child sexual exploitation, including using a child to produce child pornography.

In the light of frightening and emotionally disturbing sexual offenses against children, experts have focused more on the level of sexual behavior, referred to subsequently as "offenses," while not differentiating the causes for that behavior in an appropriate and adequate way. Concerning sexual abuse against children, two groups can be distinguished: first, those who show no sexual preference disorder, but whom, for various reasons, sexually abuse children. Reasons include sexually inexperienced adolescents, people with developmental disabilities, and those with antisocial personality disorders (ASPDs), or perpetrators within general traumatizing family constellations, which seek surrogate partners in children (Rice and Harris, 2002; Greenberg et al., 2005). These individuals are most likely diagnosed with various impulse-control disorders, accounting for their engaging in child sexual abuse without a specific sexual preference for prepubescent children (Allnutt et al., 1996; Greenberg et al., 2005). Second, there are those who do display a sexual preference disorder, namely pedophilia (i.e., the sexual preference for prepubescent minors) and/or **hebephilia** (i.e., the sexual preference for pubescent minors) (Seto et al., 1999).[10]

Although this preference increases the risk of engaging in child sexual abuse, only about 50% of all individuals who do sexually abuse children are pedophilic (Blanchard et al., 2001; Schaefer et al., 2010) and not every pedophilic individual actually has abused children. The other 50% of individuals that have abused children are those who do so without a sexual attraction to children; i.e., they lack the necessary social skills to develop and maintain emotional and sexual relationships with appropriately aged peers and look to "replacement partners" in children as a kind of "surrogate" (Beier, 1998; Seto, 2008; Mokros et al., 2012b).[11]

There are motives for child sexual abuse that are unrelated to pedophilia, such as stress, marital problems, the unavailability of an adult partner, general anti-social tendencies, high sex drive, or alcohol use. As child sexual abuse is not automatically an indicator that its perpetrator is a pedophile, offenders can be separated into two types: pedophilic and non-pedophilic (or preferential and situational). In sex crimes, a situational sex offender is one whose offense is associated with situational sexual behavior, i.e., sexual behavior different from the person's usual habits. This term is in an opposition to the **preferential offender**, whose offense is associated with the person's preferential behavior (the case of pedophilia). For example, a preferential child molester is exclusively involved with children, whereas the situational ones are normally engaged in sexual behavior within their peer group.

Estimates for the rate of pedophilia in detected child molesters generally range between 25% and 50%. A 2006 study found that 35% of its sample of child molesters were pedophilic. Pedophilia appears to be less common in incest offenders, especially fathers and step-fathers. According to a U.S. study on 2,429 adult male sex offenders who were categorized as pedophiles, 7% identified themselves as exclusive, which indicates that many or most child sexual abusers may fall into the *non-exclusive category.*

Again, some pedophiles do not molest children. Little is known about this population because most studies of pedophilia use criminal or clinical samples, which may not be representative of pedophiles in general. Researcher Michael Seto (2009) suggests that pedophiles who commit child sexual abuse do so because of other anti-social traits in addition to their sexual attraction. He states that pedophiles who are "reflective, sensitive to the feelings of others, averse to risk, abstain from alcohol or drug use, and endorse attitudes and beliefs supportive of norms and the laws" may be unlikely to abuse children.

According to Abel, Mittleman, and Becker (1985) and Ward *et al.* (1995), there are generally large distinctions between the characteristics of pedophilic and non-pedophilic molesters. They state that non-pedophilic offenders tend to offend at times of stress; have a later onset of offending; and have fewer, often familial, victims. Pedophilic offenders often start offending at an early age, often have a larger number of victims who are frequently extrafamilial, are more inwardly driven to offend, and have values or beliefs that strongly support an offense lifestyle. One study found that pedophilic molesters had a median of 1.3 victims for those with girl victims and 4.4 for those with boy victims. Child molesters, pedophilic or not, employ a variety of methods to gain sexual access to children. Some groom their victims into compliance with attention and gifts while others use threats, alcohol or drugs, or physical force.

Controversies and Complications Surrounding the Diagnosis of Paraphilic Disorders

Many complications of paraphilia and paraphilic disorders can arise. Living with these intense and abnormal desires and behaviors can be troublesome and cause much turmoil to those individuals suffering from them. When the paraphilia becomes a paraphilic disorder, individuals suffer harm, distress, and functional impairment. Furthermore, legal ramifications often ensue, leading to a life of incarceration and/or probation, and forensic psychiatric hospital confinement as well as permanent induction onto the sexual offender registry. Dependent on jurisdiction, the requirements of being on the sex offender registry include address notification; making living location public knowledge; housing limitations; and restrictions on being in the presence of underage persons, living in proximity to a school or daycare center, internet use, or even owning toys or other items that may suggest involvement with children.

Overall, the majority of patients suffering from paraphilic disorders rarely seek treatment voluntarily, as many individuals feel indignity, culpability, or discomfiture while others focus on the difficulty and lack of desire to halt efforts of achieving intense sexual pleasure and ultimate satisfaction. Furthermore, the very nature of paraphilic disorders in many individuals leads to victimization and often legal repercussions. These consequences will only further deter patients from seeking treatment. Those patients actually involved in treatment plans are most often either mandated legally or convinced by family, friends, or sexual partners.

Despite the medications used in the management of paraphilic disorders, better preventative measures are deemed necessary to reduce victimization, as many of those with paraphilic disorders will only be under evaluation upon incarceration after the harm has been inflicted.

Childhood sexual abuse is considered to be a leading cause of pedophilia, among other paraphilias and resultant paraphilic disorders. Maybe preventative measures should start here. Many psychiatrists and mental healthcare providers feel that the only cure for pedophilic disorder is incarceration. As paraphilias and paraphilic disorders are deeply engrained thoughts and irresistible behaviors of the individual, whether viewed as right or wrong, it is currently deemed as merely impossible to fix. [footnote]Fisher KA, Marwaha R. Paraphilia. [Updated 2020 Mar 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/N...25/[/footnote]

Lastly, sexual preference itself cannot be considered a mental disorder similar to how a homosexual orientation was considered in the 1970s in the United States of America (Green, 2002). Separating sexual preference from psychosocial impairment, thus allowing for the practice of various sexual behaviors with consenting partners, has been applied within the new DSM-5 with the other paraphilias as well, including fetishism, bondage/dominance-sadism/masochism, and is therefore not specific to pedophilia (Wright, 2010, 2014).[12]

Watch It

This video explains some of the complications surrounding the diagnosis of pedophilic disorder.



You can view the transcript for “Should pedophilia be treated as mental illness?” here (opens in new window).

Glossary

chemical castration: castration via drugs to reduce libido and sexual activity

child sexual abuse (child molestation): a form of child abuse in which an adult or older adolescent uses a child for sexual stimulation

contact-driven: behavior directly related to contact sex offending (e.g., as a behavioral substitute, facilitatory factor, or product of a contact offense)

egosyntonic: refers to the behaviors, values, and feelings that are in harmony with or acceptable to the needs and goals of the ego, or consistent with one's ideal self-image

egodystonic: refers to thoughts and behaviors (dreams, compulsions, desires, etc.) that are in conflict, or dissonant, with the needs and goals of the ego, or, further, in conflict with a person's ideal self-image

ephebophilia: sexual interest in mid-to-late adolescents, generally ages 15 to 19

fantasy-driven: behavior confined to engagement with images (e.g., as a facilitator of sexual fantasy or collecting behavior).

hebephilia: sexual preference for pubescent minors

non-pedophilic offenders: non-exclusive offenders that are sexually attracted to both children and adults, and can be sexually aroused by both, though a sexual preference for one over the other in this case may also exist

penile plethysmography (PPG) (or phallometry): a method that measures genital sexual arousal through sexual stimuli and is based on the relative change in penile response

preferential offender: one whose offense is associated with the person's preferential behavior

relapse therapy: a technique where the patient is taught to identify and respond to potentially risky situations based on principles used for treating addictions

situational offender: sexual behavior is different from the person's usual habits

surgical castration: the gonads are removed through an incision in the body

true pedophiles: exclusive pedophiles that are sexually attracted to prepubescent children and only prepubescent children

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10.13: Sexual Violence, Crime, and Abuse

Learning Objectives

- Discuss problems and concerns related to the effects of rape and sexual abuse

As mentioned earlier, there is some controversy surrounding the diagnosis of a paraphilic disorder and what constitutes deviant sexual behavior. The definitions of these disorders can vary depending on time and culture, and the complication in diagnosing paraphilic disorders is compounded by the fact that several of the disorders could be associated with criminal behavior.[1] In this section, we will take a closer look at the criminal forms of sexual violence and abuse (that may or may not stem from a paraphilic disorder).

Sexual assault is an act in which a person intentionally sexually touches another person without that person's consent or coerces or physically forces a person to engage in a sexual act against their will. It is a form of sexual violence, which includes child sexual abuse, groping, **rape** (forced vaginal, anal, or oral penetration or a drug-facilitated sexual assault), or the torture of the person in a sexual manner.

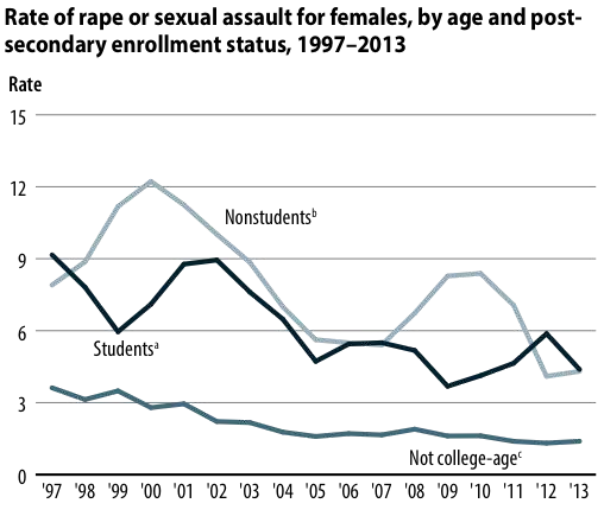


Figure 10.13.1: Reported rates of sexual assault in the United States (BJS)

Victims of Sexual Assault

Age

- 15% are under the age of 12
- 29% are age 12–17
- 44% are under age 18
- 80% are under age 30
- 12–34 are the highest risk years
- Girls ages 16–19 are four times more likely than the general population to be victims of rape, attempted rape, or sexual assault.

By Gender

A study from 1998 found that

- 88.7% of rape victims are women, the other 11.3% are men.
- 17.6% of women have been victims of attempted (2.8%) or completed (14.8%) rape during their lifetime.
- 3% of men have been victims of attempted or completed rape during their lifetime.
- 17.7 million women have been victims of attempted or completed rape during their lifetime.
- 2.78 million men have been victims of attempted or completed rape during their lifetime.

Largely because of child and prison rape, approximately 10% of reported rape victims are male.

The National Crime Victimization Survey conducted by the U.S. Justice Department (Bureau of Justice Statistics) found that from 1995 to 2013, men represented 17% of victims of sexual assault and rape on college campuses, and 4% of non-campus sexual assaults and rapes.

Child Sexual Abuse

Approximately 15–25% of women and five to 15% of men were sexually abused when they were children. The median age for sexual abuse is eight or nine years for both boys and girls (Finkelhor et. al. 1990). Most boys and girls are sexually abused by a male. **Childhood sexual abuse** is defined as any sexual contact between a child and an adult or a much older child. **Incest** refers to sexual contact between a child and family members. In each of these cases, the child is exploited by an older person without regard for the child's developmental immaturity and inability to understand the sexual behavior (Steele, 1986).

Sexual abuse can create feelings of self-blame, betrayal, and feelings of shame and guilt (Valente, 2005). The effects of child sexual abuse include depression, post-traumatic stress disorder, anxiety, propensity to re-victimization in adulthood, physical injury to the child, and increased risk for future interpersonal violence perpetration among males, among other problems. Additionally, victims of sexual abuse may suffer from problems with intimacy and suicide (Valente, 2005). Studies suggest that children who have been sexually abused have an increased risk of eating disorders and sleep disturbances.

Sexual abuse is particularly damaging when the perpetrator is someone the child trusts. As stated above, sexual abuse by a family member is a form of incest. It is more common than other forms of sexual assault on a child and can result in more serious and long-term psychological trauma, especially in the case of parental incest. Most sexual abuse offenders are acquainted with their victims. Approximately 30% of the perpetrators are relatives of the child—most often brothers, fathers, mothers, sisters, and uncles or cousins. Around 60% are other acquaintances such as friends of the family, babysitters, or neighbors. Strangers are the offenders in approximately 10% of child sexual abuse cases. Many states have criminalized sexual contact between teachers or school administrators and students, even if the student is over the age of consent.

Often, sexual assault on a child is not reported by the child for several of the following reasons:

- children are too young to recognize their victimization or put it into words
- they were threatened or bribed by the abuser
- they feel confused by fearing the abuser
- they are afraid no one will believe them
- they blame themselves or believe the abuse is a punishment
- they feel guilty for consequences to the perpetrator

Although rates of sexual abuse are higher for girls than for boys, boys may be less likely to report abuse because of the cultural expectation that boys should be able to take care of themselves and because of the stigma attached to homosexual encounters (Finkelhor et. al. 1990). Girls are more likely to be victims of incest and boys are more likely to be abused by someone outside the family.



Figure **10.13.2**: Abuse can lead to low self-esteem in girls.

Being sexually abused as a child can have a powerful impact on self-concept. The concept of **false self-training** (Davis, 1999) refers to holding a child to adult standards while denying the child's developmental needs. Sexual abuse is just one example of false self-training. Children are held to adult standards of desirableness and sexuality while their level of cognitive, psychological, and emotional immaturity is ignored. Consider how confusing it might be for a nine-year-old girl who has physically matured early to be thought of as a potential sex partner. Her cognitive, psychological, and emotional state do not equip her to make decisions about

sexuality or, perhaps, to know that she can say no to sexual advances. She may feel like a nine-year-old in all ways and be embarrassed and ashamed of her physical development. Girls who mature early have problems with low self-esteem because of the failure of others (family members, teachers, ministers, peers, advertisers, and others) to recognize and respect their developmental needs. Overall, youth are more likely to be victimized because they do not have control over their contact with offenders (parents, babysitters, etc.) and have no means of escape (Finkelhor and Dzuiba-Leatherman, in Davis, 1999).

Child grooming is befriending and establishing an emotional connection with a child, and sometimes the family, to lower the child's inhibitions with the objective of sexual abuse. To establish a good relationship with a child and the child's family, child groomers might do several things: They might try to gain the child's or parents' trust by befriending them, with the goal of easy access to the child. Sexual grooming of children also occurs on the internet. Some abusers will pose as children online and make arrangements to meet with them in person. Online grooming of children is most prevalent within the 13–17 age group (99% of cases), and particularly 13–14 (48%). The majority of targeted children are girls, and most victimization occurs with mobile-phone support. Children and teenagers with behavioral issues such as "high attention seeking" have a much higher risk than others. Contact-driven pedophiles and predators use online grooming to carry out cybersex trafficking crimes. The internet provides adults and children with a medium for professional and personal communication throughout the world. As widespread communication in cyberspace grows exponentially, the potential for internet-related crimes, such as cyberstalking, has accelerated. Children are particularly vulnerable to sexual predators, such as pedophiles, on the internet.

? Sexual Abuse in the Church

Catholic Church sexual abuse cases are cases of child sexual abuse by Catholic priests, nuns, and members of religious orders. In the 20th and 21st centuries, the cases have involved many allegations, investigations, trials, convictions, and revelations about decades of attempts by Church officials to cover up reported incidents. The abused include mostly boys but also girls, some as young as three years old, with the majority between the ages of 11 and 14. Many of these cases allege decades of abuse, frequently made by adults or older youths years after the abuse occurred. Cases have also been brought against members of the Catholic hierarchy who covered up sex abuse allegations and moved abusive priests to other parishes, where abuse continued. By the 1990s, the cases began to receive significant media and public attention in countries including Canada, the United States, Chile, Australia, Ireland, and much of Europe. From 2001 to 2010, the Holy See examined sex abuse cases involving about 3,000 priests, some of which dated back fifty years. Members of the Church's hierarchy have argued that media coverage was excessive and disproportionate and that such abuse also takes place in other religions and institutions, a stance that dismayed critics who saw it as a device to avoid resolving the abuse problem within the Church. Hofstra University researcher Charol Shakeshaft, the author of a report on sexual offenses in schools, said sexual violence is much more prevalent in schools than in the Church. According to the report, up to 422,000 students from California will be victims of sexual violence in the future.

In *Sexual Addiction andCompulsivity*: *The Journal of Treatment and Prevention*, Cimbolic & Cartor (2006) noted that because of the large share of post-pubescent male minors among cleric victims, there is need to further study the differential variables related to **ephebophilia** (sexual interest in mid-to-late adolescents, generally ages 15 to 19) versus pedophilia (sexual interest in prepubescent children, generally those 13 years of age or younger) offenders. Cartor, Cimbolic & Tallon (2008) found that 6% of the cleric offenders in the *John Jay Report* are pedophiles, 32% ephebophiles, 15% attracted to 11- and 12-year-olds only (both male and female), 20% indiscriminate, and 27% mildly indiscriminate.

In the United States, following ***Kansas v. Hendricks,*** sex offenders who have certain mental disorders, including pedophilia, can be subject to indefinite civil commitment under various state laws (generically called SVP laws or Sexually Violent Predator laws) and the federal Adam Walsh Child Protection and Safety Act of 2006. Similar legislation exists in Canada. Generally speaking, ***SVP laws*** have three elements: (1) that the person has been convicted of a sexually violent offense; (2) that the person suffers from a mental abnormality and/or personality disorder, which causes them serious difficulty controlling their sexually violent behavior; and (3) that this mental abnormality and/or personality disorder makes the person likely to engage in predatory acts of sexual violence if not confined in a secure facility.

The **Adam Walsh Child Protection and Safety Act** is a federal statute that was signed into law by U.S. President George W. Bush on July 27, 2006. The Walsh Act organizes sex offenders into three tiers according to the crime committed, and mandates that Tier

3 offenders (the most serious tier) update their whereabouts every three months with lifetime registration requirements. Tier 2 offenders must update their whereabouts every six months with 25 years of registration, and Tier 1 offenders must update their whereabouts every year with 15 years of registration. Failure to register and update information is a felony under the law. States are required to publicly disclose information of Tier 2 and Tier 3 offenders, at minimum. As stated above, it also contains civil commitment provisions for sexually dangerous people.

The Act also creates a **national sex offender registry** and instructs each state and territory to apply identical criteria for posting offender data on the internet (i.e., offender's name, address, date of birth, place of employment, photograph, etc.). The Act was named after Adam Walsh, an American boy who was abducted from a Florida shopping mall and later found murdered.

In *Kansas v. Hendricks*, the U.S. Supreme Court upheld as constitutional a Kansas law, the **Sexually Violent Predator Act,** under which Hendricks, a pedophile, was found to have a "mental abnormality" defined as a "congenital or acquired condition affecting the emotional or volitional capacity that predisposes the person to commit sexually violent offenses to the degree that such person is a menace to the health and safety of others," which allowed the State to confine Hendricks indefinitely irrespective of whether the State provided any treatment to him. In *United States v. Comstock*, this type of indefinite confinement was upheld for someone previously convicted on child pornography charges; this time a federal law was involved—the Adam Walsh Child Protection and Safety Act. The Walsh Act does not require a conviction on a sex offense charge, but only that the person be a federal prisoner, and one who "has engaged or attempted to engage in sexually violent conduct or child molestation and who is sexually dangerous to others," and who "would have serious difficulty in refraining from sexually violent conduct or child molestation if released."

In the United States, offenders with pedophilia are more likely to be recommended for civil commitment than non-pedophilic offenders. About half of committed offenders have a diagnosis of pedophilia. Psychiatrist Michael First (2008), in "*Use of DSM Paraphilia Diagnoses in Sexually Violent Predator Commitment Cases*" writes that, since not all people with a paraphilia have difficulty controlling their behavior, the evaluating clinician must present additional evidence of volitional impairment instead of recommending commitment based on pedophilia alone.

Deviant Sexual Preferences

Research focusing on the deviant sexual preferences propensity model suggests that people who perpetrate acts of sexual violence are sexually aroused by non-consensual sexual interactions more than consensual sexual interactions. The research that seeks to support this model (penile plethysmography) has not been able to reliably find differences in the two different groups of males (those who have committed acts of sexual aggression and those who have not). Instead, the studies are providing more evidence for the cognitive, attitudinal, neuropsychological, and lifestyle differences that impact sexual arousal in certain situations, rather than a deviant sexual preference leading to a higher propensity for sexual violence.

The antisocial personality lens stems from a study done by Hall and Hirschman (1991) and emphasizes the subsequent consequences of experiencing adversities/abuses in childhood, which can lead to the development of antisocial personality traits in adulthood. The antisocial personality traits combine with contextual, emotional (rage/anger), cognitive (irrational thoughts that influence emotions), and physiological (deviant sexual arousal) factors that increase the probability of committing sexual violence.

Domestic Violence

Domestic violence is violence or other abuse by one person against another in a domestic setting, such as in marriage or cohabitation. It is strongly correlated with sexual assault. Not only can domestic abuse be emotional, physical, psychological, and financial, it can also be sexual. Some of the signs of sexual abuse are similar to those of domestic violence. Domestic sexual violence includes all forms of unwanted sexual activity. It is considered abuse even if the victim may have previously engaged in consensual sexual activities with the perpetrator. Men and women can both fall victim to this type of abuse. A 2006 WHO study on physical and sexual domestic violence against women conducted across ten countries, found that the prevalence of sexual domestic violence ranges on average between 10 and 50%. Domestic sexual violence is also considerably less common than other forms of domestic violence.

Rape

Outside of law, the term *rape* (sexual intercourse or other forms of sexual penetration carried out against a person without that person's consent) is often used interchangeably with sexual assault. Although closely related, the two terms are technically distinct

in most jurisdictions. Sexual assault typically includes rape and other forms of non-consensual sexual activity.

Abbey *et al.* state that female victims are much more likely to be assaulted by an acquaintance, such as a friend or co-worker, a dating partner, an ex-boyfriend, or a husband or other intimate partner than by a complete stranger. In a study of hospital emergency room treatments for rape, Kaufman *et al.* stated that the male victims as a group sustained more physical trauma and were more likely to have been a victim of multiple assaults from multiple assailants. It was also stated that male victims were more likely to have been held captive longer.

In the U.S., rape is a crime committed primarily against youth. A national telephone survey on violence against women conducted by the National Institute of Justice and the Centers for Disease Control and Prevention found that 18% of women surveyed had experienced a completed or attempted rape at some time in their lives. Of these, 22% were younger than 12 years and 32% were between 12 and 17 years old when they were first raped.

In the United States, several studies since 1987 have indicated that one in four college women have experienced rape or attempted rape at some point in their lifetime. These studies are based on anonymous surveys of college women, not reports to the police, and the results are disputed.

The removal of a condom during intercourse without the consent of the sex partner, known as **stealthing,** may be treated as a sexual assault or rape.



Figure **10.13.3**: For many victims, emotional trauma lasts for years after the assault takes place.

Emotional Effects

Aside from physical traumas, rape and other sexual assault often result in long-term emotional effects, particularly in child victims. These can include, but are not limited to denial, learned helplessness, genophobia, anger, self-blame, anxiety, shame, nightmares, fear, depression, flashbacks, guilt, rationalization, mood swings, numbness, promiscuity, loneliness, social anxiety, difficulty trusting oneself or others, and difficulty concentrating. Being the victim of sexual assault may lead to the development of post-traumatic stress disorder, addiction, major depressive disorder, or other psychopathologies. Family and friends experience emotional scarring including a strong desire for revenge, a desire to “fix” the problem and/or move on, and a rationalization that “it wasn’t that bad.”

Physical Effects

While sexual assault, including rape, can result in physical trauma, many people who experience sexual assault will not suffer any physical injury. Rape myths suggest that the stereotypical victim of sexual violence is a bruised and battered young woman. The central issue in many cases of rape or other sexual assault is whether or not both parties consented to the sexual activity or whether or not both parties had the capacity to do so. Thus, the physical force resulting in visible physical injury is not always seen. This stereotype can be damaging because people who have experienced sexual assault but have no physical trauma may be less inclined to report to the authorities or to seek health care. However, women who experienced rape or physical violence by a partner were more likely than people who had not experienced this violence to report frequent headaches, chronic pain, difficulty sleeping, activity limitation, poor physical health, and poor mental health.

Glossary

Adam Walsh Child Protection and Safety Act: a law that does not require a conviction on a sex offense charge, but only that the person be a federal prisoner, and one who “has engaged or attempted to engage in sexually violent conduct or child molestation and who is sexually dangerous to others,” and who “would have serious difficulty in refraining from sexually violent conduct or child molestation if released”

Catholic Church sexual abuse cases: cases of child sexual abuse by Catholic priests, nuns, and members of religious orders

child sexual abuse (child molestation): a form of child abuse in which an adult or older adolescent uses a child for sexual stimulation

child grooming: befriending and establishing an emotional connection with a child, and sometimes the family, to lower the child's inhibitions with the objective of sexual abuse

childhood sexual abuse: defined as any sexual contact between a child and an adult or a much older child

false self-training: refers to holding a child to adult standards while denying the child's developmental needs

incest: refers to sexual contact between a child and family members

rape: forced vaginal, anal, or oral penetration or a drug-facilitated sexual assault

sexual assault: an act in which a person intentionally sexually touches another person without that person's consent or coerces or physically forces a person to engage in a sexual act against their will

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1. McManus, M. A., Hargreaves, P., Rainbow, L., & Alison, L. J. (2013). Paraphilias: definition, diagnosis, and treatment. F1000prime reports, 5, 36. https://doi.org/10.12703/P5-36↵

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10.14: Psychological Perspectives and Treatments for Sexual Deviations

Learning Objectives

- Examine psychological perspectives and treatment methods for sexual deviations
- Describe and evaluate the conventional and controversial treatments provided for sex offenders and rapists

Psychodynamic Perspectives

Freud's psychoanalytic understanding of the paraphilias was the dominant psychological perspective throughout the 20th century. He believed that these disorders were perversions representing both biological and psychological factors in early development [1]. **Perversion** is a type of human behavior that deviates from that which is understood to be orthodox or normal. Although the term *perversion* can refer to a variety of forms of deviation, it is most often used to describe sexual behaviors that are considered particularly abnormal, repulsive, or obsessive. Perversion differs from deviant behavior, in that the latter covers areas of behavior (such as petty crime) for which *perversion* would be too strong a term. It is often considered derogatory, and, in psychological literature, the term *paraphilia* has been used as a replacement, though this term is controversial, and *deviation* is sometimes used in its place.

The majority of the psychological literature on paraphilic disorders focuses on pedophilic disorder. Various psychological theories exist in the literature to explain the behavior of men who commit child sex offenses, including the belief that child sexual abuse (CSA) is a predisposing factor for the transition from victim to the offender. These theories are, however, unable to explain the fact that while most victims of CSA are female, most perpetrators of CSA are male. The sex specificity of CSA in terms of victims and offenders suggests that the experience of CSA and its psychosocial effects may be different for boys compared to girls. And contrary to these theories is the fact that most abuse victims do not go on to abuse or molest children. On the other hand, some people with pedophilic disorder who were abused as children show an age preference that matches their age when they were abused, suggesting that they could be replicating the abuse they experienced as children.

Paraphilic Lovemaps

A **lovemap** is a person's emotional, internal blueprint for their ideal erotic, sexual situations. The concept was originated by sexologist John Money in his discussions of how people develop their sexual preferences. Money defined it as "a developmental representation or template in the mind and in the brain depicting the idealized lover and the idealized program of sexual and erotic activity projected in imagery or actually engaged in with that lover."

Money describes the formation of an individual's lovemap as similar to the acquisition of a native language, in that it becomes established at an early age and bears the mark of the person's unique individuality, like an accent in a spoken language. According to Money, lovemaps are not present at birth, but begin to develop shortly thereafter, and manifest in full after puberty. The individual may not discover certain aspects of their lovemap until triggered by a relevant experience (such as pornography use) later in life. **Vandalized lovemaps**—according to Money—occur when the lovemapping process becomes traumatized, as when a young child is either exposed to, or forced to participate in such inappropriate behaviors as child sexual abuse, incest, or sadomasochism. He states that such a lovemap is typically formed between the ages of five and eight. A vandalized lovemap may be paraphilic or hyposexual.

In paraphilic disorders, lust is attached to fantasies and practices that are socially forbidden, disapproved of, ridiculed, or penalized. According to Money, it can result from lust being displaced from a "vandalized" area of an ordinary lovemap following abuse. Alternatively, Money suggests that a paraphilic lovemap could arise from a non-sexual childhood experience that induced genital arousal in the child.

Sociocultural Perspectives on Sexual Deviations

There is no stereotypical profile of sexually violent persons. Perpetrators may come from various backgrounds and they may be someone known by the victim, like a friend, a family member, an intimate partner, or an acquaintance or they may be a complete stranger. The primary motivators behind sexually violent acts are believed to be power and control, and not, as it is widely perceived, a sexual desire. Sexual violence is rather a violent, aggressive, and hostile act aiming to degrade, dominate, humiliate,

terrorize, and control the victim. Some of the reasons for committing sexual violence are that it reassures the offender about his or her sexual adequacy, it discharges frustration, compensates for feelings of helplessness, and achieves sexual gratification.

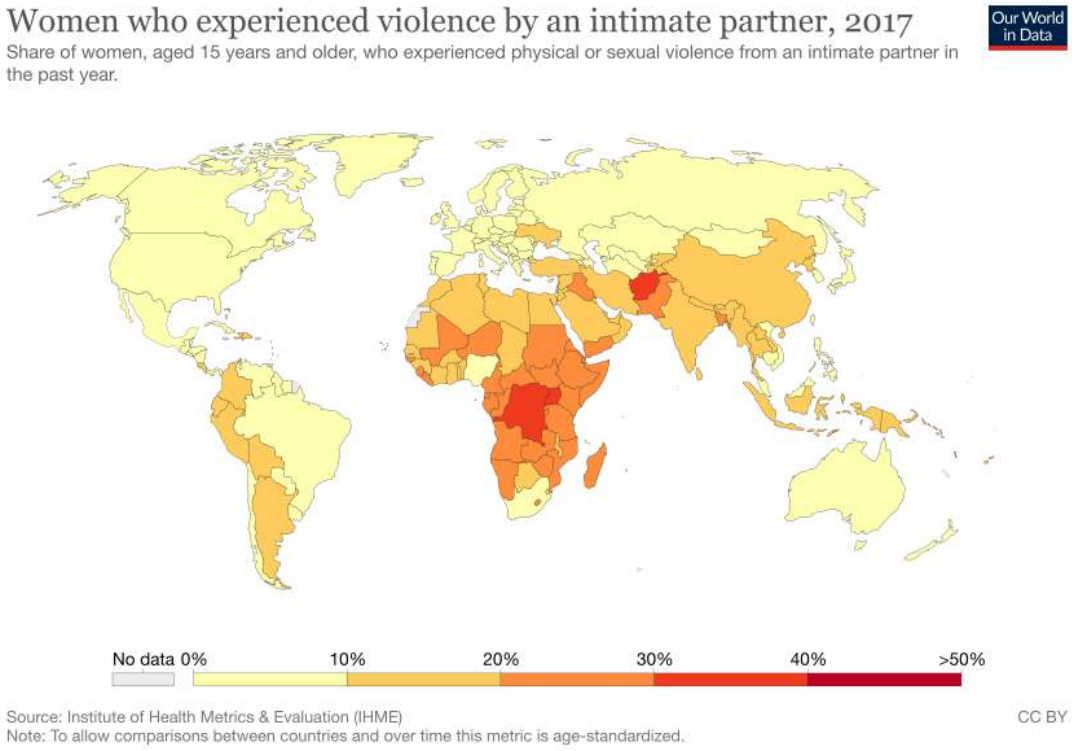


Figure **10.14.1**: This map shows us that sexual violence occurs all over the world and at different rates in different countries.

Data on sexually violent men are somewhat limited and heavily biased towards apprehended rapists, except in the United States, where research has also been conducted on male college students. Despite the limited amount of information on sexually violent men, it appears that sexual violence is found in almost all countries (though with differences in prevalence), in all socioeconomic classes, and in all age groups from childhood onwards. Data on sexually violent men also show that most direct their acts at women whom they already know. Among the factors increasing the risk of a man committing rape are those related to attitudes and beliefs as well as behavior arising from situations and social conditions that provide opportunities and support for abuse.

Developmental and Behavioral Perspectives

There is evidence to suggest that sexual violence is also a learned behavior in some adults, particularly in regard to child sexual abuse. Studies on sexually abused boys have shown that around one in five continue in later life to molest children themselves.

Childhood environments that are physically violent, emotionally unsupportive, and characterized by competition for scarce resources have been associated with sexual violence. Sexually aggressive behavior in young men, for instance, has been linked to witnessing family violence and having emotionally distant and uncaring fathers. Men raised in families with strongly patriarchal structures are also more likely to become violent, to rape and use sexual coercion against women, and to abuse their intimate partners than men raised in homes that are more egalitarian.

With regard to early childhood environments, research has stressed the importance of encouraging nurturing, with better and more gender-balanced parenting, to prevent sexual violence. A **prevention model** that adopts a developmental approach, with interventions before birth, during childhood, and in adolescence and young adulthood has been developed and is used by some. In this model, the prenatal element would include discussions of parenting skills, the stereotyping of gender roles, stress, conflict, and violence. In the early years of childhood, health providers would pursue these issues and introduce child sexual abuse and exposure to violence in the media to the list of discussion topics, as well as promoting the use of non-sexist educational materials. In later childhood, health promotion would include modeling behaviors and attitudes that avoid stereotyping, encouraging children to

distinguish between good and bad touching, and enhancing their ability and confidence to take control over their own bodies. This intervention would allow room for talking about sexual aggression. During adolescence and young adulthood, discussions would cover myths about rape, how to set boundaries for sexual activity, and breaking the links between sex, violence, and coercion.

Treatment for Sexual Deviations

The treatment and management of paraphilias and paraphilic disorders pose extreme difficulty due to a multitude of factors. Despite the egosyntonic and egodystonic dual nature of paraphilias in general, the overall majority of patients rarely seek treatment voluntarily. Many individuals may feel indignity, culpability, or discomfiture, while others focus on the difficulty and lack of desire to halt efforts of achieving intense sexual pleasure and ultimate satisfaction. Furthermore, many may fear the legal repercussions of coming forward for treatment. Those patients in treatment or seeking treatment are most often either mandated legally or convinced by family, friends, or sexual partners.



Figure **10.14.2**: Psychotherapy combined with group therapy may be particularly useful for patients with a paraphilic disorder.

The management of paraphilic disorders falls into two main categories, incorporating both psychological and biological constituents. The psychological approach, which includes psychotherapy, but more importantly, CBT, yields an overall positive outcome in terms of efficacy, regardless of the type of diagnosed paraphilic disorder.

Treatments within the psychological perspective seem most effective when combining individual with group therapy. The cognitive-behavioral perspective is particularly useful in helping clients recognize their distortions and denial. At the same time, these clients benefit from training in empathy, so that they can understand how their victims are feeling. Adding to the equation within the psychological perspective, clinicians may also train clients in learning to control their sexual impulses. [2] However, due to the patient's reluctance to seek treatment or the legal obligation to obtain treatment, psychiatrists are often forced to focus efforts on protection against potential victimization and limit focus on the reduction of distress in the patient. The predisposition of committing sexual offenses demonstrates the significance of biological treatments for paraphilic disorders for not only the suffering individual, but also for the greater good of society. However, specialized management, with a comprehensive treatment plan encompassing both psychological and pharmacological components, proves to be the optimal therapeutic option overall.

The three main classifications of pharmacological agents used in the management of paraphilic disorders involve selective serotonin reuptake inhibitors (SSRIs), synthetic steroidal analogs, and antiandrogens. The therapeutic choice is dependent upon previous medical history and medication compliance, along with the intensity of both the sexual fantasy and the risk of sexual violence.

Literature suggests that each of the three drug categories mentioned above help to target diverse physiological pathways and subsequent psychological attributes through their unique mechanism of action. Studies have shown SSRIs to be particularly useful in the adolescent population and milder paraphilias, including exhibitionism, as well as in patients suffering from comorbidities of obsessive-compulsive disorders (OCD) or depression. SSRIs have also been used in the attempted alleviation of hypersexuality, but strong evidence of actual efficacy has yet to be established. Antiandrogens, with emphasis on gonadotropin-releasing hormone (GnRH) analogs, have shown to considerably reduce the frequency and intensity of both deviant sexual arousal and behavior. Gonadotropin-releasing hormone (GnRH) analogs are also considered to be among the most promising pharmacological management for those sex offenders at high risk of particularly violent acts, particularly serial rapists or those individuals with pedophilic disorder. Of note, informed consent is obligatory before initiating antiandrogenic therapy.

Treatment for the Sexually Violent

Sexually violent men have been shown to be more likely to consider victims responsible for the rape and are less knowledgeable about the impact of rape on victims. Such men may misread cues given out by women in social situations and may lack the

inhibitions that act to suppress associations between sex and aggression. They may have coercive sexual fantasies and overall are more hostile towards women than are men who are not sexually violent. In addition to these factors, sexually violent men are believed to differ from other men in terms of impulsivity and antisocial tendencies. They also tend to have an exaggerated sense of masculinity.

The research on convicted rapists has found several important motivational factors in the sexual aggression of males. Those motivational factors repeatedly implicated are having anger at women and having the need to control or dominate them. A study by Marshall *et al.* (2001) found that male rapists had less empathy toward women who had been sexually assaulted by an unknown assailant and more hostility toward women than non-sex-offenders and nonoffender males/females. Meta-analyses indicate that convicted rapists demonstrate greater sexual arousal to scenes of sexual coercion involving force than do non-rapists.

Psychotherapy can also be helpful in treatment, though research is unclear about its effectiveness for long-term prevention.[3]

? Applied criminal psychology

The effect of psychological and social factors on the functioning of our brain is the central question forensic or criminal psychologists deal with due to the fact it is the seed of all our actions. For forensic psychiatry, the main question is, “Which patient becomes an offender?” or “Which offender becomes a patient?” Another main question asked by these psychiatrists is, “What came first, the crime or the mental disorder?” Psychologists also look at environmental factors along with genetics to determine the likeliness (profiling) of a particular person to commit a crime.

Criminal and forensic psychologists may also consider the following questions:

1. Is a mental disorder present now? Was it present during the time of the crime?
2. What is the level of responsibility of the offender for the crime?
3. What is the risk of reoffending and which risk factors are involved?
4. Is treatment possible to reduce the risk of reoffending?

Accordingly, individual psychiatric evaluations are resorted to measuring personality traits by psychological testing that have good validity for the purpose of the court.



Figure **10.14.3**: Incarceration alone does not guarantee that offenders won’t commit acts of sexual violence again.

Therapists use various methods to assess individual sex offenders’ recidivism risk. Risk assessment tools consider factors that have been empirically linked by research to sexual recidivism risk. Researchers and practitioners consider some factors as **static**, such as age, the number of prior sex offenses, victim gender, relationship to the victim, and indicators of psychopathy and deviant sexual arousal, and some other factors as **dynamic**, such as an offender’s compliance with supervision and treatment. By examining both types of factors, a more complete picture of the offender’s risk can emerge, compared with static or dynamic factors used alone.

Recidivism is a major concern, in the treatment of paraphilia, especially in pedophilia. Most people recognize that incarceration alone will not solve sexual violence. Treating the offenders is critical in an approach to preventing sexual violence and reducing victimization.

Behavior modification programs have been shown to reduce recidivism in sex offenders. Often, such programs use principles of **applied behavior analysis (ABA)**, also called behavioral engineering, which is a scientific technique concerned with applying empirical approaches based upon the principles of respondent and operant conditioning to change behavior of social significance**.** Two such approaches from this line of research have promise. The first uses *operant*

conditioning approaches(which use reward and punishment to train new behavior, such as problem-solving) and the second uses **respondent conditioning** or *classical conditioning* procedures, such as **aversion therapy** (a form of treatment in which the patient is exposed to a stimulus while simultaneously being subjected to some form of discomfort). Many of the behaviorism programs use **covert sensitization** (eliminating the unwanted behavior by creating a profound and lasting association between the behavior and a highly disturbing *covert*, i.e., imagined, stimulus or consequence) and/or odor aversion: both are forms of aversion therapy, which have had ethical challenges. Such programs are effective in lowering recidivism by 15–18%. The use of aversion therapy remains controversial, and is an ethical issue related to the professional practice of behavior analysis. Previous research in the early 1990s has shown covert conditioning to be effective with sex offenders as part of a behavior modification treatment package. Clinical studies continue to find it effective with some generalization from office to natural environment with this population.

Castration as a treatment for men with paraphilic disorder, particularly pedophilic disorder, is intended to destroy the body's production of testosterone through surgical castration (removal of the testes) or chemical castration, in which the individual receives medications that suppress the production of testosterone. **Chemical castration** is also used in some countries, including the United States, to treat male sex offenders. Unlike physical castration, it is reversible by stopping the medication. For male sex offenders with severe or extreme paraphilias, physical castration appears to be effective, although more radical. It results in a 20-year re-offense rate of less than 2.3% (versus 80% in the untreated control group), according to a large 1963 study involving a total of 1,036 sex offenders by the German researcher A. Langelüddeke. This rate was much lower than otherwise expected compared with overall sex offender recidivism rates.

Programs for Perpetrators

There are few programs outside of the criminal justice system that target perpetrators of sexual violence. They are generally aimed at men convicted of male-on-female sexual assault, who form a significant portion of criminal cases of sexual violence. A common response of men who commit sexual violence is to deny both that they are responsible and that what they are doing is violent. These programs work with male perpetrators to make them admit responsibility. One way of achieving this admittance is for programs that target male perpetrators of sexual violence to collaborate with support services for victims, but this could potentially be a revictimization of rape victims and be a poor choice of action unless the rape perpetrator is highly apologetic.

In 2007, the Texas State Auditor released a report showing that sex offenders who completed the Texas Sex Offender Treatment Program (SOTP) were 61% less likely to commit a new crime.[4] Oftentimes psychological treatment programs are mandated in sentences, but unlike the successes shown in this Texas report, their efficacy is contested.[5]

Glossary

applied behavior analysis (ABA): also called behavioral engineering, is a scientific technique concerned with applying empirical approaches based upon the principles of respondent and operant conditioning to change behavior of social significance

aversion therapy: a form of treatment in which the patient is exposed to a stimulus while simultaneously being subjected to some form of discomfort.

behavior modification: used to change behavior or reduce maladaptive behavior by means of techniques that include negative and positive reinforcement, imposing environmental limitations, goal setting, and conditioning

castration: a treatment intended to destroy the body's production of testosterone through the removal of the testes

chemical castration: a treatment in which the individual receives medications that suppress the production of testosterone

covert sensitization: eliminating the unwanted behavior by creating a profound and lasting association between the behavior and a highly disturbing *covert*" (i.e., imagined) stimulus or consequence

dynamic factors: include an offender's compliance with supervision and treatment.

lovemap: a person's emotional, internal blueprint for their ideal erotic, sexual situations

perversion: a type of human behavior that deviates from that which is understood to be orthodox or normal.

prevention model to sexual violence: a model that adopts a developmental approach, with interventions before birth, during childhood, and in adolescence and young adulthood

respondent conditioning: known also as classical conditioning or Pavlovian, conditioning that occurs when we link or pair a previously neutral stimulus with a stimulus that is unlearned or inborn, called an unconditioned stimulus

static factors: include age, number of prior sex offenses, victim gender, relationship to the victim, and indicators of psychopathy and deviant sexual arousal

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10.15: Case Studies- Examining Sexual Deviations and Dysfunctions

Learning Objectives

- Identify sexual deviation and dysfunction in case studies

Case Study: Derek

Derek and Camilla met online and hit it off immediately. Derek knew Camilla had a nine-year-old daughter named Maggie, and when Camilla introduced the two of them, they also hit it off. No one was surprised when they decided to marry, even if they had only known each other shortly. Derek and Camilla were visibly in love, and they all seemed to be a happy family. Not too long after his marriage, Derek began developing an inappropriately close relationship with Maggie, now his stepdaughter. Seemingly innocent back rubs turned into extra time to give her bubble baths. He found it harder and harder to control urges he was having when left alone with the child. After only two months of life together under the same roof, Derek's behavior stepped outside the limits of normal parental physical affection. When Camilla, a nurse, left for work early each morning, Derek invited Maggie to the parental bedroom on the pretext that she could watch cartoons with him on the bed. He would stroke Maggie's hair and gradually proceed to massage her body while encouraging her to touch his genitals, saying that it was "OK for her to learn about what daddies are like." Maggie was confused by his behavior and even a little scared, but she did as she was told. Derek reinforced compliance to this behavior by offering Maggie gifts. He also encouraged her to keep their secret, and stated that even if she told anyone, he would deny it. One day Camilla returned home unexpectedly because she had forgotten something and caught him engaging in this behavior. She later learned that it had been going on for over a year.

Treatment

As evident in Derek's case, individuals with pedophilic disorder often do not feel distressed as a result of their urges and may never seek treatment. Thus, the first issue to address in psychotherapy would be to help Derek realize the harm he had done to both Maggie and Camilla; in other words, making his disorder ego-dystonic. The hope would be that it would become increasingly difficult for Derek to deny the problems his urges caused and recognize that he would have to work to control his urges throughout the rest of his life. He might eventually accept that his disorder was harmful to others and to his relationship with his wife, and that there could have been legal ramifications. Therapy would focus on controlling his urges and fantasies about children, in combination with the antiandrogen medication to reduce his fantasies and urges to have sex with children.

Case Study: Anne

For a number of years, Anne and Jodie have mutually enjoyed BDSM (bondage, domination/discipline, sadism/submission, and masochism) sex parties as a part of a community. Lately, Anne has developed a strong interest in maintaining control, both in regard to sex (e.g., tying up her girlfriend, Jodie) and in her life. She has insisted on submitting Jodie to demeaning and abusive sexual behavior. In the early years of their relationship, Jodie's requests involved relatively innocent pleas that Anne pinch her and bite her while they were sexually intimate. Over time, however, behaviors that involved pain increased and the nature of the pain changed. At present, they engage in what they call "private parties," during which Anne handcuffs Jodie to the bed and inflicts various forms of torture. Jodie goes along with and likes Anne's ideas of new ways to inflict pain. Now Anne has a repertoire of behaviors, ranging from burning Jodie's skin with matches to cutting her with razor blades. Anne and Jodie no longer have an interest in sexual intimacy other than that involving pain.

? Controversies in Diagnosing Paraphilias

As mentioned in this module, deciding what is "normal" in the area of sexuality is an issue fraught with difficulty and controversy. Critics of the DSM argue that several of the paraphilic disorders in DSM-5 pathologize nonnormative sexual behavior. These critics maintain that even breaking the law is not a sufficient basis for determining that an individual engaging in a paraphilic behavior has a psychological disorder. This criticism also applies to exhibitionistic, voyeuristic, and frotteuristic disorders, which don't involve victims in the same sense as do the other paraphilic disorders.[1]

1. Hinderliter, A. C. (2010). Disregarding science, clinical utility, and the DSM's definition of mental disorder: The case of exhibitionism, voyeurism, and frotteurism. *Archives of Sexual Behavior*, 39, 1235–1237. ↵

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10.16: Putting It Together- Sexual Deviations and Dysfunctions



In this chapter, you learned about sexual deviations and dysfunctions, such as gender dysphoria, disorders of sexual function and paraphilic disorders. At this point, you hopefully have a better understanding of how each diagnosis is unique in its presentation of symptoms, how the different disorders are treated, and which biopsychosocial perspectives prevail in the etiology of these disorders.

The module started out with a discussion on the distress a person may feel when there is a mismatch between their gender identity and their sex assigned at birth. You now know that this condition is referred to as gender dysphoria. You then had a thorough review of various types of sexual dysfunction and how sexual functioning is a complex bio-psycho-social process. The module ended with a somewhat disturbing review of deviant or disordered sexual behavior, from the different types of paraphilias to the different types of sexual violence. Interestingly enough, the same psychologist who baffled the scientific community on his treatment of David Reimer also proposed a plausible viewpoint that paraphilias are the expression of **lovemaps** gone awry or vandalized, hypothesizing that an individual is, in a sense, programmed to act out fantasies that are socially unacceptable and often harmful.

It's important to take time to think critically on what has been presented, and also to reflect on the concepts presented in the module and how each one has firm roots in empirical research.

Here's a review of the disorders discussed in this module:

- **Gender dysphoria** is a diagnostic category in DSM-5 that describes individuals who do not identify as their biological gender. This dysphoria must persist for at least six months and result in significant distress or dysfunction to meet DSM-5 diagnostic criteria. In order for children to be assigned this diagnostic category, they must verbalize their desire to become the other gender.
- **Early-onset gender dysphoria** is behaviorally visible in childhood. Sometimes gender dysphoria will desist in this group and they will identify as gay or homosexual for a period of time, followed by recurrence of gender dysphoria. This group is usually sexually attracted to members of their natal sex in adulthood.
- **Late-onset gender dysphoria** does not include visible signs in early childhood, but some report having had wishes to be the opposite sex in childhood that they did not report to others.
- **Male hypoactive sexual desire disorder (MHSDD)** is described as persistently or recurrently deficient (or absent) sexual/erotic thoughts or fantasies and desire for sexual activity, as judged by a clinician with consideration for the patient's age and cultural context, and persisting for at least six months.
- **Female sexual interest/arousal disorder (FSIAD)** is described as a lack of interest or significantly reduced interest in sexual activity. At least three of the following symptoms occur for a minimum of six months: reduced interest in sex, reduced sexual thought or fantasies, reduced initiation of sexual activity, reduced excitement during sexual activity, reduced excitement to sexual cues, and reduced sensation during sexual encounters.
- **Erectile disorder (ED**) is described as difficulty in obtaining or maintaining an erection during sexual activity 75–100% of the time.
- **Delayed ejaculation** is described as a marked delay in ejaculation or infrequency/absence of ejaculation in 75–100% of partnered sexual activity.
- **Premature ejaculation** is described as a persistent and undesirable premature ejaculation that occurs around one minute or less of vaginal penetration.

- **Female orgasmic disorder** is described as a delay in, absence of, or markedly reduced intensity of orgasms during 75–100% of sexual activity.
- **Genito-pelvic pain/penetration disorder (GPPPD)** was formerly called dyspareunia and vaginismus, and is characterized by difficulty or pain during vaginal penetration during intercourse. This disorder may also include fear or anxiety about penetration or tensing and tightening of pelvic floor muscles during intercourse.
- **Paraphilias** are persistent and recurrent sexual interests, urges, fantasies, or behaviors of marked intensity involving objects, activities, or even situations that are atypical in nature.
- **Fetishism** is the use of nonliving objects, most commonly shoes and undergarments, for sexual pleasure.
- **Transvestic fetishism** is the derivation of sexual arousal from cross-dressing or dressing in clothes of the opposite sex.
- **Exhibitionism** is the exposure of an individual's genitalia to unsuspecting strangers for sexual satisfaction.
- **Voyeurism** is the viewing of an unsuspecting person engaging in disrobing or sexual activity.
- **Frotteurism** is the touching of or rubbing against a nonconsenting person.
- **Sexual masochism** is the derivation of sexual arousal from being the recipient of physical or mental abuse and/or humiliation.
- **Sexual sadism** is when sexual arousal is gained from inflicting mental or physical suffering on a nonconsenting person.
- **Pedophilia** is any sexual activity with a prepubescent child, where the offender/patient is at least sixteen years of age, and the victim is at least five years younger.
- **Sexual assault** is an act in which a person intentionally sexually touches another person without that person's consent or coerces or physically forces a person to engage in a sexual act against their will. It is a form of sexual violence, which includes child sexual abuse, groping, **rape** (forced vaginal, anal, or oral penetration or a drug-facilitated sexual assault), or the torture of the person in a sexual manner.
- **Childhood sexual abuse** is defined as any sexual contact between a child and an adult or a much older child.
- **Incest** refers to sexual contact between a child and family members. In each of these cases, the child is exploited by an older person without regard for the child's developmental immaturity and inability to understand the sexual behavior

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CHAPTER OVERVIEW

11: Schizophrenia Spectrum and Other Psychotic Disorders

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11.1: Why It Matters- Schizophrenia Spectrum and Other Psychotic Disorders

Why learn about schizophrenia spectrum and other psychotic disorders?



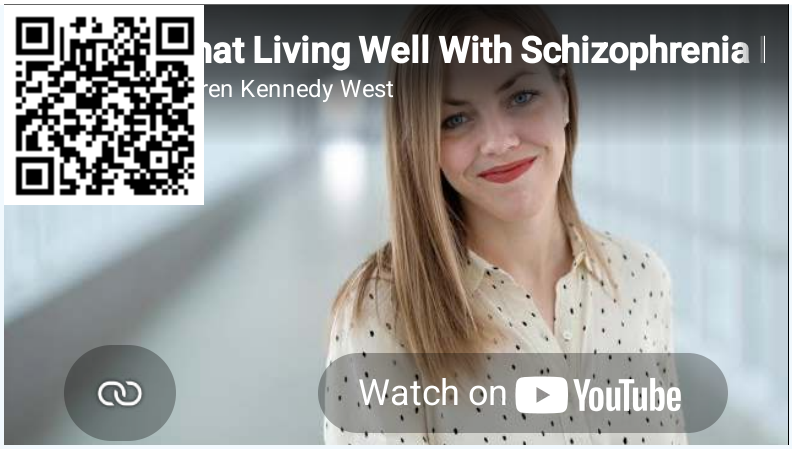
Schizophrenia and other psychotic disorders are some of the most impairing forms of psychopathology, frequently associated with a profound negative effect on the individual's educational, occupational, and social function. Sadly, these disorders often manifest right at the transition from adolescence to adulthood, just as young people are evolving into independent young adults. The spectrum or family of psychotic disorders includes schizophrenia, schizoaffective disorder, delusional disorder, schizotypal personality disorder, schizophreniform disorder, brief psychotic disorder, and psychosis associated with substance use or medical conditions. In this module, we summarize the primary clinical features of these disorders, describe the known cognitive and neurobiological changes associated with schizophrenia, describe potential risk factors and/or causes for the development of schizophrenia, and describe treatments for schizophrenia.

Perhaps you have encountered someone with schizophrenia or seen someone experiencing a psychotic episode. Individuals may have been talking to themselves or yelling at someone you could not see. If you tried to speak to them, they may have been difficult to follow or understand, or they may have acted paranoid or started telling a bizarre story about the people who were plotting against them. These behaviors are characteristic of the types of delusions, hallucinations, or eccentric behaviors exhibited by someone during a psychotic episode. Psychosis is a condition where an individual is out of touch with reality in some way, such as experiencing hallucinations (perceiving something that is not actually there such as hearing voices) and delusions (false beliefs based on faulty interpretations of reality). You have likely met people with schizophrenia without ever knowing it, as they may manage their symptoms in a way that you never know about it, or they may stay isolated to protect themselves from the frightening things they see, hear, or believe are operating in the outside world.

While individuals with schizophrenia are often portrayed as dangerous in media, those with schizophrenia are rarely violent and pose a much greater danger to themselves than others. These stereotypes and the accompanying stigma, however, are so accepted by most people in society, that persons with these disorders are frequently discriminated against with regard to relationships (even within their own families), housing, employment, and many other opportunities. Some people with schizophrenia have been famous for their impressive accomplishments, like NFL player and announcer Lionel Aldridge, or mathematician and Nobel Prize winner John Nash. Read on to learn more about it.

Watch It

Youtuber Lauren runs a YouTube channel about her experiences with schizophrenia and schizoaffective disorder. In this short clip, you'll see that Lauren speaks openly about her experiences and is an advocate for living well with the disorder.



You can view the transcript for “What Living Well With Schizophrenia Means To Me” here (opens in new window).

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11.2: Introduction to Schizophrenic Disorders

What you'll learn to do: explain the characteristics, symptoms, and etiology of schizophrenic disorders

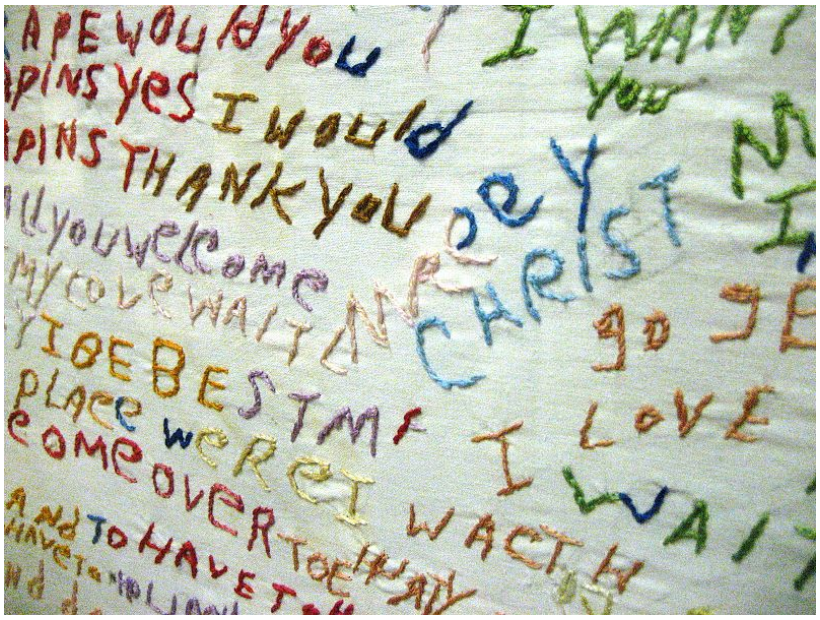


Figure **11.2.1**: Speaking in word salad, or random words strung together without meaning, is sometimes characteristic of schizophrenia.

Schizophrenia is a severe disorder characterized by a breakdown in a person's ability to function in life. People with schizophrenia experience hallucinations, delusions, or both and may have extreme difficulty regulating their emotions and behavior. Thinking tends to be incoherent and disorganized, the ability to communicate may be affected, some behaviors may be unusual by social standards, expression of emotions can be flattened, and motivation to engage in most basic life activities may be lacking. More often than not, an individual diagnosed with schizophrenia will require hospitalization, especially when they are first manifesting symptoms, in order to evaluate and treat them accurately; some persons will need ongoing additional support while others will respond to treatment and be able to work and be successful in life.

Schizophrenia is not to be confused with dissociative identity disorder, which confusion is a common mistake. The main characteristic of dissociative disorders is that people become dissociated from their sense of self, resulting in memory and identity disturbances; dissociative symptoms do not include psychotic symptoms nor the emotional and communication problems characteristic of schizophrenia spectrum disorders—these are very different types of disorders. Schizophrenia spectrum disorders are marked by at least two or more of the following symptoms: delusions, hallucinations, disorganized thinking, abnormal motor behavior, or other negative symptoms. Other psychotic disorders that include some degree of these characterizing features include schizotypal personality disorder, delusional disorder, brief psychotic disorder, schizophreniform disorder, schizoaffective disorder, and psychotic disorder related to substance abuse or other medical conditions.

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11.3: Schizophrenia

Learning Objectives

- Identify and describe the diagnostic criteria and major symptoms of schizophrenia
- Differentiate between the positive and negative symptoms of schizophrenia

Schizophrenia is a complex and significant psychological disorder characterized by major disturbances in thought, perception, emotion, and behavior. About 1% of the population experiences schizophrenia in their lifetime (i.e., over three million people in the United States alone), and usually, the disorder is first diagnosed during early adulthood (early to mid-20s). Most people with schizophrenia experience significant difficulties in many day-to-day activities, such as holding a job, paying bills, caring for oneself (grooming and hygiene), and maintaining relationships with others. However, contrary to common assumptions, a recent review of studies on schizophrenia (Vita & Barlati, 2018[1]) found a wide range of outcomes for persons who have schizophrenia, ranging from persons with severe symptoms and repeated episodes of remission and subsequent hospitalization to persons who experience a single episode that meets criteria followed by complete remission (although they usually continue to participate in treatment). Vita and Barlati (2018) also found that possibly up to half of the individuals diagnosed with schizophrenia either recovered or demonstrated significant improvement over time. They recommended that clinicians and society focus on two outcomes of consideration for persons with schizophrenia: clinical remission (significant reduction of symptoms and severity) and social functioning (e.g., ability to work, to function as a family member or in relationships, enjoy recreation, and dwell in independent living) in thinking about recovery.

Unlike other conditions such as depression or anxiety that almost all people can relate to in some ways, it is more difficult for most people to see the symptoms of schizophrenia and other psychotic disorders as part of the normal continuum of human experiences. However, the types of psychotic symptoms that characterize disorders like schizophrenia are on a continuum with normal mental experiences. For example, work by Jim van Os in the Netherlands has shown that a surprisingly large percentage of the general population (10%+) experience psychotic-like symptoms, though many fewer have multiple experiences and most will not continue to experience these symptoms in the long run (Verdoux & van Os, 2002). Similarly, work in a general population of adolescents and young adults in Kenya has also shown that a relatively high percentage of individuals experience one or more psychotic-like experiences (~19%) at some point in their lives (Mamah et al., 2012; Ndetei et al., 2012), though again most will not go on to develop a full-blown psychotic disorder.

So, what is schizophrenia? It is important to realize that schizophrenia is *not* a condition involving a split personality; that is, schizophrenia is not the same as dissociative identity disorder (previously known as multiple personality disorder). These disorders are sometimes confused because the word *schizophrenia* first coined by the Swiss psychiatrist Eugen Bleuler in 1911, derives from Greek words that refer to a “splitting” (schizo) of the mind (phrene) (Green, 2001). In the case of schizophrenia, the “split” is usually interpreted as between cognition (thinking and communicating) and emotions (see the discussion of flat affect below). Schizophrenia is considered a psychotic disorder (while dissociative disorders are not), which impairs a person’s thoughts, perceptions, and behaviors to the point where that individual is not able to function normally in life. Individuals who suffer from psychotic disorders experience a major disconnection with the world around them and do not share in the normal perception of the external environment. In other words, terms like *psychosis* or *psychotic* do not have anything to do with violence, serial killers, or other common misunderstandings; psychosis refers specifically to the presence of hallucinations (sensory distortions), delusions (unusual beliefs), or disorganized thought processes and can also occur during severe instances with other disorders.

Schizophrenia was once classified into distinct subtypes, such as paranoid, catatonic, disorganized, residual, or undifferentiated, but that method has since been replaced by approaching schizophrenia as a spectrum of disorders with varying degrees of severity and displaying several aspects of the subtypes. Now, schizophrenia subtypes are not listed in the DSM-5, as the subtypes would often change or coexist, but clinicians still sometimes specify a dominant type of subtype, such as “schizophrenia with paranoia.” The spectrum of psychotic disorders includes schizophrenia, schizoaffective disorder, delusional disorder, schizotypal personality disorder, schizophreniform disorder, brief psychotic disorder, and psychosis associated with substance use or medical conditions. These are all disorders of psychosis, with schizophrenia and schizoaffective disorder (schizophrenia combined with a mood disorder) being the most severe and personality disorders being less severe.

Symptoms of Schizophrenia

The main symptoms of schizophrenia can be categorized as either positive symptoms or negative symptoms. **Positive symptoms** are symptoms of addition, meaning they add something atypical or unusual to what other individuals experience, do, or think. Examples include hallucinations, delusions, and disorganized thinking and behaviors. **Negative symptoms** are those that result in noticeable decreases or absences in common behaviors, emotions, or drives (APA, 2013; Green, 2001). Examples include flattened emotional expression, lack of motivation for self-care, or significant social withdrawal.

Positive Symptoms

A **hallucination** is a perceptual experience that occurs in the absence of external stimulation. Auditory hallucinations (hearing voices) occur in roughly two-thirds of patients with schizophrenia and are by far the most common form of hallucination (Andreasen, 1987). The auditory voices may be familiar or unfamiliar, they may have a conversation or argue, or the voices may provide a running commentary on the person's behavior (Tsuang, Farone, & Green, 1999).

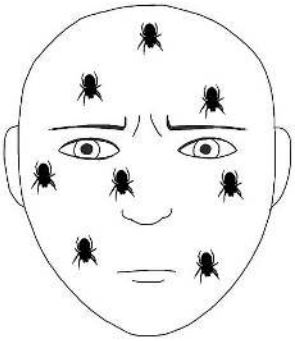


Figure **11.3.1**: Tactile hallucinations, like that of imaginary spiders crawling on the skin, are another type of hallucination.

Less common are visual hallucinations (seeing things that are not there) and olfactory hallucinations (smelling odors that are not actually present). In interacting with persons with psychotic symptoms, it is helpful to remember that although you may not hear what they are hearing nor see what they are seeing or experiencing, they *are* having those sensory experiences. To them, these experiences seem as real as you seeing a car drive by on the street or hearing a neighbor's dog barking. Telling them they are wrong or that those things are not happening does not improve your ability to relate to them or help them reconnect to the world around them. Instead, try to understand what they are experiencing and demonstrate empathy and understanding.

Delusions are beliefs that are contrary to reality and are firmly held even in the face of contradictory evidence. Many of us hold beliefs that some would consider odd, but a delusion is easily identified because it is absurd according to normal social and cultural standards. A person with schizophrenia may believe that his mother is plotting with the FBI to poison his coffee or that his neighbor is an enemy spy who wants to kill him. These kinds of delusions are known as **paranoid delusions**, which involve the false belief that other people or agencies are plotting against the person. People with schizophrenia also may hold **grandiose delusions**, which are beliefs that one holds special power, unique knowledge, or is extremely important. For example, the person who claims to be Jesus Christ, or who claims to have knowledge going back 5,000 years, or who claims to be a great philosopher is experiencing grandiose delusions. Other delusions include the belief that one's thoughts are being removed from their head (thought withdrawal) or thoughts have been placed inside one's head (thought insertion). Another type of delusion is a **somatic delusion**, which is the belief that something highly abnormal and improbable is happening to one's body (e.g., that one's kidneys are being eaten by cockroaches).

Disorganized thinking refers to disjointed and incoherent thought processes—usually detected by what a person says. Individuals might ramble, exhibit loose associations (jump from topic to topic), or talk in a way that is so disorganized and incomprehensible that it seems as though the person is randomly combining words. Disorganized thinking is also exhibited by blatantly illogical remarks (e.g., "Fenway Park is in Boston. I live in Boston. I live at Fenway Park.") and by tangentiality: responding to others' statements or questions by remarks that are either barely related or unrelated to what was said or asked. For example, if a person diagnosed with schizophrenia is asked if she is interested in receiving special job training, she might state that she once rode on a train somewhere. To a person with schizophrenia, the tangential (slightly related) connection between job *training* and riding a *train* are sufficient enough to cause such a response.

As another example, at the beginning of an interview, a clinician remarked in passing that he forgot to bring his pen to take notes. The patient begins to talk about living on a farm as a child and taking care of pigs which was tangential to the focus of the conversation. However, there is a linguistic association between "pen" (writing tool) and an animal enclosure (pen) on a farm. In persons without thought disorder or disorganized thinking, the brain would light up with these language associations, but would quickly sort through them and prioritize those that match the context. For someone with schizophrenia, these filters and the ability to determine the appropriate context are usually impaired.

Disorganized or abnormal motor behavior refers to unusual behaviors and movements: becoming unusually active, exhibiting silly child-like behaviors (giggling and self-absorbed smiling), engaging in repeated and purposeless movements, or displaying odd facial expressions and gestures. In some cases, the person will exhibit **catatonic** behaviors that show decreased reactivity to the external environment, such as posturing, in which the person maintains a rigid and bizarre posture for long periods of time, or catatonic stupor, a complete lack of movement and verbal behavior. Another way catatonia is displayed is through **waxy flexibility**, which occurs when another person places an individual with schizophrenia in an unusual or uncomfortable position and they remain in that position, sometimes for hours.

Negative Symptoms

Unlike positive symptoms, negative symptoms are symptoms where ordinary and expected behaviors may be reduced or absent. A person who exhibits diminished emotional expression displays little emotion in his facial expressions, speech, or movements, even when such expressions are normal or expected (also known as **flat affect** where *affect* is a noun meaning the display of emotion). It is important to recognize, however, that although the person may have difficulty expressing their emotions the way most people do, they still experience the full range of normal human emotions[2]. Avolition (lack of volition) is characterized by a lack of motivation to engage in self-initiated and meaningful activity, including the most basic of daily living tasks such as bathing and grooming. Alogia (lack of speech; from the Greek *logos* meaning word or speech) refers to reduced speech output; in simple terms, patients do not speak or respond much in interactions with others. Another negative symptom is asociality, or social withdrawal and lack of interest in engaging in social interactions with others (this is differentiated from antisocial activity such as that of persons with antisocial personality disorder who are "against" or "anti" society). A final negative symptom, anhedonia, refers to an inability to experience pleasure. One who exhibits anhedonia expresses little interest in what most people consider to be pleasurable activities, such as hobbies, recreation, or sexual activity.

In their review of schizophrenia research, Vita and Barlati (2018) note that positive symptoms receive much attention, but negative and cognitive symptoms are often not treated effectively, leading persons to not achieve "functional" (or daily living) levels of remission. In addition to the use of some of the newer antipsychotics that may help with negative symptoms, psychosocial treatments are important in reducing negative symptoms and improving the person's ability to function well in their life.

Diagnostic Criteria

The diagnostic criteria for schizophrenia are listed below:

A. Two (or more) of the following must each present for a significant portion of time during a one-month period (or less if successfully treated). At least one of these must be (1), (2), or (3):

1. delusions
2. hallucinations
3. disorganized speech (e.g., frequent derailment or incoherence)
4. grossly disorganized or catatonic behavior
5. negative symptoms (i.e., diminished emotional expression or avolition)

B. For a significant portion of the time since the onset of the disturbance, level of functioning in one or more major areas, such as work, interpersonal relations, or self-care, is markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, there is failure to achieve expected level of interpersonal, academic, or occupational functioning).

C. Duration: continuous signs of the disturbance persist for at least six months. This six-month period must include at least one month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by

only negative symptoms or two or more symptoms listed in Criterion A present in an attenuated form (e.g., odd beliefs or unusual perceptual experiences).[3]

D. Schizoaffective disorder and depressive or bipolar disorder with psychotic features have been ruled out because either 1) no major depressive or manic episodes have occurred concurrently with the active-phase symptoms or 2) if mood episodes have occurred during active-phase symptoms, they have been present for a minority of the total duration of the active and residual periods of the illness.

E. The disturbance is not attributable to the physiological effect of a substance (e.g., drug abuse or a medication) or other medical condition.

F. If there is a history of autism spectrum disorder or communication disorder of childhood onset, the additional diagnosis of schizophrenia is made only if prominent delusions or hallucinations, in addition to the other required symptoms of schizophrenia, are also present for at least one month (or less if successfully treated).[4]

Link to Learning

Watch this video and try to identify which classic symptoms of schizophrenia are shown. See if you can describe the positive and negative symptoms that this individual exhibits.

Watch it

Watch this video for an overview of schizophrenia, including the causes and symptoms you've learned about thus far.



You can view the transcript for "Schizophrenia – causes, symptoms, diagnosis, treatment & pathology" here (opens in new window).

Risk Factors for Developing Schizophrenia

Consistent with the biopsychosocial model, several factors contribute to the risk of developing schizophrenia:

- **Genetics:** It is clear that there are important genetic contributions to the likelihood that someone will develop schizophrenia, with consistent evidence from family, twin, and adoption studies. (Sullivan, Kendler, & Neale, 2003). However, there is no "schizophrenia gene" and it is likely that the genetic risk for schizophrenia reflects the summation of many different genes that each contribute something to the likelihood of developing psychosis (Gottesman & Shields, 1967; Owen, Craddock, & O'Donovan, 2010). Further, schizophrenia is a very heterogeneous disorder, which means that two different people with schizophrenia may each have very different symptoms (e.g., one has hallucinations and delusions, the other has disorganized speech and negative symptoms). This makes it even more challenging to identify specific genes associated with risk for psychosis. Importantly, many studies also now suggest that at least some of the genes potentially associated with schizophrenia are also associated with other mental health conditions, including bipolar disorder,

depression, and autism (Gejman, Sanders, & Kendler, 2011; Y. Kim, Zerwas, Trace, & Sullivan, 2011; Owen et al., 2010; Rutter, Kim-Cohen, & Maughan, 2006).

- **Environment**: There are also a number of environmental factors that are associated with an increased risk of developing schizophrenia. For example, problems during pregnancy such as increased stress, infection, malnutrition, and/or diabetes have been associated with increased risk of schizophrenia. In addition, complications that occur at the time of birth and which cause hypoxia (lack of oxygen) are also associated with an increased risk for developing schizophrenia (M. Cannon, Jones, & Murray, 2002; Miller et al., 2011). Children born to older fathers are also at a somewhat increased risk of developing schizophrenia. Further, using cannabis increases risk for developing psychosis, especially if you have other risk factors (Casadio, Fernandes, Murray, & Di Forti, 2011; Luzi, Morrison, Powell, di Forti, & Murray, 2008). The likelihood of developing schizophrenia is also higher for kids who grow up in urban settings (March et al., 2008) and for some minority ethnic groups (Bourque, van der Ven, & Malla, 2011). Both of these factors may reflect higher social and environmental stress in these settings. Unfortunately, none of these risk factors is specific enough to be particularly useful in a clinical setting, and most people with these risk factors do not develop schizophrenia. However, together they are beginning to give us clues as the neurodevelopmental factors that may lead someone to be at an increased risk for developing this disease.
- **Brain structure and function**: Scientists think that differences in brain structure, function, and interactions among neurotransmitters may contribute to the development of schizophrenia. For example, differences in the volumes of specific components of the brain, in the way regions of the brain are connected and work together, and in neurotransmitters, such as dopamine, are found in people with schizophrenia. Differences in brain connections and brain circuits seen in people with schizophrenia may begin developing before birth. Changes to the brain that occur during puberty may trigger psychotic episodes in people who are vulnerable due to genetics, environmental exposures, or the types of brain differences mentioned above.

An important research area on risk for psychosis has been work with individuals who may be at clinical high risk. These are individuals who are showing attenuated (milder) symptoms of psychosis that have developed recently and who are experiencing some distress or disability associated with these symptoms. When people with these types of symptoms are followed over time, about 35% of them develop a psychotic disorder (T. D. Cannon et al., 2008), most frequently schizophrenia (Fusar-Poli, McGuire, & Borgwardt, 2012). In order to identify these individuals, a new category of diagnosis, called “Attenuated Psychotic Syndrome,” was added to Section III (the section for disorders in need of further study) of the DSM-5 (APA, 2013). However, adding this diagnostic category to the DSM-5 created a good deal of controversy (Batstra & Frances, 2012; Fusar-Poli & Yung, 2012). Many scientists and clinicians have been worried that including risk states in the DSM-5 would create mental disorders where none exist, that these individuals are often already seeking treatment for other problems, and that it is not clear that we have good treatments to stop these individuals from developing to psychosis. However, the counterarguments have been that there is evidence that individuals with high-risk symptoms develop psychosis at a much higher rate than individuals with other types of psychiatric symptoms, and that the inclusion of Attenuated Psychotic Syndrome in Section III will spur important research that might have clinical benefits. Further, there is some evidence that non-invasive treatments such as omega-3 fatty acids and intensive family intervention may help reduce the development of full-blown psychosis (Preti & Cella, 2010) in people who have high-risk symptoms.

Treatments and Therapies

The causes of schizophrenia are complex and are not fully understood, so current treatments focus on managing symptoms and solving problems related to day to day functioning.

Antipsychotic Medications

Antipsychotic medications can help reduce the intensity and frequency of psychotic symptoms. The medications are usually taken daily in pill or liquid forms. Some antipsychotic medications are given as injections once or twice a month, which some individuals find to be more convenient than daily oral doses. Patients whose symptoms do not improve with standard antipsychotic medication typically receive clozapine. People treated with clozapine must undergo routine blood testing to detect a potentially dangerous side effect that occurs in 1%-2% of patients.

Many people taking antipsychotic medications have side effects such as weight gain, dry mouth, restlessness, and drowsiness when they start taking these medications. Some of these side effects subside over time, but others may persist, which may cause some people to consider stopping their antipsychotic medication. Suddenly stopping medication can be dangerous and it can make schizophrenia symptoms worse. People should not stop taking antipsychotic medication without talking to a health care provider first.

Psychosocial Treatments

Cognitive behavioral therapy (CBT), behavioral skills training, supported employment, and cognitive remediation interventions may help address the negative and cognitive symptoms of schizophrenia. A combination of these therapies and antipsychotic medication is common. Psychosocial treatments can be helpful for teaching and improving coping skills to address the everyday challenges of schizophrenia. These treatments may help people pursue their life goals, such as attending school, working, or forming relationships. Individuals who participate in regular psychosocial treatment are less likely to relapse or be hospitalized.

Family Education and Support

Educational programs for family members, significant others, and friends offer instruction about schizophrenia symptoms and treatments, and strategies for assisting the person with the illness. Increasing key supporters' understanding of psychotic symptoms, treatment options, and the course of recovery can lessen their distress, bolster coping and empowerment, and strengthen their capacity to offer effective assistance. Family-based services may be provided on an individual basis or through multi-family workshops and support groups.

Coordinated Specialty Care

Coordinated specialty care (CSC) is a general term used to describe recovery-oriented treatment programs for people with first-episode psychosis, an early stage of schizophrenia. A team of health professionals and specialists deliver coordinated specialty care (CSC), which includes psychotherapy, medication management, case management, employment and education support, and family education and support. The person with early psychosis and the team work together to make treatment decisions, involving family members as much as possible. Compared to typical care for early psychosis, coordinated specialty care (CSC) is more effective at reducing symptoms, improving quality of life, and increasing involvement in work or school.

Assertive Community Treatment

Assertive community treatment (ACT) is designed especially for individuals with schizophrenia who are at risk for repeated hospitalizations or homelessness. The key elements of assertive community treatment (ACT) include a multidisciplinary team, a clinician who prescribes medication, a shared caseload among team members, direct service provision by team members, a high frequency of patient contact, low patient to staff ratios, and outreach to patients in the community. Assertive community treatment (ACT) reduces hospitalizations and homelessness among individuals with schizophrenia.

Try It

? Forensic Psychology

In August 2013, 17-year-old Cody Metzker-Madsen attacked five-year-old Dominic Elkins on his foster parents' property. Believing that he was fighting goblins and that Dominic was the goblin commander, Metzker-Madsen beat Dominic with a brick and then held him face down in a creek. Dr. Alan Goldstein, a clinical and forensic psychologist, testified that Metzker-Madsen believed that the goblins he saw were real and was not aware that it was Dominic at the time. He was found not guilty by reason of insanity and was not held legally responsible for Dominic's death (Nelson, 2014). Cody was also found to be a

danger to himself or others. He will be held in a psychiatric facility until he is judged to be no longer dangerous. This does not mean that he “got away with” anything. In fact, according to the American Psychiatric Association, individuals who are found not guilty by reason of insanity are often confined to psychiatric hospitals for as long or longer than they would have spent in prison for a conviction.

Hollywood depictions and news reports to the contrary, most people with schizophrenia are not violent. Only 3%-5% of violent acts are committed by individuals diagnosed with severe mental illness, whereas individuals with severe mental illnesses are more than 10 times as likely to be victims of crime (MentalHealth.gov, 2017). The most common conditions linked to violence are psychopathic personality (severe antisocial personality disorder), bipolar disorder, and persons who are abusing drugs (especially alcohol). The psychologists who work with individuals such as Metzker-Madsen are part of the subdiscipline of forensic psychology. Forensic psychologists are involved in psychological assessment and treatment of individuals involved with the legal system. They use their knowledge of human behavior and mental illness to assist the judicial and legal system in making decisions in cases involving such issues as personal injury suits, workers’ compensation, competency to stand trial, and pleas of not guilty by reason of insanity.

Glossary

catatonic behavior: decreased reactivity to the environment; includes posturing and catatonic stupor

delusion: belief that is contrary to reality and is firmly held, despite contradictory evidence

disorganized/abnormal motor behavior: highly unusual behaviors and movements (such as child-like behaviors), repeated and purposeless movements, and displaying odd facial expressions and gestures

disorganized thinking: disjointed and incoherent thought processes, usually detected by what a person says

dopamine hypothesis: theory of schizophrenia that proposes that an overabundance of dopamine or dopamine receptors is responsible for the onset and maintenance of schizophrenia

grandiose delusion: characterized by beliefs that one holds special power, unique knowledge, or is extremely important

hallucination: a perceptual experience that occurs in the absence of external stimulation, such as the auditory hallucinations (hearing voices) common to schizophrenia

negative symptom: characterized by decreases and absences in certain normal behaviors, emotions, or drives, such as an expressionless face, lack of motivation to engage in activities, reduced speech, lack of social engagement, and inability to experience pleasure

paranoid delusion: characterized by beliefs that others are out to harm them

prodromal symptom: in schizophrenia, one of the early minor symptoms of psychosis

schizophrenia: a severe disorder characterized by major disturbances in thought, perception, emotion, and behavior with symptoms that include hallucinations, delusions, disorganized thinking and behavior, and negative symptoms

somatic delusion: belief that something highly unusual is happening to one’s body or internal organs

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11.4: Delusional Disorder

Learning Objectives

- Explain the symptoms of, diagnosis of, etiology of, and treatment options for delusional disorder

A **delusion** is a fixed false belief based on an inaccurate interpretation of an external reality despite evidence to the contrary. The belief is not congruent with one's culture or subculture, and almost everyone else agrees it is false. An individual with delusional disorder has a difficult time discerning, or distinguishing, between external reality and their own belief. Similar to schizophrenia, delusional disorder causes a major disconnect between an individual's internal world (thoughts, feelings, and perceptions) and an individual's external world (relationships, work, and hobbies). Unlike schizophrenia, individuals with delusional disorder do not suffer from disorganized thinking or moderate to severe hallucinations that impact the ability to function and interact with others.

One interesting aspect of delusional disorders is that overall functioning is more or less preserved as compared to other psychotic disorders such as schizophrenia. Individuals with delusional disorder often continue to function at home and at work and relate to others in a normal manner, and their functionality is not impacted and behavior is not obviously bizarre as long as interactions do not lead to discussions of topics related to the delusion. However, the preoccupation with their delusional ideas can disrupt their overall life. Aside from discussing the specific delusion, it would not be easy to tell that an individual has a diagnosis of delusional disorder. Impairment can be significant in one's occupation, however, making it difficult for those with delusional disorder to hold a steady job if they are unable to focus on other issues than the delusional thought content. Additionally, there may be social isolation which can lead to additional psychological symptoms such as depression.

Diagnosis

The diagnosis of a **delusional disorder** occurs when a person has one or more delusional thoughts for one month or more, that has no explanation by another physiological, substance-induced, or medical condition or any other mental health condition. An individual's cultural and religious beliefs merit consideration before coming to the diagnosis. Cultural beliefs may also impact the content of delusions. In some cultures, a specific delusion can be perceived as possible, and therefore is not considered to be an impairment on a person's life, nor abnormal. A person also cannot have met Criterion A for schizophrenia at any point (two or more of hallucinations, delusions, disorganized speech, grossly disorganized behavior, or negative symptoms of schizophrenia).

There are two main classifications of delusions: **bizarre** and **non-bizarre**. Bizarre delusions include delusions involving a phenomenon that is impossible, not understandable, and unrelated to normal life. An example of a bizarre delusion would be an individual believing their organs have been removed and replaced overnight while they were asleep. A bizarre delusion such as this is not only impossible, but can be demonstrated as false by exhibiting no signs of invasion or physical marks. Even after their delusions are proven to be untrue, or impossible, individuals experiencing delusions will continue their belief with certainty and conviction. Non-bizarre delusions involve situations that are possible, such as being manipulated or harmed, that remain fixed false beliefs even without evidence.

Types of Delusions

Some of the most frequently encountered types of delusions are

- **delusional jealousy.** This delusion can be displayed by the belief that one's romantic, or sexual partner, is unfaithful, accompanied by abnormal or socially unacceptable behavior that is related to these thoughts. Also known as *Othello syndrome*, this type is more common in males; it can sometimes correlate with suicidal or homicidal ideations, and hence safety is an important consideration in evaluation and management. It is important to note that if an individual has been in prior relationships where their significant other had been unfaithful, this belief would not merit a delusional disorder as there is a real reason for that concern, fear, and initial mistrust. In such a situation, initial skepticism or mistrust of a partner might be considered normal.
- **erotomanic.** A delusion that another person, more frequently someone of higher status, is in love with the individual. Also known as *psychose passionelle*. These patients are usually socially withdrawn, dependent, sexually inhibited with a poor level of social and/or occupational functioning. Paradoxical conduct is an important characteristic wherein all denials of affection are rationalized as affirmations. Males with this type of delusion tend to be more aggressive than their female counterparts.

- **grandiose.** A conviction that one has great talent; made a great discovery; or has power, knowledge or a relationship with someone famous or a deity. This is also known as megalomania, which is the delusion of increased self-importance. Grandiose delusions often have religious content.
- **persecutory.** The central theme is being conspired against, threatened, attacked, harassed, or obstructed in pursuit of long-term goals. This is one of the most common types of delusions and patients can be anxious, irritable, aggressive, or even assaultive at times, often believing they need to protect themselves
- **somatic.** These delusions involve imaginary bodily functions and sensations. Also called monosymptomatic hypochondriacal psychosis, somatic delusions can severely impair reality. The patient is unarguably convinced of the severity of symptoms, even after seeing a medical professional or doctor who lets them know they are healthy and their concern is not a reality. The most common type of somatic delusions is that the person emits a foul odor or severe bad breath. Others include beliefs of infestation, for example with parasites, or body dysmorphic delusion (that parts of the body are distorted or misshapen). These patients also tend to have high levels of anxiety and nervousness.
- **thought broadcasting.** This is the delusion that one's thoughts are projected and can be perceived by others.
- **thought insertion.** A delusion that an external source or entity has inserted thoughts into one's mind and one's thoughts are not their own.
- **mixed.** No single theme is prevalent, but rather a mix of two or more delusions.



Figure **11.4.1**: A body dysmorphic delusion may lead someone to believe that body parts are distorted, but more common types of somatic delusions have someone concerned about the way they smell.

Differential Diagnosis

Differential diagnosis involves ruling out other causes of delusions such as drug-induced conditions, dementia, head injury or cancer, infections, metabolic disorders, and endocrine disorders. Other psychiatric disorders must then be ruled out. In delusional disorder, mood symptoms tend to be brief or absent, and unlike schizophrenia, delusions are usually non-bizarre, and hallucinations are minimal or absent. Interviews are important tools to obtain information about the patient's life situation and past history to help make a diagnosis. Clinicians generally review earlier medical records to gather a full history. Clinicians also try to interview the patient's immediate family, as interviews can be helpful in determining the presence of delusions. The mental status examination (MSE) is used to assess the patient's current mental condition. A psychological questionnaire sometimes used in the diagnosis of the delusional disorder is the Peters Delusion Inventory (PDI), which focuses on identifying and understanding delusional thinking. However, this questionnaire is more likely to be used in research than in clinical practice.

Etiology

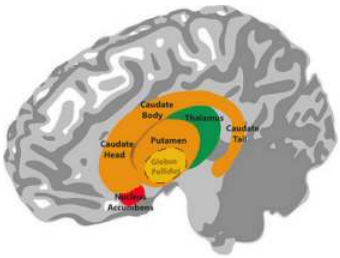


Figure **11.4.2**: The basal ganglia is located deep within the brain under the cerebral cortex.

Delusions can be caused in several ways, including from both substance use and medical conditions including traumatic brain injury and other forms of damage to brain tissue including strokes and dementia. Neurological conditions involving the limbic

system and basal ganglia in those with intact cortical functioning can also cause delusions (Joseph & Siddiqui, 2020). The main functions of the limbic system involve processing emotion, memory, motivation, and learning. The basal ganglia is a structure that plays a role in emotional and cognitive functioning, but is mainly responsible for motor control and movement.

Hypersensitive persons and ego defense mechanisms like reaction formation, projection, and denial are some psychodynamic theories for delusional disorder. Social isolation, envy, distrust, suspicion, and low self-esteem are some of the factors, which when becoming intolerable, lead to a person seeking an explanation and thus form a delusion as a solution. Immigrants with language barriers, deaf and visually impaired persons, as well as the elderly, are special populations who are more vulnerable to delusions due to a greater sense of vulnerability.

Epidemiology

The lifetime risk of delusional disorder in the general population has been estimated to range from 0.05%-0.1%, based on data from various sources, including case registries, case series, and population-based samples. According to the DSM-5, the lifetime prevalence of delusional disorder is about 0.02%. The prevalence of delusional disorder is much rarer than other conditions like schizophrenia, bipolar disorder, and other mood disorders, possibly due to under-reporting, as those with delusional disorder may not seek mental health attention unless forced by family or friends. The mean age of onset is about 40 years, but the range is from 18 years to 90 years. The persecutory and jealous types of delusion are more common in males, while the erotomanic variety is more common in females (Joseph & Siddiqui, 2020).

Treatment

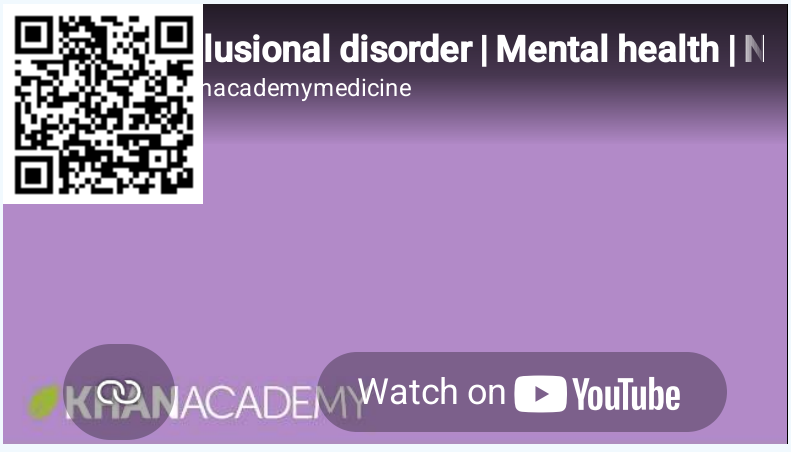
The treatment of delusional disorder is difficult considering the lack of insight. A good doctor-patient relationship is a key to treatment success. Treatment includes psychotherapy by establishing trust and building a therapeutic alliance.

A patient's history of medication compliance is the best guide to select appropriate antipsychotic medication. An antipsychotic should be started for a trial period of six weeks after which there is an evaluation of the effectiveness of the medication. Start a low dose and gradually increase the dosage as needed. Another drug from another class can be tried after six weeks if no benefit is noted from the initial treatment.

Some delusional disorders may respond well to antipsychotic medications; often the best approach is a combined treatment that includes antipsychotics and psychotherapy.

Watch It

This video explains delusional disorder and types of delusions.



You can view the transcript for "Delusional disorder | Mental health | NCLEX-RN | Khan Academy" here (opens in new window).

Glossary

bizarre delusions: delusions that involve phenomenon that are impossible, not understandable, and unrelated to normal life

delusion: a fixed false belief based on an inaccurate interpretation of an external reality despite evidence to the contrary

delusional jealousy: the belief that one's romantic, or sexual partner, is unfaithful, accompanied by abnormal or socially unacceptable behavior that's related to these thoughts

erotomanic delusions: a delusion that another person, more frequently someone of higher status, is in love with the individual

grandiose delusions: a conviction of great talent, discovery, inflated self-worth, power, knowledge or relationship with someone famous or deity

non-bizarre delusions: involve situations that are possible, such as being manipulated or harmed, but remain fixed false beliefs even after proven false.

persecutory delusions: central theme is being conspired against, attacked, harassed, obstructed in pursuit of long-term goals

somatic delusions: delusions involving imaginary bodily functions and sensations

thought broadcasting: the delusion that one's thoughts are projected and perceived by others

thought insertion: a delusion that an external source or entity has inserted thoughts into one's mind and one's thoughts are not their own

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11.5: Brief Psychotic Disorder

Learning Objectives

- Describe the symptoms, etiology, and management of brief psychotic disorder

Brief psychotic disorder according to DSM-5 is the sudden onset of psychotic behavior that lasts at least one day but less than one month followed by complete remission with possible future relapses. It is differentiated from schizophreniform disorder and schizophrenia by the duration of the psychosis. Brief psychotic disorder is an acute, often very intense, but transient disorder with the onset of one or more of the following psychotic symptoms:

- delusions
- hallucinations
- disorganized speech
- grossly disorganized or catatonic behavior

At least one of these symptoms must be delusions, hallucinations, or disorganized speech. The symptoms in brief psychotic disorder last between one day to one month, with a complete return to previous level of functioning after the disease course in response to antipsychotic medications. The disturbance in behavior cannot be better accounted for by schizophrenia, schizoaffective disorder, mood disorder with psychotic features, or be a direct result of a drug, medication, or medical condition like thyrotoxicosis (excessive levels of thyroid hormone in the blood), sarcoidosis (strong immune system inflammatory response), or syphilis (sexually transmitted bacterial infection). Other relevant medical conditions include brain tumors and head injury. The term *sudden onset* in this case refers to symptoms appearing within two weeks of a major stressful event.

Etiology

Although unclear, the underlying etiology of brief psychotic disorder is often a severely stressful event or trauma. There may be a genetic, neurological, or environmental component to brief psychotic disorder as well. The specific trigger of brief psychotic disorder, if present, must be specified as follows:

- Brief psychotic disorder with marked stressor(s) is also referred to as **brief reactive psychosis**. It is the onset of psychotic symptoms that occur in response to a traumatic event that would be stressful for anyone in similar circumstances in the same culture, such as the sudden loss of a loved one.
- Brief psychotic disorder without marked stressor(s) is the onset of psychotic symptoms that occur in the absence of a traumatic event.
- Brief psychotic disorder with **postpartum** onset is defined as the onset of psychotic symptoms that occur within four weeks postpartum.

Epidemiology

Reliable data on the frequency of brief psychotic disorder is not available, mostly because of its low incidence and variation based on the population under study. However, increased frequency of the disorder generally occurs in populations known to be under high stress such as immigrants, refugees, earthquake victims, etc. A study researching the Finnish population found the prevalence of brief psychotic disorder to be 0.05%. Another study in rural Ireland found 10 cases of brief psychotic disorder among 196 first-admission psychosis cases.

Compared to developed countries, reports show a higher incidence of brief psychotic disorder in developing countries. Data drawn from the World Health Organization (WHO) Determinants of Outcome Study also found that the incidence of brief psychotic disorder in developing countries was ten times as much as that in industrialized countries. Brief psychotic disorder is also thought to be more common in women and those with a personality disorder (such as schizotypal or borderline personality disorders).

Etiology

The exact cause of brief psychotic disorder is not known. One theory suggests a genetic link, because the disorder is more common in people who have family members with mood disorders, such as depression or bipolar disorder. Another theory suggests that the

disorder is caused by poor coping skills as a defense against or escape from a particularly frightening or stressful situation. These factors may create a vulnerability to develop brief psychotic disorder. In most cases, the disorder is triggered by a major stress or traumatic event.

In females, a low estrogen state (which may occur premenstrual, postpartum, or perimenopausal) can trigger sudden, short-lived psychosis. The psychosis is often linked to an underlying bipolar or schizophrenic condition. Such psychosis (when diagnosed as such) is often considered premenstrual exacerbation, menstrual psychosis, or postpartum psychosis. Childbirth may trigger the disorder in some women. Approximately one in 10,000 women experience brief psychotic disorder shortly after childbirth.[1]

Evaluation

There are no particular lab studies or psychological testing instruments that are useful in diagnosing brief psychotic disorder. The most appropriate tests and imaging to be done would rule out other potential diagnoses or causes for the behavioral disturbances. Hence, it would be apt to do a serum pregnancy test in women to evaluate any underlying triggers for the patient's behavioral disturbances. Other potential tests to consider ordering would be ECG, electrolytes, glucose level, liver function tests, thyroid function tests, and urinalysis. Urine toxicology tests can help exclude any potential drug or medication intoxication or withdrawal. CT scans and brain MRIs may also be performed to evaluate for any underlying structural causes for the symptoms.

Treatment/Management

It is important to first and foremost decide the appropriate level of care and whether the patient should be hospitalized or treated on an outpatient basis. The basis for decisions regarding treatment should be on multiple factors such as the patient's presenting symptoms, socioeconomic stability, the presence of supporting individuals or family, and the presence of homicidal or suicidal ideation. Because of the limited number of clinical trials evaluating the efficacy of specific treatment modalities in patients with brief psychotic disorder, current recommendations for treatment of brief psychotic disorder relies on pharmacological and psychotherapeutic interventions known to be effective in patients with other psychotic disorders.

Antipsychotics, especially second-generation (e.g., Clozaril, Zyprexa, Seroquel), are the first-line treatment for brief psychotic disorder. Although brief psychotic disorder characteristically shows complete resolution of symptoms within one month of symptom onset, it is suggested to continue treatment with antipsychotics for one to three months after symptom remission. Although oral formulations are preferable as first-line treatment for brief psychotic disorder, intramuscular formulations may have to be used in patients during immediate assessments and treatment, especially in emergency settings.

Pharmacotherapy

During the treatment process, the patient should be monitored on a long-term basis to assess for relapse or presence of residual symptoms that may necessitate referral to a specialist. It is essential to support the patient to maintain medication adherence as a lack of adherence may facilitate symptom relapse. The overall treatment plan for brief psychotic disorder should ideally include both pharmacological and psychosocial interventions. The biological, psychological, and social dimensions of the patient's life should in unison dictate the eventual treatment decisions made.

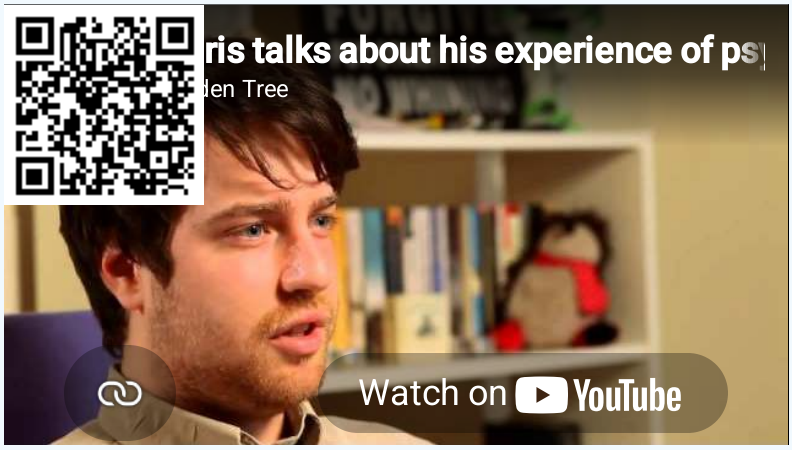
Prognosis

Given the nature of this condition, the prognosis is considerably good, as symptoms subside within a month. However, the symptoms may reoccur especially in the setting of a stressful psychosocial environment. Some positive indicators for the brief psychotic disorder are the absence of genetically related individuals with schizophrenia or brief psychotic disorder, sudden symptom onset, the presence of stressful triggers, and a short duration of symptoms.

Prognosis is notably worse for individuals diagnosed with brief psychotic disorder who have then been able to meet criteria for other disorders characterized by psychosis. A study conducted in Suffolk County, New York, in 2000 found that only 2% of the first-admission psychosis patients met the criteria for brief psychotic disorder at the six-month mark. Per the Suffolk County study, consisting of 11 patients initially given the diagnosis of brief psychotic disorder, three retained the diagnosis of brief psychotic disorder while the remaining nine received diagnoses of mood disorder, schizophrenia, schizophreniform disorder, and other disorders involving psychosis.[2]

Watch It

In this video, Chris speaks about his experience and recovery from brief psychotic disorder.



You can view the transcript for “Chris talks about his experience of psychosis and his recovery journey” here (opens in new window).

Glossary

brief psychotic disorder:: the sudden onset of psychotic behavior that lasts less than one month followed by complete remission with possible future relapses

brief psychotic disorder with postpartum: the onset of psychotic symptoms that occur within four weeks postpartum

brief reactive psychosis: brief psychotic disorder with marked stressor(s)

postpartum: period of time following the birth of a new baby

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11.6: Schizophreniform Disorder

Learning Objectives

- Describe the symptoms and management of schizophreniform disorder

Schizophreniform Disorder

Schizophreniform disorder is a mental disorder diagnosed when symptoms of schizophrenia are present for a significant portion of time (at least a month), but signs of disturbance are not present for the full six months required for the diagnosis of schizophrenia.

The exact cause of the disorder remains unknown, and relatively few studies have focused exclusively on the etiology of schizophreniform disorder. Like other psychotic disorders, a diathesis-stress model has been proposed, suggesting that some individuals have an underlying multifactorial genetic vulnerability to the disorder that can be triggered by certain environmental factors. Schizophreniform disorder is more likely to occur in people with family members who have schizophrenia or bipolar disorder.

If the symptoms have persisted for at least one month, a provisional diagnosis of schizophreniform disorder can be made while waiting to see if recovery occurs. If the symptoms resolve within six months of onset, the provisional qualifier is removed from the diagnosis. However, if the symptoms persist for six months or more, the diagnosis of schizophreniform disorder must be revised. The diagnosis of brief psychotic disorder may be considered when the duration of symptoms is less than one month.

The main symptoms of both schizophreniform disorder and schizophrenia may include

- delusions.
- hallucinations.
- disorganized speech resulting from thought disorder.
- disorganized or catatonic behavior.
- negative symptoms, such as:
  - an inability to express a range of emotions (flat affect),
  - an inability to experience pleasure (anhedonia),
  - impaired or decreased speech (aphasia),
  - a lack of desire to form relationships (asociality), and
  - a lack of motivation (avolition).

In order to be diagnosed with schizophreniform disorder, the person must be experiencing at least two of the symptoms listed, and at least one of them must be delusional thinking, hallucinations, or disorganized speech/thought disorder.

Epidemiology

Schizophreniform disorder is equally prevalent among men and women. The most common ages of onset are 18–24 for men and 18–35 for women. While the symptoms of schizophrenia often develop gradually over a period of years, the diagnostic criteria for schizophreniform disorder require a much more rapid onset.

Available evidence suggests variations in incidence across sociocultural settings. In the United States and other developed countries, the incidence is low, possibly fivefold less than that of schizophrenia. In developing countries, the incidence is substantially higher, especially for the subtype with good prognostic features. *Good prognostic features* means that two of the following are met:

- symptom onset within four weeks of unusual behavior
- confusion or perplexity
- good pre-illness social and work functioning
- absence of a flat affect

Treatment

Various modalities of treatment, including pharmacotherapy, psychotherapy, and various other psychosocial and educational interventions, are used in the treatment of schizophreniform disorder. Pharmacotherapy is the most commonly used treatment modality as psychiatric medications can act quickly to both reduce the severity of symptoms and shorten their duration. The medications used are largely the same as those used to treat schizophrenia, with an atypical antipsychotic as the usual drug of choice. Patients who do not respond to the initial atypical antipsychotic may benefit from being switched to another atypical antipsychotic; other alternative approaches include the addition of a mood stabilizer such as lithium or an anticonvulsant to current medications, or being switched to a typical (first-generation) antipsychotic.

Treatment of schizophreniform disorder can occur in inpatient, outpatient, and partial hospitalization settings. In selecting the treatment setting, the primary aims are to minimize the psychosocial consequences for the patient and maintain the safety of the patient and others. While the need to quickly stabilize the patient's symptoms almost always exists, consideration of the patient's severity of symptoms, family support, and perceived likelihood of compliance with outpatient treatment can help determine if stabilization can occur in the outpatient setting. Patients who receive inpatient treatment may benefit from a structured intermediate environment, such as a sub-acute unit, step-down unit, partial hospital, or day hospital, during the initial phases of returning to the community.

As improvement progresses during treatment, help with coping skills, problem-solving techniques, psychoeducational approaches, and eventually occupational therapy and vocational assessments are often beneficial for patients and their families. Virtually all types of individual psychotherapy are used in the treatment of schizophreniform disorder, except for insight-oriented therapies as patients often have limited insight as a symptom of their illness.

Since schizophreniform disorder has such rapid onset of severe symptoms, patients are sometimes in denial about their illness, which also would limit the efficacy of insight-oriented therapies. Supportive forms of psychotherapy, such as interpersonal psychotherapy, supportive psychotherapy, and CBT, are particularly well suited for the treatment of the disorder. Group psychotherapy is usually not indicated for patients with schizophreniform disorder because they may be distressed by the symptoms of patients with more advanced psychotic disorders.

Watch It

Watch the end of this video (start at the 2:17 mark) to review ways to differentiate between psychotic disorders. Brief psychotic episodes last less than one month, schizophreniform disorder is the diagnosis for psychotic symptoms lasting between one and six months, and schizophrenia is the diagnosis for psychotic symptoms lasting longer than six months. Schizoaffective disorder, which we'll learn about next, is different in that it includes a mood disorder.



You can view the transcript for "Schizophrenia, Schizoaffective, and Schizophreniform" here (opens in new window).

Glossary

schizophreniform disorder: the diagnosis for psychotic behavior that lasts between one and six months

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11.7: Schizoaffective Disorder

Learning Objectives

- Describe the symptoms, etiology, and management of schizoaffective disorder

Schizoaffective disorder (SD) is a mental disorder characterized by both ongoing psychosis and significant symptoms of mood disorders. The diagnosis is made when the person has symptoms of both schizophrenia (usually psychosis) and a mood disorder—either bipolar disorder or depression. The main criterion for the schizoaffective disorder diagnosis that separates it from a mood disorder diagnosis is the presence of psychotic symptoms for at least two weeks without any mood symptoms present. The "main" disorder most resembles schizophrenia, and while the mood disorder may come and go, elements of psychosis are always present. This differs from a mood disorder with psychotic features, which is predominately a mood disorder where elements of psychosis sometimes flare up, such as during a manic episode.[1] Schizoaffective disorder can often be misdiagnosed when the correct diagnosis may be a major depressive episode or bipolar disorder with mood-congruent or mood-incongruent psychotic features.



Figure **11.7.1**: Schizoaffective disorder includes typical features of schizophrenia, combined with features of major mood episodes.

Diagnosis

The specific DSM-5 criteria for schizoaffective disorder are as follows: [2]

> A. An uninterrupted duration of illness during which there is a major mood episode (manic or depressive) in addition to criterion A for schizophrenia; the major depressive episode must include depressed mood. Criterion A for schizophrenia is as follows:
>
> Two or more of the following presentations, each present for a significant amount of time during a one-month period (or less if successfully treated). At least one of these must be from the first three below:

- delusions
- hallucinations
- disorganized speech (e.g., frequent derailment or incoherence).
- grossly disorganized or catatonic behavior
- negative symptoms (i.e., diminished emotional expression or avolition)

> B. Hallucinations and delusions for two or more weeks in the absence of a major mood episode (manic or depressive) during the entire lifetime duration of the illness.
>
> C. Symptoms that meet the criteria for a major mood episode are present for the majority of the total duration of the active as well as residual portions of the illness.
>
> D. The disturbance is not the result of the effects of a substance (e.g., drug abuse or a medication) or another underlying medical condition.

The following are specifiers based on the primary mood episode as part of the presentation:

- bipolar type: includes episodes of mania and sometimes major depression
- depressive type: includes only major depressive episodes

A patient must meet the criteria for A–D above to be diagnosed with schizoaffective disorder.

Schizoaffective disorder is one of the most misdiagnosed psychiatric disorders in clinical practice. In fact, some researchers have proposed revisions to the diagnostic criteria, while others have suggested removing the diagnosis from the DSM-5. The challenges lie within the diagnostic criteria itself, since the disorder is part of a spectrum that shares criteria with many other prominent psychiatric disorders found in clinical practice.

Differential Diagnosis

Due to having criteria that encompass both psychotic and mood symptoms, schizoaffective disorder is easy to mistake for other mental disorders. Disorders that must be ruled out during the workup of schizoaffective disorder include:

- schizophrenia
- major depressive disorder (MDD) with psychotic features
- bipolar disorder with psychotic features

There has to be a definite period of at least two weeks in which there are only psychotic symptoms (delusions and hallucinations) without mood symptoms to diagnose schizoaffective disorder. However, a major mood episode (depression or mania) is present for the majority of the total duration of the illness. If the psychotic symptoms predominate the majority of the total duration of the illness, the diagnosis leans towards schizophrenia. Also, schizophrenia requires six months of prodromal or residual symptoms; schizoaffective disorder does not require this criterion.

Patients with major depression with psychotic features (MDD with PF), only experience psychotic features during their mood episodes. In contrast, schizoaffective requires at least two weeks in which there are only psychotic symptoms (delusions and hallucinations) without mood symptoms. Patients with MDD with psychotic features do not meet criterion A of schizoaffective disorder.

Similar to the contrasts with MDD with psychotic features, patients with bipolar disorder with psychotic features only experience psychotic features (delusions and hallucinations) during a manic episode. Again, schizoaffective disorder requires a period of at least two weeks in which there are only psychotic symptoms without mood symptoms. Psychotic features in bipolar disorder do not meet criterion A of schizoaffective disorder.

Watch It

This video explains more about the diagnostic criteria and symptoms of schizoaffective disorder.



You can view the transcript for “Schizoaffective Disorder?…What is it?” here (opens in new window).

Etiology

The term *schizoaffective disorder* first appeared as a subtype of schizophrenia in the first edition of the DSM. Schizoaffective disorder eventually became its own diagnosis despite a lack of evidence for unique differences in etiology or physiological processes. Therefore, there have been no conclusive studies on the etiology of the disorder. However, investigating the potential causes of mood disorders and schizophrenia as individual disorders allows for further discussion.

Some studies show that as high as 50% of people with schizophrenia also have comorbid depression. The etiology of both mood disorders and schizophrenia is multifactorial (remember the biopsychosocial model) and covers a range of risk factors including genetics, social factors, trauma, and stress. Among people with schizophrenia, there is a possible increased risk for first-degree relatives to develop schizoaffective disorder and vice-versa; there may also be increased risk among individuals for schizoaffective disorder who have a first-degree relative with bipolar disorder, schizophrenia, or schizoaffective disorder.

Treatment/Management

The treatment of schizoaffective disorder typically involves both pharmacotherapy and psychotherapy. The mainstay of most treatment regimens should include an antipsychotic, but the choice of treatment should be tailored to the individual. A study that reported obtained data on treatment regimens for schizoaffective showed that 93% of patients received an antipsychotic. Twenty percent of patients received a mood-stabilizer in addition to an antipsychotic, while 19% received an antidepressant along with an antipsychotic. [3] Prior to initiating treatment, if a patient with schizoaffective disorder is a danger to themselves or others, inpatient hospitalization should be considered; this includes patients who are neglecting activities of daily living or those who are disabled well below their baseline in terms of functioning.

Pharmacotherapy



Figure **11.7.2**: Antipsychotics medications can come as syrups, pills, or injections.

Antipsychotics

Antipsychotics are used to target psychosis and aggressive behavior in schizoaffective disorder. Other symptoms include delusions, hallucinations, negative symptoms, disorganized speech, and behavior. Most first- and second-generation antipsychotics block dopamine receptors while second-generation antipsychotics have further actions on serotonin receptors. Antipsychotics include, but are not limited to paliperidone (FDA approved for schizoaffective disorder), risperidone, olanzapine, quetiapine, ziprasidone, aripiprazole, and haloperidol. Clozapine is a consideration for refractory cases, much like in schizophrenia.

Mood-stabilizers

Patients who have periods of distractibility, indiscretion, grandiosity, a flight of ideas, increased goal-directed activity, and decreased need for sleep, and who are hyper-verbal fall under the bipolar specifier for schizoaffective disorder. Consider the use of mood stabilizers if the patient has a history of manic or hypomanic symptoms. These include medications such as lithium, valproic acid, carbamazepine, oxcarbazepine, and lamotrigine, which target mood dysregulation.

Antidepressants

Used to target depressive symptoms in schizoaffective disorder. Selective-serotonin reuptake inhibitors (SSRIs) are preferred due to lower risk for adverse drug effects and tolerability when compared to tricyclic antidepressants and selective norepinephrine reuptake inhibitors. SSRIs include fluoxetine, sertraline, citalopram, escitalopram, paroxetine, and fluvoxamine. It is vital to rule out bipolar disorder before starting an antidepressant due to the risk of exacerbating a manic episode.

Psychotherapy

Patients who have schizoaffective disorder can benefit from psychotherapy, as is the case with most mental disorders. Treatment plans should incorporate individual therapy, family therapy, and psychoeducational programs. The aim is to develop their social skills and improve cognitive functioning to prevent relapse and possible rehospitalization. This treatment plan includes education about the disorder, etiology, and treatment.

Individual therapy aims to normalize thought processes and better help the patient understand the disorder to reduce the burden of symptoms. Sessions focus on everyday goals, social interactions, social skills training, and vocational training.

Family involvement is crucial in the treatment of this schizoaffective disorder. Family education aids in compliance with medications and appointments, and helps provide structure throughout the patient's life given the dynamic nature of the schizoaffective disorder. Supportive group programs can also help if the patient has been in social isolation and provides a sense of shared experiences among participants.

Electroconvulsive Therapy



Figure **11.7.3**: A modern ECT machine.

Electroconvulsive therapy (ECT) is usually a last resort treatment. ECT is typically administered by a psychiatrist, an anesthesiologist, and a nurse, as it consists of electrical stimulation to the brain while the patient is under anesthesia. However, not only has ECT been used in urgent cases and treatment resistance, but it should also merit consideration in augmentation of current pharmacotherapy. The most common indicated symptoms are catatonia and aggression. ECT is safe and effective for most chronically hospitalized patients.

Prognosis

Given that the diagnostic criteria of schizoaffective disorder change periodically, prognostic studies have been challenging to conduct. However, a study by Harrison et al., 2001 on the overall prognosis of those with psychotic illness showed that 50% of cases showed favorable outcomes.[4] This study defined a favorable outcome as minimal or no symptoms, and/or employment. These outcomes were highly reliant on early initiation of treatment and optimized treatment regimens as described above.

Left untreated, schizoaffective disorder has many ramifications in both social functioning and activities of daily living, including unemployment, isolation, impaired ability to care for self, etc. Untreated mental disorders have more than just social and functional consequences. Some studies show that as many as 5% of people with a psychotic illness will commit suicide over their lifetime.[5] Research has shown that among all completed suicides, 10% are attributable to those with a psychotic illness.

Watch It

This video tells the story of Juan, who talks about how he's managed his schizoaffective disorder diagnosis through an early treatment program.



You can view the transcript for "Schizoaffective disorder: Juan's Story" here (opens in new window).

Glossary

electroconvulsive therapy: last resort treatment which involves a brief electrical stimulation of the brain while the patient is under anesthesia

schizoaffective disorder: a mental disorder characterized by abnormal thought processes and unstable moods

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11.8: Substance/Medication-Induced Psychotic Disorder

Learning Objectives

- Describe substance/medication-induced psychotic disorder

Substance/Medication-induced psychotic disorder (SIPD) involves the onset of the presence of psychotic features that have been triggered by the use of a substance or medication. It is a psychosis that results from the effects of chemicals or drugs, including those produced by the body itself. Various psychoactive substances have been implicated in either causing or worsening psychosis in some users. In these situations, psychosis manifests as disorientation, visual hallucinations, and/or haptic hallucinations (involving touch and sensations of the skin). In this state, a person's mental capacity to recognize reality, communicate, and relate to others is impaired and interferes with the capacity to deal with daily tasks and living.



Figure 11.8.1: Some people who smoke marijuana experience hallucinations.

There are a variety of chemical substances and drugs that have been known to induce psychosis:

- **Alcohol** is a common cause of psychotic disorders or episodes, which may occur through acute intoxication, chronic alcoholism, withdrawal, exacerbation of existing disorders, or acute idiosyncratic reactions. Often times, alcohol-induced psychosis is misdiagnosed as schizophrenia or another related mental illness with similar features and symptoms.
- **Cannabis** and **opioids** have also been found to cause psychosis, specifically hallucinations.
- **Sedatives** and **hypnotics** such as benzodiazepines and barbiturates can also cause depression, aggressiveness, and symptoms that can be misdiagnosed as psychosis.
- **Stimulants** such as cocaine and amphetamines have also been reported to cause psychosis.
- Additionally, medications such as fluoroquinolone drugs (antibiotic drugs like Cipro) and other prescription drugs such as antidepressants, L-dopa, prednisone, and anticholinergic drugs (medications such as Cogentin and Ditropan) have all been found to potentially induce psychosis, some of which are reported to be irreversible and permanent.
- Illicit substances, such as hallucinogenic or psychedelic substances, can also induce psychosis such as MDMA, PCP, and ketamine.

Stimulants are drugs that increase the body's functions by increasing the speed of activity in the central nervous system (CNS). These drugs increase the amount of dopamine in the brain, resulting in an increase in heart rate, alertness, and energy. Stimulants can be beneficial in treating medical conditions such as attention-deficit/hyperactivity disorder, narcolepsy, obesity, and depression. Familiar examples of stimulants include caffeine, cocaine, amphetamines, methamphetamines, and nicotine. All these substances share common side effects due to their similar influence on the CNS. These include, but are not limited to, appetite suppression, sleep disturbances, increased blood pressure, agitation, and psychosis. The research has focused on acute stimulant use resulting in psychosis.

Diagnostic Criteria

The DSM-5 defines **substance/medication-induced psychotic disorder** as the presence of delusions and/or hallucinations, with symptoms occurring soon after the intoxication or withdrawal of a substance or soon after exposure to a medication. The substance must have the potential to produce delusions or hallucinations that result in clinically significant impairment. Impairment as a result of delusions or hallucinations is a prevalent side effect of stimulants, and one reason why people who use them are more frequently referred to the emergency department or psychiatric ward than those who do not.



Figure 11.8.2: Substance/medication-induced psychotic disorder is diagnosed as the presence of delusions and/or hallucinations, with symptoms occurring soon after the intoxication or withdrawal of a substance or soon after exposure to a medication.

Acute psychosis induced by stimulants occurs within a period of four to five days after intoxication. The symptoms typically resolve with abstinence. However, recovery may be incomplete. Japan, where methamphetamines were first developed, has experienced major epidemics of stimulant-induced psychosis. This has provided further knowledge on the subject and has led to the realization that patients suffering from this disorder can be separated into three groups.[1] The first group includes patients experiencing transient psychosis after stimulant use that lasts for a period of four to five days following intoxication. The second group consists of patients who experience psychotic symptoms for as long as one month. The third group comprises patients who will not fully recover from their symptoms. In the Yui et al. study, 64% of patients gained full recovery from their psychosis within 10 days, 82% recovered within a month, and 18% suffered symptoms for over a month. In a more recent study by Zarrabi et al., the percentage of patients who experienced persistent symptoms for over one month was 31.6%. This phenomenon has resulted in a necessary increase of beds and resources available to health care systems worldwide.[2]

It should be noted that numerous studies have found a comorbidity of mental illness with stimulant-induced psychosis. However, the cause of stimulant-induced psychosis where there is no family history or prior personal history of mental illness remains unclear.

More research identifying the patient population most susceptible to stimulant-induced psychosis and/or a more exact determination of the minimum dosage of each stimulant drug necessary to trigger psychotic symptoms would assist in providing better patient care through a determination of whether the benefits of the prescription outweigh the risks. Answers to these questions could also alert those patients susceptible to acute psychosis—along with their family members—to watch for changes in behavior while the patient is on the medication. Additionally, if the general population is made more aware of the risk factors associated with psychosis, it may help prevent those who are vulnerable from abusing stimulants.

? Drugs and the Transition to Schizophrenia

A 2019 systematic review and meta-analysis by Murrie et al. estimated that the rate of persons who transitioned from substance-induced psychosis to a later diagnosis of schizophrenia was 25%, compared with 36% for a diagnosis of brief, atypical, and not otherwise specified psychoses.[3] Type of substance was the primary predictor of transition from drug-induced psychosis to schizophrenia, with the highest rates associated with cannabis (six studies, 34%), hallucinogens (three studies, 26%), and amphetamines (five studies, 22%). Lower rates were reported for opioid- (12%), alcohol- (10%), and sedative- (9%) induced psychoses. Transition rates were slightly lower in older cohorts, but were not affected by sex, country of the study, hospital or community location, urban or rural setting, diagnostic methods, or duration of follow-up.

Etiology

It is important to note that the consumption of medicines and drugs will not cause psychosis in everyone who uses them. As the diathesis-stress model suggests, there are genetic factors at play, which may make it more likely that an individual with a genetic predisposition to a mental disorder, especially that of schizophrenia and similar diagnoses, will be at greater risk for substance/medication-induced psychosis. Other factors such as duration of use, withdrawal, and the presence of other mental health disorders influence whether or not an individual will experience psychosis due to substance or medication use.

These conclusions are supported by research that has found that individuals with schizophrenia and schizophreniform disorder were at a greater risk for substance use disorders than non-psychotic individuals. There is also an increased risk for psychosis among substance-dependent individuals and a link between adolescent substance use and the onset of psychosis in young adulthood.[4]

Having a genetic risk (diathesis) for developing schizophrenia may also be associated with a stronger psychotic response to certain drugs. In a study of patients admitted to the hospital with first-episode psychosis, 74% had been diagnosed with a substance use

disorder at some point in their lives, and 62% met the criteria for a substance use disorder at the time of the psychotic episode.[5]

Watch It

This video explains some of the known causes of schizophrenia and psychosis, one of which is neurotransmitter dysfunction. Neurotransmitter dysfunction is affected by drug use, which helps to explain how drugs may lead to psychosis in some individuals.



https://youtu.be/ZP0oAh5WC-A

Alcohol-Induced Psychosis

The majority of patients presenting with psychosis for the first time have some history of substance abuse. A detailed history is important in the assessment and evaluation of alcohol-related psychosis; specifically, it is imperative to determine the patient's alcohol use history. It may be difficult to determine whether a patient's psychotic symptoms are due to a primary psychotic disorder or due to substance use, including alcohol. Diagnosis may be especially difficult in the emergency department where records of prior history are frequently lacking. No family history of psychotic disorder in a patient who has a clear history of alcohol use supports the diagnosis of alcohol-related psychosis.

Alcohol-related psychosis must be differentiated from other causes of psychosis and specifically from schizophrenia. When compared to schizophrenia, patients with alcohol-related psychosis tend to have significantly lower education levels, an onset of psychosis at an older age, more intense depressive and anxiety symptoms, and fewer negative and disorganized symptoms. Patients with alcohol-related psychosis also usually have better insight and judgment.

The DSM-5 states that the diagnosis of substance-induced psychotic disorder requires the presence of significant hallucinations or delusions. There must be evidence that the hallucinations or delusions started during or soon after substance intoxication or withdrawal or the substance used is known to cause the disturbance. The symptoms are not better explained by a psychotic disorder unrelated to substance use. The psychosis does not occur only with delirium (an abrupt state of mental and emotional confusion). The symptoms cause clinically significant distress or difficulty with normal activity such as work or social interactions.

The first priority in treating an individual with alcohol-induced psychosis is to stabilize the patient, paying close attention to airway, breathing, and vital signs. If the patient requires sedation due to alcohol-related psychosis, first-generation antipsychotics (neuroleptics), such as haloperidol, have been considered the first-line medications for treatment. Benzodiazepines, such as lorazepam, are used if there is a concern for alcohol withdrawal and seizures. Certain atypical antipsychotics, such as ziprasidone and olanzpine, have also been used to help sedate patients with acute psychosis. Some patients may require the use of physical restraints to protect the patient as well as the staff. Patients with alcohol-related psychosis must also be evaluated for suicidality since it is associated with higher rates of suicidal behaviors. The prognosis for alcohol-related psychosis is less favorable than earlier studies had speculated. However, if the patient can abstain from alcohol, the prognosis is good. If patients are unable to abstain from alcohol, the risk of recurrence is high.

Treatment

Acute psychotic symptoms may be managed through the use of medications such as antipsychotics. Because treatment often involves treating both the psychosis and the co-occurring disorders (called a *dual diagnosis*), then comprehensive treatment for both the drug addiction and the mental illness is recommended. This sometimes includes residential treatment and detox medications.

Psychotherapy such as CBT can help individuals with the disorder learn what may trigger a psychotic episode. CBT may help them to avoid relapse and develop healthier coping mechanisms. Family therapy programs focus on the entire family as a whole and not just on the individual battling mental illness and/or substance abuse. This type of therapy can help to restore stability and positive connection within the family unit. Recovery support groups are also usually incorporated into treatment and recommended as an ongoing relapse prevention strategy once treatment ends. These groups provide a continuous connection to the sober community and can help provide an environment where you can be honest about your journey and any struggles along the way.[6]

Watch It

This video details some of the complications in treating a drug-induced psychosis. Dr. Bakti tells of a young man who develops psychosis due to his excessive Adderall dependency.



You can view the transcript for "Drug-Induced Psychosis – Steven Batki, M.D." here (opens in new window).

Try It

Glossary

depressant: drug that tends to suppress central nervous system activity

hallucinogen: one of a class of drugs that results in profound alterations in sensory and perceptual experiences, often with vivid hallucinations

stimulant: drug that tends to increase overall levels of neural activity; includes caffeine, nicotine, amphetamines, and cocaine

substance/medication-induced psychotic disorder: the presence of delusions and/or hallucinations, with symptoms occurring soon after the intoxication or withdrawal of a substance or soon after exposure to a medication

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11.9: Introduction to Perspectives and Treatments Related to Schizophrenia Spectrum Disorders

What you'll learn to do: examine various perspectives and treatment methods for schizophrenia spectrum disorders



Now that you have learned about the major types of schizophrenia spectrum and psychotic disorders, let's examine a little bit more about their etiology and treatment. What factors lead to these disorders and how are they explained from the various psychological perspectives? What treatment methods are most effective? These are the types of questions we will consider in this next section.

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11.10: Biological Explanations for Schizophrenia and Other Psychotic Disorders

Learning Objectives

- Explain genetic studies, twin and adoption research, and biological explanations for schizophrenia

Causes of Schizophrenia

There is considerable evidence suggesting that genetic inheritance has a strong influence on the development of schizophrenia. For example, the risk of developing schizophrenia is nearly six times greater if one has a parent with schizophrenia than if one does not (Goldstein, Buka, Seidman, & Tsuang, 2010). Additionally, one's risk of developing schizophrenia increases as genetic relatedness to family members diagnosed with schizophrenia increases (Gottesman, 2001).

Genes



Figure **11.10.1**: Twin and adoptions studies estimate that the heritability of schizophrenia may be up to 80%.

When considering the role of genetics in schizophrenia, as in any disorder, conclusions based on family and twin studies have some inherent flaws because family members who are closely related (such as siblings) are more likely to share similar environments than are family members who are less closely related (such as cousins); further, identical twins may be more likely to be treated similarly by others than might fraternal twins. Thus, family and twin studies cannot completely rule out the possible effects of shared environments and experiences. Such problems can be corrected by using adoption studies, in which children are separated from their parents at an early age. One of the first adoption studies of schizophrenia conducted by Heston (1966) followed 97 adoptees, including 47 who were born to mothers with schizophrenia, over a 36-year period. Five of the 47 adoptees (11%) whose mothers had schizophrenia were later diagnosed with schizophrenia, compared to none of the 50 control adoptees. Other adoption studies have consistently reported that for adoptees who are later diagnosed with schizophrenia, their biological relatives have a higher risk of schizophrenia than do adoptive relatives (Shih, Belmonte, & Zandi, 2004).

Individual twin studies and meta-analyses of twin studies have estimated the heritability of risk for schizophrenia to be approximately 80% (this refers to the proportion of variation between individuals in a population that is influenced by genetic factors, not the degree of genetic determination of individual risk), but the heritability estimate varies from 41 to 87%. Concordance rates (the percentage of twins whose co-twin has the same condition) between monozygotic twins vary in different studies, with an average estimate of approximately 50%; whereas dizygotic twins was 17%. Some twin studies have found rates as low as 11.0%–13.8% among monozygotic twins, and 1.8%–4.1% among dizygotic twins, however. Genetics do appear to play a strong role in the etiology of schizophrenia, but others factors are also clearly involved as predicted by the biopsychosocial model.

? Genetics and Schizophrenia

Read this explanatory message, "Piecing Together the Genetic Puzzle of Schizophrenia," by Joshua Gordon, the director of the National Institute of Mental Health. Remember that genes may interact with each other, some of them suppressing other genes' activity and others enhancing them; epigenetic research also teaches us that some genes also interact with the environment, and that interaction shapes the outcomes of the genes' functions. Also, everyone (with the exception of twins) has his or her own genome; out of the many genetic variations involved in schizophrenia, a given person may inherit only a portion of the problematic genes. This complex situation may help us understand why many experts consider schizophrenia, other psychotic disorders, and even personality disorders, such as schizoid or schizotypal personality disorders as part of a family or

spectrum, a range of disorders that may all relate back to hundreds of interacting genetic variations. No two people have the same symptoms even if they have the same schizophrenia diagnosis.

Genetics does play a substantial role in the origins of schizophrenia, a role that is both pretty straightforward and incredibly complicated. The straightforward part: genetic differences account for a significant portion of our risk of having a mental illness. The complicated part: determining *how* these genetic differences contribute to risk is anything but straightforward.

One way to think about it is to consider two very different kinds of genetic traits: (red) hair color and height. Both run in families, but in very different ways. These differences can help us think about the kinds of genetic differences that underlie schizophrenia.

Most people with red hair have it because of a known alteration, or mutation, in the gene for a protein called the melanocortin-1 receptor (MC1R). If you have one or two copies of the **un**altered MC1R gene, you will have brown or black hair. But if you have two copies of the altered gene, you'll have red hair. There are some exceptions to this rule, but, for the vast majority of people, this one gene determines whether they have red hair or not.

The story is very different for height. According to the National Human Genome Research Institute, there are more than 700 genes that can contribute to determining how tall a person will be. It's the combined action of some of these individual genetic differences, interacting with environmental factors (epigenetics) such as diet and exercise, that influence how tall someone is likely to be when fully grown.

It has become clear that the genetics of schizophrenia is less like that of red hair and more like that of height. Over the past six years, NIMH-funded investigators and collaborators around the globe have amassed an impressively large genetics dataset, thanks to the generous participation of more than 60,000 individuals with schizophrenia and almost 100,000 individuals without the disorder. Comparing data from individuals in these two groups, scientists have identified over **250** places in the genome that contribute to the overall risk for schizophrenia. As with height, each one of these genetic risk factors plays a small role in influencing individual risk. Collectively, however, they play a significant role.

These findings are really good news, as each of these 250 risk factors represents an important clue into the biology of schizophrenia. However, the sheer number of risk factors, plus the fact that we still don't have a clear understanding of how these risk factors interact at the individual level, poses a challenge for those of us who would like to translate these genetic findings into better treatments. How can we study the relevant effects of these genes when we don't know which combinations are important? Although we have some ideas about how to proceed — for example, we can look for common biological mechanisms shared by multiple genes — it will take a while for us to sort out the best way to learn about schizophrenia from these "small-effect-size" variations.

But is schizophrenia all "small-effect-size" variations? In the past few months, researchers have started to find some rare, larger effect-size differences in single genes using a method called whole-exome sequencing.

Whole-exome sequencing involves reading the DNA sequences of all "protein-coding" genes — that is, all of the genetic information that gets transcribed into messenger RNA and used to make proteins. The exome is a prime target for investigation because we have a good understanding of how the genetic code is translated into proteins and, most importantly, how changes in protein structure can lead to dysfunction. This allows scientists to home in on the genetic differences that are likely to impact the function of proteins and cause disease.

With support from NIMH and other research funding organizations, psychiatric geneticists have been able to sequence the exomes of thousands of people with schizophrenia, and the effort is starting to pay off. A collaborative effort led by Ben Neale, Ph.D., a genetics researcher at the Broad Institute of MIT and Harvard and Massachusetts General Hospital, examined data from more than 2,700 people with schizophrenia and found the strongest evidence to date that rare genetic mutations increase the likelihood of developing schizophrenia. The findings, recently published in *Nature Neuroscience*, add to unpublished evidence presented at the most recent meeting of the World Congress of Psychiatric Genetics. Presenting on behalf of the SCHEMA (Schizophrenia Exome Meta-Analysis) consortium, Broad Institute researcher Tarjinder Singh, Ph.D., reported findings from a larger exome sequencing study that found several rare genetic variants with large-effect-size changes.

None of these gene variants will lead to schizophrenia in the way that the MC1R variation leads to red hair — an individual's risk of having schizophrenia is influenced by the combination of many genetic differences with small and large effects, all

working in concert. Nonetheless, these whole-exome sequencing findings are game-changing. Why? One reason is that it is much more straightforward to study the biological mechanisms underlying large-effect-size genetic differences in protein-coding genes — researchers can learn about the effects by introducing single gene mutations in human cell lines or model organisms and then looking for changes in molecular and biological processes.

In addition, the biological pathways affected by large-effect-size variations can suggest novel therapeutic targets. One quick example — two of the genes identified in the research presented at the World Congress of Psychiatric Genetics are known to code for receptors for the neurotransmitter glutamate. Researchers had previously hypothesized that drugs affecting the glutamate system may be useful in the treatment of schizophrenia; these new findings give greater credence and specificity to efforts to develop glutamatergic agents as novel therapeutics.

Although adoption studies have supported the hypothesis that genetic factors contribute to schizophrenia, they have also demonstrated that the disorder most likely arises from a combination of genetic and environmental factors rather than just genes themselves. For example, investigators in one study examined the rates of schizophrenia among 303 adoptees (Tienari et al., 2004). A total of 145 of the adoptees had biological mothers with schizophrenia; these adoptees constituted the high genetic risk group. The other 158 adoptees had mothers with no psychiatric history; these adoptees composed the low genetic risk group. The researchers managed to determine whether the adoptees' families were either healthy or disturbed. The adoptees were considered to be raised in a disturbed family environment if the family exhibited a lot of criticism, conflict, and a lack of problem-solving skills. The findings revealed that adoptees whose mothers had schizophrenia (high genetic risk) *and* who had been raised in a disturbed family environment were much more likely to develop schizophrenia or another psychotic disorder (36.8%) than were adoptees whose biological mothers had schizophrenia but who had been raised in a healthy environment (5.8%), or than adoptees with a low genetic risk who were raised in either a disturbed (5.3%) or healthy (4.8%) environment. Since the adoptees who were at high genetic risk were likely to develop schizophrenia *only* if they were raised in a disturbed home environment, this study supports a biopsychosocial diathesis-stress interpretation of schizophrenia—both genetic vulnerability and environmental stress are necessary for schizophrenia to develop; genes alone do not show the complete picture.

The diathesis-stress model of schizophrenia explains how an environmental trigger, such as severe stress or drug use, can catalyze the onset of a disease in a genetically susceptible person. This model does not mean that without the specific environmental trigger the person would be safe from developing schizophrenia, but it does display the importance of the environment on genetic expression. For example, a young woman in her early to mid-twenties who has a genetic propensity to develop schizophrenia can go through a traumatic relationship, and that experience then catalyzes the initial onset of schizophrenic symptoms known as the prodromal phase.

Neurotransmitters

If we accept that schizophrenia is at least partly genetic in origin, as it seems to be, it makes sense that the next step should be to identify biological abnormalities commonly found in people with the disorder. Perhaps not surprisingly, a number of neurobiological factors have indeed been found to be related to schizophrenia. One such factor that has received considerable attention for many years is the neurotransmitter dopamine. Interest in the role of dopamine in schizophrenia was stimulated by two sets of findings: drugs that increase dopamine levels can produce schizophrenia-like symptoms and medications that block dopamine activity reduce positive symptoms (Howes & Kapur, 2009). These findings were the oldest biologically focused hypothesis about the etiology of schizophrenia that began with research first conducted in the 1950s.

The **dopamine hypothesis** of schizophrenia proposed that an overabundance of dopamine or too many dopamine receptors are responsible for the onset and maintenance of schizophrenia (Snyder, 1976). More recent work in this area suggests that abnormalities in dopamine vary by brain region and thus contribute to symptoms in unique ways. In general, this research has suggested that an overabundance of dopamine in the limbic system may be responsible for some symptoms, such as hallucinations and delusions, whereas low levels of dopamine in the prefrontal cortex might be responsible primarily for the negative symptoms (avolition, alogia, asociality, and anhedonia) (Davis, Kahn, Ko, & Davidson, 1991). In recent years, serotonin has received attention and newer antipsychotic medications used to treat the disorder work by blocking serotonin receptors (Baumeister & Hawkins, 2004). As noted above in the summary by Dr. Gordon, large effect-size variations studies are now also suggesting an important role for the neurotransmitter glutamate.

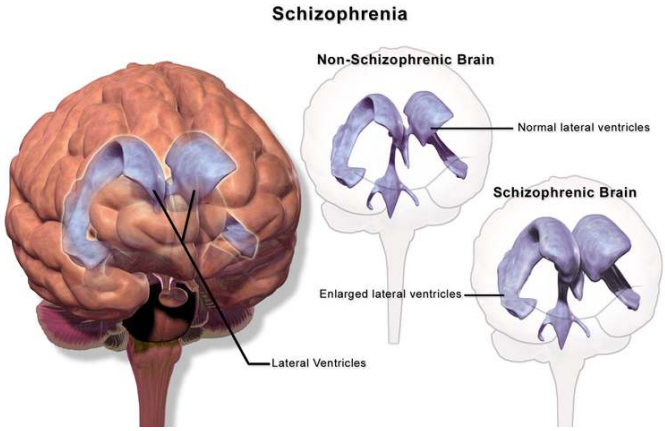


Figure **11.10.2**: Schizophrenia is associated with enlarged ventricles in the brain.

Brain imaging studies reveal that people with schizophrenia have enlarged **ventricles**, the cavities within the brain that contain cerebral spinal fluid (Green, 2001). This finding is important because larger than normal ventricles suggests that various brain regions are reduced in size, thus implying that schizophrenia is associated with a loss of brain tissue. In addition, many people with schizophrenia display a reduction in gray matter (cell bodies of neurons) in the frontal lobes (Lawrie & Abukmeil, 1998), and many show less frontal lobe activity when performing cognitive tasks (Buchsbaum et al., 1990). The frontal lobes are important in a variety of complex cognitive functions, such as planning and executing behavior, attention, speech, movement, and problem-solving. Hence, abnormalities in this region provide merit in explaining why people with schizophrenia experience deficits in these areas. We do not yet understand the mechanisms or processes involved in the apparent atrophy of brain tissue in the brains of persons with schizophrenia or when this process takes place as a person grows and matures.

The Biological Perspective: Neuroimaging

The advent of neuroimaging techniques like structural and functional magnetic resonance imaging and positron emission tomography opened up the ability to try to understand the brain mechanisms of the symptoms of schizophrenia as well as the cognitive impairments found in psychosis. For example, a number of studies have suggested that delusions in psychosis may be associated with problems in salience detection mechanisms supported by the ventral striatum (Jensen & Kapur, 2009; Jensen et al., 2008; Kapur, 2003; Kapur, Mizrahi, & Li, 2005; Murray et al., 2008) and the anterior prefrontal cortex (Corlett et al., 2006; Corlett, Honey, & Fletcher, 2007; Corlett, Murray, et al., 2007a, 2007b). These are regions of the brain that normally increase their activity when something important (a.k.a. *salient*) happens in the environment. If these brain regions misfire, individuals with psychosis may mistakenly attribute importance to irrelevant or unconnected events.

Further, there is good evidence that problems in working memory and cognitive control in schizophrenia are related to problems in the function of a region of the brain called the dorsolateral prefrontal cortex (DLPFC) (Minzenberg, Laird, Thelen, Carter, & Glahn, 2009; Ragland et al., 2009). These problems include changes in how the dorsolateral prefrontal cortex (DLPFC) works when people are doing working-memory or cognitive-control tasks, and problems with how this brain region is connected to other brain regions important for working memory and cognitive control, including the posterior parietal cortex (e.g., Karlsgodt et al., 2008; J. J. Kim et al., 2003; Schlosser et al., 2003), the anterior cingulate (Repovs & Barch, 2012), and temporal cortex (e.g., Fletcher et al., 1995; Meyer-Lindenberg et al., 2001).

In terms of understanding episodic memory problems in schizophrenia, many researchers have focused on medial temporal lobe deficits, with a specific focus on the hippocampus (e.g., Heckers & Konradi, 2010) because there is much data from humans and animals showing that the hippocampus is important for the creation of new memories (Squire, 1992). However, it has become increasingly clear that problems with the dorsolateral prefrontal cortex (DLPFC) also make important contributions to episodic memory deficits in schizophrenia (Ragland et al., 2009), probably because this part of the brain is important for controlling our use of memory.

In addition to problems with regions such as the DLFPC and medial temporal lobes in schizophrenia described above, magnitude resonance neuroimaging studies have also identified changes in cellular architecture, white matter connectivity, and gray matter

volume in a variety of regions that include the prefrontal and temporal cortices (Bora et al., 2011). People with schizophrenia also show reduced overall brain volume. Reductions in brain volume as people get older may be larger in those with schizophrenia than in healthy people (Olabi et al., 2011). Taking antipsychotic medications or using drugs such as marijuana, alcohol, and tobacco may cause some of these structural changes. However, these structural changes are not completely explained by medications or substance use alone. Further, both functional and structural brain changes are seen, again to a milder degree, in the first-degree relatives of people with schizophrenia (Boos, Aleman, Cahn, Pol, & Kahn, 2007; Brans et al., 2008; Fusar-Poli et al., 2007; MacDonald, Thermenos, Barch, & Seidman, 2009), again suggesting that neural changes associated with schizophrenia are related to a genetic risk for this illness.

Environmental Risk Factors

As mentioned previously, some environmental factors are associated with a slight risk of developing schizophrenia in later life, such as concerns during prenatal development, including oxygen deprivation, infection, prenatal maternal stress, and malnutrition. Some studies found that the mother having the flu during the first or second trimesters increases the risk of a child having schizophrenia, but those results have been questioned.[1] Both maternal stress and infection have been demonstrated to alter fetal neurodevelopment through an increase of pro-inflammatory cytokines. There is a slighter higher risk associated with being born in the winter or spring possibly due to vitamin D deficiency or a prenatal viral infection. Other infections during pregnancy or around the time of birth that have been linked to an increased risk include infections by toxoplasma gondii and chlamydia. Viral infections of the brain during childhood are also linked to a risk of schizophrenia during adulthood.

Living in an urban environment during childhood or as an adult has consistently been found to increase the risk of schizophrenia by a factor of two, even after taking into account drug use, ethnic group, and size of social group. A possible link between the urban environment and pollution has been suggested to be the cause of the elevated risk of schizophrenia.

Other risk factors of importance include social isolation, immigration related to social adversity and racial discrimination, family dysfunction, unemployment, and poor housing conditions. Having a father older than 40 years old or parents younger than 20 years old are also associated with schizophrenia. It has been suggested that apart from gene-environment interactions, environment-environment interactions also be taken into account as each environmental risk factor on its own is not enough.

Link to Learning

This PBS News Hour clip highlights some research conducted by Steven McCarroll, an associate professor of genetics at Harvard University, that identified specific genetic markers connected to schizophrenia.

Watch It

Watch this video to learn more about the biological explanations of schizophrenia.



You can view the transcript for “Biological basis of schizophrenia | Behavior | MCAT | Khan Academy” here (opens in new window).

Glossary

dopamine-hypothesis: the oldest and most widely accepted biochemical model of schizophrenia that suggests overactivity of dopamine in the brain; newer studies are now exploring the role and interaction of other neurotransmitter systems

ventricles: hollow spaces in the brain

1. Siegmann A. E. (1976). A classification of sociomedical health indicators: perspectives for health administrators and health planners. *International journal of health services : planning, administration, evaluation*, 6(3), 521–538. https://doi.org/10.2190/MY7U-4BGM-9QFY-N0TN↵

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11.11: Perspectives on Schizophrenia

Learning Objectives

- Describe how various psychological perspectives view and explain schizophrenia

Psychodynamic Perspectives on Psychosis

Early psychoanalytic conceptions of psychosis explained psychotic symptoms as a manifestation of the conscious mind being invaded by the unconscious and by dreams (Federn, 1928/1952). More contemporary approaches underline the importance of early relationship patterns (e.g., Bion, 1962; Winnicott, 1991). Internal representations of experiences with significant others and current relationships are assumed to result in tension and psychotic symptoms are considered to be a constructive way of dealing with this tension (von Haebler & Freyberger, 2013). Psychodynamic therapy focuses on these processes and helps the patient to gain self-awareness and understanding of the influence of the past on present behavior, and it fosters new positive relationship experiences. An empathic, respectful, and supportive attitude allows re-enactment of internalized relational patterns in the therapist–patient interaction (Lempa, Montag, & von Haebler, 2013). Some early theories of psychoanalytic thought argued that psychosis could result from poor parenting behaviors (e.g., the schizophrenogenic mother stereotype) and the concept of double-bind communication, which refers to parental communication that is contradictory (rejecting while demanding affection), have not been supported in later research.[1] Additionally, studies have generally shown that insight-oriented forms of psychotherapy do not typically work well with most persons with schizophrenia because of their difficulty in self-reflection and abstract thinking due to thought disorder.

Humanistic Perspectives on Psychosis

In client-centered or humanistic therapy, unconditional positive regard, accurate empathy, and genuineness are assumed to help a patient to increase the congruence between the real self and the ideal self (Rogers, Gendlin, Kiesler, & Truax, 1967). Rogers and colleagues' concept of actualizing tendency points to an inherent tendency to achieve personal growth and reach one's full potential. In this framework, psychotic symptoms are understood as a distortion of this actualizing tendency. Client-centered therapy focuses on personal experiences whereas relieving specific symptoms is secondary. Thus, no specific therapeutic strategies have been established for psychosis. However, this perspective recommends therapists pay particular attention to understanding the client's perspective, ensuring that the patient is being heard and emphasizing the personal relationship (Gendlin, 1962).[2]

The Cognitive Perspective of Schizophrenia

When we think of the core symptoms of psychotic disorders such as schizophrenia, we think of an individual who may hear voices, see visions, and have false beliefs about reality (i.e., delusions). However, problems in cognitive function are also a critical aspect of psychotic disorders and of schizophrenia in particular. This emphasis on cognition in schizophrenia is in part due to the growing body of research suggesting that cognitive problems in schizophrenia are a major source of disability and loss of functional capacity (Green, 2006; Nuechterlein et al., 2011). The cognitive deficits that are present in schizophrenia are widespread and can include problems with episodic memory (the ability to learn and retrieve new information or episodes in one's life), working memory (the ability to maintain information over a short period of time, such as 30 seconds), and other tasks that require one to control or regulate one's behavior (Barch & Ceaser, 2012; Bora, Yucel, & Pantelis, 2009a; Fioravanti, Carlone, Vitale, Cinti, & Clare, 2005; Forbes, Carrick, McIntosh, & Lawrie, 2009; Mesholam-Gately, Giuliano, Goff, Faraone, & Seidman, 2009). Individuals with schizophrenia also have difficulty with what is referred to as *processing speed* and are frequently slower than healthy individuals on almost all tasks. Importantly, these cognitive deficits are present prior to the onset of the illness (Fusar-Poli et al., 2007) and are also present, albeit in a milder form, in the first-degree relatives of people with schizophrenia (Snitz, Macdonald, & Carter, 2006).

These findings suggest that cognitive impairments in schizophrenia reflect part of the risk for the development of psychosis, rather than only being an outcome of developing psychosis. Further, people with schizophrenia who have more severe cognitive problems also tend to have more severe negative symptoms and more disorganized speech and behavior (Barch, Carter, & Cohen, 2003; Barch et al., 1999; Dominguez Mde, Viechtbauer, Simons, van Os, & Krabbendam, 2009; Ventura, Hellemann, Thames, Koellner, & Nuechterlein, 2009; Ventura, Thames, Wood, Guzik, & Hellemann, 2010). In addition, people with more cognitive problems

have worse functioning in everyday life (Bowie et al., 2008; Bowie, Reichenberg, Patterson, Heaton, & Harvey, 2006; Fett et al., 2011).

The Cognitive-Behavioral Perspective

Cognitive-behavioral interventions for psychosis (CBTp) build on the assumption that psychotic symptoms lie on a continuum with normal experiences. They are also informed by research suggesting that psychotic experiences result from normal, though exaggerated, mechanisms of perception and reasoning. This understanding has formed the basis for cognitive models of psychosis. As one of the most influential of these models, Garety, Kuipers, Fowler, Freeman, & Bebbington (2001) postulate that psychotic symptoms develop when stressors overload a person, causing them to have unusual experiences. According to this model, the unusual experience itself is not crucial, but its appraisal—how it is understood or evaluated by the person—is. Most descriptions within the cognitive-behavioral interventions for psychosis (CBTp) framework converge in stressing the importance of building a stable therapeutic relationship through the process of listening and validating, of taking a collaborative approach, and of working with an individual case formulation. The use of cognitive and behavioral interventions for working with psychotic symptoms as well as for changing dysfunctional beliefs and interventions to prevent relapse are also essential elements.[3]

Social Cognition

Some people with schizophrenia also show deficits in what is referred to as social cognition, though it is not clear whether such problems are separate from the cognitive problems described above or the result of them (Hoe, Nakagami, Green, & Brekke, 2012; Kerr & Neale, 1993; van Hooren et al., 2008). This deficit of social cognition includes problems with the recognition of emotional expressions on the faces of other individuals (Kohler, Walker, Martin, Healey, & Moberg, 2010) and problems inferring the intentions of other people (theory of mind) (Bora, Yucel, & Pantelis, 2009b). Individuals with schizophrenia who have more problems with social cognition also tend to have more negative and disorganized symptoms (Ventura, Wood, & Hellemann, 2011) as well as worse community function (Fett et al., 2011).

Diathesis-Stress Model

The **diathesis-stress model** helps to settle the debate of nature versus nurture; it explains how the two have a bidirectional relationship and a dual influence on the development of many mental health illnesses, especially schizophrenia. The **diathesis** refers to the genetic predisposition or risk an individual has of developing a certain disorder. This predisposition comes from the individual's unique genetic makeup as well as the increased risk if a first-degree blood relative such as parent or sibling has been diagnosed with a disorder. The diathesis is the nature component of the model, reflecting the biological vulnerability an individual possesses. An environmental **stressor** can trigger the onset of a disorder, especially in those genetically vulnerable to developing the disorder. If an individual is greatly susceptible to developing a disorder, only a small level of stress is needed to catalyze the onset of the disorder. Extreme trauma or the use of a drug such as cannabis can serve as environmental stressors and aspects of nurture that influence the onset of schizophrenia and related disorders.

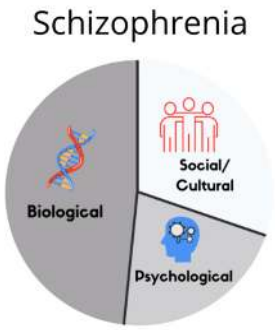


Figure 11.11.1: We know that biological and genetic components play a large role in influencing the development of schizophrenia, although biological factors alone cannot explain why a person may develop the disorder.

Childhood trauma has specifically been shown to be a predictor of adolescent and adult psychosis. Approximately 65% of individuals with psychotic symptoms have experienced childhood trauma (e.g., physical or sexual abuse and physical or emotional neglect). Increased individual vulnerability toward psychosis may interact with traumatic experiences promoting an onset of future

psychotic symptoms, particularly during sensitive developmental periods. Importantly, the relationship between traumatic life events and psychotic symptoms appears to be dose-dependent, in which multiple traumatic life events accumulate, compounding symptom expression and severity. This relationship suggest trauma prevention and early intervention may be an important target for decreasing the incidence of psychotic disorders and ameliorating its effects.

Sociocultural Perspective



Figure **11.11.2**: In some cultures, symptoms of schizophrenia may not be considered abnormal.

There are also a number of environmental factors that are associated with an increased risk of developing schizophrenia. For example, problems during pregnancy such as increased stress, infection, malnutrition, and/or diabetes have been associated with increased risk of schizophrenia. In addition, complications that occur at the time of birth and cause hypoxia (lack of oxygen) are also associated with an increased risk for developing schizophrenia in the child (M. Cannon, Jones, & Murray, 2002; Miller et al., 2011). Children born to older fathers are also at a somewhat increased risk of developing schizophrenia. Further, using cannabis increases risk for developing psychosis, especially if when other risk factors are present (Casadio, Fernandes, Murray, & Di Forti, 2011; Luzi, Morrison, Powell, di Forti, & Murray, 2008). The likelihood of developing schizophrenia is also higher for kids who grow up in urban settings (March et al., 2008) and for some minority ethnic groups (Bourque, van der Ven, & Malla, 2011). Both of these factors may reflect higher social and environmental stress in these settings. Unfortunately, none of these risk factors is specific enough to be particularly useful in a clinical setting, and most people with these risk factors do not develop schizophrenia. However, together they are beginning to give us clues as the neurodevelopmental factors that may lead someone to be at an increased risk for developing this disorder.

Cross-Cultural Perspectives and Cultural Influences

Culture plays a role in the way we view mental health disorders and their corresponding features. There are cultures around the world, such as in Peru, who do not perceive features of schizophrenia like hearing voices (hallucinations) as abnormal. Rather, they may even be seen as special abilities and connections to the spirit realm, where the individual who hears voices could be the community Shaman, or medicine man. These individuals actually help to provide insight and healing to themselves and to others.

In Western societies, the same feature of hearing voices would be considered to be abnormal and a symptom of an underlying disease such as schizophrenia. An individual experiencing these symptoms would not be placed in a position of reverence or admiration, but would most likely be placed in a treatment facility or hospital for further care and treatment to manage and reduce the experienced symptoms. Even in Western society, however, there have been advocates, like Dorothea Dix and Philippe, who emphasized respecting and admiring those with mental disorders.

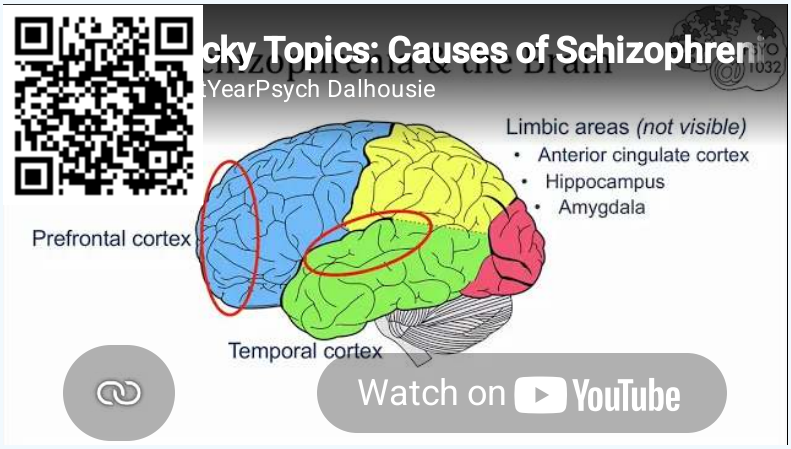
? Cross-Cultural Studies

The International Pilot Study of Schizophrenia revealed some interesting data about how schizophrenia differs across cultures. Among all cultures, paranoid schizophrenia was the most common subtype (40% of persons diagnosed). The content and themes of delusions vary between the background experiences and beliefs of individuals with schizophrenia—religious delusions are more common in Christian societies, while magical religious delusions are more common in rural areas. In Islamic Pakistan, there were lower rates of religious delusions, grandiose delusions, and delusions of guilt, while these were more common in African countries.

Visual hallucinations are more common in African countries and non-European patients. Auditory hallucinations are common everywhere. Negative symptoms are also more common than positive symptoms, though there are differences between countries as to which types of negative symptoms are most distressing.[4]

Watch It

Watch this video (starting at the 3:35 mark) to learn about various explanations for the etiology of schizophrenia.



You can view the transcript for "Tricky Topics: Causes of Schizophrenia" here (opens in new window).

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11.12: Treatment for Schizophrenia and Other Psychotic Disorders

Learning Objectives

- Discuss the use of antipsychotic medications in the treatment of schizophrenia and concerns regarding drug treatments
- Describe the process and effectiveness of psychotherapies used to treat schizophrenia and other psychotic disorders

Treatment of Schizophrenia

The currently available treatments for schizophrenia leave much to be desired, and the search for more effective treatments for both the positive psychotic symptoms of schizophrenia (e.g., hallucinations and delusions) as well as cognitive deficits and negative symptoms is a highly active area of research. Despite this lack, treatment methods have improved drastically in recent years.

Schizophrenia was long considered a lifelong illness with little or no hope of recovery. Following the introduction of antipsychotics more than sixty years ago, treatment prospects were initially modest, with clinicians settling for outcomes such as behavioral control, symptom control, or stability. However, advances in pharmacological treatment and psychosocial interventions have heightened expectations for outcomes. Indeed, there has been a progression of treatment goals from containment to remission through response, and more recently, to recovery. Recovery became a focus of attention when it was recognized that symptom reduction alone was not sufficient, and that functionality and outcomes that are most meaningful to patients and families need to be considered. There is growing acknowledgment that people with schizophrenia do not inevitably experience deterioration over time, and most have the potential to experience considerable symptomatic improvement and achieve a substantial degree of recovery.

A South African study of 98 individuals with schizophrenia studied over two years showed that most improvement occurred within the first six months of treatment with antipsychotic medications, and symptom remission was achieved by 70% of patients. Over half of the patients met the criteria for functional remission and good quality of life, though only 29% met the researchers' full criteria for recovery. Patients who met the recovery criteria had better premorbid adjustment, were less likely to be of mixed ethnicity, and less likely to use substances.[1]

Emergency room (ER) visits and subsequent hospitalization for schizophrenia is common, especially for first psychotic episodes. An analysis of National Hospital Ambulatory Medical Care Survey data indicates that during 2009–2011, an average of 382,000 ER visits related to schizophrenia occurred each year among adults aged 18–64, corresponding to an overall visit rate of 20.1 per 10,000 adults. This same study concluded that of the ER visits related to schizophrenia, 32% resulted in a hospital admission, which is higher than ER visits not related to schizophrenia (10.3%). An additional 16.7% of ER visits related to schizophrenia resulted in a transfer to a psychiatric hospital. This was higher than the percentage of ER visits not related to schizophrenia (0.7%).[2]

Voluntary or involuntary admittance to hospitals may be needed to treat a severe psychotic episode; however, hospital stays are as short as possible. In-patient treatment methods aim to manage psychotic symptoms and ensure that needs, such as safety and nutrition needs, are met. Once symptoms are medications are stable, the goal is to help patients live independently, though in some cases, individuals remain in psychiatric care facilities.

Antipsychotic Medications

The first line of treatment for schizophrenia and other psychotic disorders is the use of antipsychotic medications.

Antipsychotic drug treatment is a key component of the schizophrenia treatment recommendations from the National Institute of Health and Care Excellence, the American Psychiatric Association, and the British Society for Psychopharmacology. The main aim of treatment with antipsychotics is to reduce the positive symptoms of psychosis that include delusions and hallucinations. There is mixed evidence to support a significant impact of antipsychotic use on negative symptoms (such as apathy, lack of emotional affect, and lack of interest in social interactions) or on the cognitive symptoms (memory impairments, confused or illogical speech, and reduced ability to plan and execute tasks). In general, the efficacy of antipsychotic treatment in reducing both positive and negative symptoms appears greater for those with more intense baseline schizophrenia symptoms. Currently, research suggests that there are at least five different types of dopamine receptors (D1 through D5), although they are often categorized as either the D1-like family (D1 and D5) or the D2-like family (D2, D3, and D4). All available antipsychotic medications work relatively the same way: by antagonizing (blocking) D2-dopamine receptors.

There are two primary types of antipsychotic medications, referred to as *typical* (first-generation) and *atypical* (second generation). The fact that typical antipsychotics helped reduce positive symptoms of schizophrenia was discovered serendipitously in the 1960s (Carpenter & Davis, 2012; Lopez-Munoz et al., 2005) and contributed to the dopamine hypothesis for the etiology of schizophrenia. In other words, the reasoning was that if excessive levels of dopamine were a primary cause of psychotic symptoms, then drugs that block dopamine binding at receptors should reduce those symptoms. Over time, as research continued, it has become clear that the etiology of psychotic symptoms is much more complex. For example, although some patients diagnosed with schizophrenia for at least a decade were found to have a 90% blockade of D2 receptors, antipsychotics did not appear to significantly reduce psychotic symptoms. Also, although these drugs may drop dopamine levels significantly within minutes, reduction in symptoms may not occur for days. Other studies have shown that atypical antipsychotics seem to reduce psychotic symptoms about as effectively as the typical antipsychotics, but they are weak antagonists for D2 receptors. Currently, many scientists believe that at least three neurotransmitters are involved in schizophrenia spectrum disorders: dopamine, serotonin, and glutamate. We are still trying to understand the interactions of these systems.

The first-generation (typical) antipsychotic medications are drugs that share the common feature of producing a strong blockade of the D2-type dopamine receptor. Although these drugs can help reduce hallucinations, delusions, and disorganized speech, they do little to improve cognitive deficits or negative symptoms and can be associated with distressing motor side effects that can become permanent over time. The newer atypical antipsychotics have more mixed mechanisms of action in terms of the receptor types that they influence, though most of them also influence D2 receptors, although not as strongly as the typical antipsychotics. These newer antipsychotics are not necessarily more helpful for schizophrenia, but they have fewer motor side effects.

First-generation antipsychotics such as haloperidol and thioridazine may produce **extrapyramidal symptoms (EPS)** side effects such as acute dystonia (involuntary movements including repetitive and twisting movements), akathisia (inability to sit still, often due to strong feelings of restlessness or muscle pain), cogwheel rigidity (a rigid form of muscle movement that occurs in ratchet-type jerking movements), and tardive dyskinesia (involuntary, writhing movements like grimacing, sticking out the tongue, smacking the lips, or jerking neck movements). Once these side effects begin, over time, some can become permanent; they can also interfere with the ability to work or carry out daily functioning. As you might imagine, given the existing stereotypes of schizophrenia spectrum disorders, the addition of these types of motor dysfunctions usually increases the stigma against the person, causing others to avoid them even more and increasing social isolation. As noted, some of these extrapyramidal symptoms (EPS) effects can also be painful. Once extrapyramidal symptoms (EPS) symptoms begin, lowering the antipsychotic dose may reduce the severity of the side effects; anticholinergic medications such as benztropine and biperiden may be added to the treatment regimen to reduce EPS side effects.[3]These side effects can be a major reason why patients may stop taking their medications, leading to relapse as a result.

Second-generation, atypical antipsychotics (SGAs) are usually prescribed in treating schizophrenia, although clozapine is not recommended as a first-line treatment because of the increased risk of agranulocytosis (severe drop in white blood cell counts, potentially leading to infections and death). Second-generation, atypical antipsychotics (SGAs) have fewer extrapyramidal symptoms than typical antipsychotics, but they may have metabolic side effects, such as weight gain, hyperlipidemia (e.g. high cholesterol), and diabetes mellitus. These side effects may cause other medical concerns that contribute to the greater risk for cardiovascular mortality in patients with schizophrenia.[4]

Watch It

This video details the treatment methods and management of schizophrenia.



You can view the transcript for "Schizophrenia treatment | Mental health | NCLEX-RN | Khan Academy" here (opens in new window).

Nonpharmacological Therapies

Goals for the treatment of schizophrenia include identifying symptoms, increasing functioning, and preventing relapse. Typically, pharmacological and non-pharmacological treatments are combined in order to help improve daily functioning and symptom management, each of them targeting their own symptoms and areas of functioning. For example, medications are used to reduce positive and sometimes negative symptoms or to manage side effects while psychotherapeutic approaches aim to improve coping skills, symptom management, daily functioning, socialization, and the ability to work and manage one's own life. Consider the typical age of onset for schizophrenia, for example. It usually manifests in the early years of adulthood as the brain finishing maturing. Thus, the disorder often interferes directly with learning and maintaining vital skills for autonomy and living, including managing finances, establishing relationships and families, living on one's own, finding work and a career, and many more essential skills. Psychotherapeutic approaches seek to teach or re-teach coping and illness management skills and other skills of daily living and include cognitive enhancement therapy, individual, and group therapy.

Even a brief yet major psychotic episode can be highly disruptive to the livelihood and functioning of an individual and his/her family and friends. Psychotherapeutic management would involve medically informing the patient and his or her family about the condition and treatment modalities employed for the particular patient. Along with emphasizing reintegration into the societal milieu, it is essential to focus on managing comorbid disorders or stressors and improving overall coping skills. These therapies can be delivered in both individual and group settings once the person's active symptoms have been stabilized.

In individual treatment, the person is the focus of the treatment and has all the attention and interaction with the therapist; insight-oriented therapies often are not effective if the person has ongoing symptoms since abstract thinking and planning are significantly impaired; some forms of individual therapy may be helpful if a person achieves significant remission. A group setting, although time and focus is divided, can be a beneficial form of therapy as individuals often can find support and comfort in hearing the stories of individuals with similar experiences and form friendships based on healing and a deep understanding.

Other support services for education, employment, and housing are usually offered for those with schizophrenia. For people suffering from severe schizophrenia and discharged from a stay in the hospital, these services are often brought together in an integrated approach to offer support in the community away from the hospital setting. In addition to medicine management, housing, and finances, assistance is given for more routine matters such as help with shopping and using public transportation. This approach is known as assertive community treatment (ACT) and has been shown to achieve positive results in symptoms, social functioning, and quality of life. Another more intense approach is known as intensive care management (ICM). Intensive care management (ICM) is a stage further than assertive community treatment (ACT) and emphasizes support of high intensity in smaller caseloads, (less than twenty). This approach is to provide long-term care in the community. Studies show that intensive care management (ICM) improves many of the relevant outcomes including social functioning.

Cognitive-Behavioral Therapy

In long-term psychoses and schizophrenia, CBT is used to complement medication and is adapted to meet individual needs. Interventions particularly related to these conditions include exploring reality testing (evaluating one's own thinking and beliefs to see how well they match the actual environment), changing delusions and hallucinations, examining factors that precipitate relapse (particularly related to stress, interpersonal conflicts, and treatment resistance), and managing relapses. While several meta-analyses suggested that CBT is effective in schizophrenia, other studies contradict these findings.[5]

Link to Learning

In this video, Jeanie Tse explains how CBT is used to treat schizophrenia.

Cognitive Enhancement Therapy (CET)

The evidence that cognitive deficits also contribute to functional impairment in schizophrenia has led to an increased search for treatments that might enhance cognitive function in schizophrenia. Unfortunately, as of yet, there are no pharmacological treatments that work consistently to improve cognition in schizophrenia, though many new types of drugs are currently under exploration. However, there is a type of psychological intervention, referred to as cognitive remediation, which has shown some evidence of boosting cognitive functioning in schizophrenia.

In particular, a version of this treatment called **cognitive enhancement therapy (CET)** has been shown to improve cognition, functional outcome, social cognition, and protection against gray matter loss (Eack et al., 2009; Eack, Greenwald, Hogarty, & Keshavan, 2010; Eack et al., 2010; Eack, Pogue-Geile, Greenwald, Hogarty, & Keshavan, 2010; Hogarty, Greenwald, & Eack, 2006) in young individuals with schizophrenia. The treatment was designed for persons whose positive and negative symptoms have been stabilized but who still struggle to function well socially and vocationally. It works by having individuals participate in structured group and computer-based exercises, with the goal of improving socialization skills and cognitive functioning (e.g., mental stamina and information processing).[6] The development of new treatments such as cognitive enhancement therapy (CET) provides some hope that we will be able to develop new and better approaches to improving the lives of individuals with this serious mental health condition and potentially even prevent it someday.

Watch It

In this video, Juno mentions a little bit about his experiences with schizophrenia and why he chooses not to take medications.



You can view the transcript for "Hearing voices and hallucinations | Juno's Story" here (opens in new window).

Third-wave-approaches are new developments in CBT that emphasize the importance of acceptance, mindfulness, and emotions, the therapeutic relationship, values, goals, and meta-cognition (Hayes & Hofmann, 2017). In psychosis, adaptations of mindfulness-based therapy, acceptance and commitment therapy (ACT) and compassion-focused therapy (CFT) have been studied most. In order to ease distress and achieve acceptance as well as to support the regaining of control, mindfulness-based interventions for psychosis guide patients to notice sensations and their own emotional and cognitive reactions to them with

awareness (Chadwick, 2014). In meditation-based practices, patients learn to observe their thoughts, feelings, and symptoms in an accepting and non-judgmental way. Mindfulness interventions for psychosis have been implemented as single treatments (e.g. Chadwick, 2014) or combined with CBT (e.g. Wright et al., 2014).

In acceptance and commitment therapy (ACT) (Hayes, Strosahl, & Wilson, 1999), experiential avoidance (avoiding one's feelings or avoiding taking productive actions) and cognitive fusion (having behavior driven by one's thoughts without questioning the validity of one's assumptions or beliefs) are suggested to be the core processes of suffering. In order to increase psychological flexibility and reduce distress associated with psychotic symptoms, patients are guided to develop a balance between committed value-guided action when solving actual problems and acceptance when control of thoughts and feelings is limited (e.g., in the case of hallucinations). Acceptance and commitment therapy (ACT) has been adapted for the treatment of psychosis (O'Donoghue, Morris, Oliver, Johns, & Hayes, 2018; combined with CBT, Wright et al., 2014).

Compassion-focused therapy (CFT) (Gilbert & Procter, 2006) encourages patients to be more compassionate towards themselves and others while reducing shame and self-criticism. Compassionate mind training includes appreciation and imagery exercises as well as aspects of mindfulness and aids the patient to experience different aspects of compassion in order to promote mental wellbeing. Compassion-focused therapy (CFT) has also been adapted for the treatment of psychosis (Brähler, Harper, & Gilbert, 2013).

Metacognitive training (MCT) (Moritz & Woodward, 2007) was designed to address positive symptoms in patients with schizophrenia. As cognitive biases have been related to positive symptoms (e.g., jumping to conclusions or externalizing attributional bias—assuming the motivations of others, see Garety & Freeman, 1999), metacognitive training (MCT) aims to extend the patient's knowledge of cognitive biases and to provide corrective experiences. Implementing a wide range of examples and exercises, patients participating in metacognitive training (MCT) group training are encouraged to identify and gain insight into these cognitive biases and reduce conviction in delusional ideas. MCT is mainly administered in group format.

Social skills training (SST) builds on the observation that patients with psychotic disorders tend to show impaired social skills. Social skills training (SST) involve therapist modeling and instructing socially confident behavior in specific situations combined with role plays. Patients receive supportive feedback from the therapist and video feedback can also be used. During the end of the training that usually takes place in group format, patients are encouraged to practice the newly learned skills in daily life.[7]

Glossary

cognitive enhancement therapy (CET): therapy shown to improve cognition, functional outcome, and social cognition and to protect against gray matter loss

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11.13: Case Studies- Schizophrenia Spectrum Disorders

Learning Objectives

- Identify schizophrenia and psychotic disorders in case studies

Case Study: Bryant

Thirty-five-year-old Bryant was admitted to the hospital because of ritualistic behaviors, depression, and distrust. At the time of admission, prominent ritualistic behaviors and depression misled clinicians to diagnose Bryant with obsessive-compulsive disorder (OCD). Shortly after, psychotic symptoms such as disorganized thoughts and delusion of control were noticeable. He told the doctors he has not been receiving any treatment, was not on any substance or medication, and has been experiencing these symptoms for about two weeks. Throughout the course of his treatment, the doctors noticed that he developed a catatonic stupor and a respiratory infection, which was identified by respiratory symptoms, blood tests, and a chest X-ray. To treat the psychotic symptoms, catatonic stupor, and respiratory infection, risperidone, MECT, and ceftriaxone (antibiotic) were administered, and these therapies proved to be dramatically effective.[1]

Case Study: Shanta

Shanta, a 28-year-old female with no prior psychiatric hospitalizations, was sent to the local emergency room after her parents called 911; they were concerned that their daughter had become uncharacteristically irritable and paranoid. The family observed that she had stopped interacting with them and had been spending long periods of time alone in her bedroom. For over a month, she had not attended school at the local community college. Her parents finally made the decision to call the police when she started to threaten them with a knife, and the police took her to the local emergency room for a crisis evaluation.

Following the administration of the medication, she tried to escape from the emergency room, contending that the hospital staff was planning to kill her. She eventually slept and when she awoke, she told the crisis worker that she had been diagnosed with attention-deficit/hyperactive disorder (ADHD) a month ago. At the time of this ADHD diagnosis, she was started on 30 mg of a stimulant to be taken every morning in order to help her focus and become less stressed over the possibility of poor school performance.

After two weeks, the provider increased her dosage to 60 mg every morning and also started her on dextroamphetamine sulfate tablets (10 mg) that she took daily in the afternoon in order to improve her concentration and ability to study. Shanta claimed that she might have taken up to three dextroamphetamine sulfate tablets over the past three days because she was worried about falling asleep and being unable to adequately prepare for an examination.

Prior to the ADHD diagnosis, the patient had no known psychiatric or substance abuse history. The urine toxicology screen taken upon admission to the emergency department was positive only for amphetamines. There was no family history of psychotic or mood disorders, and she didn't exhibit any depressive, manic, or hypomanic symptoms.

The stimulant medications were discontinued by the hospital upon admission to the emergency department and the patient was treated with an atypical antipsychotic. She tolerated the medications well, started psychotherapy sessions, and was released five days later. On the day of discharge, there were no delusions or hallucinations reported. She was referred to the local mental health center for aftercare follow-up with a psychiatrist.[2]

Watch It

Another powerful case study example is that of Elyn R. Saks, the associate dean and Orrin B. Evans professor of law, psychology, and psychiatry and the behavioral sciences at the University of Southern California Gould Law School.

Saks began experiencing symptoms of mental illness at eight years old, but she had her first full-blown episode when studying as a Marshall scholar at Oxford University. Another breakdown happened while Saks was a student at Yale Law School, after which she "ended up forcibly restrained and forced to take anti-psychotic medication." Her scholarly efforts thus include taking a careful look at the destructive impact force and coercion can have on the lives of people with psychiatric illnesses,

whether during treatment or perhaps in interactions with police; the Saks Institute, for example, co-hosted a conference examining the urgent problem of how to address excessive use of force in encounters between law enforcement and individuals with mental health challenges.

Saks lives with schizophrenia and has written and spoken about her experiences. She says, “There’s a tremendous need to implode the myths of mental illness, to put a face on it, to show people that a diagnosis does not have to lead to a painful and oblique life.”

In recent years, researchers have begun talking about mental health care in the same way addiction specialists speak of recovery—the lifelong journey of self-treatment and discipline that guides substance abuse programs. The idea remains controversial: managing a severe mental illness is more complicated than simply avoiding certain behaviors. Approaches include “medication (usually), therapy (often), a measure of good luck (always)—and, most of all, the inner strength to manage one’s demons, if not banish them. That strength can come from any number of places…love, forgiveness, faith in God, a lifelong friendship.” Saks says, “We who struggle with these disorders can lead full, happy, productive lives, if we have the right resources.”



You can view the transcript for “A tale of mental illness | Elyn Saks” here (opens in new window).

1. Bai, Y., Yang, X., Zeng, Z., & Yang, H. (2018). A case report of schizoaffective disorder with ritualistic behaviors and catatonic stupor: successful treatment by risperidone and modified electroconvulsive therapy. *BMC psychiatry*, 18(1), 67. https://doi.org/10.1186/s12888-018-1655-5↵
2. Henning A, Kurtom M, Espiridion E D (February 23, 2019) A Case Study of Acute Stimulant-induced Psychosis. Cureus 11(2): e4126. doi:10.7759/cureus.4126 ↵

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11.14: Putting It Together- Schizophrenia Spectrum Disorders



Overall, schizophrenia and other psychotic disorders are some of the most difficult to understand and to treat. With common features of psychosis, specifically hallucinations, delusions, and disorganized speech, individuals with schizophrenia experience a disconnect from the external world around them. It is challenging for loved ones and caretakers as well, as it is difficult to put yourself in the shoes of an individual experiencing psychosis to truly understand how their world is experienced. Continued scientific research studies on the risk factors, treatment procedures, and biological and environmental influences will help with diagnosis and treatment in the future.

The good news is that as research into the field increases our understanding of schizophrenia and other psychotic disorders, there is a greater effort to understand and manage the illnesses in a way that allows those with the illness to live a full and robust life, as in the case of Elyn Saks. At the beginning of the module, we met Lauren, who advocates in her YouTube channel that it is possible to live well with schizophrenia. Visit her channel and watch some more videos to learn more about how she manages living with her diagnosis of schizoaffective disorder.

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CHAPTER OVERVIEW

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12.1: Why It Matters- Personality Disorders

Why learn about personality disorders?

When we observe people around us, one of the first things that strikes us is how different people are from one another. Some people are very talkative while others are very quiet. Some are active whereas others are couch potatoes. Some worry a lot, others almost never seem anxious. Each time we use one of these words, words like *talkative*, *quiet*, *active*, or *anxious*, to describe those around us, we are talking about a person's personality—the characteristic ways that people differ from one another.



Everybody has their own unique personality; that is, their characteristic manner of thinking, feeling, behaving, and relating to others (John, Robins, & Pervin, 2008). Some people are typically introverted, quiet, and withdrawn, whereas others are more extraverted, active, and outgoing. Some individuals are invariably conscientious, dutiful, and efficient; whereas others might be characteristically undependable and negligent. Some individuals are consistently anxious, self-conscious, and apprehensive whereas others are routinely relaxed, self-assured, and unconcerned. Personality traits refer to these characteristic, routine ways of thinking, feeling, and relating to others. There are signs or indicators of these traits in childhood, but they become particularly evident when the person is an adult. Personality traits are integral to each person's sense of self because they involve what people value; how they think and feel about things; what they like to do; and, basically, what they are like most every day throughout much of their lives.

There are three criteria that characterize personality traits: (1) consistency, (2) stability, and (3) individual differences.

1. To have a personality trait, individuals must be somewhat consistent across situations in their behaviors related to the trait. For example, if they are talkative at home, they tend also to be talkative at work.
2. Individuals with a trait are also somewhat stable over time in behaviors related to the trait. If they are talkative, for example, at age 30, they will also tend to be talkative at age 40.
3. People differ from one another on behaviors related to the trait. Using speech is not a personality trait and neither is walking on two feet—virtually all individuals do these activities, and there are almost no individual differences. But people differ on how frequently they talk and how active they are, and thus personality traits such as talkativeness and activity level do exist.

When personality traits result in significant distress, social impairment, and/or occupational impairment, they are considered to be a personality disorder (American Psychiatric Association, 2013). The DSM provides a common language and standard criteria for the classification and diagnosis of mental disorders. DSM-5 includes 10 personality disorders grouped into Cluster A, B, or C: antisocial, avoidant, borderline, dependent, histrionic, narcissistic, obsessive-compulsive, paranoid, schizoid, and schizotypal.

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12.2: Introduction to Cluster A Personality Disorders

What you'll learn to do: describe personality disorder clusters and compare characteristics between Cluster A personality disorders



Individuals with personality disorders exhibit a personality style that is inflexible, causes distress and impairment, and creates problems for themselves and others. The DSM-5 recognizes 10 personality disorders, organized into three clusters.

Cluster A is described as the *odd/eccentric cluster* and consists of paranoid personality disorder, schizoid personality disorder, and schizotypal personality disorder. The common feature of these three disorders is social awkwardness and social withdrawal (APA, 2013).

Cluster B is typically described as the *dramatic*, *emotional*, *or erratic cluster* and consists of antisocial personality disorder, borderline personality disorder, histrionic personality disorder, and narcissistic personality disorder. Individuals with these personality disorders often experience problems with impulse control and emotional regulation (APA, 2013).

Cluster C is characterized as the *anxious/fearful cluster* and consists of avoidant personality disorder, dependent personality disorder, and obsessive-compulsive personality disorder. As you read through the descriptions of these disorders, you will see an overlap with symptoms of anxiety and depressive disorders.

In this section, you'll learn about Cluster A, the odd or eccentric types of personality disorders: paranoid, schizoid, and schizotypal personality disorders.

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12.3: Personality Disorders

Learning Objectives

- Define personality disorders and distinguish between the three clusters of personality disordersObjective 1

The term *personality* refers loosely to one's stable, consistent, and distinctive way of thinking about, feeling, acting, and relating to the world. People with **personality disorders** exhibit a personality style that differs markedly from the expectations of their culture, is pervasive and inflexible, begins in adolescence or early adulthood, and causes distress or impairment (APA, 2013). Generally, individuals with these disorders exhibit enduring personality styles that are extremely troubling and often create problems for them and those with whom they come into contact. Individual maladaptive personality styles frequently bring them into conflict with others, disrupt their ability to develop and maintain social relationships, and prevent them from accomplishing realistic life goals.

Understanding Personality

There are literally hundreds of different personality traits. All of these traits can be organized into the broad dimensions referred to as the Five-Factor Model (John, Naumann, & Soto, 2008). These five broad domains are inclusive; there does not appear to be any traits of personality that lie outside of the Five-Factor Model. This model even applies to traits that you may use to describe yourself. Table 1 provides illustrative traits for both poles of the five domains of this model of personality (you can remember them

using the acronym *OCEAN*). A number of the traits that you see in this table may describe you. If you can think of some other traits that describe yourself, you should be able to place them somewhere in this table.

Table 1. Personality traits in the five-factor model.

Openness: dreamer, unrealistic, imaginative, aberrant, aesthetic, self-aware, eccentric, strange, odd, peculiar, creative, permissive, broad-minded	OR	**Closedness:** practical, concrete, uninvolved, no aesthetic interest, constricted, unaware, alexyhymic, routine, predictable, habitual, stubborn, pragmatic, rigid, traditional, inflexible, dogmatic
Conscientiousness: perfectionistic, efficient, ordered, methodical, organized, rigid, reliable, dependable, workaholic, ambitious, dogged, devoted, cautious, ruminative, reflective	OR	**Disinhibition:** lax, negligent, haphazard, disorganized, sloppy, casual, undependable, unethical, aimless, desultory, hedonistic, negligent, hasty, careless, rash
Extraversion: cordial, affectionate, attached, sociable, outgoing, dominant, forceful, vigorous, energetic, active, reckless, daring, high-spirited, excitement-seeking	OR	**Introversion:** cold, aloof, indifferent, withdrawn, isolated, unassuming, quiet, resigned, passive, lethargic, cautious, monotonous, dull, placid, anhedonic
Agreeableness: gullible, naïve, trusting, confiding, honest, sacrificial, giving, docile, cooperative, meek, self-effacing, humble, soft, empathetic	OR	**Antagonism:** skeptical, cynical, suspicious, paranoid, cunning, manipulative, deceptive, stingy, selfish, greedy, exploitative, oppositional, combative, aggressive,

		confident, boastful, arrogant, tough, callous, ruthless
Neuroticism (emotional instability): fearful, apprehensive, angry, bitter, pessimistic, glum, timid, embarrassed, tempted, urgency, helpless, fragile	OR	**Emotional Stability:** relaxed, unconcerned, cool, even-tempered, optimistic, self-assured, glib, shameless, controlled, restrained, clear-thinking, fearless, unflappable

Classifying Personality Disorders

The DSM-5 defines personality disorders as "an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment." The consideration of cultural context is perhaps the most significant change in this definition from earlier editions. The DSM-5 suggests that the personality disorders can be grouped into three clusters, plus a "not otherwise specified" category, for a total of 11 specific diagnoses, the authors caution that the identified clusters have not been consistently validated and that individuals may present combinations of personality disorders from different clusters. **Cluster A**, the odd or eccentric types, are paranoid, schizoid, and schizotypal personality disorders. **Cluster B**, the dramatic, emotional, or erratic types, are antisocial, borderline, histrionic, and narcissistic personality disorders. And finally, **Cluster C**, the anxious or fearful types, are the avoidant, dependent, and obsessive-compulsive personality disorders.

The table provides a description of each of the DSM-5 personality disorders:

Table 1. DSM-5 Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Paranoid	harbors a pervasive and unjustifiable suspiciousness and mistrust of others; reluctant to confide in or become close to others; reads hidden demeaning or threatening meaning into benign remarks or events; takes offense easily and bears grudges; not due to schizophrenia or other psychotic disorders	A
Schizoid	lacks interest and desire to form relationships with others; aloof and shows emotional coldness and detachment; indifferent to approval or criticism of others; lacks close friends or confidants; not due to schizophrenia or other psychotic disorders, not an autism spectrum disorder	A
Schizotypal	exhibits eccentricities in thought, perception, emotion, speech, and behavior; shows suspiciousness or paranoia; has unusual perceptual experiences; speech is often idiosyncratic; displays inappropriate emotions; lacks friends or confidants; not due to schizophrenia or other psychotic disorder, or to autism spectrum disorder	A
Antisocial	continuously violates the rights of others; history of antisocial tendencies prior to age 15; often lies, fights, and has problems with the law; impulsive and fails to think ahead; can be deceitful and manipulative in order to gain profit or pleasure; irresponsible and often fails to hold down a job or pay financial debts; lacks feelings for others and remorse over misdeeds	B
Histrionic	excessively overdramatic, emotional, and theatrical; feels uncomfortable when not the center of others' attention; behavior is often inappropriately seductive or provocative; speech is highly emotional but often vague and diffuse; emotions are shallow and often shift rapidly; may alienate friends with demands for constant attention	B
Narcissistic	overinflated and unjustified sense of self-importance and preoccupied with fantasies of success; believes self is entitled to special treatment from others; shows arrogant attitudes and behaviors; takes advantage of others; lacks empathy	B

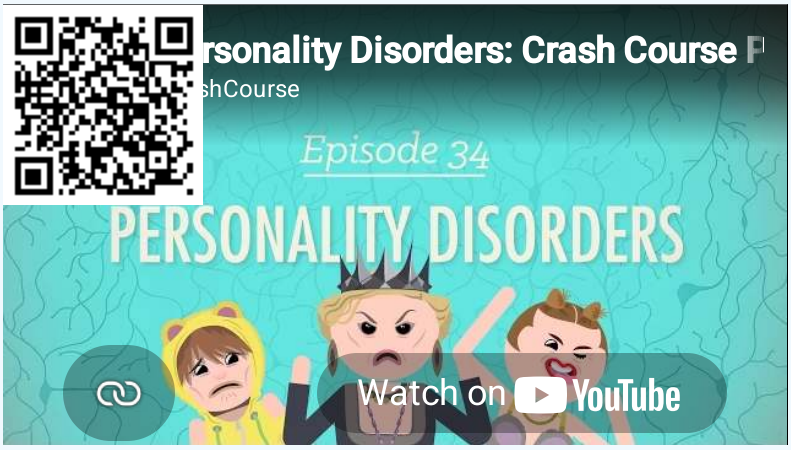
DSM-5 Personality Disorder	Description	Cluster
Borderline	unstable in self-image, mood, and behavior; cannot tolerate being alone and experiences chronic feelings of emptiness; unstable and intense relationships with others; behavior is impulsive, unpredictable, and sometimes self-damaging; shows inappropriate and intense anger; makes suicidal gestures	B
Avoidant	socially inhibited and oversensitive to negative evaluation; avoids occupations that involve interpersonal contact because of fears of criticism or rejection; avoids relationships with others unless guaranteed to be accepted unconditionally; feels inadequate and views self as socially inept and unappealing; unwilling to take risks or engage in new activities if they may prove embarrassing	C
Dependent	allows others to take over and run his or her life; is submissive, clingy, and fears separation; cannot make decisions without advice and reassurance from others; lacks self-confidence; cannot do things on his or her own; feels uncomfortable or helpless when alone	C
Obsessive-Compulsive	pervasive need for perfectionism that interferes with the ability to complete tasks; preoccupied with details, rules, order, and schedules; excessively devoted to work at the expense of leisure and friendships; rigid, inflexible, and stubborn; insists things be done his or her way; miserly with money	C

This list of 10 though does not fully cover all the different ways in which a personality can be maladaptive. DSM-5 also includes a "wastebasket" diagnosis of other specified personality disorder (OSPD) and unspecified personality disorder (UPD). This diagnosis is used when a clinician believes that a patient has a personality disorder but the traits that constitute this disorder are not well covered by one of the 10 existing diagnoses. OSPD and UPD, or as they used to be referred to in previous editions *PDNOS* (personality disorder not otherwise specified), are often one of the most frequently used diagnoses in clinical practice, suggesting that the current list of 10 is not adequately comprehensive (Widiger & Trull, 2007).

Slightly over 9% of the U.S. population suffers from a personality disorder, with avoidant and schizoid personality disorders the most frequent (Lezenweger, Lane, Loranger, & Kessler, 2007). Two of these personality disorders, borderline personality disorder and antisocial personality disorder, are regarded by many as especially problematic.

Watch It

Watch the following CrashCourse psychology video for an overview of personality disorders:



You can view the transcript for "Personality Disorders: Crash Course Psychology #34" here (opens in new window).

Description

Each of the 10 DSM-5 (and DSM-IV-TR) personality disorders is a constellation of maladaptive personality traits, rather than just one particular personality trait (Lynam & Widiger, 2001). In this regard, personality disorders are *syndromes*. For example, avoidant personality disorder is a pervasive pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation (APA, 2013), which is a combination of traits from introversion (e.g., socially withdrawn, passive, and cautious) and neuroticism (e.g., self-consciousness, apprehensiveness, anxiousness, and worrisome). Dependent personality disorder includes submissiveness, clinging behavior, and fears of separation (APA, 2013), for the most part a combination of traits of neuroticism (anxious, uncertain, pessimistic, and helpless) and maladaptive agreeableness (e.g., gullible, guileless, meek, subservient, and self-effacing). Antisocial personality disorder is, for the most part, a combination of traits from antagonism (e.g., dishonest, manipulative, exploitative, callous, and merciless) and low conscientiousness (e.g., irresponsible, immoral, lax, hedonistic, and rash). See the 1967 movie *Bonnie and Clyde*, starring Warren Beatty, for a nice portrayal of someone with antisocial personality disorder.



Some of the DSM-5 personality disorders are confined largely to traits within one of the basic domains of personality. For example, obsessive-compulsive personality disorder is largely a disorder of maladaptive conscientiousness, including such traits as workaholism, perfectionism, punctilious, ruminative, and dogged; schizoid is confined largely to traits of introversion (e.g., withdrawn, cold, isolated, placid, and anhedonic); borderline personality disorder is largely a disorder of neuroticism, including such traits as emotionally unstable, vulnerable, overwhelmed, rageful, depressive, and self-destructive (watch the 1987 movie *Fatal Attraction*, starring Glenn Close, for a nice portrayal of this personality disorder); and histrionic personality disorder is largely a disorder of maladaptive extraversion, including such traits as attention-seeking, seductiveness, melodramatic emotionality, and strong attachment needs (see the 1951 film adaptation of Tennessee William's play *Streetcar Named Desire*, starring Vivian Leigh, for a nice portrayal of this personality disorder).

It should be noted though that a complete description of each DSM-5 personality disorder would typically include at least some traits from other domains. For example, antisocial personality disorder (or psychopathy) also includes some traits from low neuroticism (e.g., fearlessness and glib charm) and extraversion (e.g., excitement-seeking and assertiveness); borderline includes some traits from antagonism (e.g., manipulative and oppositional) and low conscientiousness (e.g., rash); and histrionic includes

some traits from antagonism (e.g., vanity) and low conscientiousness (e.g., impressionistic). Narcissistic personality disorder includes traits from neuroticism (e.g., reactive anger, reactive shame, and need for admiration), extraversion (e.g., exhibitionism and authoritativeness), antagonism (e.g., arrogance, entitlement, and lack of empathy), and conscientiousness (e.g., acclaim-seeking). Schizotypal personality disorder includes traits from neuroticism (e.g., social anxiousness and social discomfort), introversion (e.g., social withdrawal), unconventionality (e.g., odd, eccentric, peculiar, and aberrant ideas), and antagonism (e.g., suspiciousness).

The APA currently conceptualizes personality disorders as qualitatively distinct conditions distinct from each other and from normal personality functioning. However, included within an appendix to DSM-5 is an alternative view that personality disorders are simply extreme and/or maladaptive variants of normal personality traits, as suggested herein. Nevertheless, many leading personality disorder researchers do not hold this view (e.g., Gunderson, 2010; Hopwood, 2011; Shedler et al., 2010). Researchers suggest that there is something qualitatively unique about persons suffering from a personality disorder, usually understood as a form of pathology in sense of self and interpersonal relatedness that is considered to be distinct from personality traits (APA, 2012; Skodol, 2012). For example, it has been suggested that antisocial personality disorder includes impairments in identity (e.g., egocentrism), self-direction, empathy, and capacity for intimacy, which are said to be different from such traits as arrogance, impulsivity, and callousness (APA, 2012).

Validity of Personality Disorders

In future revisions of the DSM, some of the personality disorders included in DSM-5 and DSM-4-TR will no longer be included. In fact, for DSM-5 it was originally proposed that four be deleted. The personality disorders that were slated for deletion were histrionic, schizoid, paranoid, and dependent (APA, 2012). The rationale for the proposed deletions was in large part because they are said to have less empirical support than the diagnoses that were at the time being retained (Skodol, 2012). There is agreement within the field with regard to the empirical support for the borderline, antisocial, and schizotypal personality disorders (Mullins-Sweat, Bernstein, & Widiger, 2012; Skodol, 2012). However, there is a difference of opinion with respect to the empirical support for the dependent personality disorder (Bornstein, 2012; Livesley, 2011; Miller, Widiger, & Campbell, 2010; Mullins-Sweat et al., 2012).

Glossary

antisocial personality disorder: a pervasive pattern of disregard and violation of the rights of others; behaviors may be aggressive or destructive and may involve breaking laws or rules, deceit, or theft

avoidant personality disorder: pervasive pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation

borderline personality disorder: a pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity

dependent personality disorder: a pervasive and excessive need to be taken care of leading to submissive and clinging behavior and fears of separation

five-factor model: five broad domains or dimensions used to describe human personality

histrionic personality disorder: a pervasive pattern of excessive emotionality and attention-seeking

narcissistic personality disorder: a pervasive pattern of grandiosity (in fantasy or behavior), need for admiration, and lack of empathy

paranoid personality disorder: a pervasive distrust and suspiciousness of others such that their motives are interpreted as malevolent

personality: characteristic, routine ways of thinking, feeling, and relating to others

personality disorders: when personality traits result in significant distress, social impairment, and/or occupational impairment

schizoid personality disorder: a pervasive pattern of detachment from social relationships and a restricted range of expression of emotions in interpersonal settings

schizotypal personality disorder: a pervasive pattern of social and interpersonal deficits marked by acute discomfort with, and reduced capacity for, close relationships as well as perceptual distortions and eccentricities of behavior

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12.4: Paranoid Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of paranoid personality disorder

Cluster A Disorders

Paranoid personality disorder (PPD) is a Cluster A personality disorder. Paranoid personality disorder is a clinically well-recognized disorder that has not been the object of a great deal of investigation. Although noted in the writings of psychiatrists since the late 1800s, the condition was first called *paranoid personality* by Kraepelin in 1921 (Akhtar, 1990).

The hallmark criteria regarding paranoid personality disorder (PPD) are distrust and suspicion of others such that others are seen as purposefully attempting to harm one in some way without any evidence to suggest this is the case. Individuals with paranoid personality disorder also may be very critical of others, argumentative, and rigid in beliefs, again stemming from harboring unwarranted suspicions about people around them. This behavior often leads to problems with relationships, both personal and in the workplace.

Table 1. DSM-5 Personality Disorders

Type of personality disorder	Description	Cluster
Paranoid	Guarded, defensive, distrustful, and suspicious. Hypervigilant to the motives of others to undermine or do harm. Always seeking confirmatory evidence of hidden schemes. Feel righteous, but persecuted. Generally difficult to work with and are very hard to form relationships with.	A
Schizoid	Apathetic, indifferent, remote, solitary, distant, humorless, contempt, and odd fantasies. Neither desire nor need human attachments. Withdrawn from relationships and prefer to be alone. Little interest in others, often seen as a loner. An uncommon condition in which people avoid social activities and consistently shy away from interaction with others. It affects more males than females. May appear somewhat dull or humorless.	A
Schizotypal	Eccentric, self-estranged, bizarre, and . Exhibit peculiar mannerisms and behaviors. Think they can read thoughts of others. Preoccupied with odd daydreams and beliefs. Blur line between reality and fantasy. People with schizotypal personality disorder are often described as odd or eccentric and usually have few, if any, close relationships.	A

Diagnosing Paranoid Personality Disorder

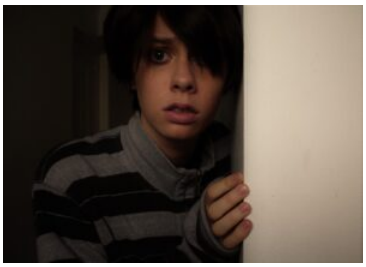


Figure 12.4.1: A person with paranoid personality disorder is suspicious and mistrustful of others but does not experience delusions or hallucinations consistent with schizophrenia.

Paranoid personality disorder (PPD) is characterized by a general suspicion and distrust of others that presents with at least four or more of the following:

- suspicion that others are exploiting or harming them
- a preoccupation with doubting the loyalty of friends
- a reluctance to share information with others for fear it will be used against them
- views harmless comments or events as threatening or demeaning
- holds grudges
- quick to defend or attack perceived threats to their reputation or character
- has recurrent suspicions about the infidelity of a partner[1]

Paranoid personality disorder must be diagnosed to the exclusion of schizophrenia, or any other psychotic disorder including psychosis in the context of a mood disorder.

Comorbidity

Individuals with paranoid personality disorder appear to have an increased likelihood of developing depression, agoraphobia, obsessive-compulsive disorder, and alcohol or substance abuse or dependence. With regard to comorbid personality disorders, there is some variation in the literature. Generally though, it has been suggested that in clinically based samples, over 75% of patients who met paranoid personality disorder criteria also met criteria for other personality disorders: the most common were found to be schizotypal and narcissistic.

One area of research is the possible relationship of post-traumatic stress disorder (PTSD) with personality disorders, with the strongest association with paranoid personality disorder, specifically paranoid personality disorder.[2] This suggests a possible link between trauma during early events in life and subsequent paranoid behavior and mistrust.

Another area that has received some attention is the relationship of violence to paranoid personality disorder. Paranoid cognitive personality style was found to increase the risk of violence in subjects with personality disorders, particularly schizophrenia spectrum disorders (Nestor, 2002). Estimates of the prevalence of paranoid personality disorder range from 2.3% to 4.4% in the United States.[3]

Etiology

There is no known cause of PPD, but researchers believe it is caused by a combination of biological, psychological, and social factors. Genetics play a role in contributing to paranoid traits and there is also a possible genetic link between PPD and schizophrenia. Using data from the Roscommon family study, an epidemiologic study conducted in Ireland, it was discovered that biological relatives of those with schizophrenia had a significantly higher amount of paranoid personality disorder compared with relatives of controls (Kendler et al. 1993).



Figure 12.4.2: Some theories argue that PPD stems from negative parental modeling.

A large, long-term Norwegian twin study found paranoid personality disorder to be modestly heritable and to share a portion of its genetic and environmental risk factors with the other Cluster A personality disorders, schizoid and schizotypal.[4]

Psychosocial theories argue that the disorder stems from early trauma or childhood abuse, possibly coming from the projection of negative internal feelings or parental modeling. Cognitive theorists believe the disorder to be a result of an underlying belief that other people are unfriendly in combination with a lack of self-awareness.

As with other disorders, cultural factors must be taken into account in diagnosing this disorder. There are some groups that might, for reasons of maltreatment, language barriers, and unfamiliarity to this society, display what could be labeled paranoid traits, including minority groups, immigrants, and refugees. In an epidemiologic study recently completed on personality disorders, minorities including Blacks, Hispanics, and Native Americans were at greater risk for having paranoid personality disorder than whites (Grant et al. 2004). Also, according to the same study, paranoid personality disorder was more common among younger people (ages 18–29), those with lower incomes, and those who were divorced or never married. Some of these findings are not surprising, taking into account the nature of paranoid personality disorder. However, this does bring up the question of which came first: are some paranoid traits the result of maltreatment by others due to socioeconomic status (SES), race, etc., or does the disorder contribute to, for example, inability to succeed professionally or remain in a relationship? There appears to be a combination of both, which can contribute to complications in diagnosing the disorder.

Course and Prognosis

PPD can be noted first in childhood; symptoms observed include solitariness, social anxiety, and odd thoughts and language. Kids who are bullied, socially unaccepted, or even abused, may be more prone to PPD. There is not a lot of data regarding the course and prognosis of the disorder. This is likely due to the fact that as it is a personality disorder, it tends to be stable over adult life and although it can cause interpersonal problems, does not often require treatment. It has been observed that the course of the disorder rarely worsens or goes into remission (Akhtar, 1990).

Treatment

There is no specific treatment or medication for PPD. When existing in conjunction with other personality disorders, i.e., borderline personality disorder, treatment may be sought, but that is primarily due to symptoms experienced in other personality disorders. There is some data on the effectiveness of day treatments for patients with personality disorders in general (Karterud et al. 2003). Treatment results, although effective for some personality disorders (i.e., borderline), were the poorest for those with paranoid, schizoid, and schizotypal personality disorders.

? Case Study: Paranoid Personality Disorder

A 36-year-old divorced worker, Nathan, developed severe depression after he was fired from his job and subsequently had severe alcohol problems. He presented himself to a general practitioner with somatic complaints, anxiety, compulsively washing his hands, fatigue, and disturbing inner feelings of hatred towards other people.

Nathan’s troubles started during his childhood. He reported that he was very aggressive towards other children and was involved in recurrent conflicts. At home he was constantly on guard. In his work relations, he was involved in severe interpersonal conflicts, reacting with aggressive attacks at the slightest offenses. The last years he spent working, he was continuously involved in conflicts with his colleagues. The only person he stayed friends with was his brother-in-law who lived a hundred miles away.

Nathan's case study illustrates important issues and characteristic features of the paranoid personality. First, they typically do not seek treatment unless they are in a crisis (fired from job) or because of additional pathology (depression). Second, when decompensated, or triggered, they most often get depression, panic attacks, OCD, or somatoform disorder (as in this case) or in other cases, an increase in alcohol abuse. His personality pathology is excessive aggression and mistrust.

Watch It

This video spells out the diagnostic characteristics of paranoid personality disorder.



You can view the transcript for "Paranoid Personality Disorder in a Minute" here (opens in new window).

Glossary

paranoid personality disorder: characterized by distrust and suspicion of others; others are seen as purposefully attempting to harm one in some way without any evidence to suggest this is the case

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12.5: Schizoid Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of schizoid personality disorder

We've learned about paranoid personality disorder, now let's take a look at another Cluster A disorder: **schizoid personality disorder**. Persons with schizoid personality disorder are considered classic loners.

Table 1. DSM-5 Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Paranoid	harbors a pervasive and unjustifiable suspiciousness and mistrust of others; reluctant to confide in or become close to others; reads hidden demeaning or threatening meaning into benign remarks or events; takes offense easily and bears grudges; not due to schizophrenia or other psychotic disorders	A
Schizoid	lacks interest and desire to form relationships with others; aloof and shows emotional coldness and detachment; indifferent to approval or criticism of others; lacks close friends or confidants; not due to schizophrenia or other psychotic disorders, not an autism spectrum disorder	A
Schizotypal	exhibits eccentricities in thought, perception, emotion, speech, and behavior; shows suspiciousness or paranoia; has unusual perceptual experiences; speech is often idiosyncratic; displays inappropriate emotions; lacks friends or confidants; not due to schizophrenia or other psychotic disorder, or to autism spectrum disorder	A

Schizoid Personality Disorder

Schizoid personality disorder (often abbreviated as SPD or SzPD) is a personality disorder characterized by a lack of interest in social relationships, a tendency toward a solitary or sheltered lifestyle, secretiveness, emotional coldness, detachment, and apathy. Affected individuals may be unable to form intimate attachments to others and simultaneously possess a rich and elaborate but exclusively internal fantasy world. Other associated features include stilted speech, a lack of deriving enjoyment from most activities, feeling as though one is an observer rather than a participant in life, an inability to tolerate emotional expectations of others, apparent indifference when praised or criticized, a degree of asexuality, and idiosyncratic moral or political beliefs. Symptoms typically start in late childhood or adolescence.



Figure 12.5.1: Schizoid personality disorder is characterized by a solitary lifestyle and detachment from others.

Schizoid personality disorder (SPD) is described as a pervasive pattern of detachment from social relationships and a restricted range of expression of emotions in interpersonal settings. Beginning by early adulthood and present in a variety of contexts, as indicated by at least four of the following:

- neither desires nor enjoys close relationships, including being part of a family
- almost always chooses solitary activities
- has little, if any, interest in having sexual experiences with another person
- takes pleasure in few, if any, activities
- lacks close friends or confidants other than first-degree relatives
- appears indifferent to the praise or criticism of others
- shows emotional coldness, detachment, or flattened affect

Those with schizoid personality disorder (SPD) may often be unable to, or will rarely, express aggressiveness or hostility, even when provoked directly. These individuals can seem vague or drifting about their goals and their lives may appear directionless.

Comorbidity

Schizoid personality disorder is often found to occur with at least one of several disorders or pathologies. Sometimes, a person with SPD may meet criteria for an additional personality disorder; when this happens, it is most often avoidant, schizotypal, or paranoid personality disorder. **Alexithymia** (the inability to identify and describe emotions) is often present in SPD.

While SPD shares several symptoms with other mental disorders, there are some important differentiating features:

Table 2. Differential Diagnosis

Psychological condition	Features
Depression	People who have SPD may also suffer from clinical depression. However, this is not always the case. Unlike a person with depression, persons with SPD generally do not consider themselves inferior to others. They may recognize instead that they are different.
Avoidant personality disorder (AvPD)	While people affected with AvPD avoid social interactions due to anxiety or feelings of incompetence, those with SPD do so because they are genuinely indifferent to social relationships. A 1989 study, however, found that "schizoid and avoidant personalities were found to display equivalent levels of anxiety, depression, and psychotic tendencies as compared to psychiatric control patients." There also seems to be some shared genetic risk between SPD and AvPD. Several sources to date have confirmed the synonymy of SPD and avoidant attachment style.
Other personality disorders	Schizoid and narcissistic personality disorders can seem similar in some respects (e.g., both show identity confusion, may lack warmth and spontaneity, and avoid deep relationships with intimacy). Another commonality observed by Akhtar is preferring ideas over people.
Autism Spectrum Disorder	There may be substantial difficulty in distinguishing milder forms of autism spectrum disorder. SPD is classified as a schizophrenia-like personality disorder. There is some overlap, as some people with autism also qualify for a diagnosis of schizotypal or SPD. However, one of the distinguishing features of SPD is a restricted affect and an impaired capacity for emotional experience and expression. Persons with autism may fail to recognize social cues such as verbal hints or body language.

Prevalence

Studies suggest that SPD disorder has a prevalence of less than 1%. There is no difference observed in the frequency between males and females.

Etiology

Although not much is definitively known regarding the etiology of schizoid personality disorder, it is assumed that genetics significantly contributes to the disorder. Twin studies using self-report questionnaires have estimated heritability rates for schizoid personality disorder to be about 30%.

Treatment

People with schizoid personality disorder rarely seek treatment for their condition. This is an issue found in many personality disorders, which prevents many people who are afflicted with these conditions from coming forward for treatment: they tend to view their condition as not conflicting with their self-image and their abnormal perceptions and behaviors as rational and appropriate.

? Case Study: Schizoid Personality Disorder

Edgar is a 26-year-old man. Despite extraordinary intelligence, Edgar was not able to complete or participate in any educational programs. He wanted to have a normal life with family and friends, but thought that he was rootless and he felt that other people thought that he was peculiar or odd. He felt that he was an outsider. As a child, he went to various schools because his parents moved around. He was thought of as a lonely wolf and did not participate in the social life, games, or sports with

his peers. During class, he was often absent-minded, being absorbed in his own thoughts and fantasies. From around the age of thirteen, he became interested in computers and was quite advanced in his understanding of mathematics. Edgar became exceedingly isolated with his computer as his sole companion and remains detached from people to this day. He has never been in a relationship and expressed no desire to be in one. Edgar is diagnosed with schizoid personality disorder because of his pervasive pattern of detachment from social relationships, his emotional flatness, his lack of desire for relationships, and his preference for solitary activities.

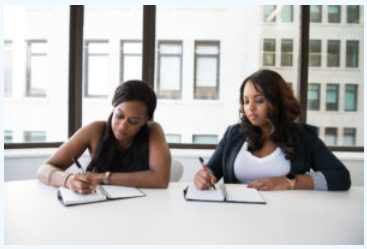


Figure 12.5.2: Some schizoid individuals can form relationships based on occupational activities.

The vignette above illustrates the difficulties for those with schizoid personality disorder in establishing stable relationships with significant others like peers and family. Often it is regarded as unusual that a person with schizoid personality disorders complains that they feel isolated. Many schizoid patients, on the contrary, claim to be quite satisfied with their loneliness and it is quite unusual that they wish to have a family. Also, schizoid persons usually accept their situation or even deny any desire for closer relationships.

People who have SPD tend to be happiest when in relationships in which their partner places few emotional or intimate demands on them and does not expect phatic or social niceties. It is not necessarily people they want to avoid, but negative or positive emotional expectations, emotional intimacy, and self-disclosure. Therefore, it is possible for individuals with SPD to form relationships with others based on intellectual, physical, familial, occupational, or recreational activities, as long as there is no need for emotional intimacy. Donald Winnicott explains this is because schizoid individuals “prefer to make relationships on their own terms and not in terms of the impulses of other people.” Failing to attain that, they prefer isolation. In general, friendship among schizoids is usually limited to one person, often also schizoid, forming what has been called a union of two eccentrics.

Watch It

This video provides a brief overview of schizoid personality disorder.



You can view the transcript for “Schizoid Personality Disorder in a Minute” here (opens in new window).

Glossary

alexithymia: the inability to identify and describe emotions

schizoid personality disorder: a personality disorder characterized by a lack of interest in social relationships, a solitary lifestyle, and detachment from others

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12.6: Schizotypal Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of schizotypal personality disorder

We have learned about paranoid personality disorder and schizoid personality disorder, now let's take a look at another Cluster A disorder: **schizotypal personality disorder**. Persons with schizotypal personality disorder usually have few, if any, close relationships and exhibit eccentric behaviors.

Table 1. DSM-5 Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Paranoid	harbors a pervasive and unjustifiable suspiciousness and mistrust of others; reluctant to confide in or become close to others; reads hidden, demeaning, or threatening meaning into benign remarks or events; takes offense easily and bears grudges; not due to schizophrenia or other psychotic disorders	A
Schizoid	lacks interest and desire to form relationships with others; aloof and shows emotional coldness and detachment; indifferent to approval or criticism of others; lacks close friends or confidants; not due to schizophrenia or other psychotic disorders, not an autism spectrum disorder	A
Schizotypal	exhibits eccentricities in thought, perception, emotion, speech, and behavior; shows suspiciousness or paranoia; has unusual perceptual experiences; speech is often idiosyncratic; displays inappropriate emotions; lacks friends or confidants; not due to schizophrenia or other psychotic disorder, or to autism spectrum disorder	A

Schizotypal Personality Disorder

Schizotypal personality disorder is characterized by severe social anxiety, thought disorder, paranoid ideation, transient psychosis, and often unconventional beliefs. People with this disorder feel extreme discomfort with maintaining close relationships with people and avoid forming them, mainly because the subject thinks their peers harbor negative thoughts towards them. The historical roots of schizotypal personality disorder (STPD) are the non-psychotic personality syndromes within the spectrum of schizophrenia, meaning that its presentation is similar to schizophrenia, but without delusions or hallucinations.



Figure 12.6.1: Common characteristics of schizotypal personality disorder.

In the DSM-5, schizotypal personality disorder (STPD) is defined as a "pervasive pattern of social and interpersonal deficits marked by acute discomfort with, and reduced capacity for, close relationships as well as by cognitive or perceptual distortions and eccentricities of behavior, beginning by early adulthood" (APA, 2010).

At least five of the following symptoms must be present:

- ideas of reference
- strange beliefs or magical thinking that influences behavior and is inconsistent with subcultural norms (e.g., superstitious beliefs; belief in clairvoyance, telepathy, or "sixth sense"; or bizarre fantasies or preoccupations)
- abnormal perceptual experiences, including bodily illusions
- strange thinking and speech (e.g., vague, circumstantial, metaphorical, overelaborate, or stereotyped)
- suspiciousness or paranoid ideation
- inappropriate or constricted affect
- strange behavior or appearance
- lack of close friends
- excessive social anxiety that does not diminish with familiarity and tends to be associated with paranoid fears rather than negative judgments about self

These symptoms must not occur only during the course of a disorder with similar symptoms (such as schizophrenia or autism spectrum disorder).

Comorbidity

There is a high rate of comorbidity with other personality disorders. McGlashan et al. (2000) stated that this high rate may be due to overlapping criteria with other personality disorders, such as avoidant personality disorder, paranoid personality disorder, and borderline personality disorder.

? Schizotypal, Schizoid, or Paranoid?

There are many similarities between schizotypal and schizoid personalities. Most notable of the similarities is the inability to initiate or maintain relationships (both friendly and romantic). The difference between the two seems to be that those labeled as schizotypal avoid social interaction because of a deep-seated fear of people. The schizoid individuals simply feel no desire to form relationships because they see no point in sharing their time with others.

Schizotypal personality disorder also differs from paranoid personality disorder in that the main feature of paranoid personality disorder is mistrust or suspicion of others, while schizotypal personality disorder (STPD) is associated with eccentric and socially dysfunctional ways.

Etiology

Schizotypal personality disorder is widely understood to have a genetic connection with a schizophrenia spectrum disorder, mood disorders, and depression. Rates of schizotypal personality disorder are much higher in relatives of individuals with schizophrenia than in the relatives of people with other mental illnesses.

There is evidence to suggest that parenting styles, early separation, trauma/maltreatment history (especially early childhood neglect) can lead to the development of schizotypal traits.

Epidemiology

The reported prevalence of schizotypal personality disorder in community studies ranges from 0.6% in a Norwegian sample to 4.6% in an American sample. A large American study found a lifetime prevalence of 3.9%, with somewhat higher rates among men (4.2%) than women (3.7%).

Treatment

Schizotypal personality disorder is rarely seen as the primary reason for treatment in a clinical setting, but it often occurs as a comorbid finding with other mental disorders. When patients with STPD are prescribed pharmaceuticals, they are most often prescribed the same antipsychotic drugs used to treat patients suffering from schizophrenia.

According to Theodore Millon, schizotypal personality disorder is one of the easiest personality disorders to identify but one of the most difficult to treat with psychotherapy. Persons with STPD usually consider themselves to be simply eccentric, productive, or nonconformist. As a rule, they underestimate the maladaptiveness of their social isolation and perceptual distortions. It is not so easy to gain rapport with people who suffer from STPD due to the fact that increasing familiarity and intimacy usually increase their level of anxiety and discomfort.

? Case Study: Schizotypal Personality Disorder

A 37-year-old, unemployed man named Tyrel complained of recurrent irrational thoughts, compulsive behavior, and social isolation. Since childhood, he had always been eccentric, withdrawn, and without real friends. He anxiously feared close relationships and was preoccupied with reading stories about Dracula. He didn't share his inner thoughts or feeling with anybody, including his parents. He never graduated high school, but maintained worked in factories, often at night. Some years earlier he started doubting if his work was accurate enough. Although he recognized these thoughts as irrational, he started spending a lot of time controlling his work over and over again. Soon these compulsive controls took so much time that he could not finish his work, and started fearing that people could notice his behavior. He started drinking alcohol to control his increasing anxiety. He adopted different peculiar strategies, which ended in new vicious circles of obsessive symptoms and suspicion.

Tyrel is diagnosed with obsessive-compulsive disorder (OCD), alcohol abuse, and schizotypal personality disorder (STPD). He had long-lasting personality difficulties like suspicion, along with odd behavior, and social anxiety prior to the OCD symptoms. STPD is rarely seen as the primary reason for treatment in a clinical setting, but often occurs as a comorbid finding with other mental disorders.

? Watch It

Watch this video to learn more details surrounding schizotypal personality disorder.



You can view the transcript for "Schizotypal personality disorder | Mental health | NCLEX-RN | Khan Academy" here (opens in new window).

Key Takeaways: Schizotypal Personality Disorder

Glossary

schizotypal personality disorder: characterized by behavior that is often described as odd or eccentric; those with SPD usually have few, if any, close relationships

schizophrenia spectrum: schizoid personality disorder, schizotypal personality disorder, and paranoid personality disorder can be considered schizophrenia-like personality disorders because of their links to the schizophrenia spectrum

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12.7: Introduction to Cluster B Personality Disorders

What you'll learn to do: describe and compare characteristics of Cluster B personality disorders



In this section, we will examine Cluster B personality disorders, which include antisocial personality disorder, histrionic personality disorder, narcissistic personality disorder, and borderline personality disorder. People with these disorders usually are impulsive, overly dramatic, highly emotional, and erratic.

Watch It

This video provides an overview of the Cluster B disorders.



You can view the transcript for "Cluster B personality disorders (antisocial, borderline, histrionic, narcissistic)" here (opens in new window).

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12.8: Antisocial Personality Disorder

Learning Objectives

- Describe the basic features of antisocial personality disorder and its etiology

Cluster B disorders include antisocial personality disorder, histrionic personality disorder, narcissistic personality disorder, and borderline personality disorder. People with these disorders usually are impulsive, overly dramatic, highly emotional, and erratic.

DSM-5 Personality Disorder	Description	Cluster
Antisocial	continuously violates the rights of others; history of antisocial tendencies prior to age 15; often lies, fights, and has problems with the law; impulsive and fails to think ahead; can be deceitful and manipulative in order to gain profit or pleasure; irresponsible and often fails to hold down a job or pay financial debts; lacks feelings for others and remorse over misdeeds	B
Histrionic	excessively overdramatic, emotional, and theatrical; feels uncomfortable when not the center of others' attention; behavior is often inappropriately seductive or provocative; speech is highly emotional but often vague and diffuse; emotions are shallow and often shift rapidly; may alienate friends with demands for constant attention	B
Narcissistic	overinflated and unjustified sense of self-importance and preoccupied with fantasies of success; feels entitled to special treatment from others; shows arrogant attitudes and behaviors; takes advantage of others; lacks empathy	B
Borderline	unstable in self-image, mood, and behavior; cannot tolerate being alone and experiences chronic feelings of emptiness; unstable and intense relationships with others; behavior is impulsive, unpredictable, and sometimes self-damaging; shows inappropriate and intense anger; makes suicidal gestures	B

Most human beings live in accordance with a moral compass, a sense of right and wrong. Most individuals learn at a very young age that there are certain things that should not be done. We learn we should not lie or cheat. We are taught it is wrong to take things that do not belong to us, and it is wrong to exploit others for personal gain. We also learn the importance of living up to our responsibilities, of doing what we say we will do. People with antisocial personality disorder, however, do not seem to have a moral compass. These individuals act as though they neither have a sense of nor care about right or wrong. Not surprisingly, these people represent a serious problem for others and for society in general.

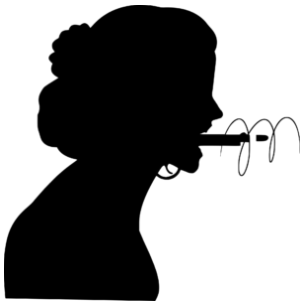


Figure 12.8.1: Individuals with ASPD may say things without thinking about how their words may affect others.

According to the DSM-5, the individual with **antisocial personality disorder (ASPD)** (sometimes referred to as psychopathy) shows no regard at all for other people's rights or feelings. This lack of regard is exhibited a number of ways and can include repeatedly performing illegal acts, lying to or conning others, impulsivity and recklessness, irritability and aggressiveness toward others, and failure to act in a responsible way (e.g., leaving debts unpaid) (APA, 2013). The worst part about antisocial personality disorder, however, is that people with this disorder have no remorse over one's misdeeds; these people will hurt, manipulate, exploit, and abuse others and not feel any guilt. Signs of this disorder can emerge early in life; however, a person must be at least 18 years old to be diagnosed with antisocial personality disorder.

People with antisocial personality disorder (ASPD) seem to view the world as self-serving and unkind. Those with ASPD seem to think they should use whatever means necessary to get by in life. They tend to view others not as living, thinking, feeling beings, but rather as pawns to be used or abused for a specific purpose. They often have an over-inflated sense of themselves and can appear extremely arrogant. They frequently display superficial charm; for example, without really meaning it, they might say exactly what they think another person wants to hear. They lack empathy: they are incapable of understanding the emotional point-of-view of others. People with this disorder may become involved in illegal enterprises, show cruelty toward others, leave their jobs with no plans to obtain another job, have multiple sexual partners, repeatedly get into fights with others, and show reckless disregard for themselves and others (e.g., repeated arrests for driving while intoxicated) (APA, 2013).

DSM-5 Criteria

Here is the DSM-5 diagnostic criteria for antisocial personality disorder (ASPD):

A. A pervasive pattern of disregard for and violation of the rights of others, since age 15 years, as indicated by three (or more) of the following:

1. failure to conform to social norms concerning lawful behaviors, such as performing acts that are grounds for arrest

- deceitfulness, repeated lying, use of aliases, or conning others for pleasure or personal profit
- impulsivity or failure to plan
- irritability and aggressiveness, often with physical fights or assaults
- reckless disregard for the safety of self or others
- consistent irresponsibility, failure to sustain consistent work behavior, or honor monetary obligat
- lack of remorse, being indifferent to or rationalizing having hurt, mistreated, or stolen from another person

B. The individual is at least age 18 years.

C. Evidence of conduct disorder typically with onset before age 15 years.

D. The occurrence of antisocial behavior is not exclusively during schizophrenia or bipolar disorder.

Persistent antisocial behavior as well as a lack of regard for others in childhood and adolescence is known as conduct disorder and considered a precursor of ASPD. About 25–40% of youths with conduct disorder will be diagnosed with ASPD in adulthood.[1]

? Differential Diagnosis

Some similar disorders to antisocial personality disorder include

- narcissistic personality disorder (cluster B personality disorder with overlap; exploitive and uncompassionate, but not aggressive or deceitful).

- borderline personality disorder (cluster B personality disorder with overlap; manipulative, but for reassurance and nurture).
- substance use disorder (Impulsivity and irresponsibility due to substance influence must be ruled out before diagnosing ASPD. ASPD can be diagnosed if substance use is co-occurring).

Etiology and Epidemiology

Although the precise etiology is unknown, both genetic and environmental factors have been found to play a role in the development of ASPD.

ASPD is observed in about 3.6% of the population; the disorder is much more common among males, with a 3:1 ratio of men to women, and it is more likely to occur in men who are younger, widowed, separated, divorced, of lower SES, live in urban areas, and live in the western United States (Compton, Conway, Stinson, Colliver, & Grant, 2005). Compared to men with ASPD, women with the disorder are more likely to have experienced emotional neglect and sexual abuse during childhood, and they are more likely to have had parents who abused substances and who engaged in antisocial behaviors themselves (Alegria et al., 2013).

The table below shows some of the differences in the specific types of antisocial behaviors that men and women with APSD exhibit (Alegria et al., 2013).

Table 1. Gender Differences in ASPD

Men with antisocial personality disorder are more likely than women with ASPD to	Women with ASPD are more likely than men with antisocial personality to
• do things that could easily hurt themselves or others • receive three or more traffic tickets for reckless driving • have their driver's license suspended • destroy others' property • start a fire on purpose • make money illegally • do anything that could lead to arrest • hit someone hard enough to injure them • hurt an animal on purpose	• run away from home overnight • frequently miss school or work • lie frequently • forge someone's signature • get into a fight that comes to blows with an intimate partner • live with others besides the family for at least one month • harass, threaten, or blackmail someone

Family, twin, and adoption studies suggest that both genetic and environmental factors influence the development of ASPD, as well as general antisocial behavior (criminality, violence, aggressiveness) (Baker, Bezdjian, & Raine, 2006). Personality and temperament dimensions that are related to this disorder, including fearlessness, impulsive antisociality, and callousness, have a substantial genetic influence (Livesley & Jang, 2008). Adoption studies clearly demonstrate that the development of antisocial behavior is determined by the interaction of genetic factors and adverse environmental circumstances (Rhee & Waldman, 2002). For example, one investigation found that adoptees of biological parents with ASPD were more likely to exhibit adolescent and adult antisocial behaviors if they were raised in adverse adoptive family environments (e.g., adoptive parents had marital problems, were divorced, used drugs, and had legal problems) than if they were raised in a more normal adoptive environment (Cadoret, Yates, Ed, Woodworth, & Stewart, 1995).

Researchers who are interested in the importance of the environment in the development of ASPD have directed their attention to such factors as the community, the structure and functioning of the family, and peer groups. Each of these factors influences the likelihood of antisocial behavior. One longitudinal investigation of more than 800 Seattle-area youth measured risk factors for violence at 10, 14, 16, and 18 years of age (Herrenkohl et al., 2000). The risk factors examined included those involving the family, peers, and community. A portion of the findings from this study are provided in Figure **12.8.2**.

Risk Factors During Adolescence That Predict Later Violence			
Risk factor	**Age 10 predictor (elementary school)**	**Age 14 predictor (middle school)**	**Age 16 predictor (high school)**
Family			
Parental violence		✕	
Parental criminality		✕	✕
Poof family management		✕	✕
Family conflict		✕	✕
Parental attitudes favorable to violence	✕		
Frequent moves			✕
Peer			
Peer delinquency	✕	✕	✕
Gang membership		✕	✕
Community			
Economic deprivation	✕		✕
Community disorganization		✕	✕
Availability of drugs	✕	✕	✕
Neighborhood adults involved in crime		✕	✕

Figure **12.8.2**: Longitudinal studies have helped to identify risk factors for predicting violent behavior.

Individuals with antisocial tendencies do not seem to experience emotions the way most other people do. These individuals fail to show fear in response to environmental cues that signal punishment, pain, or noxious stimulation. For instance, they show less skin conductance (sweatiness on hands) in anticipation of electric shock than do people without antisocial tendencies (Hare, 1965). Skin conductance is controlled by the sympathetic nervous system and is used to assess autonomic nervous system functioning. When the sympathetic nervous system is active, people become aroused and anxious, and sweat gland activity increases. Thus, increased sweat gland activity, as assessed through skin conductance, is taken as a sign of arousal or anxiety. For those with ASPD, a lack of skin conductance may indicate the presence of characteristics such as emotional deficits and impulsivity that underlie the propensity for antisocial behavior and negative social relationships (Fung et al., 2005).

While emotional deficits may contribute to antisocial personality disorder, so too might an inability to relate to others' pain. In a recent study, 80 prisoners were shown photos of people being intentionally hurt by others (e.g., someone crushing a person's hand in an automobile door) while undergoing brain imaging (Decety, Skelly, & Kiehl, 2013). Prisoners who scored high on a test of antisocial tendencies showed significantly less activation in brain regions involved in the experience of empathy and feeling concerned for others than did prisoners with low scores on the antisocial test. Notably, the prisoners who scored high on the antisocial test showed greater activation in a brain area involved self-awareness, cognitive function, and interpersonal experience. The investigators suggested that the heightened activation in this region when watching social interactions involving one person harming another may reflect a propensity or desire for this kind of behavior.

? Case Study

Consider the case study of a 27-year-old male, Joseph, who committed murder at age 17. He stayed in a high-security hospital for 10 years and started individual treatment after being released. He was an intelligent boy who did well in school until his

peers began to tease him. This teasing made Joseph feel helpless and unable to defend himself. At home, however, he felt strong and supportive of his mother. His father lived with another woman. He experienced himself as a loser among his peers but as a winner with his mother.

At the end of elementary school, his father, who then had accumulated substantial wealth, returned home, and the parents resumed their marriage and intimacy. Joseph's situation at school changed as he became popular and the teasing stopped, but he still felt insecure and uneasy. With his newfound popularity, Joseph found it easy to get others to do things for him and enjoyed organizing mischief in his neighborhood. At this same time, he decided to attend karate school to gain a sense of power.

In high school, a peer introduced Joseph to a gang where he felt accepted and appreciated. During a robbery, he became incredibly angry and physically violent without really understanding why. The victim died as a consequence of his attack, and Joseph was sent to prison for two years, followed by a high-security hospital for treatment. While he accepted his prison sentence, he protested treatment in the psychiatric hospital. He was suspicious, remained non-relative, and was often restrained due to anger outbursts. Joseph was diagnosed with ASPD. A therapist confronted him with the fact that his behavior could lead to a prolonged hospital stay and pointed to his choice of future inside or outside the hospital. This was a turning point that made him focus on goals and training for a future with a life outside of an institution. After discharge, he continued to work on self-esteem and trustworthiness; shame and guilt; and how to understand, control, and come to terms with his anger. Two years later, he was married with a son and pursuing a career as a teacher.

Treatment

No current diagnostic modalities, including serology tests, are currently accepted standards in diagnosing ASPD. However, genetic testing and neuroimaging have been used to evaluate potential causes and patterns with ASPD. Patients with ASPD are at a higher risk of contracting certain viral infections and sexually transmitted diseases associated with high-risk behavior, including hepatitis C and human immunodeficiency virus, as well as increased mortality rates due to accidents, traumatic injuries, suicides, and homicides.

Literature suggests early treatment intervention with conduct disorder in children as the least costly and most effective in treating ASPD. Most of the needs of ASPD are addressable in the outpatient setting. No pharmacological intervention has been shown to treat ASPD, but medications are highly recommended to treat co-occurring conditions.

Watch It

Learn more about behavioral characteristics of ASPD.



You can view the transcript for "Antisocial Personality Disorder.. What is it?" here (opens in new window).

Glossary

antisocial personality disorder (ASPD): characterized by a lack of regard for others' rights, impulsivity, deceitfulness, irresponsibility, and lack of remorse over misdeeds

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12.9: Histrionic Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of histrionic personality disorder

Cluster B personality disorders include those related to impulsive, dramatic, emotional, or erratic behavior. These include antisocial personality disorder (ASPD), borderline personality disorder (BPD), histrionic personality disorder (HPD), and narcissistic personality disorder (NPD). We will review histrionic personality disorder on this page.

Table 1. DSM-5 Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Antisocial	continuously violates the rights of others; history of antisocial tendencies prior to age 15; often lies, fights, and has problems with the law; impulsive and fails to think ahead; can be deceitful and manipulative in order to gain profit or pleasure; irresponsible and often fails to hold down a job or pay financial debts; lacks feelings for others and remorse over misdeeds	B
Histrionic	excessively overdramatic, emotional, and theatrical; feels uncomfortable when not the center of others' attention; behavior is often inappropriately seductive or provocative; speech is highly emotional but often vague and diffuse; emotions are shallow and often shift rapidly; may alienate friends with demands for constant attention	B
Narcissistic	overinflated and unjustified sense of self-importance and preoccupied with fantasies of success; feels entitled to special treatment from others; shows arrogant attitudes and behaviors; takes advantage of others; lacks empathy	B
Borderline	unstable in self-image, mood, and behavior; cannot tolerate being alone and experiences chronic feelings of emptiness; unstable and intense relationships with others; behavior is impulsive, unpredictable, and sometimes self-damaging; shows inappropriate and intense anger; makes suicidal gestures	B

Histrionic Personality Disorder

Histrionic personality disorder (HPD) is a personality disorder characterized by a pattern of excessive attention-seeking behaviors, usually beginning in early childhood, including inappropriate seduction and an excessive need for approval. People diagnosed with the disorder are said to be lively, dramatic, vivacious, enthusiastic, and flirtatious.

People with a histrionic personality disorder (HPD) may feel underappreciated or disregarded when they are not the center of attention. People with this disorder are typically “the life of the party” and have a “larger than life” presence. People with histrionic personality disorder (HPD) have a high need for attention, make loud and inappropriate appearances, exaggerate their behaviors and emotions, and crave stimulation. They may exhibit sexually provocative behavior, express strong emotions with an impressionistic style, and can be easily influenced by others.

Diagnostic Criteria



Figure **12.9.1**: Individuals with histrionic personality disorder crave stimulation and need to be the center of attention.

The DSM-5 defines histrionic personality disorder as a pervasive pattern of excessive emotionality and attention-seeking, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following characteristics:

- is uncomfortable in situations in which they are not the center of attention
- interaction with others is often characterized by inappropriate sexually seductive or provocative behavior
- displays rapidly shifting and shallow expression of emotions
- consistently uses physical appearance to draw attention to self
- has a style of speech that is excessively impressionistic and lacking in detail
- shows self-dramatization, theatricality, and exaggerated expression of emotion
- is suggestible, i.e., easily influenced by others or circumstances
- considers relationships to be more intimate than they actually are

Differential Diagnosis

The **differential diagnosis** (unique attributes that differentiate a disorder from others with similar features) for histrionic personality disorder includes narcissistic personality disorder, borderline personality disorder, dependent personality disorder, somatic symptom disorder, and illness anxiety disorder.

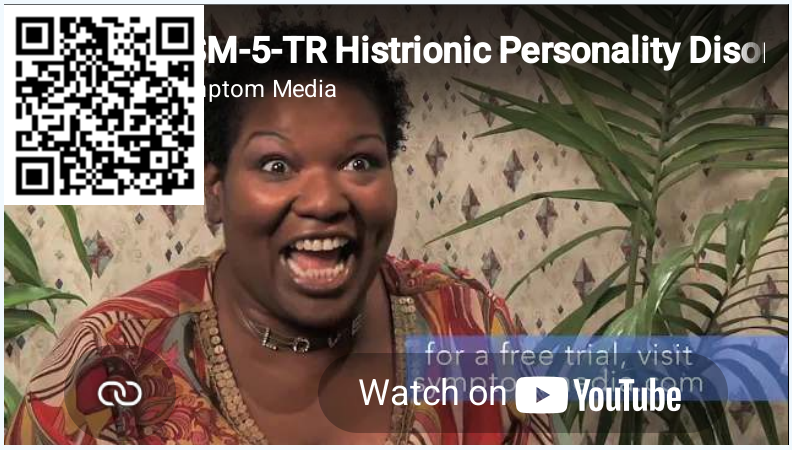
- Like histrionic personality disorder, patients with narcissistic personality disorder prefer to be the center of attention. However, patients with narcissistic personality disorder want attention, which results from admiration or veneration, whereas people with histrionic personality disorder are not particular about what type of attention they garner.
- Patients with histrionic personality disorder are similar to those with borderline personality because both demographics of patients experience intense emotions; however, patients with borderline personality disorder usually dislike themselves.
- Dependent personality disorder should be considered in the differential diagnosis for histrionic personality disorder as with both personality disorders; the patient will prefer to be around others. However, patients with dependent personality disorder tend to be more submissive, and their behavior is more inhibited as they are preoccupied with fears of rejection.
- Somatic symptom disorder and illness anxiety disorder are also included in the differential for histrionic personality disorder as patients with a histrionic personality disorder may use physical symptoms and complaints to gain attention from others.

Comorbidity

Most people with histrionic personality disorder also have other mental disorders. Comorbid conditions include antisocial, dependent, borderline, and narcissistic personality disorders as well as depression, anxiety disorders, panic disorder, somatoform disorders, anorexia nervosa, substance use disorder, and attachment disorders.

Watch It

Watch this clip to see how histrionic personality disorder may be demonstrated.



You can view the transcript for "DSM 5 Histrionic Personality Disorder Criteria Example, Symptoms Video" here (opens in new window).

Epidemiology

The prevalence of histrionic personality disorder in the general population runs about 2%-3%. It is possible for people to have more than one personality disorder. Women are four times more likely to be diagnosed with histrionic personality disorder than men. However, research suggests that women may be overly diagnosed with this disorder when compared to men due to sexual-forwardness being less socially acceptable for women. Furthermore, men may be less likely to report their symptoms and thereby be under-diagnosed. Histrionic personality disorder tends to be ego-syntonic, meaning people with this disorder typically consider their behavior to be normal and struggle to identify a problem.

Etiology

Histrionic personality disorder probably develops as a combination of both learned and inherited factors. One hypothesis is that histrionic personality disorder may develop as a result of trauma experienced during childhood. Children may endure their trauma by coping with their environment in ways that may ultimately lead to a personality disorder. Personality disorders in childhood may originate as an adaptation to cope with a traumatic situation or traumatic environment.

Parenting styles may also influence the likelihood of developing a histrionic personality disorder. Parenting that lacks boundaries is over-indulgent, or inconsistent may predispose children to develop a histrionic personality disorder. Because histrionic personality disorder tends to run in families, there is some consideration that there is genetic susceptibility for this disorder that may be inherited. As with all psychiatric disorders, having a family history of personality disorders, psychiatric illness, or substance use disorders is a risk factor for histrionic personality disorder.

Treatment

Supportive psychotherapy is a recommended modality of treatment for patients with histrionic personality disorder, as this approach is found to be encouraging, reassuring, and non-threatening. Supportive psychotherapy aims to reduce emotional distress, improve self-esteem, and to enhance the patient's coping skills, all through attentive and sympathetic listening.

Psychodynamic psychotherapy (also called insight-oriented therapy) has also proven to be a successful approach in treating patients with histrionic personality disorder. The goal of this therapy is to alter an aspect of a patient's dysfunctional personality by integrating crucial developmental milestones a patient may have missed during previous stages of emotional maturation. Psychodynamic psychotherapy aims to resolve underlying, unconscious conflicts in an effort for patients to understand themselves and their behaviors better. The patients are encouraged to substitute excessively dramatic speech for a more adaptive action or behavior, to promote better communication with others. Through psychodynamic psychotherapy, patients learn to recognize that hyper-sexual, attention-seeking behaviors are maladaptive, and discover new, healthier ways to develop self-esteem.

While the gold standard for treating personality disorders is psychotherapy, patients with histrionic personality disorder may be profoundly symptomatic. Patients may experience affective dysregulation, where they frequently endure mood swings, anger, tearfulness, anxiety, and depression. While there are no FDA-approved medications for the treatment of histrionic personality disorder, affective dysregulation may be treated with antidepressants, mood stabilizers, and antipsychotics. Antidepressants that have proven to be effective include desipramine, fluoxetine, amitriptyline, and fluvoxamine. The mood stabilizers with proven therapeutic benefits include lamotrigine, carbamazepine, topiramate, valproate, and lithium. Research has demonstrated that antipsychotics such as risperidone, aripiprazole, olanzapine, and haloperidol have been useful in treating affective dysregulation. Patients with histrionic personality disorder may struggle with impulse control and regulation of their behaviors. Clinical trials have demonstrated that mood stabilizers specifically can target these symptoms.

Learning Objectives

histrionic personality disorder: characterized by a pattern of excessive attention-seeking behaviors, usually beginning in early childhood, including inappropriate seduction and an excessive need for approval

personality disorder: group of DSM-5 disorders characterized by an inflexible and pervasive personality style that differs markedly from the expectations of one's culture and causes distress and impairment

1. Novais F, Araújo A and Godinho P (2015) Historical roots of histrionic personality disorder. *Front. Psychol*. 6:1463. doi: 10.3389/fpsyg.2015.01463 ↵

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12.10: Narcissistic Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of narcissistic personality disorder

Now we will review another Cluster B personality disorder: narcissistic personality disorder.

Table 1. Cluster B Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Antisocial	continuously violates the rights of others; history of antisocial tendencies prior to age 15; often lies, fights, and has problems with the law; impulsive and fails to think ahead; can be deceitful and manipulative in order to gain profit or pleasure; irresponsible and often fails to hold down a job or pay financial debts; lacks feelings for others and remorse over misdeeds	B
Histrionic	excessively overdramatic, emotional, and theatrical; feels uncomfortable when not the center of others' attention; behavior is often inappropriately seductive or provocative; speech is highly emotional but often vague and diffuse; emotions are shallow and often shift rapidly; may alienate friends with demands for constant attention	B
Narcissistic	overinflated and unjustified sense of self-importance and preoccupied with fantasies of success; feels entitled to special treatment from others; shows arrogant attitudes and behaviors; takes advantage of others; lacks empathy	B
Borderline	unstable in self-image, mood, and behavior; cannot tolerate being alone and experiences chronic feelings of emptiness; unstable and intense relationships with others; behavior is impulsive, unpredictable, and sometimes self-damaging; shows inappropriate and intense anger; makes suicidal gestures	B

Narcissistic personality disorder (NPD) is characterized by the personality traits of persistent grandiosity, an excessive need for admiration, and a personal disdain and lack of empathy for other people. As such, an individual with narcissistic personality disorder (NPD) usually displays arrogance and a distorted sense of personal superiority and seeks to establish abusive power and control over others. Self-confidence (a strong sense of self) is a personality trait different from the traits of narcissistic personality disorder; thus, people with narcissistic personality disorder (NPD) typically value themselves over others, to the extent of openly disregarding the wishes and feelings of anyone else, and expect to be treated as superior, regardless of their actual status or

achievements. Socially, the person with NPD usually exhibits a fragile ego (self-concept), intolerance of criticism, and a tendency to belittle other people in order to validate his or her own superiority.



Figure **12.10.1**: Individuals with NPD typically value themselves over anyone else.

The DSM-5 indicates that a person with NPD possesses at least five of the following nine criteria, typically without possessing the commensurate personal qualities or accomplishments for which he or she demands respect and status:

- has a grandiose sense of self-importance (e.g. exaggerates achievements and talents, expects to be recognized as superior without commensurate achievements).
- is preoccupied with fantasies of unlimited success, power, brilliance, beauty, or ideal love.
- believes that they are special and unique and can only be understood by, or should associate with, other special or high-status people (or institutions).
- requires excessive admiration.
- has a sense of entitlement (i.e., unreasonable expectations of especially favorable treatment or automatic compliance with his or her expectations).
- is interpersonally exploitative (i.e., takes advantage of others to achieve his or her own ends).
- lacks empathy: is unwilling to recognize or identify with the feelings and needs of others.
- is often envious of others or believes that others are envious of him or her.
- shows arrogant, haughty behaviors or attitudes.

Narcissistic Personality Disorder

NPD is a personality disorder characterized by a long-term pattern of exaggerated feelings of self-importance, an excessive craving for admiration, and struggles with empathy. People with NPD often spend much time daydreaming about achieving power and success, or on their appearance. People with the diagnosis in recent years have spoken out about its stigma in media, and possible links to abusive situations and childhood trauma. Such narcissistic behavior typically begins by early adulthood and occurs across a broad range of situations.

Epidemiology

The lifetime rates of narcissistic personality disorder are estimated at 1% in the general population; and between two to 16% in the clinical population.

Narcissistic personality disorder usually develops either in adolescence or in early adulthood, and it is common for children and adolescents to display personality traits that resemble NPD, but such occurrences are usually transient, and register below the clinical criteria for a formal diagnosis of NPD. True symptoms of NPD are pervasive, apparent in varied social situations, and rigidly consistent over time. Severe symptoms of NPD can significantly impair the person's mental capabilities to develop meaningful human relationships, such as friendship, kinship, and marriage. The DSM-5 indicates that, in order to qualify as symptomatic of NPD, the person's manifested personality traits must substantially differ from the cultural norms of society.

Etiology

The causes of narcissistic personality disorder are unknown, but theorized to be linked to certain types of traumas. A combination of genetic, environmental, and social factors are involved in narcissistic personality disorder.

Narcissistic personality disorder usually develops either in adolescence or in early adulthood, and it is common for children and adolescents to display personality traits that resemble NPD, but such occurrences are usually transient and register below the clinical criteria for a formal diagnosis of NPD. True symptoms of NPD are pervasive, apparent in varied social situations, and are rigidly consistent over time. Severe symptoms of NPD can significantly impair the person's mental capabilities to develop meaningful human relationships, such as friendship, kinship, and marriage. Generally, the symptoms of NPD also impair the person's psychological abilities to function socially, either at work, or at school, or within important societal settings. The DSM-5 indicates that, in order to qualify as symptomatic of NPD, the person's manifested personality traits must substantially differ from the cultural norms of society.

Treatment



Figure 12.10.2: NPD can give individuals inflated perceptions about themselves.

Narcissistic personality disorder is rarely the primary reason for which people seek mental health treatment. Therapy is difficult because people with narcissistic personality disorder usually do not consider their own issues as symptoms, despite mental distress. When people with NPD enter treatment, they usually are prompted by difficulties in their lives, or are seeking relief from some other disorder of their mental health, such as a major depressive disorder, drug addiction, or manic depression.

The reason for such an indirect path to psychotherapy is partly because an individually with NPD generally possess poor insight, and are unaware that their actions produced their mentally unhealthy circumstance, and so fail to recognize that their perceptions and behaviors are socially inappropriate and problematic, because of their very positive self-image (inflated self-concept).

Comorbidity

The occurrence of narcissistic personality disorder presents a high rate of comorbidity with other mental disorders. People with NPD are prone to bouts of psychological depression, often to the degree that meets the clinical criteria for a co-occurring depressive disorder. Moreover, the occurrence of NPD is further associated with the occurrence of bipolar disorder and substance use disorders, especially cocaine use disorder.

Watch It

This video further explains and gives examples of narcissism.



You can view the transcript for "The psychology of narcissism – W. Keith Campbell" here (opens in new window).

Case Study: Narcissistic Personality Disorder

A 42-year-old male professional in public office, Edgar, was forced to resign after being arrested when visiting a brothel. In the aftermath, he suffered from depression, considerable alcohol consumption, and was admitted for a three-month treatment. He stopped drinking, but his depression remained unresponsive to anti-depressant medication. Still, without meaningful activities, he felt empty, restless, and was eventually referred to psychotherapy.

Developmental history indicates that at age five, Edgar's father left the family and they did not meet again until he was in law school. He was always ahead of his age and went through school without difficulty. In law school, he got high marks without hard work. He had many acquaintances, but no close friendships, and always felt like an outsider. He got married and had two children. Reaching his mid-thirties, Edgar felt bored. He had everything: house, career, and family. He was respected and accomplished but felt he didn't belong. He started drinking heavily and visiting brothels.

The psychotherapist found him self-assured, easily irritated, and quick to make devaluing remarks. Interactions during weekly appointments were extremely difficult. Unwilling to explore his situation or his feelings, he blamed the therapist for the impasse and told the therapist that he would not change and that the therapist could not help. The therapist dreaded the appointments, while the patient, despite finding the sessions unhelpful, always showed up. When the therapist announced a three-week break, Edgar suggested the treatment end and did not return. Nine months later, Edgar informed the therapist that he moved to another city, had a leading position working with international trade, and was greeted as a king. He said nothing about his wife and children. Nor did he indicate how he felt about the treatment.

Glossary

narcissistic personality disorder: a Cluster B personality disorder characterized by a pattern of grandiosity, a need for admiration, and a lack of empathy.

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12.11: Borderline Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of borderline personality disorder

So far, we have revied the Type B personality disorders related to impulsive, dramatic, emotional, or erratic behavior. Now we'll take a closer look at borderline personality disorder (BPD).

Table 1. DSM-5 Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Antisocial	continuously violates the rights of others; history of antisocial tendencies prior to age 15; often lies, fights, and has problems with the law; impulsive and fails to think ahead; can be deceitful and manipulative in order to gain profit or pleasure; irresponsible and often fails to hold down a job or pay financial debts; lacks feelings for others and remorse over misdeeds	B
Histrionic	excessively overdramatic, emotional, and theatrical; feels uncomfortable when not the center of others' attention; behavior is often inappropriately seductive or provocative; speech is highly emotional but often vague and diffuse; emotions are shallow and often shift rapidly; may alienate friends with demands for constant attention	B
Narcissistic	overinflated and unjustified sense of self-importance and preoccupied with fantasies of success; feels entitled to special treatment from others; shows arrogant attitudes and behaviors; takes advantage of others; lacks empathy	B
Borderline	unstable in self-image, mood, and behavior; cannot tolerate being alone and experiences chronic feelings of emptiness; unstable and intense relationships with others; behavior is impulsive, unpredictable, and sometimes self-damaging; shows inappropriate and intense anger; makes suicidal gestures	B

Borderline Personality Disorder

Borderline personality disorder (BPD) is a mental illness characterized by a long-term pattern of unstable relationships, distorted sense of self, and strong emotional reactions. Those affected often engage in self-harm and other dangerous behavior. They may also struggle with a feeling of emptiness, fear of abandonment, and detachment from reality.



Figure 12.11.1: Those with borderline personality disorder may quickly change their mood or behavior.

The DSM-5 diagnostic criteria for borderline personality disorder is

A. A pervasive pattern of instability of interpersonal relationships, self-image, and affects as well as marked impulsivity beginning by early adulthood and present in a variety of contexts as indicated by five or more of the following:

1. frantic efforts to avoid real or imagined abandonment (note: do not include suicidal or self-mutilating behavior covered in Criterion 5)

- a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation
- identity disturbance marked by a persistently unstable self-image or sense of self
- impulsivity in at least two areas that are potentially self-damaging, for example, spending, sex, substance abuse, reckless driving, binge eating (note: do not include suicidal or self-mutilating behavior covered in Criterion 5)
- affective instability due to a marked reactivity of mood, for example, intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days
- chronic feelings of emptiness
- inappropriate, intense anger or difficulty controlling anger, for example, frequent displays of temper, constant anger, recurrent physical fights
- transient, stress-related paranoid ideation or severe dissociative symptoms

Differential Diagnosis

When considering a diagnosis of borderline personality disorder, the differential diagnosis should always include other personality disorders since overlap is common, especially within Cluster B. Other diagnostic considerations include

- substance use disorder
- non-suicidal self-injury disorder (a time-limited phenomenon in many patients)
- bipolar disorder (five times less prevalent, but comorbidity approaches 15%)
- autism spectrum disorder (patients with congenital deficits in theory of mind may also show severe mood dysregulation)

Watch It

This video provides an overview of borderline personality disorder and also explains how it is differentiated from bipolar disorder.



You can view the transcript for "What is Borderline Personality Disorder?" here (opens in new window).

Epidemiology

The behavior in BPD typically begins by early adulthood and occurs across a variety of situations. Substance abuse, depression, and eating disorders are commonly associated with BPD. Approximately 10% of people affected with the disorder commit suicide. There is a genetic predisposition for the disorder. Twin studies show over 50% heritability (greater than that for major depression). Twin studies performed in 2000 and 2008 both demonstrated higher concordance of the rate of borderline personality disorder for monozygotic versus dizygotic twins. Environmental factors that have been identified as contributing to the development of borderline personality disorder include primarily childhood maltreatment (physical, sexual, or neglect), found in up to 70% of people with BPD, as well as maternal separation, poor maternal attachment, inappropriate family boundaries, parental substance abuse, and serious parental psychopathology.

Approximately 1.6% of people have BPD in a given year, with some estimates as high as 6%. Women are diagnosed about three times as often as men. The disorder appears to become less common among older people. Up to half of those with BPD improve over a 10-year period.

Etiology

The causes of BPD are unclear but seem to involve genetic, environmental, and social factors. It occurs about five times more often in a person who has an affected close relative. Adverse life events appear to also play a role.

As is the case with other mental disorders, the causes of BPD are complex and not fully agreed upon. Evidence suggests that BPD and PTSD may be related in some way. Most researchers agree that a history of childhood trauma can be a contributing factor.

Comorbidity



Figure **12.11.2**: BPD can exist simultaneously with other disorders in a patient.

Patients with borderline personality disorder have been shown to have high rates of comorbid disorders:

- mood disorders: 80% to 96%
- anxiety disorders: 88%
- substance abuse disorders: 64%
- eating disorders: 53%
- attention deficit hyperactivity disorder (ADHD): 10–30%
- bipolar disorder: 15%
- somatoform disorders: 10%

Treatment

Treatment of borderline personality disorder relies on psychotherapy. Three evidence-based therapies are effective for patients with borderline personality disorder. First, mentalizing-based therapy (MBT) helps patients manage emotion dysregulation by feeling understood, allowing them to be more curious and make fewer assumptions about the intentions of the people around them. A second therapy, dialectical behavior therapy (DBT) combines mindfulness practices with concrete interpersonal and emotion regulation skills. Third, transference-focused psychotherapy (TFP) focuses on using the patient–therapist relationship to develop the patient's awareness of problematic interpersonal dynamics. MBT and DBT each incorporate individual and group treatment over 12 to 18 months. For adolescents, family therapy may be an appropriate substitute for group therapy, though not always.

Key Takeaways: Borderline Personality Disorder

DSM-V Criteria:

Characterized by hypersensitivity to rejection and resulting instability of interpersonal relationships, self-image, affect, and behavior.

Epidemiology:

The prevalence of borderline personality disorder in the general population is 1.6% with a lifetime prevalence of 5.9%.

Etiology:

Borderline personality disorder is multifactorial in etiology, including genetic, environmental, and social factors.

Treatment:

Long-term psychotherapy is currently the treatment of choice for BPD.

Case Study: BPD

A 23-year-old woman, Nikki, reacted with depressive symptoms and suicidal thoughts after the death of her grandfather. She was treated with antidepressant medication without addressing the loss. Three years later and after a suicidal attempt, she was admitted to a hospital where she first presented with depressed mood and suicidal thoughts, but quickly engaged in vivid conversations with the other patients. Nikki was discharged with the diagnosis of personality disorder, but soon re-admitted because of suicidal thoughts, and referred to an outpatient program specialized in the treatment of personality disorders.

Since childhood, Nikki has displayed unstable moods, aggressive temperament, and self-destructive behaviors (headbanging). At the age of 10, she was sexually abused by an older man. Suicidal thoughts and urges to kill herself were first experienced at age 11. Since age 13, she has had multiple sexual partners but also a seven-year long relationship, which was quite unstable with frequent conflicts and impulsive acts. Nikki dropped out of school and has been living on disability benefits, interrupted by short periods of employment.

In a two-year psychoanalytic treatment program, with one individual session and one group session each week, Nikki worked together with other patients on identifying and understanding the characteristic features of borderline personality disorder. Her self-destructive behavior tapered off after three months as she began to process her feelings of aggression and sadness. The pharmacological treatment terminated after six months and she quickly became less sedated and anxious. Nikki resumed school towards the end of the first year of treatment, with the intention of taking a degree in teaching. The relationship with her boyfriend stabilized. Contacts with class became more satisfying, and conflicts with her teachers stopped. Her ability to begin to contain feelings increased dramatically.

Glossary

borderline personality disorder: characterized by instability in interpersonal relationships, self-image, and mood, as well as impulsivity; key features include intolerance of being alone and fear of abandonment, unstable relationships, unpredictable behavior and moods, and intense and inappropriate anger

personality disorders: a class of mental disorders characterized by enduring maladaptive patterns of behavior, cognition, and inner experience, exhibited across many contexts and deviating from those accepted by the individual's culture; patterns develop early, are inflexible, and are associated with significant distress or disability

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12.12: Introduction to Cluster C Personality Disorders

What you'll learn to do: describe and compare characteristics of Cluster C personality disorders



In this section, you will learn about Cluster C personality disorders, which include avoidant, dependent, and obsessive-compulsive personality disorders. These Cluster C disorders are characterized by anxious, fearful, or clingy behaviors.

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12.13: Avoidant Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of avoidant personality disorder

Cluster C personality disorders include avoidant, dependent, and obsessive-compulsive personality disorders. Individuals with these disorders often appear anxious, fearful, or clingy (a mnemonic to help remember Cluster C disorders are the three C's—cowardly, clingy, and compulsive). Avoidant personality disorders exhibit a pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation. Dependent personality disorders show a pattern of submissive and clinging behavior that evidence an excessive need to be taken care of. Lastly, obsessive-compulsive personality disorders manifest a preoccupation with orderliness, perfectionism, and control.

Table 1. DSM-5 Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Avoidant	socially inhibited and oversensitive to negative evaluation; avoids occupations that involve interpersonal contact because of fears of criticism or rejection; avoids relationships with others unless guaranteed to be accepted unconditionally; feels inadequate and views self as socially inept and unappealing; unwilling to take risks or engage in new activities if they may prove embarrassing	C
Dependent	allows others to take over and run their life; is submissive, clingy, and fears separation; cannot make decisions without advice and reassurance from others; lacks self-confidence; cannot do things on his or her own; feels uncomfortable or helpless when alone	C
Obsessive-Compulsive	pervasive need for perfectionism that interferes with the ability to complete tasks; preoccupied with details, rules, order, and schedules; excessively devoted to work at the expense of leisure and friendships; rigid, inflexible, and stubborn; insists things be done a certain way; miserly with money	C

Avoidant Personality Disorder

People with avoidant personality disorder (AvPD) often consider themselves to be socially inept or personally unappealing and avoid social interaction for fear of being ridiculed, humiliated, rejected, or disliked. They often avoid becoming involved with others unless they are certain they will be liked. As the name suggests, the main coping mechanism of those with avoidant personality disorder (AvPD) is avoidance of feared stimuli.

Those affected display a pattern of severe social anxiety, social inhibition, feelings of inadequacy and inferiority, extreme sensitivity to negative evaluation and rejection, and avoidance of social interaction despite a strong desire for intimacy.

According to the DSM-5, avoidant personality disorder (AvPD) diagnosis refers to a widespread pattern of inhibition around people, feeling inadequate, and being very sensitive to negative evaluation. Symptoms begin by early adulthood and occur in a range of situations.



Figure **12.13.1**: People with AvPD often have a negative perception of themselves.

Four of the following seven specific symptoms should be present:

- avoids occupational activities that involve significant interpersonal contact, because of fears of criticism, disapproval, or rejection
- is unwilling to get involved with people unless certain of being liked
- shows restraint within intimate relationships because of the fear of being shamed or ridiculed
- is preoccupied with being criticized or rejected in social situations
- is inhibited in new interpersonal situations because of feelings of inadequacy
- views self as socially inept, personally unappealing, or inferior to others
- is unusually reluctant to take personal risk or to engage in any new activities because they may prove embarrassing

Differential Diagnosis

In contrast to social anxiety disorder, a diagnosis of AvPD also requires that the general criteria for a personality disorder are met. According to DSM-5, AvPD must be differentiated from similar personality disorders such as dependent, paranoid, schizoid, and schizotypal. But these disorders can also occur together; this is particularly likely for AvPD and dependent personality disorder. Thus, if criteria for more than one personality disorder are met, all can be diagnosed. There is also an overlap between avoidant and schizoid personality traits, and AvPD may have a relationship to the schizophrenia spectrum.

Comorbidity

AvPD is reported to be especially prevalent in people with anxiety disorders, although estimates of comorbidity vary widely due to differences in (among others) diagnostic instruments. Research suggests that approximately 10–50% of people who have panic disorder with agoraphobia have avoidant personality disorder, as well as about 20–40% of people who have social anxiety disorder. In addition, AvPD is more prevalent in people who have comorbid social anxiety disorder and generalized anxiety disorder than in those who have only one of the aforementioned conditions. Some studies report prevalence rates of up to 45% among people with generalized anxiety disorder and up to 56% of those with obsessive-compulsive disorder. Post-traumatic stress disorder is also commonly comorbid with avoidant personality disorder.

Epidemiology

Data from the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions indicates a prevalence rate of 2.36% in the American general population. It appears to occur with equal frequency in males and females.

Etiology



Figure **12.13.2**: Childhood neglect is a risk factor for AvPD.

Causes of AvPD are not clearly defined, but appear to be influenced by a combination of social, genetic, and psychological factors. The disorder may be related to temperamental factors that are inherited.

Childhood emotional neglect (in particular, the rejection of a child by one or both parents) and peer group rejection are associated with an increased risk for its development; however, it is possible for AvPD to occur without any notable history of abuse or neglect.

Treatment

Treatment of avoidant personality disorder can employ various techniques, such as social skills training, psychotherapy, cognitive therapy, exposure treatment to gradually increase social contacts, group therapy for practicing social skills, and sometimes drug therapy.

? Case Study: Avoidant Personality Disorder

The patient was 35-year-old, unmarried data technician William, referred to a specialized treatment program for personality disorders from an outpatient drug addiction service. His personality pathology was considered more devastating than his substance abuse. Presenting complaints included low self-esteem, loneliness, a sense of emptiness, suicidal ideation, social isolation, substance abuse, and general dissatisfaction with life. Present complaints had been chronic in nature, dating back to childhood.

William recalled having daily suicidal thoughts for several years in his early youth. His avoidant behavior was more prominent than his level of experienced anxiety. William's most prominent feature was a pervasive fear of being ridiculed when interacting with others, and he was diagnosed with avoidant personality disorder.

In a group-based treatment program lasting for 20 weeks, he was a regular, but somewhat detached participant. The therapists encountered a series of problems related to passivity: he postponed most of his obligations, resisted sorting out practical affairs, did not pay his bills, and avoided contacting people who could be helpful.

? Watch It

Watch this video to learn more about behaviors associated with avoidant personality disorder.



You can view the transcript for “Avoidant Personality Disorder.. What is it?” here (opens in new window).

Glossary

avoidant personality disorder: personality disorder characterized by patterns of social withdrawal, inadequacy, and fear of criticism

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12.14: Dependent Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of dependent personality disorder

Dependent Personality Disorder

Dependent personality disorder (DPD) is a cluster C personality disorder characterized by a pervasive psychological dependence on other people.

Table 1. DSM-5 Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Avoidant	socially inhibited and oversensitive to negative evaluation; avoids occupations that involve interpersonal contact because of fears of criticism or rejection; avoids relationships with others unless guaranteed to be accepted unconditionally; feels inadequate and views self as socially inept and unappealing; unwilling to take risks or engage in new activities if they may prove embarrassing	C
Dependent	allows others to take over and run their life; is submissive, clingy, and fears separation; cannot make decisions without advice and reassurance from others; lacks self-confidence; cannot do things on his or her own; feels uncomfortable or helpless when alone	C
Obsessive-Compulsive	pervasive need for perfectionism that interferes with the ability to complete tasks; preoccupied with details, rules, order, and schedules; excessively devoted to work at the expense of leisure and friendships; rigid, inflexible, and stubborn; insists things be done a certain way; miserly with money	C

Dependent personality disorder (DPD) is a long-term condition in which people depend on others to meet their emotional and physical needs, with only a minority achieving normal levels of independence. Dependent personality disorder (DPD) is characterized by excessive fear and anxiety. Dependent personality disorder (DPD) begins by early adulthood, is present in a variety of contexts, and is associated with inadequate functioning. Symptoms can include anything from extreme passivity, devastation, or helplessness when relationships end; avoidance of responsibilities; and severe submission.

People who have dependent personality disorder are overdependent on other people when it comes to making decisions. They cannot make a decision on their own as they need constant approval from other people. Consequently, individuals diagnosed with DPD tend to place the needs and opinions of others above their own as they do not have the confidence to trust their decisions. This kind of behavior can explain why people with DPD tend to show passive and clingy behavior. These individuals display a fear of separation and cannot stand being alone. When alone, they experience feelings of isolation and loneliness due to their overwhelming dependence on other people.

DSM-5 Criteria

DSM-5 refers to dependent personality disorder as a pervasive and excessive need to be taken care of, which leads to submissive and clinging behavior and fears of separation. This behavior begins by early adulthood and can be present in a variety of contexts.



Figure 12.14.1: People with DPD can be clingy and may constantly need the approval of others.

According to the DSM-5, the disorder is indicated by at least five of the following factors:

1. has difficulty making everyday decisions without an excessive amount of advice and reassurance from others.
2. needs others to assume responsibility for most major areas of their life.
3. has difficulty expressing disagreement with others because of fear of loss of support or approval.
4. has difficulty initiating projects or doing things on their own (because of a lack of self-confidence in judgment or abilities rather than a lack of motivation or energy).
5. goes to excessive lengths to obtain nurturance and support from others, to the point of volunteering to do things that are unpleasant.
6. feels uncomfortable or helpless when alone because of exaggerated fears of being unable to care for themselves.
7. urgently seeks another relationship as a source of care and support when a close relationship ends.
8. is unrealistically preoccupied with fears of being left to take care of themselves.

Differential Diagnosis

There are similarities between individuals with dependent personality disorder and individuals with borderline personality disorder in that they both have a fear of abandonment. Those with dependent personality disorder do not exhibit impulsive behavior, unstable affect, and poor self-image experienced by those with borderline personality disorder.

The following conditions are commonly comorbid with dependent personality disorder:

- mood disorders
- anxiety disorders
- adjustment disorder
- borderline personality disorder
- avoidant personality disorder
- histrionic personality disorder

Epidemiology

Based on a survey of 43,093 Americans, 0.49% of adults meet diagnostic criteria for DPD (National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; Grant et al., 2004). Traits related to DPD, like most personality disorders, emerge in childhood or early adulthood. DPD is more common among women compared to men as 0.6% of women have DPD compared to 0.4% of men. A 2004 twin study suggests a heritability of 0.81 for developing dependent personality disorder. Because of this, there is significant evidence that this disorder runs in families. Children and adolescents with a history of anxiety disorders and physical illnesses are more susceptible to acquiring this disorder.

Etiology



Figure **12.14.2**: Overprotective parenting behavior is a risk factor for DPD.

The exact cause of dependent personality disorder is unknown. A study in 2012 estimated that between 55% and 72% of the risk of the condition is inherited from one's parents. The difference between a dependent personality and a dependent personality disorder is somewhat subjective, which makes diagnosis sensitive to cultural influences such as gender role expectations. There is a higher frequency of the disorder seen in women than men.

Dependent traits in children tended to increase with parenting behaviors and attitudes characterized by overprotectiveness and authoritarianism. Traumatic or adverse experiences early in an individual's life, such as neglect and abuse or serious illness, can increase the likelihood of developing personality disorders, including dependent personality disorder, later on in life. This likelihood is especially prevalent for those individuals who also experience high interpersonal stress and poor social support.

Treatment

People who have DPD are generally treated with psychotherapy. The main goal of this therapy is to make the individual more independent and help them form healthy relationships with the people around them. This is done by improving their self-esteem and confidence.

Medication can be used to treat patients who suffer from depression or anxiety because of their DPD, but this does not treat the core problems caused by DPD.

? Case Study: DEPENDENT PERSONALITY DISORDER

This case study looks at the patient of a 27-year-old, white female named Sally. She works as an administrative assistant whose work required extensive data entry. Sally's mother used her network to get this stable job for her daughter. Sally is consumed with pleasing her mother and even asks for her mother's advice on what to wear to the office each day. Sally consistently worries about pleasing others and her colleagues at work, even at the expense to herself, which leads to the diagnosis of dependent personality disorder. The degree to which her self-destructive passivity and compliance at work stemmed from her early experiences within the family are unclear, but her parents' overprotectiveness likely played some role in the etiology of her personality pathology.

? Watch It

This video describes the diagnostic criteria and treatment options for dependent personality disorder.



You can view the transcript for "What is Dependent Personality Disorder?" here (opens in new window).

Learning Objectives

dependent personality disorder (DPD): a personality disorder characterized by a pervasive and excessive need to be taken care of, leading to a lack of independent action

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12.15: Obsessive-Compulsive Personality Disorder

Learning Objectives

- Describe the characteristics and diagnosis of obsessive-compulsive personality disorder

Now we will explore the final cluster C personality disorder, obsessive-compulsive personality disorder (OCPD). OCPD is different from the previously covered obsessive-compulsive disorder (OCD).

Table 1. DSM-5 Personality Disorders

DSM-5 Personality Disorder	Description	Cluster
Avoidant	socially inhibited and oversensitive to negative evaluation; avoids occupations that involve interpersonal contact because of fears of criticism or rejection; avoids relationships with others unless guaranteed to be accepted unconditionally; feels inadequate and views self as socially inept and unappealing; unwilling to take risks or engage in new activities if they may prove embarrassing	C
Dependent	allows others to take over and run their life; is submissive, clingy, and fears separation; cannot make decisions without advice and reassurance from others; lacks self-confidence; cannot do things on his or her own; feels uncomfortable or helpless when alone	C
Obsessive-Compulsive	pervasive need for perfectionism that interferes with the ability to complete tasks; preoccupied with details, rules, order, and schedules; excessively devoted to work at the expense of leisure and friendships; rigid, inflexible, and stubborn; insists things be done a certain way; miserly with money	C

Obsessive-Compulsive Personality Disorder



Figure **12.15.1**: People with OCPD may obsess over lists and rules.

Obsessive–compulsive personality disorder (OCPD) is a Cluster C personality disorder marked by an excessive need for orderliness, neatness, and perfectionism. Symptoms are usually present by the time a person reaches adulthood, and are visible in a variety of situations.

Obsessive-compulsive personality disorder (OCPD) is marked by an excessive obsession with rules, lists, schedules, and order; a need for perfectionism that interferes with efficiency and the ability to complete tasks; a devotion to productivity that hinders interpersonal relationships and leisure time; rigidity and zealousness on matters of morality and ethics; and an inability to delegate responsibilities or work to others. People with obsessive-compulsive personality disorder (OCPD) tend to be obsessed with controlling their environments; to satisfy this need for control, they become preoccupied with trivial details, lists, procedures, rules, and schedules.

Individuals with OCPD are at one extreme of the conscientiousness continuum. While conscientiousness is a desirable trait generally, its extreme presentation for those with OCPD leads to interpersonal problems. OCPD individuals present as over-controlled and this characteristic extends to the relationships they have with other people. Individuals with OCPD are referential to authority and rules. OCPD individuals may therefore punish those who violate their strict standards. The inability to accept differences in beliefs or behaviors from others often leads to high conflict and controlling relationships with coworkers, spouses, and children.

Differential Diagnosis

There are several mental disorders in the DSM-5 that are listed as differential diagnoses for OCPD. They are as follows:

- **obsessive-compulsive disorder.** OCD and OCPD may have a similar name wh,ich may cause confusion; however, OCD can be easily distinguished from OCPD by the absence of true obsessions or compulsions.
- **hoarding disorder.** A diagnosis of hoarding disorder is only considered when the hoarding behavior exhibited is causing severe impairment in the functioning of the person, such as an inability to access rooms in a house due to excessive hoarding.
- **narcissistic personality disorder (NPD).** Individuals with narcissistic personality disorder may believe that they are perfect and that no one else can be as perfect or right as them; however, people with narcissistic personality disorder usually believe that they have achieved perfection and cannot get better, whereas those with OCPD do not believe that they have achieved perfection and are self-critical. Those with NPD tend to be stingy and lack generosity; however, they are usually generous when spending on themselves, unlike those with OCPD who hoard money and are miserly towards themselves and others.
- **antisocial personality disorder.** Similarly, individuals with anti personality disorder are not generous and miserly around others, although they usually over-indulge themselves and are sometimes reckless in spending.
- **schizoid personality disorder.** Schizoid personality disorder and OCPD may both display restricted affectivity and coldness; however, in OCPD, this is usually due to a controlling attitude, whereas in SPD, it occurs due to a lack of ability to experience emotion and display affection.
- **Other personality traits.** Obsessive-compulsive personality traits may be particularly useful and helpful, especially in productive environments. Only when these traits become extreme, maladaptive, and cause clinically significant impairment in several aspects of one’s life should a diagnosis of OCPD be considered.
- **personality change due to another medical condition.** OCPD must be differentiated from a personality change due to a medical condition, which affects the central nervous system, and may cause changes in behavior and traits.
- **substance use disorders.** Substance use may cause the advent of obsessive-compulsive traits. It is necessary that this is distinguished from underlying and persistent behavior, which must occur when a person is not under influence of a substance.



Figure **12.15.2**: While obsessive-compulsive personality traits can sometimes be useful, they are usually excessive and can be damaging to ones life and relationships.

Etiology

The cause of OCPD is thought to involve a combination of genetic and environmental factors, namely problems with attachment. The cause of OCPD is thought to involve a combination of genetic and environmental factors. There is clear evidence to support the theory that OCPD is genetically inherited; however, the relevance and impact of genetic factors varies with studies placing it somewhere between 27% and 78%.[1] Too few studies have dealt with the specific gene involved in the disorder's heritability, and more research is required to ascertain the exact genes.

Other studies have found links between attachment theory and the development of OCPD. According to this hypothesis, those with OCPD have never developed a secure attachment style, had overbearing parents, were shown little care, and were unable to develop empathetically and emotionally.

Epidemiology

Estimates for the prevalence of OCPD in the general population range from 3%-8%, making it the most common personality disorder. Some studies show no gender differences, but others show OCPD more prevalent among men.

A diagnosis of OCPD is common with anxiety disorders, substance use disorders, and mood disorders. OCPD is also highly comorbid with Cluster A personality disorders, especially paranoid and schizotypal personality disorders. OCPD has also been linked to a higher relapse in those who are treated for major depressive disorder, and a higher risk of suicidal behavior. OCPD is also linked to hypochondriasis, with some studies estimating a rate of co-occurrence as high as 55.7%. In people with eating disorders, 13% also have OCPD.

Treatment

Treatment involves psychotherapy, such as CBT, and sometimes antidepressants.

✔ Obsessive-Compulsive Personality Disorder is Not Obsessive-Compulsive Disorder

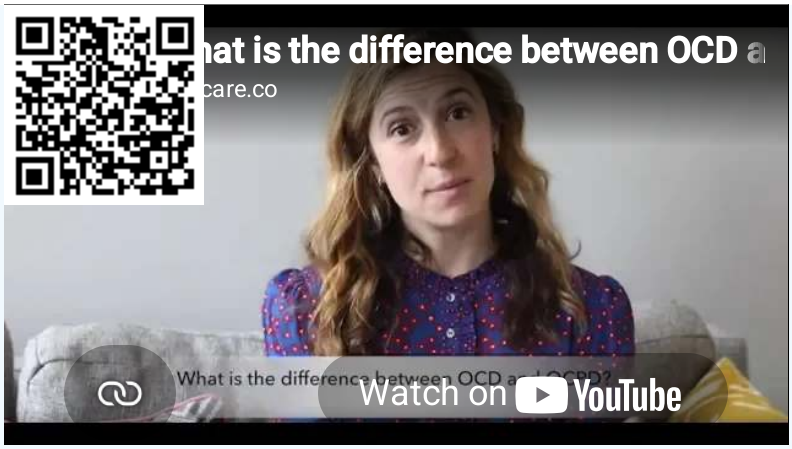
OCPD is often confused with OCD. Despite the similar names, they are two distinct disorders. Some OCPD individuals do have OCD, and the two can be found in the same family, sometimes along with eating disorders. People with OCPD do not generally feel the need to repeatedly perform ritualistic actions—a common symptom of OCD—and usually find pleasure in perfecting a task whereas people with OCD are often more distressed after their actions.

People with OCPD tend to be obsessed with controlling their environments; to satisfy this need for control, they become preoccupied with trivial details, lists, procedures, rules, and schedules. Symptoms are usually present by the time a person reaches adulthood, and are visible in a variety of situations.

Both disorders may share outside similarities; rigid and ritual-like behaviors, for example. Attitudes toward these behaviors differ between people affected with either of the disorders: for people with OCD, these behaviors are unwanted and involuntary, being the product of anxiety-inducing and involuntary thoughts. On the other hand, for people with OCPD, the person perceives them as rational and wanted, being the result of, for example, strong adherence to routines, a desire for control, or a need for perfection. OCPD is highly comorbid with other personality disorders, Asperger's syndrome, eating disorders, anxiety, mood disorders, and substance use disorders.

Obsessive-compulsive personality disorder (OCPD) is marked by an excessive obsession with rules, lists, schedules, and order; a need for perfectionism that interferes with efficiency and the ability to complete tasks; a devotion to productivity that hinders interpersonal relationships and leisure time; rigidity and zealousness on matters of morality and ethics; and an inability to delegate responsibilities or work to others.

This video explains the differences between OCD and OCPD:



You can view the transcript for “What is the difference between OCD and OCPD?” here (opens in new window).

Case Study: OCPD

Mark is a 42-year-old, single male who lives with his parents. He has been unemployed for some time because he has had difficulty maintaining employment. Most recently, he was not able to meet production demands at his factory job because he got consumed with making each package perfect before moving on to the next package. One weekend, when his family planned to visit the grandparents, Mark started packing on Wednesday afternoon but did not finish the packing until Saturday, when it was too late to go. Mark’s employment and personal relationships are impacted by his rigidity and extreme attention to detail. Mark was diagnosed with obsessive-compulsive personality disorder.

In therapy, Mark was very punctual in treatment and never missed a session; he talked freely and in great detail. The initial part of therapy mainly dealt with family relationships. When the time came to leave the sessions, he would often continue talking and delaying even when the therapist was standing at the door.

Glossary

obsessive-compulsive personality disorder: characterized by a preoccupation with orderliness, perfectionism, and rigidity in behaviors and relationships

obsessive-compulsive disorder: a pervasive pattern of preoccupation with orderliness, perfectionism, and mental and interpersonal control at the expense of flexibility, openness, and efficiency

1. Diedrich A, Voderholzer U (February 2015). "Obsessive-compulsive personality disorder: a current review". *Current Psychiatry Reports*. 17 (2): 2. doi:10.1007/s11920-014-0547-8. PMID 25617042. S2CID 20999600. ↵

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12.16: Introduction to Perspectives and Treatments Related to Personality Disorders

What you'll learn to do: examine various perspectives and treatment methods related to personality disorders



As we have learned, individuals diagnosed with personality disorders exhibit rigid and long term patterns of unhealthy thought, behavior, and functioning. These characteristics often cause significant challenges in personal and professional relationships.

The disorders in Cluster A include those characterized by a personality style that is odd and eccentric. Cluster B includes personality disorders characterized chiefly by a personality style that is impulsive, dramatic, highly emotional, and erratic, and those in Cluster C are characterized by a nervous and fearful personality style.

Now that we have identified the ten personality disorders, let's explore the different perspectives and treatment options.

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12.17: Perspectives on Personality Disorders

Learning Objectives

- Describe viewpoints from the major psychological perspectives related to the etiology of personality disorders

A Historical Understanding of Personality Disorders

As you have learned in this module, the DSM-5 defines personality disorders as "an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment." This definition has evolved over time.

The consideration of cultural context is perhaps the most significant change in this definition from the earlier DSM-3. Although the DSM-4-TR made a point of adding cultural context to the definition of personality disorders, it wasn't until the publication of the DSM-5 that much was said in the manual about culture. Not only is there limited research regarding the influence of culture on personality disorders, there is likewise only limited research on cultural influences on normal personality.

Biological Perspectives

Currently, genetic research for the understanding of the development of personality disorders is severely lacking. However, there are a few possible risk factors currently in discovery. Researchers are currently looking into genetic mechanisms for traits such as aggression, fear, and anxiety, which are associated with diagnosed individuals. More research is being conducted into disorder-specific mechanisms.

There are some genetic factors connected to biological or genetic components, but no specific mechanisms have been identified. This means that there is likely a biological predisposition for some personality disorders, but more research needs to be done to determine the precise connections.

Psychodynamic Perspectives



Figure **12.17.1**: An individual's personality can be influenced by things they went through as a child.

The psychodynamic theory on personality proposes events that happen to us in our childhood impacts our lives as we mature. Even if childhood events remain in our unconscious, significant problems may arise with our personalities in adolescence and adulthood. Psychodynamic theory views personality as exhibited behaviors and characteristics caused by unconscious factors that we do not have control over.

Cognitive Perspectives

Cognitive theorists propose a link between personality and disruptive and disordered thought patterns. Individuals with a personality disorder may develop inaccurate perceptions of others (Beck, 2015). There may be a relationship between biological factors and adverse environmental factors. Disordered patterns of perceiving and relating to the world may develop as protective measures during childhood. These patterns of behavior may be reflected as characteristics of a personality disorder.

Behavioral Perspectives

Behavioral theorists believe people behave in a consistent manner and view personality as being shaped by reinforcements and consequences. We learn to behave in certain ways, increasing behaviors that lead to positive consequences and decreasing behaviors that lead to negative outcomes. We have to consider modeling, reinforcement, and the inability to develop normal social skills. In modeling, an individual may learn dysfunctional social patterns and behaviors by directly observing family members

engaging in similar behaviors. With reinforcement, individuals may be rewarded by gaining their desired outcomes when they successfully manipulate others. Additionally, when there is exaggerated praise, individuals may develop a grandiose sense of self, similar to a narcissistic personality (Millon, 2011).

Although genetic studies on personality disorders are relatively rare, many researchers agree avoiding certain social situations may contribute to the development of personality disorders. It's not clear if lacking social skills leads to the avoidance of social settings or if lacking social skills develops as a result of avoiding social situations (APA, 2013).

Sociocultural Perspectives

We also have to consider how life experiences factor into the development of personality disorders. Trauma and abuse play key roles in possible causes of the ten personality disorders.



Figure **12.17.2**: There is a correlation between child neglect and the development of personality disorders in adulthood.

Child abuse and neglect consistently show up as risk factors to the development of personality disorders in adulthood. A study looked at retrospective reports of abuse of participants that had demonstrated psychopathology throughout their life and were later found to have past experience with abuse. In a study of 793 mothers and children, researchers asked mothers if they had screamed at their children, told them that they did not love them, or threatened to send them away. Children who had experienced such verbal abuse were three times as likely as other children (who did not experience such verbal abuse) to have borderline, narcissistic, obsessive-compulsive, or paranoid personality disorders in adulthood.[1] The sexually abused group demonstrated the most consistently elevated patterns of psychopathology. Officially verified physical abuse showed an extremely strong correlation with the development of antisocial and impulsive behavior.

Socioeconomic status has also been looked at as a potential cause for personality disorders. There is a strong association between low parental/neighborhood socioeconomic status and personality disorder symptoms. In a recent study comparing parental SES and a child's personality, it was seen that children who were from higher socioeconomic backgrounds were more altruistic, less risk-seeking, and had overall higher IQs.[2] A study looking at female children who were detained for disciplinary actions found that psychological problems were most negatively associated with socioeconomic problems.

Parenting

Evidence shows personality disorders may begin with parental personality issues. These cause the parent to have his or her own difficulties in adulthood, such as difficulties reaching higher education, obtaining jobs, and securing dependable relationships. By either genetic or modeling mechanisms, children can pick up these traits. Additionally, poor parenting appears to have symptom elevating effects on personality disorders. More specifically, lack of maternal bonding has also been correlated with personality disorders. In a study comparing 100 healthy individuals to 100 borderline personality disorder patients, analysis showed that BPD patients were significantly more likely not to have been breastfed as a baby (42.4% in BPD vs. 9.2% in healthy controls).[3] These researchers suggested this act may be essential in fostering maternal relationships. When left unfostered, other attachment and interpersonal problems occur later in life ultimately leading to the development of personality disorders.

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2. Deckers, Thomas (February 2015). "How does Socio-Economic Status Shape a Child's Personality?" (PDF). *Human Capital and Economic Opportunity Global Working Group*. ↵
3. Schwarze, Cornelia E.; Hellhammer, Dirk H.; Stroehle, Verena; Lieb, Klaus; Mobascher, Arian (23 September 2014). "Lack of Breastfeeding: A Potential Risk Factor in the Multifactorial Genesis of Borderline Personality Disorder and Impaired Maternal

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12.18: Treatments for Personality Disorders

Learning Objectives

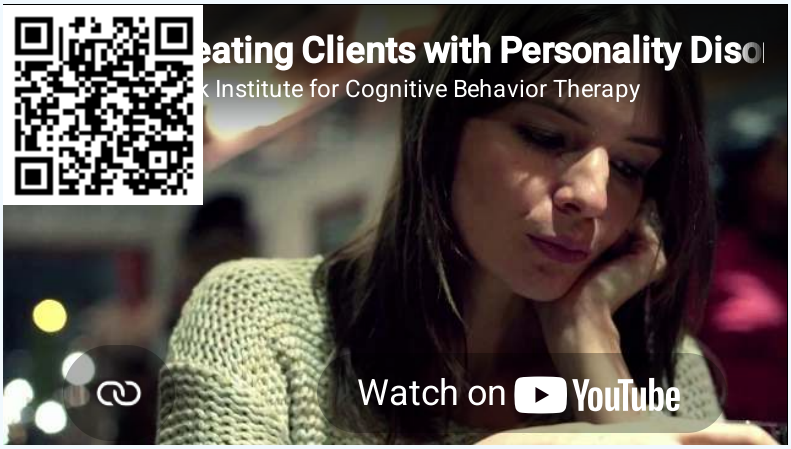
- Describe treatment options and complications in treating personality disorders

Personality disorders are oftentimes difficult to treat. Your personality, the characteristics that define you, is unique to you and you alone. What happens when it is your personality that actually causes challenges in life? There may not be a baseline of healthy behaviors or relationships as a result of the personality. More than likely, it would be a challenge to admit there is a problem and to seek treatment.

Most people are relatively unique because they are often **ego-syntonic**; that is, most people are largely comfortable with themselves, with their characteristic manner of behaving, feeling, and relating to others. As a result, people rarely seek treatment for their antisocial, narcissistic, histrionic, paranoid, and/or schizoid personality disorder. There are times when personality disorders are revealed, however, when people seek treatment for anxiety, mood, or substance use disorders.

Watch It

This video explains how some negative beliefs, often entrenched in those with personality disorders, make it difficult to treat the disorders.



You can view the transcript for "Treating Clients with Personality Disorders" here (opens in new window).

Treatment for Personality Disorders

It is a misnomer, though, to suggest that personality disorders cannot themselves be treated. Personality disorders are among the most difficult of disorders to treat because they involve well-established behaviors that can be integral to a client's self-image (Millon, 2011). Nevertheless, much has been written on the treatment of personality disorder (e.g., Beck, Freeman, Davis, & Associates, 1990; Gunderson & Gabbard, 2000), and there is empirical support for clinically and socially meaningful changes in response to psychosocial and pharmacologic treatments (Perry & Bond, 2000). The development of an ideal or fully healthy personality structure is unlikely to occur through the course of treatment, but given the considerable social, public health, and personal costs associated with some of the personality disorders, such as the antisocial and borderline, even just moderate adjustments in personality functioning can represent quite significant and meaningful change.

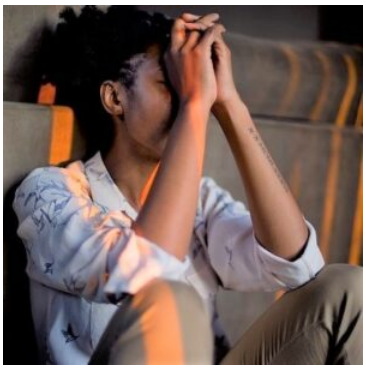


Figure 12.18.1: Many people with personality disorders do not seek treatment. Those with borderline personality disorder and avoidant personality disorder are exceptions. High levels of neuroticism and emotional pain may motivate them to seek help. [Image: CC0 Public Domain, https://goo.gl/m25gce]

Some modalities of treatment used for personality disorders include

- individual psychotherapy;
- family therapy, including couples therapy;
- group therapy for personality dysfunction;
- psychological-education and self-help groups;
- psychiatric medications for treating symptoms of personality dysfunction or co-occurring conditions;
- Milieu therapy, a kind of group-based residential approach, has a history of use in treating personality disorders, including therapeutic communities; and
- the practice of mindfulness that includes developing the ability to be nonjudgmentally aware of unpleasant emotions appears to be a promising clinical tool for managing different types of personality disorders.

There are different specific theories or schools of therapy within many of these modalities. They may, for example, emphasize psychodynamic techniques or cognitive or behavioral techniques. In clinical practice, many therapists use an eclectic approach, taking elements of different schools when they seem to fit to an individual client. There is also often a focus on common themes that seem to be beneficial regardless of techniques, including attributes of the therapist (e.g., trustworthiness, competence, and caring), processes afforded to the client (e.g., ability to express and confide difficulties and emotions), and the match between the two (e.g., aiming for mutual respect, trust, and boundaries).

Response of Patients with Personality Disorders to Biological and Psychosocial Treatments

Cluster	Evidence for Brain Dysfunction	Response to Biological Treatments	Response to Psychosocial Treatments
A	Evidence for relationship to schizophrenia; otherwise none known	Schizotypal patients may improve on antipsychotic medication; otherwise not indicated	Poor. Supportive psychotherapy may help. They are often distanced or distrustful of the therapist and therapy moves slow.
B	Evidence for relationship to bipolar disorder; otherwise none known	Antidepressants, antipsychotics, or mood stabilizers may help for borderline personality; otherwise not indicated	Responses are poor in treating antisocial personality disorder. Varies in treating borderline, narcissistic, and histrionic personality disorders. DBT is known to treat BPD.
C	Evidence for relationship to generalized anxiety disorder; otherwise none known	No direct response. Medications may help with comorbid anxiety and depression	Patients often seek out treatment for other symptoms, such as depression or anxiety. Responses to treatment vary.

Manualized and/or empirically validated treatment protocols have been developed for only one personality disorder: borderline (APA, 2001).

? Treatment of Borderline Personality Disorder

Dialectical behavior therapy (DBT) (Lynch & Cuyper, 2012) and mentalization therapy (Bateman & Fonagy, 2012): Dialectical behavior therapy is a form of cognitive-behavior therapy that draws on principles from Zen Buddhism, dialectical philosophy, and behavioral science. The treatment has four components, individual therapy, group skills training, telephone coaching, and a therapist consultation team, and will typically last a full year. As such, it is a relatively expensive form of treatment, but research has indicated that its benefits far outweighs its costs, both financially and socially.

The main skills taught in DBT include mindfulness, interpersonal effectiveness, emotion regulation, and distress tolerance. Within DBT, mindfulness refers to the capacity to pay attention, nonjudgmentally, to the present moment and living in the moment, experiencing one's emotions and senses fully, yet with perspective. The practice of mindfulness can also be intended to make people more aware of their environments through their five senses: touch, smell, sight, taste, and sound. Mindfulness relies heavily on the principle of acceptance, sometimes referred to as "radical acceptance." Acceptance skills rely on the patient's ability to view situations with no judgment, and to accept situations and their accompanying emotions. These skills cause less distress overall, which can result in reduced discomfort and symptomology.

This video from the Centre for Addiction and Mental Health (CAMH) discusses how DBT is used to treat BPD and highlights how they are working on shorter treatment programs to enable greater access to treatment.

It is unclear why specific and explicit treatment manuals have not been developed for other personality disorders. This may reflect a regrettable assumption that personality disorders are unresponsive to treatment. It may also reflect the complexity of their treatment. As noted earlier, each DSM-5 disorder is a heterogeneous constellation of maladaptive personality traits. In fact, a person can meet diagnostic criteria for antisocial, borderline, schizoid, schizotypal, narcissistic, and avoidant personality disorders and yet have only one diagnostic criterion in common. For example, only five of nine features are necessary for the diagnosis of borderline personality disorder; therefore, two persons can meet criteria for this disorder and yet have only one feature in common. In addition, patients meeting diagnostic criteria for one personality disorder will often meet diagnostic criteria for another. This degree of diagnostic overlap and heterogeneity of membership hinders tremendously any effort to identify a specific etiology, pathology, or treatment for a respective personality disorder as there is so much variation within any particular group of patients sharing the same diagnosis (Smith & Zapolski, 2009).

Of course, this diagnostic overlap and complexity did not prevent researchers and clinicians from developing dialectical behavior therapy and mentalization therapy. A further reason for the weak progress in treatment development is that, as noted earlier, persons rarely seek treatment for their personality disorder. It would be difficult to obtain a sufficiently large group of people with, for instance, narcissistic or obsessive-compulsive disorder to participate in a treatment outcome study, one receiving the manualized treatment protocol, the other receiving treatment as usual.



Figure **12.18.2**: For Cluster A disorders, one-on-one therapy is recommended over group therapy.

The best strategy for a clinician to implement is to help the patient develop new behavior in the face of adversity. For each disorder, the clinician should emphasize different tactics. For Cluster A disorders, group therapy may not be the wisest as patients tend to be suspicious and distrustful; instead, individual social skills training may be most beneficial.

One clear exception though is borderline personality disorder (and perhaps avoidant personality disorder as well). Neuroticism is the domain of general personality structure that concerns inherent feelings of emotional pain and suffering, including feelings of

distress, anxiety, depression, self-consciousness, helplessness, and vulnerability. Persons who have very high elevations on neuroticism (i.e., persons with borderline personality disorder) experience life as one of pain and suffering, and they will seek treatment to alleviate this severe emotional distress. People with avoidant personality may also seek treatment for their high levels of neuroticism (anxiousness and self-consciousness) and introversion (social isolation). In contrast, narcissistic individuals will rarely seek treatment to reduce their arrogance; paranoid persons rarely seek treatment to reduce their feelings of suspiciousness; and antisocial people rarely (or at least willfully) seek treatment to reduce their disposition for criminality, aggression, and irresponsibility.

Nevertheless, maladaptive personality traits will be evident in many individuals seeking treatment for other mental disorders, such as anxiety, mood, or substance use. Many of the people with a substance use disorder will have antisocial personality traits; many of the people with mood disorder will have borderline personality traits. The prevalence of personality disorders within clinical settings is estimated to be well above 50% (Torgersen, 2012). As many as 60% of inpatients within some clinical settings are diagnosed with borderline personality disorder (APA, 2000). Antisocial personality disorder may be diagnosed in as many as 50% of inmates within a correctional setting (Hare et al., 2012). It is estimated that 10–15% of the general population meets criteria for at least one of the 10 DSM-4-TR personality disorders (Torgersen, 2012), and quite a few more individuals are likely to have maladaptive personality traits not covered by one of the 10 DSM-5 diagnoses.

The presence of a personality disorder will often have an impact on the treatment of other mental disorders, typically inhibiting or impairing responsivity. Antisocial persons will tend to be irresponsible and negligent; borderline persons can form intensely manipulative attachments to their therapists; paranoid patients will be unduly suspicious and accusatory; narcissistic patients can be dismissive and denigrating; and dependent patients can become overly attached to and feel helpless without their therapists.

Glossary

dialectical behavior therapy: a form of CBT developed by Marsha M. Linehan to help people increase their emotional and cognitive regulation by learning about the triggers that lead to reactive states and helping to assess which coping skills to apply

ego-syntonic: the behaviors, values, and feelings that are in harmony with or acceptable to the needs and goals of the ego, or consistent with one's ideal self-image

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12.19: Case Studies- Personality Disorders

Learning Objectives

- Identify personality disorders in case studies

Case Study: Latasha

Latasha was a 20-year-old college student who lived in the dorms on campus. Classmates described Latasha as absent-minded and geeky because she didn't interact with others and rarely, if ever, engaged with classmates or professors in class. She usually raced back to her dorm as soon as classes were over. Latasha primarily stayed in her room, did not appear to have any friends, and had no interest in the events happening on campus. Latasha even asked for special permission to stay on campus when most students went home for Thanksgiving break.

Now let's examine some fictional case studies.

Case Study: The Mad Hatter



The Mad Hatter, from *Alice in Wonderland*, appears to be living in a forest that is part of Alice's dream. He appears to be in his mid-thirties, is Caucasian, and dresses vibrantly. The Mad Hatter climbs on a table, walks across it, and breaks plates and teacups along the way. He is rather protective of Alice; when the guards of the Queen of Hearts come, he hides Alice in a tea kettle. Upon making sure that Alice is safe, Mad Hatter puts her on his hat, after he had shrunk her, and takes her for a walk. While walking, he starts to talk about the Jabberwocky and becomes enraged when Alice tells him that she will not slay the Jabberwocky. Talking to Alice about why she needs to slay the Jabberwocky, the Mad Hatter becomes emotional and tells Alice that she has changed.

The Mad Hatter continues to go to lengths to protect Alice; he throws his hat with her on it across the field, so the Queen of Heart's guards do not capture her. He lies to the Queen and indicates he has not seen Alice, although she is clearly sitting next to the Queen. He decides to charm the Queen, by telling her that he wants to make her a hat for her rather large head. Once the White Queen regained her land again, the Mad Hatter is happy.

Case Study: The Grinch



The Grinch, who is a bitter and cave-dwelling creature, lives on the snowy Mount Crumpits, a high mountain north of Whoville. His age is undisclosed, but he looks to be in his 40s and does not have a job. He normally spends a lot of his time alone in his cave. He is often depressed and spends his time avoiding and hating the people of Whoville and their celebration of Christmas. He disregards the feelings of the people, knowingly steals and destroys their property, and finds pleasure in doing so. We do not know

his family history, as he was abandoned as a child, but he was taken in by two ladies who raised him with a love for Christmas. He is green and fuzzy, so he stands out among the Whos, and he was often ridiculed for his looks in school. He does not maintain any social relationships with his friends and family. The only social companion the Grinch has is his dog, Max. The Grinch had no goal in his life except to stop Christmas from happening. There is no history of drug or alcohol use.

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12.20: Putting It Together- Personality Disorders



The term *personality disorder* encompasses a wide range of issues, each with a different level of severity or disability; thus, personality disorders can require fundamentally different approaches, understandings, and possible treatments. In this module, you learned about three different clusters and ten unique disorders: paranoid, schizoid, schizotypal, antisocial, borderline, histrionic, narcissistic, avoidant, dependent, and obsessive-compulsive personality disorder. It is evident that all individuals have a personality, as indicated by their characteristic way of thinking, feeling, behaving, and relating to others. For some people, these traits result in a considerable degree of distress and/or impairment, constituting a personality disorder. To be diagnosed with a personality disorder, the following criteria must be met:

- The individual has an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture. This pattern is manifested in two (or more) of the following areas:
  - cognition (i.e., ways of perceiving and interpreting self, other people, and events).
  - affectivity (i.e., the range, intensity, lability, and appropriateness of emotional response).
  - interpersonal functioning.
  - impulse control.
- The enduring pattern is inflexible and pervasive across a broad range of personal and social situations.
- The enduring pattern leads to clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- The pattern is stable and of long duration, and its onset can be traced back at least to adolescence or early adulthood.
- The enduring pattern is not better explained as a manifestation or consequence of another mental disorder.
- The enduring pattern is not attributable to the physiological effects of a substance (e.g., drug abuse or a medication) or another medical condition (e.g., head trauma).

A considerable body of research has accumulated to help understand the etiology, pathology, and/or treatment for some personality disorders (i.e., antisocial, schizotypal, borderline, dependent, and narcissistic), but not so much for others (e.g., histrionic, schizoid, and paranoid). However, researchers and clinicians are now shifting toward a more dimensional understanding of personality disorders wherein each is understood as a maladaptive variant of general personality structure, thereby bringing to bear all that is known about general personality functioning to an understanding of these maladaptive variants.

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CHAPTER OVERVIEW

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13.1: Why It Matters- Disorders of Childhood and Adolescense

Why learn about disorders of childhood and adolescence?

Childhood mental and developmental disorders encompass neurodevelopmental, emotional, and behavioral disorders that have broad and serious adverse impacts on psychological and social well-being. Children with these disorders typically require significant additional support from families and the educational system.



Figure 13.1.1: Children with mental health and developmental disorders are more likely to experience a compromised developmental trajectory, with an increased need for medical and disability services as well as increased risk of contact with law enforcement agencies.

Childhood mental and developmental disorders are an emerging challenge to health care systems globally. Two contributing factors are the increases in the proportion of children and adolescents in the populations of low- to middle-income countries, which is a result of reduced mortality of children under age five years and the fact that the onset of many adult mental and developmental disorders occurs in childhood and adolescence.

The consequences of these disorders include the impact during childhood and the persistence of mental ill-health into adult life. In childhood, the impact is broad, encompassing the individual suffering of children, as well as the potential negative effects on their families and peers. This impact may include aggression toward other children and the distraction of peers from learning. These children are at higher risk of mental and physical health problems in adulthood, as well as increased likelihood of unemployment, contact with law enforcement agencies, and need for disability support.

Child maltreatment is a well-established risk factor for mental and developmental disorders in children, including any form of physical or emotional ill-treatment, sexual abuse, neglect or negligent treatment, or commercial or other exploitation that results in actual or potential harm to a child's health, survival, development, or dignity in the context of a relationship of responsibility, trust, or power. Legislation to address child maltreatment requires the support of well-integrated systems that increase public awareness and enable incident reporting to a constituted authority with investigative and interventional expertise and the ability to prosecute.

Children are our future and we have a responsibility to protect the next generation by ensuring their survival—not simply their physical well-being, but their emotional and mental health as well.

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13.2: Introduction to Neurodevelopmental Disorders

What you'll learn to do: examine and explain neurodevelopmental disorders



Neurodevelopmental disorders are a group of disorders that are typically diagnosed during childhood and are characterized by developmental deficits in personal, social, academic, and intellectual realms; these disorders include attention deficit/hyperactivity disorder (ADHD) and autism spectrum disorder as well as other intellectual disabilities and learning disorders. ADHD is characterized by a pervasive pattern of inattention and/or hyperactive and impulsive behavior that interferes with normal functioning. Genetic and neurobiological factors contribute to the development of ADHD, which can persist well into adulthood and is often associated with poor long-term outcomes. The major features of autism spectrum disorder include deficits in social interaction and communication and repetitive movements or interests. As with ADHD, genetic factors appear to play a prominent role in the development of autism spectrum disorder; exposure to environmental pollutants such as mercury have also been linked to the development of this disorder. Although it is believed by some that autism is triggered by the MMR vaccination, evidence does not support this claim.

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13.3: Intellectual Disabilities

Learning Objectives

- Describe intelligence and how it is measured
- Explain diagnostic criteria for intellectual disabilities

Understanding Intelligence

Intellectual functioning, generally called **intelligence**, includes a wide range of mental activities such as the ability of logical reasoning and practical intelligence (problem-solving), ability in learning, verbal skills, and so on. It manifests and expresses itself through a numerous set of capabilities, behaviors, thoughts, and emotions. In other words, intellectual functioning is definable as the global ability that allows the individual to understand reality and interact with it.



Figure **13.3.1**: Children with high scores on intelligence tests tend to learn more of what is taught in school than their lower-scoring peers

Intellectual functioning is commonly measured by the **intelligence quotient** (IQ), which represents a total score obtained from standardized tests (IQ tests) developed for evaluating human intelligence. IQ test score has a median of 100 and a standard deviation of 15.

Intellectual Disability

Intellectual disability (ID), also called intellectual developmental disorder or a general learning disability, is a generalized neurodevelopmental disorder characterized by significantly impaired intellectual and adaptive functioning. Intellectual disability (ID) is defined by an IQ under 70 (two standard deviations below the median), in addition to deficits in two or more adaptive behaviors that affect everyday, general living.

Once focused almost entirely on cognition, the definition now includes both a component relating to mental functioning and one relating to an individual's functional skills in their daily environment. As a result of this focus on the person's abilities in practice, a person with an unusually low IQ may still not be considered to have Intellectual disability (ID).

Intellectual disability is subdivided into **syndromic intellectual disability**, in which intellectual deficits associated with other medical and behavioral signs and symptoms are present, and **non-syndromic intellectual disability**, in which intellectual deficits appear without other abnormalities. Down syndrome and fragile X syndrome are examples of syndromic intellectual disabilities.

Intellectual disability affects about 2%-3% of the general population, of which 75–90% of the affected people have mild intellectual disability.

Intellectually Disabled Criminals and Capital Punishment

The case of *Atkins v. Virginia* was a landmark case in the United States Supreme Court. On August 16, 1996, two men, Daryl Atkins and William Jones, robbed, kidnapped, and then shot and killed Eric Nesbitt, a local airman from the U.S. Air Force. A clinical psychologist evaluated Atkins and testified at the trial that Atkins had an IQ of 59. The mean IQ score is 100. The psychologist concluded that Atkins was mildly intellectually disabled. The jury found Atkins guilty, and he was sentenced to death. Atkins and his attorneys appealed to the Supreme Court. In June 2002, the Supreme Court reversed a previous decision and ruled that executions of mentally retarded criminals are "cruel and unusual punishments" prohibited by the Eighth Amendment. The court wrote in their decision:

> Clinical definitions of mental retardation require not only subaverage intellectual functioning, but also significant limitations in adaptive skills. Mentally retarded persons frequently know the difference between right and wrong and are competent to stand trial. Because of their impairments, however, by definition they have diminished capacities to understand and process information, to communicate, to abstract from mistakes and learn from experience, to engage in logical reasoning, to control impulses, and to understand others' reactions. Their deficiencies do not warrant an exemption from criminal sanctions, but diminish their personal culpability (*Atkins v. Virginia*, 2002, par. 5).
>
> The court also decided that there was a state legislature consensus against the execution of the mentally retarded and that this consensus should stand for all the states. The Supreme Court ruling left it up to the states to determine their own definitions of mental retardation and intellectual disability. The definitions vary among states as to who can be executed. In the Atkins case, a jury decided that because he had many contacts with his lawyers and thus was provided with intellectual stimulation, his IQ had reportedly increased, and he was now smart enough to be executed. He was given an execution date and then received a stay of execution after it was revealed that lawyers for co-defendant, William Jones, coached Jones to "produce a testimony against Mr. Atkins that did match the evidence" (Liptak, 2008). After the revelation of this misconduct, Atkins was re-sentenced to life imprisonment. *Atkins v. Virginia* (2002) highlights several issues regarding society's beliefs around intelligence. In the Atkins case, the Supreme Court decided that intellectual disability *does* affect decision-making and therefore should affect the nature of the punishment such criminals receive. Where, however, should the lines of intellectual disability be drawn? In May 2014, the Supreme Court ruled in a related case (*Hall v. Florida*) that IQ scores cannot be used as a final determination of a prisoner's eligibility for the death penalty (Roberts, 2014).

Measuring Intelligence

The value of IQ testing is most evident in educational or clinical settings. Children who seem to be experiencing learning difficulties or severe behavioral problems can be tested to ascertain whether the child's difficulties can be partly attributed to an IQ score that is significantly different from the mean for her age group. Without IQ testing—or another measure of intelligence—children and adults needing extra support might not be identified effectively. In addition, IQ testing is used in courts to determine whether a defendant has special or extenuating circumstances that preclude him from participating in some way in a trial. People also use IQ testing results to seek disability benefits from the Social Security Administration.

Bell Curves

The results of intelligence tests follow the bell curve, a graph in the general shape of a bell. When the bell curve is used in psychological testing, the graph demonstrates a normal distribution of a trait, in this case, intelligence, in the human population. Many human traits naturally follow the bell curve. For example, if you lined up all your female schoolmates according to height, it is likely that a large cluster of them would be the average height for an American woman: 5'4"–5'6". This cluster would fall in the center of the bell curve, representing the average height for American women. There would be fewer women who stand closer to 4'11". The same would be true for women of above-average height: those who stand closer to 5'11".

The trick to finding a bell curve in nature is to use a large sample size. Without a large sample size, it is less likely that the bell curve will represent the wider population. A **representative sample** is a subset of the population that accurately represents the general population. If, for example, you measured the height of the women in your classroom only, you might not actually have a representative sample. Perhaps the women's basketball team wanted to take this course together, and they are all in your class. Because basketball players tend to be taller than average, the women in your class may not be a good representative sample of the population of American women. But if your sample included all the women at your school, it is likely that their heights would form a natural bell curve.

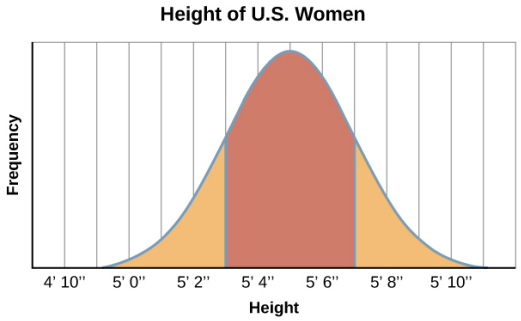


Figure **13.3.2**: Are you of below-average, average, or above-average height?

The same principles apply to intelligence tests scores. Individuals earn a score called an intelligence quotient (IQ). Over the years, different types of IQ tests have evolved, but the way scores are interpreted remains the same. The average IQ score on an IQ test is 100. **Standard deviations** describe how data are dispersed in a population and give context to large data sets. The bell curve uses the standard deviation to show how all scores are dispersed from the average score (Figure **13.3.3**). In modern IQ testing, one standard deviation is 15 points. So a score of 85 would be described as "one standard deviation below the mean." How would you describe a score of 115 and a score of 70? Any IQ score that falls within one standard deviation above and below the mean (between 85 and 115) is considered average, and 82% of the population has IQ scores in this range. An IQ score of 130 or above is considered a superior level.

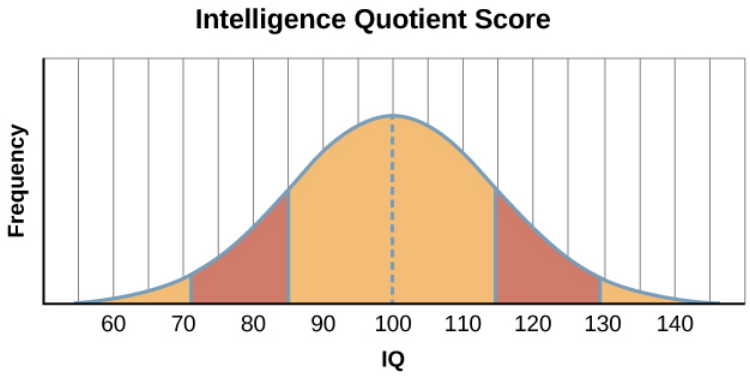


Figure **13.3.3**: The majority of people have an IQ score between 85 and 115.

Only 2.2% of the population has an IQ score below 70 (American Psychological Association [APA], 2013). A score of 70 or below indicates significant cognitive delays, major deficits in adaptive functioning, and difficulty meeting "community standards of personal independence and social responsibility" when compared to same-aged peers (APA, 2013, p. 37). An individual in this IQ range would be considered to have an intellectual disability and exhibit deficits in intellectual functioning and adaptive behavior (American Association on Intellectual and Developmental Disabilities, 2013). Formerly known as mental retardation, the accepted term now is intellectual disability, and it has four subtypes: mild, moderate, severe, and profound. The DSM lists criteria for each subgroup (APA, 2013).

Table 1. Characteristics of Cognitive Disorders

Intellectual Disability Subtype	Percentage of Intellectually Disabled Population	Description
Mild	85%	third- to sixth-grade skill level in reading, writing, and math; may be employed and live independently
Moderate	10%	Basic reading and writing skills; functional self-care skills; requires some oversight
Severe	5%	Functional self-care skills; requires oversight of daily environment and activities
Profound	<1%	May be able to communicate verbally or nonverbally; requires intensive oversight

Diagnostic Criteria

According to both the American Association on Intellectual and Developmental Disabilities (Intellectual Disability: Definition, Classification, and Systems of Supports (11th Edition) and the DSM-5, three criteria must be met for a diagnosis of intellectual disability:

- significant limitation in general mental abilities (intellectual functioning),
- significant limitations in one or more areas of adaptive behavior across multiple environments (as measured by an adaptive behavior rating scale, i.e., communication, self-help skills, interpersonal skills, and more)
- evidence that the limitations became apparent in childhood or adolescence.

It is formally diagnosed by an assessment of IQ and adaptive behavior. A third condition requiring onset during the developmental period is used to distinguish intellectual disability from other conditions, such as traumatic brain injuries and dementias (including Alzheimer's disease).

Levels of intellectual disability vary greatly in children. Children with intellectual disability might have a hard time letting others know their wants and needs, and taking care of themselves. Intellectual disability could cause a child to learn and develop more slowly than other children of the same age. It could take longer for a child with intellectual disability to learn to speak, walk, dress, or eat without help, and they could have trouble learning in school.

Intellectual disability can be caused by a problem that starts any time before a child turns 18 years old—even before birth. It can be caused by injury, disease, or a problem in the brain. For many children, the cause of their intellectual disability is not known. Some of the most common known causes of intellectual disability—like Down syndrome, fetal alcohol syndrome, fragile X syndrome, genetic conditions, birth defects, and infections—happen before birth. Others happen while a baby is being born or soon after birth. Still other causes of intellectual disability do not occur until a child is older; these might include serious head injury, stroke, or certain infections.

What are some of the signs of intellectual disability?

Usually, the more severe the degree of intellectual disability, the earlier the signs can be noticed. However, it might still be hard to tell how young children will be affected later in life.

There are many signs of intellectual disability. For example, children with intellectual disability may

- sit up, crawl, or walk later than other children;
- learn to talk later or have trouble speaking;
- find it hard to remember things;
- have trouble understanding social rules;
- have trouble seeing the results of their actions; or
- have trouble solving problems.

Dig Deeper: What's in a Name? Mental Retardation

In the past, individuals with IQ scores below 70 and significant adaptive and social functioning delays were diagnosed with mental retardation. When this diagnosis was first named, the title held no social stigma. In time, however, the degrading word *retard* sprang from this diagnostic term. *Retard* was frequently used as a taunt, especially among young people, until the words *mentally retarded* and *retard* became an insult. As such, the DSM-5 now labels this diagnosis as *intellectual disability*. Many states once had a Department of Mental Retardation to serve those diagnosed with such cognitive delays, but most have changed their name to Department of Developmental Disabilities or something similar in language. The Social Security Administration still uses the term *mental retardation* but is considering eliminating it from its programming (Goad, 2013). Earlier in the module, we discussed how language affects how we think. Do you think changing the title of this department has any impact on how people regard those with developmental disabilities? Does a different name give people more dignity, and if so, how? Does it change the expectations for those with developmental or cognitive disabilities? Why or why not?

Glossary

intelligence: one's intellectual functioning, the overall ability to gain and apply knowledge, including a wide range of mental activities such as logical reasoning, problem-solving, learning, verbal skills, etc.

intellectual disability: a generalized neurodevelopmental disorder characterized by significantly impaired intellectual and adaptive functioning. It is defined by an IQ under 70 (two standard deviations below the median), in addition to deficits in two or more adaptive behaviors that affect everyday, general living

intelligence quotient (IQ): score on a test designed to measure intelligence

non-syndromic intellectual disability: intellectual deficits that appear without other abnormalities

norming: administering a test to a large population so data can be collected to reference the normal scores for a population and its groups

representative sample: subset of the population that accurately represents the general population

standard deviation: measure of variability that describes the difference between a set of scores and their mean

standardization: method of testing in which administration, scoring, and interpretation of results are consistent

syndromic intellectual disability: intellectual deficits associated with other medical and behavioral signs and symptoms that are present, such as in the case of those with Down syndrome or fragile X syndrome

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13.4: Etiology and Treatment for Intellectual Developmental Disorders

Learning Objectives

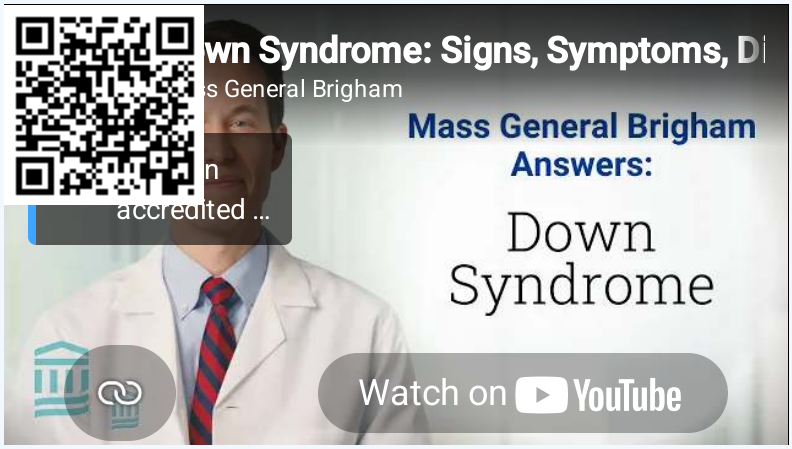
- Describe common causes of intellectual disability, including fetal alcohol syndrome, Down syndrome, and fragile X syndrome
- Examine the management and treatment of intellectual developmental disorders

Etiology

While many causes of intellectual disability are not known, the etiology of intellectual disability mainly divides into genetic abnormalities and environmental exposure. Genetic abnormality can be a single gene mutation, copy number variation, or chromosomal abnormality that causes an inborn error of metabolism, neurodevelopmental defect, and neurodegeneration. Environment exposure can be maternal exposure to toxin/infectious agents, uncontrolled maternal medical conditions, delivery complications, and postnatal trauma and exposure to toxin/infectious agents. The most common known preventable or environmental cause of intellectual disability is **fetal alcohol syndrome**, the most common chromosomal cause is **Down syndrome**, and the most common genetic cause is **fragile X syndrome**.

Watch It: Down Syndrome

Watch this video for an overview of the causes and symptoms of Down syndrome.



Genetic

The genetic abnormality may cause an inborn error of metabolism neurodevelopmental defect, or neurodegeneration. An inborn error of metabolism, toxic by-products accumulate, causing intellectual disability and other behavioral problems. Phenylketonuria (PKU) is one of the inborn errors of metabolism that occurs in approximately 0.01% of the newborn. Phenylketonuria (PKU) most commonly results from phenylalanine hydroxylase defect inherited in an autosomal recessive fashion. The defect disables the liver from converting phenylalanine to para-tyrosine. The consequent accumulation of phenylalanine is the culprit of intellectual disability. Neurological damage from phenylketonuria (PKU) is irreversible but preventable. Therefore, newborn screening of PKU is mandatory in the United States and other countries. Early screening followed by prompt initiation of a low phenylalanine diet before the age of three may prevent intellectual disability. Recently, different rare types of hyperphenylalaninemia were described, including the deficiency of the enzyme dihydropyridine reductase and a deficiency of a cofactor, biopterin.

Lesch-Nyhan syndrome is an *X*-linked inborn error of metabolism caused by purine metabolism enzyme deficiency. This condition results from a mutation in HGPRT. This mutation leads to a buildup of uric acid, which causes severe self-mutilating behavior of biting mouth and finger as well as intellectual disability. Other known inborn errors of metabolism that result in intellectual disability are Niemann-Pick disease, Hunter disease, Hurler disease, maple syrup urine disease, Hartnup disease, homocystinuria, and galactosemia.

A neurodevelopmental defect presents in fragile X syndrome, a leading genetic cause of intellectual disability. It results from a single gene mutation in FMR1 (Xq27.3) gene. In most cases, CGG repeat of the FMR1 gene expands to over 200 times. The expansion leads to a phosphorylated CG pattern, causing methylation imprinting of the gene, thereby silencing FMR1 gene expression. FMR1 is a transcription factor of hundreds of genes expressed in the central nervous system, and its disruption causes intellectual disability as well as behavioral disturbance, and seizure. Neurodevelopment defect also presents in neurofibromatosis type 1, also known as von Recklinghausen syndrome, an autosomal dominant condition, caused by mutations of NF1. Its characteristic presentation is abnormal neural cell migration leading to cafe au lait spot, movement disorder, and intellectual disability.

Watch It: Fragile X Syndrome

Watch this video for an overview of the causes and symptoms of Fragile X syndrome.



You can view the transcript for "Fragile X Syndrome – causes, symptoms, diagnosis, treatment, pathology" here (opens in new window).

Neurodegeneration leading to intellectual disability presents in Rett syndrome (RS), an *X*-linked dominant degenerative condition only seen in female secondary to mutation of the MeCP2 gene. In patients with Rett syndrome (RS), cerebral atrophy (the loss of brain cells over time) occurs at a part of the midbrain known as the substantia nigra, causing defects in the way dopamine is released throughout areas of the brain, starting at six to 18 months of age.

IQ alterations can also occur as part of a clinical picture in other genetic syndromes. In many cases, the intellectual deficit appears to be limited and can occur at different stages of neurodevelopment, also in terms of cognitive decline. For example, in chromosome 22q11.2 deletion syndrome, DiGeorge syndrome, or velocardiofacial syndrome, which is one of the most common multiple anomaly syndromes in humans, it is usually described as a cognitive decline rather than an early onset intellectual disability.

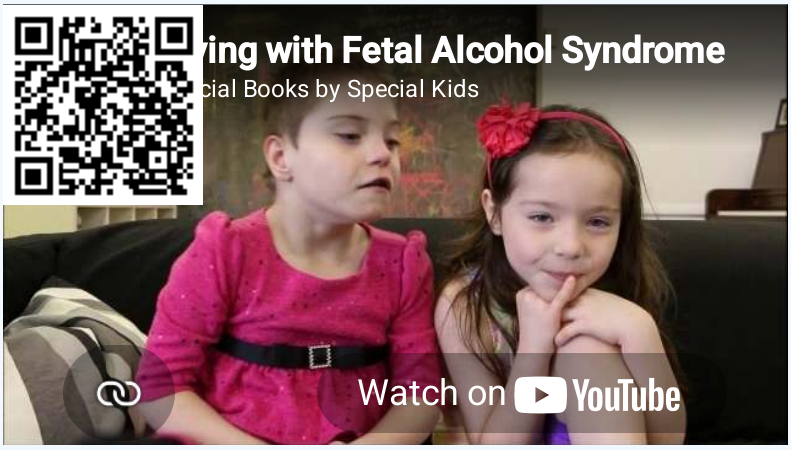
Environmental

Environmental exposure during pregnancy may lead to intellectual disability, which can be caused by maternal exposure to a toxin, infectious agent, uncontrolled maternal condition, and birth complications. One common toxic substance that leads to an intellectual disability during pregnancy includes alcohol. Alcohol exposure, indeed, commonly causes intellectual disability along with other developmental abnormalities, in a condition known as fetal alcohol syndrome.

One study looked at the effects of moderate alcohol exposure by studying pregnant rats who were given 5% ethanol for four hours per day; the results showed that the rats born to mothers exposed to ethanol had deficiencies in various aspects of social behavior, tongue protrusion, and issues with cognitive processes.[1] Fetal exposure to alcohol inhibits the production of retinoic acid, which is an essential, signaling molecule for the development of the nervous system. Even a small amount of alcohol at any trimester of pregnancy may cause fetal alcohol syndrome. Exposure to opioids, cocaine, and teratogenic medications may also lead to intellectual disability.

Watch It: Fetal Alcohol Syndrome

This video introduces us to Natalie, who lives with fetal alcohol syndrome.



You can view the transcript for "Living with Fetal Alcohol Syndrome" here (opens in new window).

To learn more about living with FAS, watch this story of Iyal and his family.

Common, well-known infectious agents that causes intellectual disability are rubella and HIV. Maternal rubella infection in the initial trimester of pregnancy leads to intellectual disability approximately 10 to 15% of the time; it can rise to above 50% with infection during the first month. Immunization may prevent the mother from rubella infection. HIV may be transferred vertically from mother to infant. Infants with HIV may develop encephalopathy, seizures, and intellectual disability within the first year of life secondary to microcephaly, immunosuppression, and Pneumocystis jiroveci pneumonitis (PCP) infection. Excessive neuro-inflammation causes overstimulation of the N-methyl-D-aspartate type receptor (NMDAR) system that leads to neuronal injury. Antiretroviral therapy to the mother and prophylactic treatment with zidovudine to newborn exposed to HIV has significantly reduced the transmission. Other known infectious exposures to mothers that may cause intellectual disability of the newborn are cytomegalic inclusion disease, syphilis, and toxoplasmosis.

Uncontrolled maternal medical conditions may lead to intellectual disability. Pregnancy hypertension, asthma, urinary tract infection, pre-pregnancy obesity, and pre-gestational diabetes were shown to increase the risk significantly. Furthermore, uncontrolled maternal diabetes, malnutrition, and obstetrical complications causing anoxia (placenta previa, placenta abruption, and umbilical cord prolapse) may also cause intellectual disability. An intellectual disability is acquirable during early childhood. Causes include infection (notably encephalitis and meningitis), head trauma, asphyxia, intracranial tumor (either directly or indirectly through seizure, surgery, and chemotherapy), malnutrition, and exposure to toxic substances.

Treatment and Management of Intellectual Disability

Management of intellectual disability must begin promptly with the goals to prevent further worsening, minimize the symptoms of disability, and improve the quality of everyday life. When initiating therapy, a healthcare provider must be aware of the various avenues of treating intellectual disability to orchestrate a multidisciplinary and individually tailored treatment appropriately. This section will discuss some examples of the multiple interventions available.



Figure **13.4.1**: Students with intellectual disabilities should be placed in less restrictive environments.

Educational support is a crucial component of intellectual disability management. Upon diagnosis of intellectual disability in children, healthcare providers must contact the school promptly to set up special education arrangements. What special education entails may vary slightly among schools, but it typically aids comprehensively with providing academic modifications as well as transition planning from childhood to adulthood with a focus on promoting self-sufficiency. It also teaches them how to seek assistance, behavioral skills, vocational skills, communication skills, functional living skills, and social skills based on individual needs in the least restrictive environment. Creating the least restrictive environment for students with an intellectual disability means to place them in general classrooms as much as tolerated. While segregated classrooms for special needs may be helpful for those who need it, implementing the least restrictive environment policy has been shown to improve student outcomes. It is important to monitor the patients' progress in school because education solely from classroom settings may not be sufficient. If an individual with an intellectual disability requires assistance beyond what is available at school, the family can meet the need at home through family education or other outside resources. For example, a disabled student who needs extra attention with social skills may be referred to participate in the Special Olympics programs that have been shown to improve social competence in intellectual disability subjects. Lastly, other causes of learning difficulties need to be ruled out, and appropriate hearing and vision screenings are necessary for individuals with intellectual disabilities.

Behavioral intervention is another important aspect of intellectual disability management, and it can occur in a few different ways. Behavioral therapy aims to encourage positive behaviors while discouraging undesirable behaviors. Providing positive reinforcement and benign punishments (e.g., time-outs) is an effective method of behavioral training. Other supplemental methods may include avoiding triggers of negative demeanor, shunning misconduct, and redirecting to prevent or curtail any troublesome behavior. Cognitive therapy is another mode of behavioral training that has been effective for eligible intellectual disability patients. Cognitive therapy has its basis on a principle that one's behavior, emotions, and cognitions are connected, and it aims to correct one's negative behaviors by identifying and adjusting negative thoughts and emotional stress. Although many other approaches to behavioral intervention exist, the implementation of behavioral therapy, cognitive therapy, or a combination of the two is the most widely used method that has shown to be an effective means of behavioral intervention for intellectual disability patients.

Vocational training helps teenagers and young adults to obtain the necessary skills to enter the labor market. In vocational training, patients carry out pre-scheduled activities under the supervision of a multi-disciplinary team consisting of a social worker, occupational therapist, teacher, counselor, and psychologist. Patients learn to keep themselves clean, wear appropriate clothes, and carry out their responsibilities. A study has shown that patients who underwent vocational training had reduced support requirements compared to their peers.

Family education is an essential service provided by healthcare providers for family members of intellectually disabled patients. The first part of this education is assisting the family members in understanding intellectual disability: definition, management, and prognosis. Then, healthcare providers can help the family through placement decisions, refer them to appropriate services and equipment, and provide caregiver training. In addition to preparing the family for the patient, physicians must recognize that family members also often bear a significant amount of stress as well. The medical team must support the whole family through psychosocial problems such as the need for respect, feeling helpless, depression, and anxiety. There are also outside resources to which the family can obtain a referral. American Association on Intellectual and Developmental Disabilities (AAIDD), The Arc of the United States, and Family-to-Family Health Information and Education Centers are some of the nationally available resources, and social workers can help with connecting the families with local resources. Establishing strong support for the family, in turn, creates a caring home environment for the patient.

Government resources are available for patients with intellectual disabilities and their families. Patients with intellectual disabilities are eligible to benefit from social security income. Patients also may benefit from various community services that provide housing and food delivery. The government also provides education and employment opportunities as outlined under the "Disability Resources" section of the U.S. Department of Labor website.

Psychopharmacologic interventions may not be the main component of intellectual disability treatment, but they play a significant role in treating behavioral abnormalities associated with intellectual disorder and comorbid conditions. Aggressive behavior is not uncommon among individuals with intellectual disabilities and causes admission to institutional settings. Risperidone is a well-documented treatment of disruptive, aggressive, and self-injurious behaviors in children with intellectual disability with good

safety and tolerability profile.[25] Risperidone is an atypical antipsychotic that is less likely to cause tardive dyskinesia, which commonly results in a typical antipsychotic. Aripiprazole is another atypical antipsychotic used to manage aggression.

Treatment of comorbid conditions is an important aspect of adequate therapy. ADHD, depression, and movement disorders are some of the comorbid conditions accompanying intellectual disability that require evaluation and treatment. Methylphenidate, clonidine, and atomoxetine are shown in randomized control trials to reduce ADHD symptoms. Methylphenidate significantly improved the maintenance of attention to complete a task; however, there was no long-term improvement in learning and social skills.

Depressive symptoms can be easily overlooked in individuals with intellectual disabilities when other behavioral problems are prominent; thus symptoms require careful evaluation. Selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine, paroxetine, and sertraline were shown in multiple studies to help with depressive symptoms in this patient population.

Involuntary movements, repetitive self-stimulatory behaviors, and obsessive-compulsive symptoms may be harmful to the patients. Antipsychotic medications have been anecdotally reported to diminish these symptoms, although there was no observation of improvement in adaptive behavior. SSRIs are useful in treating obsessive-compulsive symptoms and stereotyped motor movements.

Studies have shown that patients with intellectual disabilities are at risk for polypharmacy, and providers should be cautious when prescribing medications as some patients may be at a higher risk of side effects and may need lower dosages.

Differential Diagnosis

Several diseases or conditions can be mistaken for intellectual disability:

- **child abuse.** Childhood abuse may cause developmental delays in language and socialization, causing a defect in adaptive function. The child may have difficulty communicating his or her needs and developing trust and social responsibility. Unlike intellectual disability, symptoms are often reversible when the environment improves.
- **debilitating medical disease.** Individuals with normal intelligence may experience depression and delay in the development of chronic, debilitating medical illness.
- **cerebral palsy.** Poor muscle coordination and problems with vision, hearing, and speaking may imitate symptoms of intellectual disability.
- **sensory disability.** Deafness or blindness may lead to a flaw in diagnostic testing, causing a false-positive diagnosis of intellectual disability.
- **speech disorder.** Expressive and receptive aphasia may be mistaken for intellectual disability.

Prognosi

The cognitive deficit of intellectual disability is permanent damage; thus, the prognosis of intellectual disability depends on the severity of cognitive impairment and the supportive environment provided. If provided adequate support, individuals with mild intellectual disabilities may achieve adequate language and social competency to live independently and raise their own families. Individuals with moderate intellectual disabilities are not likely to achieve academic levels greater than second grade; however, they may acquire language and communicate their needs. They may be able to get a job under supervision. Individuals with a severe intellectual disability usually have difficulty acquiring language. They may be able to develop alternative communication skills and recognize words that are critical to functioning. Adults with a severe intellectual disability usually require supervised living situations, such as group homes, and supervision to perform any work-related tasks. Individuals with profound intellectual disabilities have significant difficulty with communication and social functioning.



Figure **13.4.2**: With the right support, many individuals with ID can have a higher quality of life.

Complications

Most of the individuals with intellectual disability have comorbid psychiatric conditions. Individuals are at higher risk of developing depression because they are prone to developing negative self-images as they have difficulty interacting with others and meeting social expectations. Other psychiatric comorbid symptoms frequently observed in these individuals are hyperactivity, self-injurious behaviors, and repetitive stereotypical behaviors.

Glossary

Down syndrome: also known as trisomy 21, a genetic disorder caused by the presence of all or part of a third copy of chromosome 21

fetal alcohol syndrome: a disorder caused by environmental factors due to ethanol exposure while developing in the womb

fragile X syndrome: a genetic disorder resulting from a single gene mutation in FMR1

1. Hamilton, D. A., Barto, D., Rodriguez, C. I., Magcalas, C. M., Fink, B. C., Rice, J. P., Bird, C. W., Davies, S., & Savage, D. D. (2014). Effects of moderate prenatal ethanol exposure and age on social behavior, spatial response perseveration errors and motor behavior. *Behavioural brain research*, 269, 44–54. https://doi.org/10.1016/j.bbr.2014.04.029↵

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13.5: Communication Disorders

Learning Objectives

- Describe the diagnosis and presentation of communication disorders
- Differentiate between communication disorders

A **communication disorder** is any disorder that affects an individual's ability to comprehend, detect, or apply language and speech to engage in discourse effectively with others. The delays and disorders can range from simple sound substitution to the inability to understand or use one's native language.

The DSM-5 categorizes five communication disorders:

- **language disorder** – The important characteristics of a language disorder are difficulties in learning and using language, which is caused by problems with vocabulary, grammar, and putting sentences together in a proper manner. Problems can both be receptive (understanding language) and expressive (producing language).
- **speech sound disorder** – Previously called phonological disorder, individuals with this disorder have problems with pronunciation and articulation of their native language.
- **childhood-onset fluency disorder (stuttering)** – Standard fluency and rhythm of speech is interrupted, often causing the repetition of whole words and syllables. May also include the prolongation of words and syllables, pauses within a word, and/or the avoidance of pronouncing difficult words and replacing them with easier words that the individual is better able to pronounce. This disorder causes many communication problems for the individual and may interfere with social communication and performance in work and/or school settings where communication is essential.
- **social (pragmatic) communication disorder** – This diagnosis describes difficulties in the social uses of verbal and nonverbal communication in naturalistic contexts, which affects the development of social relationships and discourse comprehension. The difference between this diagnosis and autism spectrum disorder is that in the latter there is also a restricted or repetitive pattern of behavior.
- **unspecified communication disorder** – This category is for those who have symptoms of a communication disorder but who do not meet all criteria, and whose symptoms cause distress or impairment.

Language Disorder

Language disorder, also called *developmental language disorder* or *DLD* (specifically when not caused by an intellectual disability) is defined purely in behavioral terms: there is no biological test. There are three points that need to be met for a diagnosis of developmental language disorder (DLD):

1. The child has language difficulties that create obstacles to communication or learning in everyday life.
2. The child's language problems are unlikely to resolve by five years of age.
3. The problems are not associated with a known biomedical condition such as brain injury, neurodegenerative conditions, genetic conditions or chromosome disorders like Down syndrome, sensorineural hearing loss, or autism spectrum disorder or intellectual disability.

Watch It

Watch this brief video on DLD.



You can view the transcript for "Developmental Language Disorder – Boys Town National Research Hospital Web" here (opens in new window).

Etiology

Developmental language disorder (DLD) is associated with aspects of the home environment, and it is often assumed that this is a causal link with poor language stimulation leading to weak language skills. Twin studies, however, show that two children in the same home environment can have very different language outcomes, suggesting we should consider other explanations for the link. Two twins growing up together are exposed to the same home environment, yet may differ radically in their language skills. Such different outcomes are, however, much more common in fraternal (non-identical) twins, who are genetically different. Identical twins share the same genes and tend to be much more similar in language ability. There can be some variation in the severity and persistence of DLD in identical twins, indicating that non-genetic factors affect the course of disorder, but it is unusual to find a child with DLD who has an identical twin with typical language. Current evidence suggests that there are many different genes that can influence language learning, and DLD results when a child inherits a particularly detrimental combination of risk factors, each of which may have only a small effect. Generally, children with DLD often grow into adults who have relatively low educational attainments, and their children may share a genetic risk for language disorder.

Epidemiology

It has long been noted that males are more affected by DLD than females, with a sex ratio of affected males to females around 3:1 or 4:1. However, the sex difference is much less striking in epidemiological samples, suggesting that similar problems may exist in females but are less likely to be detected. The reason for the sex difference is not well understood. Prevalence studies have shown DLD is found in about 7% of five-year-olds, approximately one in every 15 children.

Table 1. Common Red Flags for children with Developmental Language Disorder

1 Year Old	2 Years Old	3 Years Old	4 Years Old	5 Years Old
No reaction to sound No babbling Difficulty feeding No imitation Limited use of gestures	Makes minimal attempts to communicate with gestures or words Has not spoken their first words Difficulty following simple directions Inconsistent response to "no"	Limited use of speech Incomprehensible speech Limited understanding of simple questions Difficulty naming objects Frustration related to communication	Uses only three-word phrases Speech is not understandable to parents Takes a long time to understand others Difficulty asking questions and finding words to express thoughts	Speaks only in simple sentences Speech is not understandable to teachers Difficulty answering questions Difficulty with complex directions Difficulty telling stories Difficulty with peer interactions

Treatment

Treatment is usually carried out by speech and language therapists/pathologists, who use a wide range of techniques to stimulate language learning. Contemporary approaches to enhancing development of language structure, for younger children at least, are more likely to adopt Milieu methods, in which the intervention is interwoven into natural episodes of communication, and the therapist builds on the child's utterances, rather than dictating what will be talked about. Interventions for older children may be more explicit, telling the children what areas are being targeted and giving explanations regarding the rules and structures they are learning, often with visual supports. Children's language tend to improve over time, and without controlled studies, it can be hard to know how much of observed change is down to a specific treatment. There is, however, increasing evidence that direct 1:1 intervention with a speech and language therapist/pathologist can be effective for improving vocabulary and expressive language.

Speech Sound Disorder



Figure **13.5.1**: Cleft lip.

Speech is the act of articulating sounds and can be impaired for all kinds of reasons—a structural problem such as cleft lip and cleft palate, a neurological problem affecting motor control of the speech apparatus **dysarthria**, or inability to perceive distinctions between sounds because of hearing loss. Some distortions of speech sounds, such as a lisp, are commonly seen in young children. These misarticulations should not be confused with language problems, which involve the ability to select and combine linguistic elements to express meanings, and the ability to comprehend meanings.

Although speech disorders can be distinguished from language disorders, they can also co-occur. Speech sound disorder (SSD) is any problem with speech production arising from any cause. The prevalence of speech sound disorders (namely, articulation disorders or phonological disorders) in young children is 8%-9%. Five percent of U.S. children ages three through 17 have a speech disorder that lasted for a week or longer during the past 12 months.

The DSM-5 diagnostic criteria for speech sound disorder are as follows:

A. A persistent difficulty with speech sound production interferes with speech intelligibility or prevents verbal communication of messages.
B. The disturbance causes limitations in effective communication that interfere with social participation, academic achievement, or occupational performance, individually or in any combination.
C. Onset of symptoms is in the early developmental period.
D. The difficulties are not attributable to congenital or acquired conditions, such as cerebral palsy, cleft palate, deafness or hearing loss, traumatic brain injury, or other medical or neurological conditions.

Speech sound disorders may be subdivided into two primary types, **articulation disorders** (also called phonetic disorders) and **phonemic disorders** (also called phonological disorders). However, some may have a mixed disorder in which both articulation and phonological problems exist. Though speech sound disorders are associated with childhood, some residual errors may persist into adulthood.

Articulation Disorders

Articulation disorders (also called *phonetic disorders*, or simply *artic disorders* for short) are based on difficulty learning to physically produce the intended phonemes. Articulation disorders have to do with the main articulators, which are the lips, teeth, alveolar ridge, hard palate, velum, glottis, and the tongue. If the disorder has anything to do with any of these articulators, then it is an articulation disorder. There are usually fewer errors than with a phonemic disorder, and distortions are more likely (though any omissions, additions, and substitutions may also be present). They are often treated by teaching the child how to physically produce

the sound and having them practice its production until it (hopefully) becomes natural. Articulation disorders should not be confused with motor speech disorders, such as dysarthria (in which there is actual paralysis of the speech musculature) or developmental verbal dyspraxia (in which motor planning is severely impaired).

Phonemic Disorders

In a phonemic disorder (also called a phonological disorders), the child is having trouble learning the sound system of the language, failing to recognize which sound-contrasts also contrast meaning. For example, the sounds /k/ and /t/ may not be recognized as having different meanings, so "call" and "tall" might be treated as homophones, both being pronounced as *tall*. This confusion is called phoneme collapse, and in some cases many sounds may all be represented by one sound—e.g., /d/ might replace /t/, /k/, and /g/. As a result, the number of error sounds is often (though not always) greater than with articulation disorders,and substitutions are usually the most common error. Phonemic disorders are often treated using minimal pairs (two words that differ by only one sound) to draw the child's attention to the difference and its effect on communication.

Some children with phonemic disorders may seem to be able to hear phoneme distinctions in the speech of others but not their own. This is called the fis phenomenon based on scenario in which a speech pathologist will say, "Did you say 'fis,' don't you mean 'fish'?" To which the child responds, "No, I didn't say 'fis,' I said 'fis'." In some cases, the sounds produced by the child are actually acoustically different, but not significantly enough for others to distinguish—because those sounds are not phonemically unique to speakers of the language.

Though phonemic disorders are often considered language disorders in that it is the language system that is affected, they are also speech sound disorders in that the errors relate to use of phonemes. This relation makes them different from specific language impairment, which is primarily a disorder of the syntax (grammar) and usage of language rather than the sound system. However, the two can coexist, affecting the same person. Other disorders can deal with a variety of different ways to pronounce consonants. Some examples are glides and liquids. Glides occur when the articulatory posture changes gradually from consonant to vowel. Liquids can include /l/ and /ɹ/ .

Mixed speech sound disorders



Figure **13.5.2**: Articulation and speech therapy can help address speech sound disorders.

Phonetic and phonemic errors may coexist in the same person. In such a case, the primary focus is usually on the phonological component but articulation therapy may be needed as part of the process, since teaching a child how to use a sound is not practical if the child does not know how to produce it.

Presentation

Errors produced by children with speech sound disorders are typically classified into four categories:

- omissions: Certain sounds are not produced—entire syllables or classes of sounds may be deleted; e.g., *fi'* for *fish* or *'at* for *cat*.
- additions (or epentheses/commissions): An extra sound or sounds are added to the intended word; e.g. puh-lane for *plane*.
- distortions: Sounds are changed slightly so that the intended sound may be recognized but sounds "wrong," or may not sound like any sound in the language.
- substitutions: One or more sounds are substituted for another; e.g., "wabbit" for *rabbit* or "tow" for *cow*.

In a typical two-year-old child, about 50% of speech may be intelligible. A four-year-old child's speech should be intelligible overall, and a seven-year-old should be able to clearly produce most words consistent with community norms for their age. Misarticulation of certain difficult sounds ([l], [ɹ], [s], [z], [θ], [ð], [t͡ʃ], [d͡ʒ], and [ʒ]) may be normal up to eight years old. Children with speech sound disorder have pronunciation difficulties inappropriate for their age, and the difficulties are not caused by hearing problems, congenital deformities, motor disorders or selective mutism.

Treatment

For most children, the disorder is not lifelong and speech difficulties improve with time and speech-language treatment. Prognosis is poorer for children who also have a language disorder, as a language disorder may be indicative of a learning disorder.

Watch It

This short video demonstrates a few different therapies used to help children with a language disorder.



You can view the transcript for "Interventions for Speech Sound Disorders in Children" here (opens in new window).

Childhood-Onset Fluency Disorder (Stuttering)

The DMS-5 characterizes stuttering as a childhood-onset "fluency disorder" (also known as *stammering* and *dysphemia*), and is a communication disorder in which the flow of speech is disrupted by involuntary repetitions and prolongations of sounds, syllables, words, or phrases as well as involuntary silent pauses or blocks in which the person who stutters is unable to produce sounds. Stuttering also encompasses the abnormal hesitation or pausing before speech, referred to by people who stutter as *blocks*, and the prolongation of certain sounds, usually vowels or semivowels. The term *stuttering* covers a wide range of severity, from barely perceptible impediments that are largely cosmetic to severe symptoms that effectively prevent oral communication.

The impact of stuttering on a person's functioning and emotional state can be severe and may include fears of having to enunciate specific vowels or consonants, fears of being caught stuttering in social situations, self-imposed isolation, anxiety, stress, shame, low self-esteem, being a possible target of bullying (especially in children), having to use word substitution and rearrange words in a sentence to hide stuttering, or a feeling of "loss of control" during speech. Stuttering is sometimes popularly seen as a symptom of anxiety, but there is no direct correlation in that direction.

Stuttering is generally not a problem with the physical production of speech sounds or putting thoughts into words. Acute nervousness and stress are not thought to cause stuttering, but they can trigger stuttering in people who have the speech disorder. Living with a stigmatized disability can result in anxiety and high allostatic stress load (chronic nervousness and stress) that reduce the amount of acute stress necessary to trigger stuttering in any given person who stutters, worsening the problem in the manner of a positive feedback system; the name *stuttered speech syndrome* has been proposed for this condition. Neither acute nor chronic stress, however, itself creates any predisposition to stuttering. The disorder is also *variable*, which means that in certain situations, such as talking on the telephone or in a large group, the stuttering might be more severe or less, depending on whether or not the person who stutters is self-conscious about their stuttering. People who stutter often find that their stuttering fluctuates and that they have good days, bad days and stutter-free days. The times in which their stuttering fluctuates can be random.



Figure 13.5.3: Most people who stutter tend to be boys, with an onset of about 2-5 years of age.

Epidemiology

Almost 70 million people worldwide stutter, about 1% of the world's population. More boys stammer than girls, in the ratio of three to four boys for every one girl, because the male hypothalamic-pituitary-adrenal (HPA) axis is more active. As males produce more cortisol than females under the same provocation, they can be tense or anxious and become repetitive.

The risk for the development of a stutter usually ends by age five, and there doesn't appear to be any effects of race, ethnicity, culture, bilingualism, or socioeconomic status (SES) on the development and prevalence of stuttering.[1]

Etiology

Although the exact etiology, or cause, of stuttering is unknown, both genetics and neurophysiology are thought to contribute. Children who have first-degree relatives who stutter are three times as likely to develop a stutter. However, twin and adoption studies suggest that genetic factors interact with environmental factors for stuttering to occur, and many stutterers have no family history of the disorder. There is evidence that stuttering is more common in children who also have concurrent speech, language, learning, or motor difficulties.

Another view is that a stutter or stammer is a complex tic. This view is held for the following reasons: it always arises from the repetition of sounds or words; young children like repetition and the tenser they are feeling, the more they like this outlet for their tension—an understandable and quite normal reaction. They are capable of repeating all types of behavior. The more tension that is felt, the less one likes change. The more change, the greater can be the repetition. So, when a three-year-old finds he has a new baby brother or sister, he may start repeating sounds. The repetitions can become conditioned and automatic and ensuing struggles against the repetitions result in prolongations and blocks in his speech.

In a 2010 article, three genes were found by Dennis Drayna and his team to correlate with stuttering: GNPTAB, GNPTG, and NAGPA. Researchers estimated that alterations in these three genes were present in 9% of those who have a family history of stuttering. For some people who stutter, congenital factors may play a role. These may include physical trauma at or around birth, learning disabilities, as well as cerebral palsy. In others, there could be added impact due to stressful situations such as the birth of a sibling, moving, or a sudden growth in linguistic ability.

There is clear empirical evidence for structural and functional differences in the brains of stutterers. Research is complicated somewhat by the possibility that such differences could be the consequences of stuttering rather than a cause, but recent research on older children confirms structural differences thereby giving strength to the argument that at least some of the differences are not a consequence of stuttering. There is also evidence of differences in linguistic processing between people who stutter and people who do not. Brain scans of adult stutterers have found greater activation of the right hemisphere, which is associated with emotions, than of the left hemisphere, which is associated with speech. In addition, reduced activation in the left auditory cortex has been observed in the brains of those who stutter.

Watch It

This clip explains a little bit more about the etiology of stuttering.



You can view the transcript for "How Do People Develop a Stutter?" here (opens in new window).

If you're interested in learning more, especially regarding the perspectives of children who stutter, watch this CBS news clip.

There are many treatments and speech therapy techniques available that may help decrease speech disfluency in some people who stutter to the point where an untrained ear cannot identify a problem; however, there is essentially no cure for the disorder at present. The severity of the person's stuttering would correspond to the amount of speech therapy needed to decrease disfluency. For severe stuttering, long-term therapy and hard work is required to decrease disfluency.

Social (Pragmatic) Communication Disorder

Social (pragmatic) communication disorder (SCD or SPCD) is a disorder where individuals have difficulties with verbal and nonverbal social communication.

The DSM-5 categorizes social (pragmatic) communication disorder (SCD) as a communication disorder within the domain of neurodevelopmental disorders, listed alongside other disorders of speech and language which typically manifest in early childhood. The DSM-5 diagnostic criteria for social communication disorder is as follows:

A. Individuals have persistent difficulties in the social use of verbal and nonverbal communication as manifested by all of the following:

- deficits in using communication for social purposes, such as greeting and sharing information, in a manner that is appropriate for the social context.
- impairment of the ability to change communication to match context or the needs of the listener, such as speaking differently in a classroom than on a playground, talking differently to a child than to an adult, and avoiding use of overly formal language.
- difficulties following rules for conversation and storytelling, such as taking turns in conversation, rephrasing when misunderstood, and knowing how to use verbal and nonverbal signals to regulate interaction.
- difficulties understanding what is not explicitly stated (e.g., making inferences) and nonliteral or ambiguous meanings of language (e.g., idioms, humor, metaphors, and multiple meanings that depend on the context for interpretation).

B. The deficits result in functional limitations in effective communication, social participation, social relationships, academic achievement, or occupational performance, individually or in combination.
C. The onset of symptoms is in the early developmental period (but deficits may not become fully manifested until social communication demands exceed limited capacities).
D. The symptoms are not attributable to another medical or neurological condition or to low abilities in the domains of word structure and grammar, and are not better explained by autism spectrum disorder, intellectual disability (intellectual developmental disorder), global developmental delay, or another mental disorder.

Individuals with social communication disorder (SCD) have particular trouble understanding the meaning of what others are saying, and they are challenged in using language appropriately to get their needs met and interact with others. Children with the disorder often exhibit

- delayed language development;

- language disorders (similar to the acquired disorder of aphasia) such as word search pauses, jargoning, word order errors, word category errors, verb tense errors;
- stuttering or cluttering speech;
- repeating words or phrases;
- tendency to be concrete or prefer facts to stories; and
- difficulties with
  - pronouns or pronoun reversal.
  - understanding questions.
  - understanding choices and making decisions.
  - following conversations or stories (conversations are “off-topic” or “one-sided”).
  - extracting the key points from a conversation or story; they tend to get lost in the details.
  - verb tenses.
  - explaining or describing an event.
  - understanding satire or jokes and contextual cues.
  - reading comprehension.
  - reading body language.
  - making and maintaining friendships and relationships because of delayed language development.
  - distinguishing offensive remarks.
  - organizational skills.

According to Bishop and Norbury (2002), children with SCD can have fluent, complex, and clearly articulated expressive language but exhibit problems with the way their language is used. These children typically are verbose. However, they usually have problems understanding and producing connected discourse, instead giving conversational responses that are socially inappropriate, tangential, and stereotyped. They often develop eccentric interests but not as strong or obsessional as people with autism spectrum disorders.

The current view, therefore, is that the disorder has more to do with communication and information processing than language. For example, children with SCD will often fail to grasp the central meaning or saliency of events. This then leads to an excessive preference for routine and “sameness” (seen in autism spectrum disorders), as children with SCD struggle to generalize and grasp the meaning of situations that are new; it also means that more difficulties occur in a stimulating environment than in a one-to-one setting.

A further problem caused by SCD is the assumption of literal communication. This would mean that obvious, concrete instructions are clearly understood and carried out, whereas simple but non-literal expressions such as jokes, sarcasm, and general social chatting are difficult and can lead to misinterpretation. Lies are also a confusing concept to children with SCD as it involves knowing what the speaker is thinking, intending, and truly meaning beyond a literal interpretation.

Differences between SCD and Autism

Communication problems are also part of the autism spectrum disorder; however, individuals with autism also show a restricted pattern of behavior, according to behavioral psychologists. The diagnosis of SCD can only be given if autism has been ruled out. It is assumed that those with autism have difficulty with the meaning of what is being said due to different ways of responding to social situations.

Prior to the release of the DSM-5 in 2013, SCD was not differentiated from a diagnosis of autism. However, there were a large number of cases of children experiencing difficulties with pragmatics that did not meet the criteria for autism. The differential diagnosis of SCD allows practitioners to account for social and communication difficulties that occur to a lesser degree than in children with autism. SCD is distinguished from autism by the absence of any history (current or past) of restricted/repetitive patterns of interest or behavior in SCD.

Epidemiology

More research will need to be conducted to determine the true prevalence of SCD, due to it’s overlap with ASD and the newness of the diagnosis in the DSM-5; however a population estimate suggests the rates of SCD among children is about 7.5% and affects

more boys than girls by a ratio of 2.6:1. Higher prevalence rates (23–33%) have been found in those with language disorders.

Treatment for SCD

Treatments for SCD are less established than for treatments for other disorders such as autism. Similarities between SCD and some aspects of autism leads some researchers to try some treatments for autism with people with SCD.

Speech therapy can help individuals who have communication disorders. Speech and language therapy treatment focuses on communication and social interaction. Speech therapists can work with clients on communication in various settings.

Watch It

Watch this video for a short explanation of social communication disorder.



You can view the transcript for “What is Social Communication Disorder” here (opens in new window).

Key Takeaways: Communication Disorders

Disorder	Criteria	Prevalence	Treatment?
Language disorder	difficulties in learning and using language, which is caused by problems with vocabulary, with grammar, and with putting sentences together in a proper manner. Problems can both be receptive (understanding language) and expressive (producing language)	approximately 7% of children	Speech and language therapists/pathologists use a wide range of techniques to stimulate language learning. There is increasing evidence that direct 1:1 intervention with an SLT/P can be effective for improving vocabulary and expressive language.
Speech sound disorder	problems with pronunciation and articulation of their native language	8%-9% of children	For most children, the disorder is not lifelong and speech difficulties improve with time and speech-language treatment.
Childhood-onset fluency disorder (stuttering)	standard fluency and rhythm of speech is interrupted, often causing the repetition of whole words and syllables	almost 70 million people worldwide stutter, about 1% of the world's population	Various speech therapy techniques are available that may help decrease speech disfluency in some people who stutter; the severity of the person's stuttering would correspond to the amount of speech therapy needed to decrease disfluency.
Social (pragmatic) communication disorder	difficulties in the social uses of verbal and nonverbal communication in naturalistic contexts, which affects the development of social relationships and discourse comprehension	about 7.5% of children	Similarities between SCD and some aspects of autism leads some researchers to try autism treatments for people with SCD.

Glossary

childhood-onset fluency disorder (stuttering): standard fluency and rhythm of speech is interrupted, often causing the repetition of whole words and syllables; may also include the prolongation of words and syllables, pauses within a word, and/or the avoidance of pronouncing difficult words and replacing them with easier words that the individual is better able to pronounce

communication disorder: any disorder that affects someone's ability to comprehend, detect, or apply language and speech to engage in discourse effectively with others

dysarthria: a collective name for a group of speech disorders resulting from disturbances in muscular control over the speech mechanism due to damage of the central or peripheral nervous system; designates problems in oral communication due to paralysis, weakness, or incoordination of the speech musculature

social (pragmatic) communication disorder (SCD): difficulties in the social uses of verbal and nonverbal communication in naturalistic contexts, which affects the development of social relationships and discourse comprehension

speech sound disorder: previously called phonological disorder, problems with pronunciation and articulation of native language

unspecified communication disorder: description for those who have symptoms of a communication disorder, but who do not meet all criteria, and whose symptoms cause distress or impairment

1. Yairi, E., & Ambrose, N. (2013). Epidemiology of stuttering: 21st century advances. Journal of fluency disorders, 38(2), 66–87. https://doi.org/10.1016/j.jfludis.2012.11.002↵

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13.6: Autism Spectrum Disorder

Learning Objectives

- Describe the symptoms, risk factors, influences, and prevalence of autism spectrum disorder

Autism Spectrum Disorder

A seminal paper published in 1943 by psychiatrist Leo Kanner described an unusual neurodevelopmental condition he observed in a group of children. Kanner called this condition early infantile autism, and it was characterized mainly by an inability to form close emotional ties with others, speech and language abnormalities, repetitive behaviors, and an intolerance of minor changes in the environment and in normal routines (Bregman, 2005). What the DSM-5 refers to as autism spectrum disorder today is a direct extension of Kanner's work.

In 2013, the DSM-5 replaced the previous subgroups of autistic disorder: Asperger syndrome, pervasive developmental disorder not otherwise specified (PDD-NOS), and childhood disintegrative disorder with the single term *autism spectrum disorder*.



Figure **13.6.1**: Few children who are correctly diagnosed with ASD are thought to lose this diagnosis due to treatment or outgrowing their symptoms. Children with poor treatment outcomes also tend to be ones that have moderate to severe forms of ASD, whereas children who appear to have responded to treatment are the ones with milder forms of ASD.

Autism spectrum disorder (ASD) is probably the most misunderstood and puzzling of **neurodevelopmental disorders**. Children with this disorder show signs of significant disturbances in three main areas: (a) deficits in social interaction, (b) deficits in communication, and (c) repetitive patterns of behavior or interests. These disturbances appear early in life and cause serious impairments in functioning (APA, 2013). The child with autism spectrum disorder might exhibit deficits in social interaction by not initiating conversations with other children or turning their head away when spoken to. These children do not make eye contact with others and seem to prefer playing alone rather than with others. In a certain sense, it is almost as though these individuals live in a personal and isolated social world others are simply not privy to or able to penetrate. Communication deficits can range from a complete lack of speech, to one-word responses (e.g., saying "Yes" or "No" when replying to questions or statements that require additional elaboration), to echoed speech (e.g., parroting what another person says, either immediately or several hours or even days later), to difficulty maintaining a conversation because of an inability to reciprocate others' comments. These deficits can also include problems in using and understanding nonverbal cues (e.g., facial expressions, gestures, and postures) that facilitate normal communication.

Repetitive patterns of behavior or interests can be exhibited in a number of ways. A child with autism spectrum disorder might engage in stereotyped, repetitive movements (rocking, head-banging, or repeatedly dropping an object and then picking it up) or might show great distress at small changes in routine or the environment. For example, the child might throw a temper tantrum if an object is not in its proper place or if a regularly-scheduled activity is rescheduled. In some cases, the person with autism spectrum disorder might show highly restricted and fixated interests that appear to be abnormal in their intensity. For instance, the person might learn and memorize every detail about something even though doing so serves no apparent purpose. Importantly, autism spectrum disorder is not the same thing as intellectual disability, although these two conditions are often comorbid. The DSM-5 specifies that the symptoms of autism spectrum disorder are not caused or explained by intellectual disability.

Life Problems from Autism Spectrum Disorder

Autism spectrum disorder is referred to in everyday language as autism; in fact, the disorder was termed *autistic disorder* in earlier editions of the DSM, and its diagnostic criteria were much narrower than those of autism spectrum disorder. The qualifier *spectrum* in *autism spectrum disorder* is used to indicate that individuals with the disorder can show a range, or spectrum, of symptoms that

vary in their magnitude and severity: some severe, others less severe. The previous edition of the DSM included a diagnosis of Asperger's disorder, generally recognized as a less severe form of autistic disorder; individuals diagnosed with Asperger's disorder were described as having average or high intelligence and a strong vocabulary, but exhibiting impairments in social interaction and social communication, such as talking only about their special interests (Wing, Gould, & Gillberg, 2011). However, because research has failed to demonstrate that Asperger's disorder differs qualitatively from autistic disorder, the DSM-5 does not include it, which is prompting concerns among some parents that their children may no longer be eligible for special services ("Asperger's Syndrome Dropped," 2012). Some individuals with autism spectrum disorder, particularly those with better language and intellectual skills, can live and work independently as adults. However, most do not because the symptoms remain sufficient to cause serious impairment in many areas of life (APA, 2013).

Watch It

This video provides an overview of what you've read, describing its diagnostic criteria and the differences between the DSM-4 and DSM-5 terms for ASD.



You can view the transcript for "Autism Spectrum Disorder | Clinical Presentation" here (opens in new window).

Epidemiology

Current estimates from the Center for Disease Control and Prevention's Autism and Developmental Disabilities Monitoring Network indicate that one in 59 children in the United States has autism spectrum disorder; the disorder is four times more common among boys (one in 38) than in girls (one in 152) (Baio et al, 2018). Rates of autistic spectrum disorder have increased dramatically since the 1980s. For example, California saw an increase of 273% in reported cases from 1987 through 1998 (Byrd, 2002); between 2000 and 2008, the rate of autism diagnoses in the United States increased 78% (CDC, 2012). Although it is difficult to interpret this increase, it is possible that the rise in prevalence is the result of the broadening of the diagnosis, increased efforts to identify cases in the community, and greater awareness and acceptance of the diagnosis. In addition, mental health professionals are now more knowledgeable about autism spectrum disorder and are better equipped to make the diagnosis, even in subtle cases (Novella, 2008).

Causes of Autism Spectrum Disorder

Early theories of autism placed the blame squarely on the shoulders of the child's parents, particularly the mother. Bruno Bettelheim (an Austrian-born American child psychologist who was heavily influenced by Sigmund Freud's ideas) suggested that a mother's ambivalent attitudes and her frozen and rigid emotions toward her child were the main causal factors in childhood autism. In what must certainly stand as one of the more controversial assertions in psychology over the last 50 years, Bettelheim wrote, "I state my belief that the precipitating factor in infantile autism is the parent's wish that his child should not exist" (Bettelheim, 1967, p. 125). As you might imagine, Bettelheim did not endear himself to a lot of people with this position; incidentally, no scientific evidence exists supporting his claims.



Figure 13.6.2: Another controversial claim suggested that watching extensive amounts of television caused autism. This hypothesis was largely based on research suggesting that the increasing rates of autism in the 1970s and 1980s were linked to the growth of cable television at this time.

The exact causes of autism spectrum disorder remain unknown despite massive research efforts over the last two decades (Meek, Lemery-Chalfant, Jahromi, & Valiente, 2013). Autism appears to be strongly influenced by genetics, as identical twins show concordance rates of 60–90%, whereas concordance rates for fraternal twins and siblings are five to 10% (Autism Genome Project Consortium, 2007). Many different genes and gene mutations have been implicated in autism (Meek et al., 2013). Among the genes involved are those important in the formation of synaptic circuits that facilitate communication between different areas of the brain (Gauthier et al., 2011). A number of environmental factors are also thought to be associated with increased risk for autism spectrum disorder, at least in part, because they contribute to new mutations. These factors include exposure to pollutants, such as plant emissions and mercury, urban versus rural residence, and vitamin D deficiency (Kinney, Barch, Chayka, Napoleon, & Munir, 2009).

Certain prenatal and perinatal complications have been reported as possible risk factors for autism. These risk factors include maternal gestational diabetes, maternal and paternal age over 30, bleeding after the first trimester, use of prescription medication (e.g. valproate) during pregnancy, and meconium in the amniotic fluid. While research is not conclusive on the relation of these factors to autism, each of these factors has been identified more frequently in children with autism compared to their siblings who do not have autism, and other typically developing youth. While it is unclear if any single factors during the prenatal phase affect the risk of autism, complications during pregnancy may be a risk. Another hypothesized risk factor is low vitamin D levels in early development.

? Child Vaccinations and Autism Spectrum Disorder

In the late 1990s, a prestigious medical journal published an article purportedly showing that autism is triggered by the MMR (measles, mumps, and rubella) vaccine. These findings were very controversial and drew a great deal of attention, sparking an international forum on whether children should be vaccinated. In a shocking turn of events, some years later, the article was retracted by the journal that had published it after accusations of fraud on the part of the lead researcher. Despite the retraction, the reporting in popular media led to concerns about a possible link between vaccines and autism persisting. A recent survey of parents, for example, found that roughly a third of respondents expressed such a concern (Kennedy, LaVail, Nowak, Basket, & Landry, 2011); and perhaps fearing that their children would develop autism, more than 10% of parents of young children refuse or delay vaccinations (Dempsey et al., 2011). Some parents of children with autism mounted a campaign against scientists who refuted the vaccine-autism link. Even politicians and several well-known celebrities weighed in; for example, actress Jenny McCarthy (who believed that a vaccination caused her son's autism) co-authored a book on the matter. However, there is no scientific evidence that a link exists between autism and vaccinations (Hughes, 2007). Indeed, a recent study compared the vaccination histories of 256 children with autism spectrum disorder with that of 752 control children across three time periods during their first two years of life (birth to three months, birth to seven months, and birth to two years) (DeStefano, Price, & Weintraub, 2013). At the time of the study, the children were between six and 13 years old, and their prior vaccination records were obtained. Because vaccines contain immunogens (substances that fight infections), the investigators examined medical records to see how many immunogens children received to determine if those children who received more immunogens were at greater risk for developing autism spectrum disorder. The results of this study, a portion of which are shown in Figure 3 and Table 1, clearly demonstrate that the quantity of immunogens from vaccines received during the first two years of life were not at all related to the development of autism spectrum disorder. There is not a relationship between vaccinations and autism spectrum disorders.

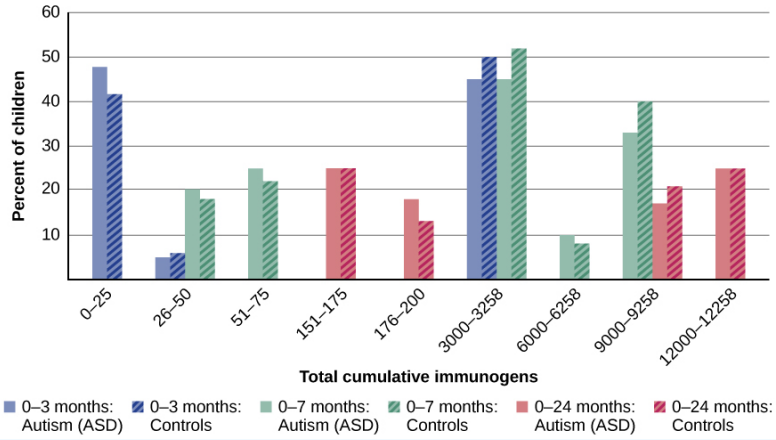


Figure **13.6.3**: In terms of their exposure to immunogens in vaccines, overall, there is not a significant difference between children with autism spectrum disorder and their age-matched controls without the disorder (DeStefano et al., 2013).

Table 1. Comparison Groups Infants 0 to 24 Months of Age With and Without Autism and Total Cumulative Immunogens Exposure Through Vaccines

Total Cumulative Immunogens	0 to 3 Months: Autism (ASD)	0 to 3 Months: Controls	0 to 7 Months: Autism (ASD)	0 to 7 Months: Controls	0 to 24 Months: Autism (ASD)	0 to 24 Months: Controls
0 to 25	48%	41%	–	–	–	–
26 to 50	5%	6%	20%	18%	–	–
51 to 75	–	–	25%	22%	–	–

Why does concern over vaccines and autism spectrum disorder persist? Since the proliferation of the internet in the 1990s, parents have been constantly bombarded with online information that can become magnified and take on a life of its own. The enormous volume of electronic information pertaining to autism spectrum disorder, combined with how difficult it can be to grasp complex scientific concepts, can make separating good research from bad challenging (Downs, 2008). Notably, the study that fueled the controversy reported that eight out of 12 children—according to their parents—developed symptoms consistent with autism spectrum disorder shortly after receiving a vaccination. To conclude that vaccines cause autism spectrum disorder on this basis, as many did, is clearly incorrect for a number of reasons, not the least of which is because correlation does not imply causation, as you've learned.

Additionally, as was the case with diet and ADHD in the 1970s, the notion that autism spectrum disorder is caused by vaccinations is appealing to some because it provides a simple explanation for this condition. Like all disorders, however, there are no simple explanations for autism spectrum disorder. Although the research discussed above has shed some light on its causes, science is still a long way from a complete understanding of the disorder.

Treatments for Autism Spectrum Disorder

Several interventions can help children with autism, and the main goals of treatment are to lessen associated deficits and family distress and to increase quality of life and functional independence. In general, higher IQs are correlated with greater responsiveness to treatment and improved treatment outcomes. Although evidence-based interventions for autistic children vary in their methods, many adopt a psychoeducational approach to enhancing cognitive, communication, and social skills while minimizing problem behaviors. It's important to keep in mind that no single treatment is best, there is no "one-size-fits-all" solution, and treatment should be tailored to the child's needs. Intensive, sustained special education or remedial education programs and behavior therapy early in life can help children acquire self-care, social, and job skills. Available approaches include applied behavior analysis (ABA), developmental models, structured teaching, speech and language therapy, social skills therapy, and occupational therapy. Among these approaches, interventions either treat autistic features comprehensively or focus treatment on a specific area of deficit.

There has been increasing attention to the development of evidence-based interventions for young children with ASD. Two theoretical frameworks outlined for early childhood intervention include applied behavioral analysis (ABA) and the developmental social-pragmatic model (DSP). Although applied behavioral analysis (ABA) therapy has a strong evidence base, particularly in regard to early intensive home-based therapy, ABA's effectiveness may be limited by diagnostic severity and IQ of the person affected by ASD.

Neurodiversity

The autism rights movement promotes the concept of *neurodiversity*, which views autism as a natural variation of the brain rather than a disorder to be cured. In response to the rising idea that autism (and other similar disorders) are an epidemic, the concept of neurodiversity shines a new perspective on the subject.



Figure 13.6.4: The symbol for the Autism Rights Movement.

Coined in 1998 by Australian sociologist Judy Singer, who helped popularize the concept along with American journalist Harvey Blume, neurodiversity emerged as a challenge to prevailing views that certain neurodevelopmental disorders are inherently pathological and instead adopts the social model of disability, in which societal barriers are the main contributing factor that disables people. The term represented a move away from previous "mother-blaming" theories about the cause of autism.

Neurodiversity advocates point out that neurodiverse people often have exceptional abilities alongside their weaknesses. For example, a person with ADHD may hyperfocus on some tasks while struggling to focus on others, or an autistic person may have exceptional memory or even savant skills. In light of these facts, advocates argue for recognition of strengths as well as weaknesses in neurodiverse people, and that a variety of neurological conditions that are currently classified as disorders are better regarded as differences. This view is especially popular within the autism rights movement.

Neurodiversity advocates denounce the framing of autism, ADHD, dyslexia, and other neurodevelopmental disorders as requiring medical intervention to "cure" or "fix" them and instead promote support systems, such as inclusion-focused services, accommodations, communication, and assistive technologies, occupational training, and independent living support. The intention is for individuals to receive support that honors authentic forms of human diversity, self-expression, and being, rather than treatment that coerces or forces them to adopt normative ideas of normality or to conform to a clinical ideal. Proponents of neurodiversity strive to reconceptualize autism and related conditions in society by the following measures: acknowledging that neurodiversity does not require a cure; changing the language from the current "condition, disease, disorder, or illness"-based nomenclature and "broaden[ing] the understanding of healthy or independent living"; acknowledging new types of autonomy; and giving non-neurotypical individuals more control over their treatment, including the type, timing, and whether there should be treatment at all.

However, the neurodiversity paradigm has been controversial among disability advocates, with opponents saying that its conceptualization doesn't reflect the realities of individuals who have high-support needs.

Glossary

autism spectrum disorder: childhood disorder characterized by deficits in social interaction and communication and repetitive patterns of behavior or interests

neurodevelopmental disorder: disorders that are first diagnosed in childhood and involve developmental problems in academic, intellectual, and social functioning

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13.7: Attention Deficit/Hyperactivity Disorder

Learning Objectives

- Describe the symptoms, risk factors, influences, and prevalence of attention deficit/hyperactivity disorder

Most of the disorders we have discussed so far are typically diagnosed in adulthood, although they can and sometimes do occur during childhood. However, there is a group of conditions that, when present, are diagnosed early in childhood, often before the time a child enters school. These conditions are listed in the DSM-5 as neurodevelopmental disorders, and they involve developmental problems in personal, social, academic, and intellectual functioning (APA, 2013). In this section, we will discuss two such disorders: attention deficit/hyperactivity disorder and autism.

Attention Deficit/Hyperactivity Disorder

Diego is always active, from the time he wakes up in the morning until the time he goes to bed at night. His mother reports that he came out the womb kicking and screaming, and he has not stopped moving since. He has a sweet disposition, but always seems to be in trouble with his teachers, parents, and after-school program counselors. Diego seems to accidentally break things constantly, he lost his jacket three times last winter, and he never seems to sit still. His teachers believe Diego is a smart child, but he never finishes anything he starts and is so impulsive that he does not seem to learn much in school.

Diego likely has **attention deficit/hyperactivity disorder (ADHD)**. The symptoms of this disorder were first described by Hans Hoffman in the 1920s. While taking care of his son while his wife was in the hospital giving birth to a second child, Hoffman noticed that the boy had trouble concentrating on his homework, had a short attention span, and had to repeatedly go over easy homework to learn the material (Jellinek & Herzog, 1999). Later, it was discovered that many hyperactive children—those who are fidgety, restless, socially disruptive, and have trouble with impulse control—also display short attention spans, problems with concentration, and distractibility. By the 1970s, it had become clear that many children who display attention problems often also exhibit signs of hyperactivity. In recognition of such findings, the DSM-3 (published in 1980) included a new disorder: attention deficit disorder with and without hyperactivity, now known as attention deficit/hyperactivity disorder (ADHD).

A child with attention deficit/hyperactivity disorder (ADHD) shows a constant pattern of inattention and/or hyperactive and impulsive behavior that interferes with normal functioning (APA, 2013). Some of the signs of inattention include great difficulty with and avoidance of tasks that require sustained attention (such as conversations or reading), failure to follow instructions (often resulting in failure to complete schoolwork and other duties), disorganization (difficulty keeping things in order, poor time management, and sloppy and messy work), lack of attention to detail, becoming easily distracted, and forgetfulness. Hyperactivity is characterized by excessive movement and includes fidgeting or squirming, leaving one's seat in situations when remaining seated is expected, having trouble sitting still (e.g., in a restaurant), running about and climbing on things, blurting out responses before another person's question or statement has been completed, difficulty waiting one's turn for something, and interrupting and intruding on others. Frequently, the hyperactive child comes across as noisy and boisterous. The child's behavior is hasty, impulsive, and seems to occur without much forethought; these characteristics may explain why adolescents and young adults diagnosed with ADHD receive more traffic tickets and have more automobile accidents than do others (Thompson, Molina, Pelham, & Gnagy, 2007).

ADHD occurs in about 5% of children (APA, 2013). On the average, boys are three times more likely to have ADHD than are girls; however, such findings might reflect the greater propensity of boys to engage in aggressive and antisocial behavior and thus incur a greater likelihood of being referred to psychological clinics (Barkley, 2006). This may also be reflective of bias in failing to appropriately diagnose girls and women who show symptoms. Children with ADHD face severe academic and social challenges. Compared to their non-ADHD counterparts, children with ADHD have lower grades and standardized test scores and higher rates of expulsion, grade retention, and dropping out (Loe & Feldman, 2007). They also are less well-liked and more often rejected by their peers (Hoza et al., 2005).

Previously, ADHD was thought to fade away by adolescence. However, longitudinal studies have suggested that ADHD is a chronic problem, one that can persist into adolescence and adulthood (Barkley, Fischer, Smallish, & Fletcher, 2002). A recent study found that 29.3% of adults who had been diagnosed with ADHD decades earlier still showed symptoms (Barbaresi et al., 2013).

Somewhat troubling, this study also reported that nearly 81% of those whose ADHD persisted into adulthood had experienced at least one other comorbid disorder compared to 47% of those whose ADHD did not persist.

Watch It

Watch this for an overview of ADHD, from diagnostic criteria to possible underlying causes and types of treatment:



You can view the transcript for "Attention deficit hyperactivity disorder (ADHD/ADD) – causes, symptoms & pathology" here (opens in new window).

For an optional video on what it's like living with ADHD, click here to watch this TED talk by Toby Shaw.

Case Studies: What is hyperactivity?

Case 1: Michael

Michael, a four-year-old boy, was referred to a child psychologist to be evaluated for ADHD. His parents reported that Michael would not comply with their instructions. They also complained that Michael would not remain seated during "quality time" with his father. The evaluating psychologist interviewed the family, and, by all accounts, Michael was noncompliant and often left his seat. Specifically, when Michael's mother asked him to prepare his preschool lunch, Michael would leave the kitchen and play with his toys soon after opening his lunch box. Further, the psychologist found that "quality time" involved Michael and his father sitting down for several hours to watch movies. In other settings, such as preschool, Michael was compliant with his teacher's requests and no more active than his peers.

In this case, Michael's parents held unrealistic expectations for a child at Michael's developmental level. The psychologist would likely educate Michael's parents about normative child development rather than diagnosing Michael with ADHD.

Case 2: Jake

Jake, a 10-year-old boy, was referred to the same psychologist as Michael. Jake's mother was concerned because Jake was not getting ready for school on time. Jake also had trouble remaining seated during dinner, which interrupted mealtime for the rest of the family. The psychologist found that in the morning, Jake would complete one or two steps of his routine before he became distracted and switched activities, despite his mother's constant reminders. During dinnertime, Jake would leave his seat between 10 and 15 times over the course of the meal. Jake's teachers were worried because Jake was only able to complete 50% of his homework. Further, his classmates would not pick Jake for team sports during recess because he often became distracted and wondered off during the game.

In this case, Jake's symptoms would not be considered developmentally appropriate for a 10-year-old child. Further, his symptoms caused him to experience impairment at home and school. Unlike Michael, Jake probably would be diagnosed with ADHD.

Life Problems from ADHD

Children diagnosed with ADHD face considerably worse long-term outcomes than do those children who do not receive such a diagnosis. In one investigation, 135 adults who had been identified as having ADHD symptoms in the 1970s were contacted decades later and interviewed (Klein et al., 2012). Compared to a control sample of 136 participants who had never been diagnosed with ADHD, those who were diagnosed as children

- had worse educational attainment (more likely to have dropped out of high school and less likely to have earned a bachelor's degree);
- had lower SES;
- held less prestigious occupational positions;
- were more likely to be unemployed;
- made considerably less in salary;
- scored worse on a measure of occupational functioning, indicating, for example, lower job satisfaction, poorer work relationships, and more firings;
- scored worse on a measure of social functioning, indicating, for example, fewer friendships and less involvement in social activities;
- were more likely to be divorced; and
- were more likely to have non-alcohol-related substance abuse problems. (Klein et al., 2012)

Longitudinal studies also show that children diagnosed with ADHD are at higher risk for substance abuse problems. One study reported that childhood ADHD predicted later drinking problems, daily smoking, and use of marijuana and other illicit drugs (Molina & Pelham, 2003). The risk of substance abuse problems appears to be even greater for those with ADHD who also exhibit antisocial tendencies (Marshal & Molina, 2006).

Causes of ADHD

Family and twin studies indicate that genetics play a significant role in the development of ADHD. Burt (2009), in a review of 26 studies, reported that the median rate of concordance for identical twins was 0.66 (one study reported a rate of 0.90), whereas the median concordance rate for fraternal twins was 0.20. This study also found that the median concordance rate for unrelated (adoptive) siblings was 0.09; although this number is small, it is greater than zero, thus suggesting that the environment may have at least some influence. Another review of studies concluded that the heritability of inattention and hyperactivity were 71% and 73%, respectively (Nikolas & Burt, 2010).



Figure **13.7.1**: The left prefrontal cortex is often affected in ADHD.

The specific genes involved in ADHD are thought to include at least two that are important in the regulation of the neurotransmitter dopamine (Gizer, Ficks, & Waldman, 2009), suggesting that dopamine may be important in ADHD. Indeed, medications used in the treatment of ADHD, such as methylphenidate (Ritalin) and amphetamine with dextroamphetamine (Adderall), have stimulant qualities and elevate dopamine activity. People with ADHD show less dopamine activity in key regions of the brain, especially those associated with motivation and reward (Volkow et al., 2009), which provides support to the theory that dopamine deficits may be a vital factor in the development this disorder (Swanson et al., 2007).

Brain imaging studies have shown that children with ADHD exhibit abnormalities in their frontal lobes, an area in which dopamine is in abundance. Compared to children without ADHD, those with ADHD appear to have smaller frontal lobe volume, and they show less frontal lobe activation when performing mental tasks. Recall that one of the functions of the frontal lobes is to inhibit our behavior. Thus, abnormalities in this region may go a long way toward explaining the hyperactive, uncontrolled behavior of ADHD.

By the 1970s, many had become aware of the connection between nutritional factors and childhood behavior. At the time, much of the public believed that hyperactivity was caused by sugar and food additives, such as artificial coloring and flavoring. Undoubtedly, part of the appeal of this hypothesis was that it provided a simple explanation of (and treatment for) behavioral problems in children. A statistical review of 16 studies, however, concluded that sugar consumption has no effect at all on the behavioral and cognitive performance of children (Wolraich, Wilson, & White, 1995).



Figure **13.7.2**: Although foods with high sugar content aren't likely to be healthy and may contribute to temporary hyperactivity even in children without ADHD, no evidence supports the idea that the avoidance of sugar prevents or cures ADHD.

Additionally, although food additives have been shown to increase hyperactivity in non-ADHD children, the effect is rather small (McCann et al., 2007). Numerous studies, however, have shown a significant relationship between exposure to nicotine in cigarette smoke during the prenatal period and ADHD (Linnet et al., 2003). Maternal smoking during pregnancy is associated with the development of more severe symptoms of the disorder (Thakur et al., 2013).

Is ADHD caused by poor parenting? Not likely. Remember, the genetics studies discussed above suggested that the family environment does not seem to play much of a role in the development of this disorder; if it did, we would expect the concordance rates to be higher for fraternal twins and adoptive siblings than has been demonstrated. All things considered, the evidence seems to point to the conclusion that ADHD is triggered more by genetic and neurological factors and less by social or environmental ones.

Treatment

The management of ADHD typically involves psychotherapy or medications either alone or in combination. While treatment may improve long-term outcomes, it does not get rid of negative outcomes entirely. Medications used include stimulants and sometimes antidepressants. In those who have trouble focusing on long-term rewards, a large amount of positive reinforcement has shown to improve task performance. ADHD stimulants also improve persistence and task performance in children with ADHD.

The use of behavioral therapies in ADHD is the recommended first-line treatment in those who have mild symptoms or are preschool-aged (and often too young for medication). Psychological therapies used include the following: psychoeducational input, behavior therapy, CBT, interpersonal psychotherapy, family therapy, school-based interventions, social skills training, behavioral peer intervention, organization training, parent management training, and neurofeedback. Training in social skills, behavioral modification, and medication may have some limited beneficial effects. The most important factor in reducing later psychological problems, such as major depression, criminality, school failure, and substance use disorders, is the formation of friendships with people who are not involved in delinquent activities.



Figure **13.7.3**: Exercising while on stimulant medication augments the effect of the medication on executive function. It is believed that these short-term effects of exercise are mediated by an increased abundance of synaptic dopamine and norepinephrine in the brain.

Regular physical exercise, particularly aerobic exercise, is an effective add-on treatment for ADHD in children and adults, particularly when combined with stimulant medication, although the best intensity and type of aerobic exercise for improving symptoms are not currently known. In particular, the long-term effects of regular aerobic exercise in ADHD individuals include better behavior and motor abilities, improved executive functions (including attention, inhibitory control, and planning, among

other cognitive domains), faster information processing speed, and better memory. Parent-teacher ratings of behavioral and socio-emotional outcomes in response to regular aerobic exercise include better overall function, reduced ADHD symptoms, better self-esteem, reduced levels of anxiety and depression, fewer somatic complaints, better academic and classroom behavior, and improved social behavior.

Stimulant medications are the pharmaceutical treatment of choice. Stimulants have at least some effect on symptoms: in the short term, in about 80% of people. Methylphenidate appears to improve symptoms as reported by teachers and parents. There is little evidence on the effects of medication on social behaviors and as of 2015, the long-term effects of ADHD medication have yet to be fully determined. MRI studies suggest that long-term treatment with amphetamine or methylphenidate decreases abnormalities in brain structure and function found in subjects with ADHD. A 2018 review found the greatest short-term benefit with methylphenidate in children and amphetamines in adults.

Dig Deeper: Why Is the Prevalence Rate of ADHD Increasing?

Many people believe that the rates of ADHD have increased in recent years, and there is evidence to support this contention. In a recent study, investigators found that the parent-reported prevalence of ADHD among children (four through 17 years old) in the United States increased by 22% during a four-year period, from 7.8% in 2003 to 9.5% in 2007 (CDC, 2010). Over time, this increase in parent-reported ADHD was observed in all sociodemographic groups and was reflected by substantial increases in 12 states (Indiana, North Carolina, and Colorado were the top three). The increases were greatest for older teens (ages 15–17), multiracial and Hispanic children, and children with a primary language other than English. Another investigation found that from 1998 to 2000 through 2007–2009 the parent-reported prevalence of ADHD increased among U.S. children between the ages of five to 17 years old, from 6.9% to 9.0% (Akinbami, Liu, Pastor, & Reuben, 2011).

A major weakness of both studies was that children were not actually given a formal diagnosis. Instead, parents were simply asked whether or not a doctor or other health-care provider had ever told them their child had ADHD; the reported prevalence rates thus may have been affected by the accuracy of parental memory. Nevertheless, the findings from these studies raise important questions concerning what appears to be a demonstrable rise in the prevalence of ADHD. Although the reasons underlying this apparent increase in the rates of ADHD over time are poorly understood and, at best, speculative, several explanations are viable: ADHD may be over-diagnosed by doctors who are too quick to medicate children as a behavior treatment. There is greater awareness of ADHD now than in the past. Nearly everyone has heard of ADHD, and most parents and teachers are aware of its key symptoms. Thus, parents may be quick to take their children to a doctor if they believe their child possesses these symptoms, or teachers may be more likely now than in the past to notice the symptoms and refer the child for evaluation. The use of computers, video games, iPhones, and other electronic devices has become pervasive among children in the early 21st century, and these devices could potentially shorten children's attention spans. Thus, what might seem like inattention to some parents and teachers could simply reflect exposure to too much technology. ADHD diagnostic criteria have changed over time.

Glossary

attention deficit/hyperactivity disorder (ADHD): childhood disorder characterized by inattentiveness and/or hyperactive, impulsive behavior

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13.8: Specific Learning Disorder

Learning Objectives

- Describe the characteristics and diagnosis of specific learning disorders

DSM-5 Diagnostic Criteria

Specific learning disorder is a classification of disorders in which a person has difficulty learning in a typical manner within one of several domains. Often referred to as learning disabilities, learning disorders are characterized by inadequate development of specific academic, language, and speech skills. Types of learning disorders include difficulties in reading (dyslexia), mathematics (dyscalculia), and writing (dysgraphia).

The diagnosis of *specific learning disorder* was added to the DSM-5 in 2013. The DSM does not require that a single domain of difficulty (such as reading, mathematics, or written expression) be identified—instead, it is a single diagnosis that describes a collection of potential difficulties with general academic skills, simply including detailed specifiers for the areas of reading, mathematics, and writing. Academic performance must be below average in at least one of these fields, and the symptoms may also interfere with daily life or work. In addition, the learning difficulties cannot be attributed to other sensory, motor, developmental, or neurological disorders.

Learning disabilities are cognitive disorders that affect different areas of cognition, particularly language or reading. It should be pointed out that learning disabilities are not the same thing as intellectual disabilities. Learning disabilities are considered specific neurological impairments rather than global intellectual or developmental disabilities. Often, learning disabilities are not recognized until a child reaches school age. One confounding aspect of learning disabilities is that they most often affect children with average to above-average intelligence. In other words, the disability is specific to a particular area and not a measure of overall intellectual ability. At the same time, learning disabilities tend to exhibit comorbidity with other disorders, like attention-deficit hyperactivity disorder (ADHD).

Link to Learning

If you are interested in further reading on learning disabilities, read this post published in Psychology Today "Gifted Kids with Learning Problems."

Dyslexia

Dyslexia, sometimes called reading disorder, is the most common learning disability; of all students with specific learning disabilities, 70–80% have deficits in reading. The term *developmental dyslexia* is often used as a catch-all term, but researchers assert that dyslexia is just one of several types of reading disabilities. A reading disability can affect any part of the reading process, including word recognition, word decoding, reading speed, prosody (oral reading with expression), and reading comprehension.



Figure **13.8.1**: People with dyslexia may have difficulties in spelling words, reading quickly, writing words, "sounding out" words in their head, pronouncing words when reading aloud, and understanding what they're reading.

Dyscalculia

Dyscalculia is a form of math-related disability that involves difficulties with learning math-related concepts (such as quantity, place value, and time), memorizing math-related facts, organizing numbers, and understanding how problems are organized on the page. People with this dyscalculia are often referred to as having poor "number sense."

Dysgraphia

The term *dysgraphia* is often used as an overarching term for all disorders of written expression. Individuals with dysgraphia typically show multiple writing-related deficiencies, such as grammatical and punctuation errors within sentences, poor paragraph organization, multiple spelling errors, and excessively poor penmanship.

An additional type of learning disability is nonverbal, also known as NVLD. Nonverbal learning disabilities represent another type of learning difficulty in which individuals demonstrate adequate vocabulary, verbal expression, or reading skills, but present difficulties with certain nonverbal activities (e.g., problem-solving, visual-spatial tasks, reading body language, and recognizing social cues).

Link to Learning

Click here to watch a short video on nonverbal learning disorder (from a 12-year-old's perspective).

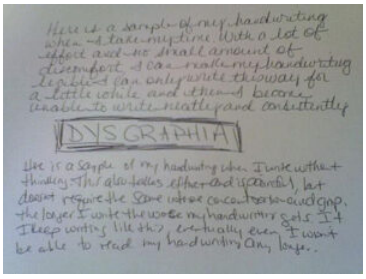


Figure **13.8.2**: Dysgraphia is often characterized by grammatical and punctuation errors within sentences, poor paragraph organization, multiple spelling errors, and excessively poor penmanship.

Etiology

The causes of learning disabilities are not well understood. However, some potential causes or contributing factors are

- heredity. Learning disabilities often run in the family—children with learning disabilities are likely to have parents or other relatives with similar difficulties.
- problems during pregnancy and birth. Learning disabilities can result from anomalies in the developing brain, illness or injury, fetal exposure to alcohol or drugs, low birth weight, oxygen deprivation, or premature or prolonged labor.
- accidents after birth. Learning disabilities can also be caused by head injuries, malnutrition, or toxic exposure (such as heavy metals or pesticides).

Epidemiology



Figure **13.8.3**: Dyscalculia tends to become more apparent as children get older; however, symptoms can appear as early as preschool. Dyscalculia is characterized by difficulties with common arithmetic tasks such as reading analog clocks; stating which of two numbers is larger; and basic adding, subtracting, multiplication, and division.

The DSM-5 estimates the prevalence of all learning disorders (including impairment in writing as well as in reading and/or mathematics) to be about five to 15% worldwide and the German S3 guideline names prevalence for reading and/or writing disorders of about 3%-8%.[1]

The percentage of people with dyslexia is unknown, but it has been estimated to be as low as 5% and as high as 17% of the population. While it is diagnosed more often in males, some believe that it affects males and females equally.

Dyscalculia is thought to be present in 3%-6% of the general population, but estimates by country and sample vary. Many studies have found prevalence rates by gender to be equivalent. Those that find a gender difference in prevalence rates often find dyscalculia higher in females, but some few studies have found prevalence rates higher in males.

The prevalence for developmental writing disorders is about seven to 15% among school-aged children, with boys being more affected than girls by two to three times.[2]

Treatment

Individuals with learning disorders face unique challenges that may persist throughout their lives. Depending on the type and severity of their disability, interventions and technology may be used to help the individual learn strategies that will foster future success. Some interventions can be quite simple while others are intricate and complex. Teachers, parents, and schools can work together to create a tailored plan for intervention and accommodation to aid an individual in successfully becoming an independent learner. School psychologists and other qualified professionals often help design and manage such interventions. Social support may also improve learning for students with learning disabilities.

Glossary

specific learning disorder: a classification of disorders in which a person has difficulty learning in a typical manner within one of several domains; often referred to as *learning disabilities*

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13.9: Introduction to Disruptive, Impulse-Control, and Conduct Disorders

What you'll learn to do: examine and explain disruptive, impulse-control, and conduct disorders



In this section, you will review and learn to identify childhood disorders characterized by irritability and defiance. These include conduct disorder and oppositional defiance disorder. You will read about the various symptoms, epidemiology, how individuals present with these problems, and a brief overview of possible causes.

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13.10: Conduct Disorder

Learning Objectives

- For an overview of disruptive, impulse control, and conduct disorders, watch this video:

Conduct disorder (CD) is a mental disorder diagnosed in childhood or adolescence that presents itself through a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate norms are violated. These behaviors are often referred to as *antisocial behaviors*. Conduct disorder (CD) is often seen as the precursor to antisocial personality disorder, which is per definition not diagnosed until the individual is 18 years old. Conduct disorder is estimated to affect 51.1 million people globally as of 2013.

Signs and Symptoms

According to DSM-5 criteria for conduct disorder (CD), there are four categories that could be present in the child's behavior: aggression to people and animals, destruction of property, deceitfulness or theft, and serious violation of rules.



Figure **13.10.1**: Conduct disorder is characterized by aggressive and destructive behavior. It is diagnosed in childhood or adolescence and is often seen as a precursor to the adult-diagnosis of antisocial personality disorder. It affects more boys than girls.

One of the symptoms of CD is a lower level of fear. Research performed on the impact of toddlers exposed to fear and distress shows that negative emotionality (fear) predicts toddlers' empathy-related response to distress. The findings support that if a caregiver is able to respond to infant cues, the toddler has a better ability to respond to fear and distress. If a child does not learn how to handle fear or distress, the child will be more likely to lash out at other children. If the caregiver is able to provide therapeutic intervention teaching children at risk better empathy skills, the child will have a lower incident level of conduct disorder.

Increased instances of violent and antisocial behavior are also associated with the condition; examples may range from pushing, hitting, and biting when the child is young, progressing towards beating and inflicted cruelty as the child becomes older.

Conduct disorder can present with limited prosocial emotions, lack of remorse or guilt, lack of empathy, lack of concern for performance, and shallow or deficient affect. Symptoms vary by individual, but the four main groups of symptoms are described below.

- aggression to people and animals
  - often bullies, threatens, or intimidates others
  - often initiates physical fights
  - has used a weapon that can cause serious physical harm to others (e.g., a bat, brick, broken bottle, knife, gun)
  - has been physically cruel to people
  - has been physically cruel to animals
  - has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, or armed robbery)
  - has forced someone into sexual activity (rape or molestation)
- destruction of property
  - has deliberately engaged in fire setting with the intention of causing serious damage
  - has deliberately destroyed others' property (other than by fire setting)
- deceitfulness or theft
  - has broken into someone else's house, building, or car

- often lies to obtain goods or favors or to avoid obligations (i.e., cons others)
- has stolen items of nontrivial value without confronting a victim (e.g., shoplifting, but without breaking and entering; forgery)

- serious violations of rules
  - often stays out at night despite parental prohibitions, beginning before age 13
  - has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning for a lengthy period)
  - is often truant from school, beginning before age 13

Currently, two possible developmental courses are thought to lead to conduct disorder. The first is known as the *childhood-onset* type and occurs when conduct disorder symptoms are present before the age of 10. This course is often linked to a more persistent life course and more pervasive behaviors. Specifically, children in this group have greater levels of ADHD symptoms, neuropsychological deficits, more academic problems, increased family dysfunction, and higher likelihood of aggression and violence.

There is debate among professionals regarding the validity and appropriateness of diagnosing young children with conduct disorder. The characteristics of the diagnosis are commonly seen in young children who are referred to mental health professionals. A premature diagnosis made in young children, and thus labeling and stigmatizing an individual, may be inappropriate. It is also argued that some children may not in fact have conduct disorder, but are engaging in developmentally appropriate disruptive behavior.

The second developmental course is known as the *adolescent-onset* type and occurs when conduct disorder symptoms are present after the age of 10 years. There is also the additional onset-type of “unspecified” when it’s not known whether symptoms began prior to age 10.

In addition to these two courses that are recognized by the DSM-5, there appears to be a relationship among oppositional defiant disorder, conduct disorder, and antisocial personality disorder. Specifically, research has demonstrated continuity in the disorders such that conduct disorder is often diagnosed in children who have been previously diagnosed with oppositional defiant disorder, and most adults with antisocial personality disorder were previously diagnosed with conduct disorder. For example, some research has shown that 90% of children diagnosed with conduct disorder had a previous diagnosis of oppositional defiant disorder. Moreover, both disorders share relevant risk factors and disruptive behaviors, suggesting that oppositional defiant disorder is a developmental precursor and milder variant of conduct disorder. However, this is not to say that this trajectory occurs in all individuals. In fact, only about 25% of children with oppositional defiant disorder will receive a later diagnosis of conduct disorder.

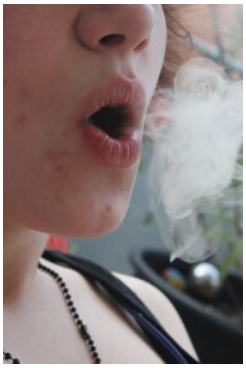


Figure **13.10.2**: Almost all adolescents who have a substance use disorder have conduct disorder-like traits, but after successful treatment for substance abuse, about half of these adolescents no longer display conduct disorder-like symptoms. Therefore, it is important to exclude a substance-induced cause and instead address the substance use disorder prior to making a psychiatric diagnosis of conduct disorder.

Correspondingly, there is an established link between conduct disorder and the diagnosis of antisocial personality disorder as an adult. In fact, the current diagnostic criteria for antisocial personality disorder requires a conduct disorder diagnosis before the age of 15 *if* the patient being diagnosed is still under 18 years old. Having a conduct disorder is a risk factor for later diagnosis of antisocial personality disorder. However, again, only 25–40% of youths with conduct disorder will develop antisocial personality disorder. Nonetheless, many of the individuals who do not meet the full criteria for antisocial personality disorder still exhibit a

pattern of social and personal impairments or antisocial behaviors. These developmental trajectories suggest the existence of antisocial pathways in certain individuals, which have important implications for both research and treatment.

Epidemiology

As stated before, this disorder is estimated to affect approximately 51 million people globally as of 2013. The percentage of children affected by conduct disorder is estimated to range from one to 10%. However, among incarcerated youth or youth in juvenile detention facilities, rates of conduct disorder are between 23% and 87%.

An epidemiological meta-analysis estimated that the worldwide prevalence of conduct disorder among children and adolescents aged six to 18 years is 3.2% and the prevalence estimate does not vary significantly across countries.

Conduct disorder can have its onset before ten years of age or in adolescence, and children with early onset conduct disorder are at greater risk for persistent difficulties. Current data indicates that the prevalence of conduct disorder is 2%-5% in children between five an 12 years old and 5%-9% in adolescents between 13 and 18 years old. Most studies show that boys are more likely to present with symptoms of conduct disorder than girls. However, this gender difference may vary somewhat across development. In young children under five years age, gender differences are small. This changes in adolescence, where both genders show an increase in the rates of conduct disorder and boys are two to three times more likely to be diagnosed than girls. Conduct disorder prevalence may or may not vary in different races and ethnicities depending on SES, neighborhood, and parenting practices. According to current data, the lifetime prevalence of conduct disorder is 6.9% for Hispanics, 4.9% for Blacks, and 5.0% for Whites. Caucasian children are more likely to be diagnosed with oppositional defiant disorder whereas Black children are more likely to be diagnosed with conduct disorder. Male teens, minorities, and children from low-income families are likely to be diagnosed with severe problems linked to neurological, attention, and conduct functioning.[1]

Etiology

The etiology of CD is complex and results from an interaction between multiple biological and psychosocial factors.

Biological

- Various studies indicate a moderate degree of heritability for antisocial behavior, impulsivity, temperament, aggression, and insensitivity to punishment.
- Evidence for low levels of plasma dopamine beta-hydroxylase supports the finding of decreased activity of the noradrenergic system in the CD.
- Low levels of 5-Hydroxy Indole acetic acid (5-HIAA) levels in cerebrospinal fluid correlates with aggression and violence in adolescence.
- High testosterone levels are also associated with aggression.

Neurological

- Some studies suggest a correlation between resting frontal brain electrical activity (EEG) and aggression in children.
- Neuropsychological insults to the brain in early life can cause deficits in language, memory, and executive functioning leading to poor judgment and inability to plan and problem solve in crisis situations.
- Developmental delays causes poor social skills, learning disability, and below average intellectual capacity, thus contributing to difficulties in learning, academic difficulties, and low self-esteem and a tendency for children to engage in disruptive behaviors.
- Any traumatic brain injury, seizures, and neurological damage can contribute to aggression.

Parental and Family

- A home environment that lacks structure and adequate supervision with frequent marital conflicts between parents and inconsistent discipline leads to maladaptive behavior and/or the following:
    - harsh parenting with verbal and physical aggression towards children
    - children exposed to frequent domestic violence
    - the family history of criminality and disruptive behaviors in caregivers
    - substance abuse particularly alcohol dependence in parents
    - living in low social, economic conditions with overcrowding and unemployment lead to economic and social stress with lack of adequate parenting.

School

- school environment with large classroom size, increased ratio of children to teachers, lack of positive feedback from teachers
- lack of supportive staff and counseling to address socio-economic difficulties in children
- exposure to increased gang violence in the community

Comorbid Conditions

- Children with a difficult temperament demonstrate poor adaptability and frequent negative emotions.
- Almost one-third of children with ADHD have symptoms of conduct disorder and other central nervous system dysfunction or damage.
- Trauma-related disorders, particularly repeated physical and sexual abuse with maltreatment in children, can lead to a diagnosis of PTSD and other anxiety disorders.
- Mood disorders that include depression and bipolar disorder can be comorbid.
- Developmental disorders can also be present.

In summary, the most important risk factors that predict conduct disorder include impulsiveness, low IQ and low school achievement, inadequate parental supervision, punitive or erratic parental discipline, cold parental attitude, child physical abuse, parental conflict, disrupted families, antisocial parents, large family size, low family income, antisocial peers, high delinquency rate schools, and high-crime neighborhoods. However, for many risk factors, it is not known whether they have causal effects. Cadoret et al. examined children who had a biological family history of antisocial personality disorder who were adopted into either stable or pathologic (unstable, disruptive) homes. They determined the highest incidence of aggression, and conduct disorder occurred in children who had both the family history of antisocial behavior and were placed in disturbed adoptive homes.[2]

Treatment & Outlook

Assessment and multiple interviews with the family, caregivers, and the child or adolescent are a critical first step and should include as many resources as possible, including educators, social workers, clinical psychologists, and family physicians. Since so many risk factors are involved, effective treatment needs to include a way to prevent or address these issues; this can involve family and social systems-based approaches and begin with education both the patient and parents/caregivers regarding the disorder, complications, and possible long-term consequences, especially if it continues untreated. CBT skill training has been found to be helpful by teaching patients skills in responding to their anger through problem-solving steps and finding alternative responses. Medication treatment is mainly used for comorbid conditions, such as ADHD, and is not a sole (or main) response to treating CD.[3]

Sadly, youth diagnosed with conduct disorder have a higher degree of distress and impairment in virtually all domains of living compared to youth with other mental disorders. Many studies have demonstrated the long-term impact of conduct disorder as a developmental precursor of antisocial behavior and criminality. Conduct disorder diagnosed in childhood acts as a strong predictor of many problems in adolescence and adulthood, including mental illness, substance abuse, legal problems, school drop-out, and academic issues and occupational problems. In two longitudinal studies, children with comorbid conduct and depressive disorders had a higher risk of late-onset criminality and antisocial behavior compared to those with only emotional issues.[4]

Watch It

This video provides a detailed explanation of conduct disorders. Note that video includes a content warning for its portrayals of violence.



You can view the transcript for “Conduct Disorders in Children : Nip in the Bud” here (opens in new window).

Glossary

conduct disorder: a mental disorder diagnosed in childhood or adolescence that presents itself through a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate norms are violated

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13.11: Oppositional Defiant Disorder

Learning Objectives

- Describe the characteristics, etiology, and treatment of oppositional defiant disorder

Oppositional defiant disorder (ODD) is listed in the DSM-5 under disruptive, impulse-control, and conduct disorders and defined as “a pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness” in children and adolescents. Unlike children with conduct disorder (CD), children with oppositional defiant disorder are not aggressive towards people or animals, do not destroy property, and do not show a pattern of theft or deceit.

Signs and Symptoms

The DSM-5 states that the child must exhibit four out of the following eight signs and symptoms to meet the diagnostic threshold for oppositional defiant disorder (ODD):

- often loses temper
- is often touchy or easily annoyed
- is often angry and resentful
- often argues with authority figures or for children and adolescents, with adults
- often actively defies or refuses to comply with requests from authority figures or with rules
- often deliberately annoys others
- often blames others for his or her mistakes or misbehavior
- has been spiteful or vindictive at least twice within the past six months

These behaviors are mostly directed towards an authority figure such as a teacher or a parent. Although these behaviors can be typical among siblings, they must be observed with individuals other than siblings for an oppositional defiant disorder (ODD) diagnosis. Children with ODD can be verbally aggressive. However, they do not display physical aggressiveness, a behavior observed in conduct disorder. Furthermore, they must be perpetuated for longer than six months and must be considered beyond a normal child’s age, gender, and culture to fit the diagnosis.

For children under five years of age, these behaviors occur on most days over a period of six months. For children over five years of age, they occur at least once a week for at least six months. It is possible to observe these symptoms in only one setting, most commonly home. Thus the severity would be mild. If it is observed in two settings, then it would be characterized as moderate; if the symptoms are observed in three or more settings, then it would be considered severe. These patterns of behavior result in impairment at school and/or other social venues.

Effects of ODD can be greatly amplified by other disorders in comorbidity such as ADHD. Other common comorbid disorders include depression and substance use disorders.

Watch It

Watch this video for a brief overview of ODD:



You can view the transcript for "What Is Oppositional Defiant Disorder? | Child Psychology" here (opens in new window).

Etiology

There is no specific element that has yet been identified as directly causing ODD. Researchers looking precisely at the etiological factors linked with ODD are limited. The literature often examines common risk factors linked with all disruptive behaviors, rather than specifically ODD. Symptoms of ODD are also often believed to be the same as conduct disorder even though the disorders have their own respective set of symptoms. When looking at disruptive behaviors such as ODD, research has shown that the causes of behaviors are multifactorial. However, disruptive behaviors have been identified as being mostly due either to biological or environmental factors.

Genetic Influences



Figure 13.11.1: There has been little research on the genetics of ODD, perhaps, because this disorder has been viewed primarily as the result of ineffective parenting. Nevertheless, the heritability of ODD has been estimated to be around 0.60 and ODD is familial among families of ADHD youth.

Research indicates that parents pass on a tendency for externalizing disorders to their children that may be displayed in multiple ways, such as inattention, hyperactivity, or oppositional and conduct problems. Research has also shown that there is a genetic overlap between ODD and other externalizing disorders. Heritability can vary by age, age of onset, and other factors. Adoption and twin studies indicate that 50% or more of the variance causing antisocial behavior is attributable to heredity for both males and females. ODD also tends to occur in families with a history of ADHD, substance use disorders, or mood disorders, suggesting that a vulnerability to develop ODD may be inherited. A difficult temperament, impulsivity, and a tendency to seek rewards can also increase the risk of developing ODD. New studies into gene variants have also identified possible gene-environment (G x E) interactions, specifically in the development of conduct problems. A variant of the gene that encodes the neurotransmitter metabolizing enzyme monoamine oxidase-A (MAOA), which relates to neural systems involved in aggression and plays a key role in regulating behavior following threatening events. Brain imaging studies show patterns of arousal in areas of the brain that are associated with aggression in response to emotion-provoking stimuli.

Prenatal Factors and Birth Complications

Many pregnancy and birth problems are related to the development of conduct problems. Malnutrition, specifically protein deficiency, lead poisoning or exposure to lead, and mother's use of alcohol or other substances during pregnancy may increase the risk of developing ODD. In numerous research, substance abuse prior to birth has also been associated with developing disruptive

behaviors such as ODD. Although pregnancy and birth factors are correlated with ODD, strong evidence of direct biological causation is lacking.

Neurobiological Factors

Deficits and injuries to certain areas of the brain can lead to serious behavioral problems in children. Brain imaging studies have suggested that children with ODD may have subtle differences in the part of the brain responsible for reasoning, judgment, and impulse control. Children with ODD are thought to have an overactive behavioral activation system (BAS), and an underactive behavioral inhibition system (BIS). The behavioral activation system (BAS) stimulates behavior in response to signals of reward or non-punishment. The behavioral inhibition system (BIS) produces anxiety and inhibits ongoing behavior in the presence of novel events, innate fear stimuli, and signals of non-reward or punishment. Neuroimaging studies have also identified structural and functional brain abnormalities in several brain regions in youths with conduct disorders. These brain regions are the amygdala, prefrontal cortex, anterior cingulate, and insula, as well as interconnected regions.

Social-Cognitive Factors

As many as 40% of boys and 25% of girls with persistent conduct problems display significant social-cognitive impairments. Some of these deficits include immature forms of thinking (such as egocentrism), failure to use verbal mediators to regulate his or her behavior, and cognitive distortions such as interpreting a neutral event as an intentional hostile act. Children with ODD have difficulty controlling their emotions or behaviors. In fact, students with ODD have limited social knowledge that is based only on individual experiences, which shapes how they process information and solve problems cognitively. This information can be linked with the social information processing model (SIP) that describes how children process information in order to respond appropriately or inappropriately in social settings. Social information processing model (SIP) explains that children will go through five stages before displaying behaviors: encoding, mental representations, response accessing, evaluation, and enactment. However, children with ODD have cognitive distortions and impaired cognitive processes. This impairment will therefore directly impact their interactions and relationship negatively. It has been shown that social and cognitive impairments result in negative peer relationships, loss of friendship, and an interruption in socially engaging in activities. Children learn through observational learning and social learning. Therefore, observations of models have a direct impact and greatly influence children's behaviors and decision-making processes. Children often learn through modeling behavior. Modeling can act as a powerful tool to modify children's cognition and behaviors.

Environmental Factors

Negative parenting practices and parent-child conflict may lead to antisocial behavior, but they may also be a reaction to the oppositional and aggressive behaviors of children. Factors such as a family history of mental illnesses and/or substance abuse as well as a dysfunctional family and inconsistent discipline by a parent or guardian can lead to the development of behavior disorders. Parenting practices not providing adequate or appropriate adjustment to situations as well as a high ratio of conflicting events within a family have been shown to be causal factors of risk for developing ODD.

Insecure parent-child attachments can also contribute to ODD. Often little internalization of parent and societal standards exists in children with conduct problems. These weak bonds with their parents may lead children to associate with delinquency and substance abuse. Family instability and stress can also contribute to the development of ODD. Although the association between family factors and conduct problems is well established, the nature of this association and the possible causal role of family factors continues to be debated. In a number of studies, low SES has also been associated with disruptive behaviors such as ODD. Other social factors such as neglect, abuse, parents that are not involved, and lack of supervision can also contribute to ODD.

Externalizing problems are reported to be more frequent among minority-status youth, a finding that is likely related to economic hardship, limited employment opportunities, and living in high-risk, urban neighborhoods. Studies have also found that the state of being exposed to violence was also a contributing factor for externalizing behaviors to occur.

Epidemiology

Oppositional defiant disorder has a wide prevalence range, from one to 11%. The average prevalence is approximately 3.3%. Gender and age play an important role in the rate of the disorder. In fact, ODD gradually develops and becomes apparent in preschool years; often before the age of eight years old. However, it is very unlikely to emerge following early adolescence. There

is a difference in prevalence between boys and girls with the ratio being 1.4 (boys) to 1 (girls). On the other hand, girls' prevalence tends to increase after puberty. When researchers observed the general prevalence of oppositional defiant disorder throughout cultures, they noticed that it remained constant. However, the sex difference in ODD prevalence is only significant in Western cultures. Two possible explanations for this difference are that in non-Western cultures there is a decreased prevalence of ODD in boys or an increased prevalence of ODD in girls. Another factor is based on the criteria used to diagnose an individual. When the disorder was first included in the DSM-3, the prevalence was 25% higher than when the DSM-4 revised the criteria of diagnosis. The DSM-5 made more changes to the criteria, grouping certain characteristics together in order to demonstrate that ODD displays both emotional and behavioral symptomatology. In addition, criteria were added to help guide clinicians in diagnosis because of the difficulty found in identifying whether the behaviors or symptoms are directly related to the disorder or simply a phase in a child's life. Consequently, future studies may obtain results indicating a decline in prevalence between the DSM-4 and the DSM-5 due to these changes.

Management

Approaches to the treatment of ODD include parent management training, individual psychotherapy, family therapy, CBT, and social skills training. According to the American Academy of Child and Adolescent Psychiatry, treatments for ODD are tailored specifically to the individual child, and different treatment techniques are applied for preschoolers and adolescents.

Psychopharmacological Treatment

Psychopharmacological treatment is the use of prescribed medication in managing oppositional defiant disorder. Prescribed medication to control ODD include mood stabilizers, anti-psychotics, and stimulants. In two controlled randomized trials, it was found that between administered lithium and the placebo group, administering lithium decreased aggression in children with conduct disorder in a safe manner. However, a third study found the treatment of lithium over a period of two weeks invalid. Other drugs seen in studies include haloperidol, thioridazine, and methylphenidate (which also is effective in treating ADHD as it is a common comorbidity).

The effectiveness of drug and medication treatment for ODD is not well established. Effects that can result in taking these medications include hypotension, extrapyramidal symptoms, tardive dyskinesia, obesity, and an increase in weight. Psychopharmacological treatment is found to be most effective when paired with another treatment plan, such as individual intervention or multimodal intervention. In one case, a 16-year-old boy was given estrogen at an L.A. juvenile jail due to allegedly having ODD due to somewhat elevated testosterone levels; he developed gynecomastia and required breast reduction surgery as a result.

Individual Interventions



Figure **13.11.2**: Parent and family treatment has a low financial cost that can yield an increase in beneficial results.

Individual interventions are focused on child-specific individualized plans. These interventions include anger control/stress inoculation, assertiveness training, and child-focused problem solving skills training program, and self-monitoring skills. Anger control and stress inoculation help prepare the child for possible upsetting situations or events that may cause anger and stress. It includes a process of steps they may go through. Assertiveness training educates individuals in keeping a balance between passivity and aggression. It is about creating a response that is controlled and fair. Child-focused problem-solving skills training program aims to teach the child new skills and cognitive processes that teach how to deal with negative thoughts, feelings, and actions.

Parent and Family Treatment

According to randomized trials, evidence shows that parent management training is most effective. It has strong influences over a longer period of time and in various environments.

Parent-child interaction training is intended to coach the parents while involving the child. This training has two phases. The first phase being child-directed interaction, whereby it is focused on teaching the child non-directive play skills. The second phase is parent-directed interaction, where the parents are coached on aspects including clear instruction, praise for compliance, and time-out noncompliance. The parent-child interaction training is best suited for elementary-aged children.

Glossary

oppositional defiant disorder (ODD): defined as a pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness in children and adolescents

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13.12: Motor Disorders

Learning Objectives

- Describe the characteristics and etiology of motor disorders

Motor Disorders

The DSM-5 motor disorders include **developmental coordination disorder**; **stereotypic movement disorder**; and the tic disorders of **Tourette's Disorder**, **persistent (chronic) motor or vocal tic disorder**, and **provisional tic disorder**.

Motor disorders are malfunctions of the nervous system that cause involuntary or uncontrollable movements or actions of the body. These disorders can cause lack of intended movement or an excess of involuntary movement. Symptoms of motor disorders include tremors, jerks, twitches, spasms, contractions, or gait problems.

Causes of Motor Disorders

Pathological changes of certain areas of the brain are the main causes of most motor disorders. Causes of motor disorders by genetic mutation usually affect the cerebrum. The way humans move requires many parts of the brain to work together to perform a complex process. The brain must send signals to the muscles instructing them to perform a certain action. There are constant signals being sent to and from the brain and the muscles that regulate the details of the movement such as speed and direction, so when a certain part of the brain malfunctions, the signals can be incorrect or uncontrollable causing involuntary or uncontrollable actions or movements.

Developmental Coordination Disorder



Figure **13.12.1**: Some possible signs of dyspraxia.

Developmental coordination disorder (DCD), also known as developmental motor coordination disorder, developmental dyspraxia, or simply dyspraxia, is a chronic neurological disorder beginning in childhood. It is also known to affect planning of movements and coordination as a result of brain messages not being accurately transmitted to the body. Impairments in skilled motor movements per a child's chronological age interfere with activities of daily living. A diagnosis of developmental coordination disorder (DCD) is then reached only in the absence of other neurological impairments like cerebral palsy, multiple sclerosis, or Parkinson's disease.

The DSM-5 criteria are as follows:

- Motor coordination will be greatly reduced, although the intelligence of the child is normal for the age.
- The difficulties the child experiences with motor coordination or planning interfere with the child's daily life.
- The difficulties with coordination are not due to any other medical condition
- If the child also experiences comorbidities such as intellectual or other developmental disability; motor coordination is still disproportionally affected.

Management

There is no cure for the condition. Instead, it is managed through therapy. Physical therapy or occupational therapy can help those living with the condition.

Some people with the condition find it helpful to find alternative ways of carrying out tasks or organizing themselves, such as typing on a laptop instead of writing by hand or using diaries and calendars to keep organized. A review completed in 2017 by Cochrane of task-oriented interventions for developmental coordination disorder (DCD) resulted in inconsistent findings and a call for further research and randomized controlled trials.

Epidemiology

DCD is a lifelong neurological condition that is more common in males than in females, with a ratio of approximately four males to every female. The exact proportion of people with the disorder is unknown since the disorder can be difficult to detect due to a lack of specific laboratory tests, thus making diagnosis of the condition one of elimination of all other possible causes/diseases. Approximatelyfive to 6% of children are affected by this condition.

Watch It

Watch this video to get a better understanding of DCD and measures taken to learn more about the disorder:



You can view the transcript for "Understanding DCD (Developmental Coordination Disorder)" here (opens in new window).

Stereotypic Movement Disorder

Stereotyped movements are common in infants and young children; if the child is not distressed by movements and daily activities are not impaired, diagnosis is not warranted. When stereotyped behaviors cause significant impairment in functioning, an evaluation for stereotypic movement disorder is needed. There are no specific tests for diagnosing this disorder, although some tests may be ordered to rule out other conditions. Stereotyped movement disorder (SMD) may occur with Lesch–Nyhan syndrome, intellectual disability, and fetal alcohol exposure or as a result of amphetamine intoxication.

When diagnosing stereotypic movement disorder, DSM-5 calls for specification of

- with or without self-injurious behavior,
- association with another known medical condition or environmental factor, and
- severity (mild, moderate, or severe).

Common repetitive movements of stereotyped movement disorder (SMD) include head banging, arm-waving, hand-shaking, rocking and rhythmic movements, self-biting, self-hitting, and skin-picking; other stereotypies are thumb-sucking, nail-biting, trichotillomania, bruxism, and abnormal running or skipping.

Epidemiology

Stereotyped movement disorder (SMD) occurs in about 3%-4% of children.[1] Stereotypies often represent a physiological and transient finding, up to 60% of neurologically typical children showing some stereotypic movements or behaviors between two and five years. Therefore, SMD are classified as *primary*, indicating their presence in an otherwise typically developing child, or *secondary*, if another of the above-mentioned neuropsychiatric disorders is present.[2] Although not necessary for the diagnosis, individuals with intellectual disability are at higher risk for SMD. It is more common in boys and can occur at any age.

Management

Though we have yet to find a cure for these disorders, some studies have looked at the effectiveness of medication therapy but thus far (based on parent reports of medication trials) there haven't been any medication therapy identified to be effective in treating symptoms. However, behavioral therapy appears to be beneficial for those with primary SMD. Researchers found that therapy that focuses on a mix of awareness and reinforcement of other behaviors helped reduce unwanted movement. Additionally, another study looked at the effectiveness of home-based, parent-administered behavioral therapy and this assessment showed significant improvement compared to the baseline.[3]

Tic Disorders

Tourette Syndrome (TS or Tourette's)

To be diagnosed with Tourette syndrome (TS or Tourette's), a person must have

- two or more motor tics (for example, blinking or shrugging the shoulders) and at least one vocal tic (for example, humming, clearing the throat, or yelling out a word or phrase), although they might not always happen at the same time.
- had tics for at least a year. The tics can occur many times a day (usually in bouts) nearly every day or off and on.
- tics that begin before age 18 years.
- symptoms that are not due to taking medicine or other drugs or due to having another medical condition (for example, seizures, Huntington disease, or postviral encephalitis).



Figure **13.12.2**: Coughing is a common tic.

Tourette syndrome or Tourette's syndrome (TS or Tourette's) is a common neurodevelopmental disorder that begins in childhood or adolescence. It is characterized by multiple movement (motor) tics and at least one vocal (phonic) tic. Common tics are blinking, coughing, throat clearing, sniffing, and facial movements. These are typically preceded by an unwanted urge or sensation in the affected muscles, can sometimes be suppressed temporarily, and characteristically change in location, strength, and frequency. Tourette's is at the more severe end of a spectrum of tic disorders. The tics often go unnoticed by casual observers.

Tourette's was once regarded as a rare and bizarre syndrome and has popularly been associated with coprolalia (the utterance of obscene words or socially inappropriate and derogatory remarks). It is no longer considered rare; about 1% of school-age children and adolescents are estimated to have Tourette's, and coprolalia occurs only in a minority. There are no specific tests for diagnosing Tourette's; it is not always correctly identified because most cases are mild and the severity of tics decreases for most children as they pass through adolescence. Therefore, many go undiagnosed or may never seek medical attention. Extreme Tourette's in adulthood, though sensationalized in the media, is rare, but for a small minority, severely debilitating tics can persist into adulthood. Tourette's does not affect intelligence or life expectancy.

Tourette syndrome was named by French neurologist Jean-Martin Charcot for his intern, Georges Gilles de la Tourette, who published in 1885 an account of nine patients with a "convulsive tic disorder." While the exact cause is unknown, it is believed to involve a combination of genetic and environmental factors. The mechanism appears to involve dysfunction in neural circuits between the basal ganglia and related structures in the brain.

Persistent (Chronic) Motor or Vocal Tic Disorder

To be diagnosed with a persistent tic disorder, a person must

- have one or more motor tics (for example, blinking or shrugging the shoulders) or vocal tics (for example, humming, clearing the throat, or yelling out a word or phrase), but not both.
- have tics that occur many times a day nearly every day or on and off throughout a period of more than a year.
- have tics that start before age 18 years.
- have symptoms that are not due to taking medicine or other drugs or due to having a medical condition that can cause tics (for example, seizures, Huntington disease, or postviral encephalitis).
- not have been diagnosed with Tourette's.

Provisional Tic Disorder

To be diagnosed with a provisional tic disorder, a person must

- have one or more motor tics (for example, blinking or shrugging the shoulders) or vocal tics (for example, humming, clearing the throat, or yelling out a word or phrase).
- have been present for no longer than 12 months in a row.
- have tics that start before age 18 years.
- have symptoms that are not due to taking medicine or other drugs or due to having a medical condition that can cause tics (for example, Huntington disease or postviral encephalitis).
- not have been diagnosed with Tourette's or persistent motor or vocal tic disorder.

Epidemiology

Tic disorders are more common among males than females. At least one in five children experience some form of tic disorder, most frequently between the ages of seven and twelve. As many as one in 100 people may experience some form of tic disorder, usually before the onset of puberty.

Treatment of Tic Disorders

There is no cure for Tourette's and no single most effective medication. In most cases, medication for tics is not necessary, and behavioral therapies are the first-line treatment. Education is an important part of any treatment plan, and explanation alone often provides sufficient reassurance that no other treatment is necessary. Among those who are referred to specialty clinics, other conditions like ADHD and OCD are more likely than in the broader population of persons with Tourette's. These co-occurring diagnoses often cause more impairment to the individual than the tics; hence it is important to correctly distinguish co-occurring conditions and treat them.

Watch It

Watch this video to learn more about Tourette's and tic disorders.



> You can view the transcript for “Tourette’s syndrome & tic disorders – definition, symptoms, diagnosis, treatment” here (opens in new window).

Disorder	Diagnostic Criteria	Prevalence
Developmental coordination disorder	• Motor coordination will be greatly reduced, although the intelligence of the child is normal for the age. • The difficulties the child experiences with motor coordination or planning interfere with the child's daily life. • The difficulties with coordination are not due to any other medical condition	The exact proportion of people with the disorder is unknown since the disorder can be difficult to detect. Approximately 5%-6% of children are affected by this condition.
Stereotypic movement disorder	• A motor disorder with onset in childhood involving repetitive, nonfunctional motor behavior (e.g., hand waving or head banging), that markedly interferes with normal activities or results in bodily injury. • To be classified as SMD, the behavior in question must not be due to the direct effects of a substance or another medical condition. <https://en.Wikipedia.org/wiki/Stereo...ement_disorder>	SMD occurs in about 3%-4% of children. Stereotypies often represent a physiological and transient finding, up to 60% of neurologically typical children showing some stereotypic movements or behaviors between two and five years.
Tourette's Disorder	• have two or more motor tics (e.g., blinking or shrugging the shoulders) and at least one vocal tic (e.g., humming, clearing the throat, or yelling out a word or phrase), although they might not always happen at the same time. • have had tics for at least a year. The tics can occur many times a day (usually in bouts) nearly every day, or off and on. • have tics that begin before age 18 years. • have symptoms that are not due to taking medicine or other drugs or due to having another medical condition (for example, seizures, Huntington disease, or postviral encephalitis).	Tic disorders are more common among males than females. At least one in five children experience some form of tic disorder, most frequently between the ages of seven and twelve.
Persistent (chronic) motor or vocal tic disorder	• have one or more motor tics or vocal tics but not both. • have tics that occur many times a day nearly every day or on and off throughout a period of more than a year. • have tics that start before age 18 years. • have symptoms that are not due to taking medicine or other drugs, or due to having a medical condition that can cause tics (for example, seizures, Huntington disease, or postviral encephalitis).	Tic disorders are more common among males than females. At least one in five children experience some form of tic disorder, most frequently between the ages of seven and 12.

Disorder	Diagnostic Criteria	Prevalence
	• not have been diagnosed with Tourette's.	
Provisional tic disorder	• have one or more motor tics or vocal tics (could be both) • have been present for no longer than 12 months in a row. • have tics that start before age 18 years. • have symptoms that are not due to taking medicine or other drugs, or due to having a medical condition that can cause tics (for example, Huntington disease or postviral encephalitis). • not have been diagnosed with Tourette's or persistent motor or vocal tic disorder.	Tic disorders are more common among males than females. At least one in five children experience some form of tic disorder, most frequently between the ages of seven and 12.

Glossary

developmental coordination disorder: a chronic neurological disorder beginning in childhood also known to affect planning of movements and co-ordination as a result of brain messages not being accurately transmitted to the body

motor disorders: malfunctions of the nervous system that cause involuntary or uncontrollable movements or actions of the body and can cause lack of intended movement or an excess of involuntary movement

stereotypic movement disorder: a motor disorder with onset in childhood involving repetitive, nonfunctional motor behavior (e.g., hand waving or head banging), that markedly interferes with normal activities or results in bodily injury

tic disorders: a motor disorder that can involve motor tics (e.g., blinking or shrugging the shoulders) or vocal tics (e.g., humming, clearing the throat, or yelling out a word or phrase), although they might not always happen at the same time

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13.13: Elimination Disorders

Learning Objectives

- Describe elimination disorders and how they are treated

Pediatric Elimination Disorders

The main categories of pediatric elimination disorders include enuresis and encopresis. **Enuresis** is defined as voiding of urine into bed/clothing in children who are at least five years of age. For the diagnosis to be given, the voiding must occur at least twice per week for at least three months. Enuresis can occur during the day or at night or both. It is believed that daytime (diurnal) enuresis is different from nocturnal enuresis in biological pathways and medical comorbidity. **Encopresis** involves either voluntary or involuntary voiding of the bowels (fecal incontinence) in inappropriate places in children who are at least four years of age (symptoms must persist for at least three months).

Enuresis

Nocturnal enuresis usually presents with voiding of urine during sleep in a child in whom it is difficult to wake. It may be accompanied by bladder dysfunction during the day that is termed *non-mono symptomatic enuresis*. Daytime enuresis, also known as urinary incontinence, may also be accompanied by bladder dysfunction. Though in some cases, children may ignore a full bladder when enjoying playtime or other activities and have an accident when they can no longer hold it in.

Clinical definition of enuresis must meet the following criteria:

- The child repeatedly voids urine into bed or clothes (whether involuntary or intentional).
- The behavior must be clinically significant as manifested by either a frequency of twice a week for at least three consecutive weeks or the presence of clinically significant distress or impairment in social, academic (occupational), or other important areas of functioning.
- The child's chronological or developmental age is at least five years of age.
- The behavior is not due exclusively to the direct physiological effect of a substance (such as a diuretic) or a general medical condition (such as diabetes, spina bifida, a seizure disorder, etc.).

All these criteria must be met in order to diagnose an individual. Generally, healthcare providers may further investigate for bladder control issues if a child is still enuretic in the daytime by age four, or if they are still enuretic at nighttime by age five or six.

Epidemiology

Approximately 10% of six- to seven-year-olds around the world experience enuresis. While 15–20% of five-year-old children experience nocturnal enuresis, which usually goes away as they grow older, approximately 2%-5% of young adults experience nocturnal enuresis. About 3% of teenagers and 0.5%-1% of adults experience enuresis or bedwetting, with the chance of enuresis resolving being lower if it is considered frequent.

Etiology



Figure 13.13.1: Currently, nocturnal enuresis is understood to be caused by three main underlying factors: excess urine production at night, lack of capacity for bladder storage, and inability to wake from sleep, with pathogenesis possibly varying based on the presence of daytime symptoms.

Bedwetting children are often normal emotionally and physically, although enuresis can be caused by other health conditions. Primary nocturnal enuresis can have multiple causes, which can make approaching a course of treatment more difficult. Enuresis can be caused by one or more of the following: caffeine consumption (which increases urine production), pattern and volume of

fluid intake (which could be remedied by drinking more throughout the day and less in the evening before bed), less bladder capacity, dysfunctional voiding (an obstruction of the bladder due to muscles controlling urine flow that do no completely relax, thus making the person feel that their bladders is always full and strain to urinate), urinary tract infection, delay in maturation and development (which can be worsened if a child experiences stress and/or anxiety), bladder instability, nocturnal polyuria (which involves altered nighttime secretions of a hormone that controls water retention in the body), sleep disorders (specifically those that may cause an inability or difficulty arousing from sleep), and genetics (although several genes are considered of interest in relation to enuresis, lack of a single gene that may cause enuresis means that individuals of a family may have differing genetic mechanisms resulting in the condition).

One particular study examined possible environmental factors leading to enuresis and concluded that a history of anxiety in the early years of childhood (as well as a parental history of anxiety symptoms) predicted the development of this disorder; the greater the anxiety and depression, the lower the functioning at age three and more likely a child was to have a longer duration of enuresis. Children with comorbid ADHD had elevated ADHD and depression symptoms. Additionally, in line with other studies that have looked at the underlying causes of enuresis, the researchers found that humiliation and lowered self-esteem may be associated with this disorder and become a risk factor for developing symptoms of depression later on in life.[1]

Effective Therapies for Pediatric Elimination Disorder

Urine alarm therapy involves use of sensors that detect moisture, either worn on the body or placed in a pad that lies atop the mattress. When moisture is detected, the child is alerted (e.g., alarm or tactile) to go use the bathroom. Via behavioral conditioning, the child learns to recognize when his or her bladder is full before the alarm sounds, thereby eliminating the need for continued use of the alarm. This approach has been extensively studied.

Dry-bed training is a multicomponent intervention. Based on operant conditioning principles, this approach pairs the urine alarm with behavioral strategies such as a frequent waking schedule and overcorrection for bedwetting (e.g., child has to change bedding). Dry-bed training is most effective when paired with the urine alarm, as opposed to use in isolation. Likewise, dry-bed training appears to be superior to the urine alarm used alone.

Full spectrum home therapy (FSHT) is a manual-based, multicomponent intervention that utilizes the urine alarm, several behavioral strategies, and graduated over-learning. Over-learning involves drinking increasing amounts of water prior to bedtime. FSHT was designed to be less burdensome on the child and family than dry-bed training (e.g., there is no frequent waking schedule).

Lifting is a Level 3 intervention. Simply, the parent lifts or walks the child to the bathroom to the toilet during the night, then returns him or her to bed. Despite evidence of a positive effect, there is some concern about parental burden with this treatment.

Hypnotherapy for enuresis has success rates equivalent to what is seen with spontaneous remission, without treatment (about 16%).

Retention control training involves encouraging the child to postpone urination for as long as possible. The goal is to increase bladder capacity.

Biofeedback for encopresis involves the use of electrodes or balloons, placed in or around the anus, which measure anorectal functioning. The goal is to help patients observe their own muscular contractions on a monitor and to teach them to tighten and relax anorectal muscles on command in order to improve bowel function.

Enhanced Toilet Training (ETT) is a behavior modification approach that utilizes education and training and defection (e.g., breathing techniques, training muscle contraction, and relaxation). Contingency-based reward can also be used in ETT.[2]

Link to Learning

Watch this video for an overview of nocturnal enuresis.

Table 1: Tested Therapies for Nocturnal Enuresis

Level One: Works well	• Urine alarm • Dry-bed training
Level Two: Works	• Full spectrum home therapy
Level Three: Might work	• Lifting
Level Four: Experimental	• None
Level Five: Tested and does not work	• Hypnotherapy • Retention control training

Table 2: Tested Therapies for Encopresis

Level One: Works well	• None
Level Two: Works	• Biofeedback • Enhanced toilet training
Level Three: Might work	• None
Level Four: Experimental	• None
Level Five: Tested and does not work	• None

Encopresis

Symptoms involve involuntary or voluntary soiling of undergarments. There are two types: with or without constipation. Those with constipation may experience a decreased appetite, abdominal pain, have pain on defecation, have fewer bowel movements, and have hard or soft stools. Those without constipation do not have these symptoms.

The DSM-r clinical diagnosis criteria are

1. erpeated passage of feces into inappropriate places (e.g., underwear or floor) whether voluntary or unintentional.
2. at least one such event a month for at least three months.
3. chronological age of at least four years (or equivalent developmental level).
4. the behavior is not exclusively due to a physiological effect of a substance (e.g., laxatives) or a general medical condition, except through a mechanism involving constipation.

The DSM-5 recognizes two subtypes: with constipation and overflow incontinence and without constipation and overflow incontinence.

Epidemiology

Between 1.5% and 7.5% of children suffer from encopresis; 25% of visits to pediatric gastroenterology clinics and 3% of visits to general pediatric clinics are due to encopresis.[3] The estimated prevalence of encopresis in four-year-olds is between 1%-3%. The disorder is thought to be more common in males than females, by a factor of six to one.

Etiology



Figure 13.13.2: The onset of encopresis is most often benign. The usual onset is associated with toilet training, demands that the child sit for long periods of time, and intense negative parental reactions to feces.

Encopresis is commonly caused by constipation, reflexive withholding of stool, due to various physiological, psychological, or neurological disorders, or from surgery (a somewhat rare occurrence). The colon normally removes excess water from feces, but if the feces or stool remains in the colon too long due to conditioned withholding or incidental constipation, so much water is removed that the stool becomes hard, and becomes painful for the child to expel in an ordinary bowel movement. A vicious cycle can develop, where the child may avoid moving his/her bowels in order to avoid the expected, painful toilet episode. This cycle can result in deeply conditioning the holding response; thus the rectal anal inhibitory response or anismus results. The rectal anal inhibitory response has been shown to occur even under anesthesia and when voluntary control is lost. The hardened stool continues to build up and stretches the colon or rectum to the point where the normal sensations associated with impending bowel movements do not occur. Eventually, softer stool leaks around the blockage and cannot be withheld by the anus, resulting in soiling. The child typically has no control over these leakage accidents, and may not be able to feel that they have occurred or are about to occur due to the loss of sensation in the rectum and the rectal anal inhibitory response. Strong emotional reactions typically result from failed and repeated attempts to control this highly aversive bodily product. These reactions, in turn, may complicate conventional treatments using stool softeners, sitting demands, and behavioral strategies.

Beginning school or preschool is another major environmental trigger with shared bathrooms. Feuding parents, siblings, moving, and divorce can also inhibit toileting behaviors and promote constipation. An initiating cause may become less relevant as chronic stimuli predominate.

Treatment

Many pediatricians will recommend the following three-pronged approach to the treatment of encopresis associated with constipation:

1. cleaning out
2. using stool softening agents
3. scheduled sitting times, typically after meals

The initial clean-out is achieved with enemas, laxatives, or both. The predominant approach today is the use of oral stool softeners like Movicol, Miralax, Lactulose, mineral oil, etc. Following that, enemas and laxatives are used daily to keep the stools soft and allow the stretched bowel to return to its normal size.

The child must be taught to use the toilet regularly to retrain his/her body. It is usually recommended that a child be required to sit on the toilet at a regular time each day and try to go for 10–15 minutes, usually soon (or immediately) after eating. Children are more likely to be able to expel a bowel movement right after eating. It is thought that creating a regular schedule of bathroom time will allow the child to achieve a proper elimination pattern. Repeated voiding success on the toilet itself helps it become a releasor stimulus for successful bowel movements.

Alternatively, when this method fails for six months or longer, a more aggressive approach may be undertaken using suppositories and enemas in a carefully programmed way to overcome the reflexive holding response and to allow the proper voiding reflex to take over. Failure to establish a normal bowel habit can result in permanent stretching of the colon. Certainly, allowing this problem to continue for years with constant assurances that the child “will grow out of it” should be avoided.

Dietary changes are an important management element. Recommended changes to the diet in the case of constipation-caused encopresis include:

1. reduction in the intake of constipating foods such as dairy, peanuts, cooked carrots, and bananas.
2. increase in high-fiber foods such as bran, whole wheat products, fruits, and vegetables.
3. higher intake of water.
4. limit drinks with caffeine, including soda and tea.
5. provide well-balanced meals and snacks, and limit fast foods/junk foods that are high in fats and sugars.

The standard behavioral treatment for functional encopresis, which has been shown to be highly effective, is a motivational system such as a contingency management system (usually an incentives-based system that rewards the child for positive behaviors and achieving goals in overcoming encopresis).

Glossary

enuresis: voiding of urine into bed/clothing in children who are at least five years of age

encopresis: voluntary or involuntary voiding of the bowels (fecal incontinence) in inappropriate places in children who are at least four years of age

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13.14: Introduction to Perspectives and Treatments Related to Childhood Disorders

What you'll learn to do: examine psychological approaches and treatments related to childhood disorders



Children are a vulnerable population and early intervention (and prevention) is often key in helping children recover from diagnoses or in developing techniques to adjust to having a mental disorder. Treatment and therapy can help children enjoy a higher qualify of life as they age into adolescence and adulthood.

Depending on one's theoretical framework (i.e., the way of thinking about a disorder) the type of treatment they prescribe (or which to receive) may vary. We'll delve more into that as well so you have a good understanding of the different ways of thinking about these neurodevelopmental disorders.

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13.15: Perspectives on Childhood Disorders

Learning Objectives

- Describe and compare viewpoints from the major psychological perspectives related to childhood disorders

The Biological Perspective

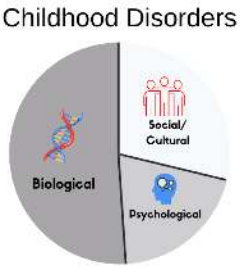


Figure **13.15.1**: Childhood disorders encompass a large category of disorders that can be explained by the interplay between biological, social/cultural, and psychological causes. Some childhood disorders, such as autism spectrum disorder, are known to have strong genetic and biological causes (autism is estimated to have a heritability rate of up to 90%, and ADHD up to 74%). Some disorders, such as learning disabilities cause by Down syndrome and fragile X syndrome can be entirely explained by biological factors. Other learning disorders and behavioral disorders have fewer biological ties and can be better explained by sociocultural and psychological factors.

As you know, the biological perspective focuses on the nature aspect regarding the etiology of childhood disorders. In other words, it looks at genetic or hereditary reasons for a disorder. Researchers and professionals who embrace this way of thinking believe that a genetic trait (something that someone is born with) is the underlying root of the mental health or development problem. Types of disorders that would fall in line with the biological perspective would be Down's syndrome, fragile X syndrome (both caused by genetic abnormalities), and motor disorders (caused by a genetic mutation affecting the cerebrum). It is believed that various genes and gene mutations may be an underlying cause for autism. In other disorders, such as stuttering, ADHD, learning disorders, and conduct disorders, there isn't enough evidence to suggest that nature alone is the reason for the development of these disorders, however, research suggests that there may be some familial heredity, indicating some genetic predisposition is at play. Twin studies and family mental health/development history can help researchers learn more about possible biological causes for disorders in children and adolescents.

The Sociocultural Perspective

The sociocultural perspective views a person's behaviors and/or symptoms in light of their culture and background. A critical part of development and growth includes social activities such as education and learning. Children learn from adults who, generally, provide an explanation of what one "can" or "can't" do, as well as providing children with a model of socially acceptable behavior; this can also be seen carried out through media and in other realms.

Psychologist Lev Vygotsky described and explained childhood disorders in light of the greater sociocultural context. For example, he viewed disorders as social abnormalities and argued that they're not actually abnormal behaviors unless they go against what is considered normal in a social context. A disability will psychologically affect someone very differently based on various social environments. Vygotsky provided an example by mentioning that a child who was blind would have a much different experience were they a young girl in America versus a young boy in Ukraine, the child of a highly affluent German, etc. According to Vygotsky, from this social perspective, the main issue for a child with a disability, is not the development, mental or neurological problem in and of itself, but rather the social implications that it bears.[1]

It is important to always consider the cross-cultural impact of research and how treatment might look in different settings with families that have different religious beliefs, social upbringing, and cultural contexts. This may be part of the neurodiverse movement as well (mentioned previously on the page discussing autism spectrum disorder). What would it look like if those with mild ASD weren't diagnosed but rather seen as functioning differently and socially accepted for who they were and how they interacted with the world?

The Psychodynamic Perspective

Psychodynamic theory suggests that there are three areas at work in determining a person's personality and behavior: the id, superego, and ego. Disorders that could possibly be viewed as being "stuck" in the id could be conduct disorders since these involve more primitive drives and urges of "acting out" versus trying to live up to standards of "right" and "wrong" within socially acceptable norms.

The Humanistic Perspective

The humanistic perspective rejects the idea of biological determinism—that regardless of nature, nurture has more influence in determining the potential outcomes of one's life. According to Rogers, parents can help their children achieve their ideal self by giving them unconditional positive regard, or unconditional love. In the development of self-concept, positive regard is key. Unconditional positive regard is an environment that is free of preconceived notions of value.

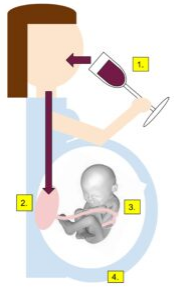


Figure **13.15.2**: FAS is caused by environmental influences that cause biological damage to a developing fetus.

According to Maslow's Hierarchy of Needs, at the very minimum, children must have their physiological, safety, and love/belonging needs met before they can achieve self-esteem and eventually, self-actualization. When the needs at the bottom of this hierarchy aren't met (due to abuse, neglect, etc.) this can lead to mental health and emotional concerns. These challenges are what could contribute to the development of disorders. Fetal alcohol syndrome (FAS) or oppositional defiance disorder, for example, may fall in line with this viewpoint since these are seen as being caused by the environment versus having a strictly biological cause. FAS is caused by environmental exposures, such as prenatal exposure to alcohol, drugs, and toxins. Though there is some research to indicate that genes, hereditary traits, and neurobiological factors may cause ODD, there are studies that show environmental risk factors that increase the risk of development for ODD. These factors include negative parenting practices, insecure parent-child attachments, and family instability—all areas that would affect the basic physiological and safety needs of a child.

The Cognitive Perspective

Cognitive psychology focuses on studying our thoughts and their relationship to our experiences and our actions. Viewed as a cognitive disorder and treated with CBT or other types of psychotherapy, ADHD symptoms have been found to be reduced; coupled with proper medication management, functioning may greatly improve.

Conduct disorders may be another category that could be viewed from this perspective. Most theories hold that, although inhibition of antisocial conduct is primarily mediated by affective empathy (i.e., vicarious affective responsiveness), cognitive dimensions of empathy such as perspective-taking skills also play a substantial role. For instance, it has been suggested that the ability to differentiate among and identify others' affective states and the ability to take their cognitive and affective perspective are prerequisites for empathizing and thereby inhibiting antisocial conduct.[2] With this prerequisites in mind, the types of treatments implicated could help children with conduct disorders possibly learn empathy and thus reduce symptoms of these disorders.

The Behavioral Perspective

Another way of looking at childhood disorders is through the lens of conditioning. If someone is *conditioned* to act or react a certain way, this conditioning could lead to the diagnosis of certain disorders. For example, a child with a learning disorder who is shamed for doing poorly in school may become averse to going to school. Some use this explanation to argue against labeling children with disorders. On the other hand, conditioning could be used to evoke the reaction a parent wishes to see in their child,

perhaps helping them sleep or concentrate better. This could be a helpful type of treatment for children with anxiety or easily distracted.[3]

Watch It

This video conveys important information about the causes of neurodevelopmental disorders and how they are influenced by multiple factors.



You can view the transcript for "Neurodevelopmental disorders: Sufficient and necessary causes | NCLEX-RN | Khan Academy" here (opens in new window).

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13.16: Treatments for Neurodevelopmental Disorders

Learning Objectives

- Examine and compare treatments for neurodevelopmental disorders

Interventions for Childhood Mental and Developmental Disorders

Neurodevelopmental disorders (ND) as grouped by DSM-5 include autism, ADHD, learning, communication and motor disorders, and intellectual disability. Neurodevelopmental disorders (ND) are defined as having an onset in the early developmental period, typically in early childhood. Neurodevelopmental disorders (ND) tend to have a steady rather than remitting and relapsing course and their core features show marked maturational changes from childhood to adult life. It is now recognized that these disorders, or at least some symptoms and impairment, persist well into adult life for many.[1] It is therefore imperative that as much prevention work and early treatment be done to mitigate these symptoms and help children grow into functioning adults with a higher quality of life.

The needs of children with ND are typically multivariate (i.e., they may need pharmacological, educational, and psychotherapeutic as well as social/economical support). Alas, health and community services for people with ND are often experienced as poorly integrated.[2] Schools have a profound influence on children, families, and communities. School-based mental health services also have the potential to bridge the gap between need and utilization by reaching children who would otherwise not have access to these services. These settings could provide an ideal environment in which programs for child mental health can be integrated in a cost-effective, culturally acceptable, and non-stigmatizing manner. Agencies in the voluntary sector (those that are nongovernment and not-for-profit) have traditionally played an important role in raising awareness of the issues faced by children with mental health difficulties and their families, as well as in reducing the associated stigma.[3]



Figure 13.16.1: Helping parents develop skills and strategies to support their children with mental disorders improves the emotional and behavioral outcomes for the children.

Providing early interventions to children with developmental disorders may optimize their developmental outcomes, and screening is necessary to identify children in need of these resource-intensive interventions. Parenting skills training aims to enhance or support the parental role through education and training, thereby improving emotional and behavioral outcomes for children. A meta-analysis identified four components of parenting skills training that were particularly effective. Increasing positive parent-child interactions, teaching parents how to communicate emotionally with their children, teaching parents the use of time out as a means of discipline, and supporting parents to consistently respond to their children's behaviors had the largest effects on reducing externalizing behaviors in children. Additionally, several systematic reviews have demonstrated the effectiveness of parenting skills training in reducing internalizing and externalizing problems in children, as well as in reducing the risk of unintentional childhood injuries and improving the mental health of parents. Childhood disruptive and externalizing behaviors may persist into adolescence, affecting peers, schools, and communities. Therefore, parenting skills interventions can reduce or prevent the onset of childhood mental disorders and subsequent adverse health and social outcomes.[4]

Autism

Early behavioral interventions or speech therapy can help children with autism gain self-care, social, and communication skills. Although there is no known cure, some autistic adults are able to live independently whereas others are not. An autistic culture has developed, with some individuals seeking a cure and others believing autism should be accepted as a difference to be accommodated instead of cured. This was also discussed in the "Neurodiversity" section on the page that reviewed ASD.

Watch It

This video explains neurodiversity and its different way of viewing autism (and similar disorders).

The main goals when treating children with autism are to lessen associated deficits and family distress and to increase the quality of life and functional independence. In general, higher IQs are correlated with greater responsiveness to treatment and improved treatment outcomes. As with most treatment for childhood disorders, no single treatment is best and treatment typically should be tailored to the child's needs. Families and the educational system are the main resources for treatment. Services should be carried out by behavior analysts, special education teachers, speech pathologists, and licensed psychologists. Studies of interventions have methodological problems that prevent definitive conclusions about efficacy, however, the development of evidence-based interventions has advanced in recent years. Intensive, sustained special education programs and behavior therapy early in life can help children acquire self-care, communication, and job skills and can often improve functioning and decrease symptom severity and maladaptive behaviors; claims that intervention by around age three years is crucial are not substantiated. While medications have not been found to help with core symptoms, they may be used for associated symptoms, such as irritability, inattention, or repetitive behavior patterns. Early, intensive, applied behavior analysis (ABA) therapy has demonstrated effectiveness in enhancing communication and adaptive functioning in preschool children; it is also well-established for improving the intellectual performance of that age group. Similarly, a teacher-implemented intervention that utilizes a more naturalistic form of ABA combined with a developmental social pragmatic approach has been found to be beneficial in improving social-communication skills in young children, although there is less evidence in its treatment of global symptoms.

ADHD

The most common method of treating ADHD is to prescribe stimulant medications such as Adderall. These medications treat many of the core symptoms of ADHD—treated children will show improved impulse control, time-on-task, and compliance with adults, and decreased hyperactivity and disruptive behavior. However, there are also negative side effects to stimulant medication, such as growth and appetite suppression, increased blood pressure, insomnia, and changes in mood (Barkley, 2006). Although these side effects can be unpleasant for children, they can often be avoided with careful monitoring and dosage adjustments.

Opinions differ on whether stimulants should be used to treat children with ADHD. Proponents argue that stimulants are relatively safe and effective, and that untreated ADHD poses a much greater risk to children (Barkley, 2006). Critics argue that because many stimulant medications are similar to illicit drugs, such as cocaine and methamphetamine, long-term use may cause cardiovascular problems or predispose children to abuse illicit drugs. However, longitudinal studies have shown that people taking these medications are not more likely to experience cardiovascular problems or to abuse drugs (Biederman, Wilens, Mick, Spencer, & Faraone, 1999; Cooper et al., 2011). On the other hand, it is not entirely clear how long-term stimulant treatment can affect the brain, particularly in adults who have been medicated for ADHD since childhood.

Finally, critics of psychostimulant medication have proposed that stimulants are increasingly being used to manage energetic but otherwise healthy children. It is true that the percentage of children prescribed stimulant medication has increased since the 1980s. This increase in use is not unique to stimulant medication, however. Prescription rates have similarly increased for most types of psychiatric medication (Olfson, Marcus, Weissman, & Jensen, 2002). As parents and teachers become more aware of ADHD, one would expect that more children with ADHD will be identified and treated with stimulant medication. Further, the percentage of children in the United States being treated with stimulant medication is lower than the estimated prevalence of children with ADHD in the general population (Nigg, 2006).



Figure **13.16.2**: Parent training may improve a number of behavioral problems including oppositional and non-compliant behaviors.

Parent Management Training

Parenting children with ADHD can be challenging. Parents of these children are understandably frustrated by their children's misbehavior. Standard discipline tactics, such as warnings and privilege removal, can feel ineffective for children with ADHD. This often leads to ineffective parenting, such as yelling at or ridiculing the child with ADHD. This cycle can leave parents feeling hopeless and children with ADHD feeling alienated from their family. Fortunately, parent management training can provide parents with a number of tools to cope with and effectively manage their child's impulsive and oppositional behavior. Parent management training teaches parents to use immediate, consistent, and powerful consequences (i.e., rewards and punishment), because children with ADHD respond well to these types of behavioral contingencies (Luman, Oosterlaan, & Sergeant, 2005). Other, more intensive, psychosocial treatments use similar behavioral principles in summer camp–based settings (Pelham, Fabiano, Gnagy, Greiner, & Hoza, 2004), and school-based intervention programs are becoming more popular.

? Treating ADHD in Schools

Succeeding at school is one of the most difficult challenges faced by children with ADHD and their parents. Teachers expect students to attend to lessons, complete lengthy assignments, and comply with rules for approximately seven hours every day. One can imagine how a child with hyperactive and inattentive behaviors would struggle under these demands, and this mismatch can lead to frustration for the student and his or her teacher. Disruptions caused by the child with ADHD can also distract and frustrate peers. Succeeding at school is an important goal for children, so researchers have developed and validated intervention strategies based on behavioral principles of contingency management that can help children with ADHD adhere to rules in the classroom (described in DuPaul & Stoner, 2003). Illustrative characteristics of an effective school-based contingency management system are described below:

Token reinforcement program

This program allows a student to earn tokens (points, stars, etc.) by meeting behavioral goals and not breaking rules. These tokens act as secondary reinforcers because they can be redeemed for privileges or goods. Parents and teachers work with the students to identify problem behaviors and create concrete behavioral goals. For example, if a student is disruptive during silent reading time, then a goal might be for him or her to remain seated for at least 80% of reading time. Token reinforcement programs are most effective when tokens are provided for appropriate behavior and removed for inappropriate behavior.

Time out

Time out can be an effective punishment when used correctly. Teachers should place a student in time out only when they fail to respond to token removal or if they engage in a severely disruptive behavior (e.g., physical aggression). When placed in time out, the student should not have access to any type of reinforcement (e.g., toys or social interaction), and the teacher should monitor their behavior throughout time out.

Daily report card

The teacher keeps track of whether or not the student meets his or her goals and records this information on a report card. This information is sent home with the student each day so parents can integrate the student's performance at school into a home-based contingency management program.

Educational services and accommodations

Students with ADHD often show deficits in specific academic skills (e.g., reading skills, math skills), and these deficits can be improved through direct intervention. Students with ADHD may spend several hours each week working one-on-one with an educator to improve their academic skills. Environmental accommodations can also help a student with ADHD be successful. For example, a student who has difficulty focusing during a test can be allowed extra time in a low-distraction setting.

The Multimodal Treatment Study

A large-scale study, the Multimodal Treatment Study (MTA) of Children with ADHD, compared pharmacological and behavioral treatment of ADHD (MTA Cooperative Group, 1999). This study compared the outcomes of children with ADHD in four different treatment conditions, including standard community care, intensive behavioral treatment, stimulant medication management, and the combination of intensive behavioral treatment and stimulant medication. In terms of core symptom relief, stimulant medication was the most effective treatment, and combined treatment was no more effective than stimulant medication alone (MTA

Cooperative Group, 1999). Behavioral treatment was advantageous in other ways, however. For example, children who received combined treatment were less disruptive at school than children receiving stimulant medication alone (Hinshaw et al., 2000). Other studies have found that children who receive behavioral treatment require lower doses of stimulant medication to achieve the desired outcomes (Pelham et al., 2005). This is important because children are better able to tolerate lower doses of stimulant medication. Further, parents report being more satisfied with treatment when behavioral management is included as a component in the program (Jensen et al., 2001). In sum, stimulant medication and behavioral treatment each have advantages and disadvantages that complement the other, and the best outcomes likely occur when both forms of treatment are used to improve children's behavior.

Learning Disorders

Various interventions can improve outcomes for children with learning disabilities, and often these can resolve on their own as children age. These include the mastery model (learners work at their own level of mastery, they practice, and gain fundamental skills before moving forward), direct instruction (careful planned lessons for small learning increments, correcting mistakes immediately, achievement-based grouping and frequent progress assessments), classroom adjustments (special seating assignment, alternative/modified assignments, quieter environment), special equipment (word processeres and spell checkers, text-to-speech/speech-to-text programs, talking calculators, computer-based activities), classroom assistants (note-takers, readers, proofreaders, scribes), and special education (such as prescribed hours or placement in a resource room, individual education plan and educational therapy).

Robert J. Sternberg, a professor of Psychology and Education at Yale University, has argued that early remediation can greatly reduce the number of children meeting diagnostic criteria for learning disabilities. He has also suggested that the focus on learning disabilities and the provision of accommodations in school fails to acknowledge that people have a range of strengths and weaknesses and places undue emphasis on academic success by insisting that people should receive additional support in this arena but not in music or sports. Other research has pinpointed the use of resource rooms as an important—yet often politicized component of educating students with learning disabilities.

? Learning Disabilities: a social construct of western concepts?

Learning disability theory is founded in the medical model of disability, in that disability is perceived as an individual deficit of biological origin. Researchers working within a social model of disability assert that there are social or structural causes of disability or the assigning of the label *disability*, and even that disability is entirely socially constructed. Since the turn of the 19th century, education in the United States has been geared toward producing citizens who can effectively contribute to a capitalistic society, with a cultural premium on efficiency and science. More agrarian cultures, for example, do not even use learning ability as a measure of adult adequacy. The diagnosis of learning disabilities is prevalent in Western capitalistic societies because of the high value placed on speed, literacy, and numeracy in both the labor force and the school system. In the bigger picture, these points demonstrate how the label of disability is socially constructed and represents a lack of fit between Western conceptions of educational institutions and proper students.

Communication Disorders

Treatment for communication disorders is usually carried out by speech and language therapists/pathologists, and/or audiologist, who use a wide range of techniques to stimulate language learning. Treatment can also include: language therapy, special education classes for children at school, and a psychologist if accompanying behavioral problems are present. These disorders can improve with time, depending on severity and early treatment.

Another way contemporary remediation differs from the past is that parents are more likely to be directly involved, but this approach is largely used with preschool children, rather than those whose problems persist into school age. For school-aged children, teachers are increasingly involved in intervention, either in collaboration with speech and language therapists/pathologists, or as the main agents of delivery of the intervention. Evidence for the benefits of a collaborative approach is emerging, but the benefits of asking education staff to be the main deliverers of speech-language therapy (SLT) intervention (the "consultative" approach) are unclear. When speech-language therapy (SLT) intervention is delivered indirectly by trained speech-language therapy (SLT) assistants, however, there are indications that this can be effective.

Motor Disorders

There is no known cure for motor disorders, but knowledge, education, and understanding are uppermost in management plans for these disorders, and psychoeducation is the first step. A child's parents are typically the first to notice the child's tics; they may feel worried, imagine that they are somehow responsible, or feel burdened by misinformation about Tourette's and other tic disorders. Parents may be overly concerned, and not realize that they, too, have tics while the child may not be bothered by the tics. In some cases, neither the parents nor their child know they have a tic; pointing out tics in this case will unnecessarily draw attention to them. In such a case, informing a parent who is unaware of their own tics can be disturbing, and they have a "right not to be informed." When their child receives a diagnosis, such as Tourette syndrome or developmental coordination disorder, both parent and child usually feel relieved, although the diagnosis can also cause distress for the parents as they confront a chronic condition that can be difficult to treat. Effectively educating parents about the diagnosis and providing social support can ease anxiety. This support can also lower the chance that their child will be unnecessarily medicated or experience an exacerbation of tics due to their parents' emotional state. Psychoeducation that encourages a matter-of-fact attitude and helps dispel misconceptions and stigma is most effective when provided to parents.

In some cases (specifically for DCD), physical or occupational therapy may be helpful. And in the case of tic disorders, behavioral therapies using habit reversal training (HRT) and exposure and response prevention (ERP) are first-line interventions, and have been shown to be effective. Because tics are somewhat suppressible, when people with Tourette's are aware of the premonitory urge that precedes a tic, they can be trained to develop a response to the urge that competes with the tic. Comprehensive behavioral intervention for tics (CBIT) is based on habit reversal training (HRT), and the best researched behavioral therapy for tics. Comprehensive behavioral intervention for tics (CBIT) has been shown with a high level of confidence to be more likely to lead to a reduction in tics than other behavioral therapies or psychoeducation.

Intellectual Disabilities

There are four broad areas of intervention that allow for active participation from caregivers, community members, clinicians, and of course, the individual(s) with an intellectual disability. These areas include psychosocial treatments, behavioral treatments, cognitive-behavioral treatments, and family-oriented strategies. Psychosocial treatments are intended primarily for children before and during the preschool years as this is the optimum time for an intervention. This early intervention should include encouragement of exploration; mentoring in basic skills; celebration of developmental advances; guided rehearsal and extension of newly acquired skills; protection from harmful displays of disapproval, teasing, or punishment; and exposure to a rich and responsive language environment. A great example of a successful intervention is the Carolina Abecedarian Project that was conducted with over 100 children from low SES families beginning in infancy through preschool years. Results indicated that by age two, the children provided the intervention had higher test scores than control group children, and they remained approximately five points higher 10 years after the end of the program. By young adulthood, children from the intervention group had better educational attainment, employment opportunities, and fewer behavioral problems than their control-group counterparts. (You can read more about this project here: https://abc.fpg.unc.edu/abecedarian-project).

Core components of behavioral treatments include language and social skills acquisition. Typically, one-to-one training is offered in which a therapist uses a shaping procedure in combination with positive reinforcements to help the child pronounce syllables until words are completed. Sometimes involving pictures and visual aids, therapists aim at improving speech capacity so that short sentences about important daily tasks (e.g., bathroom use, eating, etc.) can be effectively communicated by the child. In a similar fashion, older children benefit from this type of training as they learn to sharpen their social skills such as sharing, taking turns, following instruction, and smiling. At the same time, a movement known as social inclusion attempts to increase valuable interactions between children with an intellectual disability and their non-disabled peers. Cognitive-behavioral treatments, a combination of the previous two treatment types, involves a learning technique that teaches children math, language, and other basic skills pertaining to memory and learning. The first goal of the training is to teach the child to be a strategical thinker through making cognitive connections and plans. Then, the therapist teaches the child to be "metastrategical" by teaching them to discriminate among different tasks and determine which plan or strategy suits each task. Finally, family-oriented strategies delve into empowering the family with the skill set they need to support and encourage their child or children with an intellectual disability. In general, this includes teaching assertiveness skills or behavior management techniques as well as how to ask for help from neighbors, extended family, or day-care staff. As the child ages, parents are then taught how to approach topics such as housing/residential care, employment, and relationships. The ultimate goal for every intervention or technique is to give the child

autonomy and a sense of independence using the acquired skills he or she has. In a 2019 Cochrane review (on beginning reading interventions for children and adolescents with intellectual disability small to moderate improvements in phonological awareness), word reading, decoding, expressive and receptive language skills and reading fluency were noted when these elements were part of the teaching intervention.

Although there is no specific medication for intellectual disability, many people with developmental disabilities have further medical complications and may be prescribed several medications. Use of psychotropic medications such as benzodiazepines in people with intellectual disabilities requires monitoring and vigilance as side effects occur commonly and are often misdiagnosed as behavioral and psychiatric problems.

Watch It

This video gives an overview of current research looking into the possible reasons for childhood issues and ways to treat these early to prevent them from worsening in adolescence and adulthood.



You can view the transcript for "Discover NIMH: Diagnosis and Treatment in Children and Adolescents" here (opens in new window).

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13.17: Case Studies- Disorders of Childhood and Adolescence

Learning Objectives

- Identify disorders of childhood and adolescence in case studies

Case Study: Jake



An 11-year-old boy, Jake, was referred to an inpatient unit of the Children's Hospital for further diagnostic evaluation and treatment by the pediatric liaison team on call. He was socially isolated at school and in the rural community where he lived. He had behavioral difficulties at home and difficulties in adhering to the boundaries set by the parents. His mother labeled him as a "troublemaker" and he was oppositional at school with inappropriate behavior. He was frequently interfering with teaching in the classroom. Although he wanted to socialize with other children, he was clumsy and aggressive in his attempts to initiate contact. Teachers and other children's parent's complaints objectified the presence of behavioral problems. His behavior was described as aggressive and violent. His play and his reactions were often inappropriate and fear-provoking to others—i.e., he performed animal amputations, made and collected poisons, destroyed objects, and set fires.

Jake was born at full term and was described as a quiet baby. In the first three months of his life, his mother became worried as he was unresponsive to cuddles and hugs. He also never cried. He has no friends and, on occasions, he has been victimized by bullying at school and in the community. His father is 44 years old and describes having had a difficult childhood; he is characterized by the family as indifferent to the children's problems and verbally violent towards his wife and son, but less so to his daughters. The mother is 41 years old, and describes herself as having a close relationship with her children and mentioned that she usually covers up for Jake's difficulties and makes excuses for his violent outbursts.[1]

Treatment

During his stay (for two and a half months) in the inpatient unit, Jake underwent psychiatric and pediatric assessments plus occupational therapy. He took part in the unit's psycho-educational activities and was started on risperidone, two mg daily. Risperidone was preferred over an anti-ADHD agent because his behavioral problems prevailed and thus were the main target of treatment. In addition, his behavioral problems had undoubtedly influenced his functionality and mainly his relations with parents, siblings, peers, teachers, and others. Risperidone was also preferred over other atypical antipsychotics for its safe profile and fewer side effects. Family meetings were held regularly, and parental and family support along with psycho-education were the main goals. Jake was aided in recognizing his own emotions and conveying them to others as well as in learning how to recognize the emotions of others and to become aware of the consequences of his actions. Improvement was made in rule setting and boundary adherence. Since his discharge, he received regular psychiatric follow-up and continues with the medication and the occupational therapy. Supportive and advisory work is done with the parents. Marked improvement has been noticed regarding his social behavior and behavior during activity as described by all concerned. Occasional anger outbursts of smaller intensity and frequency have been reported, but seem more manageable by the child with the support of his mother and teachers.

In the case presented here, the history of abuse by the parents, the disrupted family relations, the bullying by his peers, the educational difficulties, and the poor SES could be identified as additional risk factors relating to a bad prognosis. Good prognostic factors would include the ending of the abuse after intervention, the child's encouragement and support from parents and teachers, and the improvement of parental relations as a result of parent training and family support by mental health professionals. Taken together, it appears that also in the case of psychiatric patients presenting with complex genetic aberrations and additional

psychosocial problems, traditional psychiatric and psychological approaches can lead to a decrease of symptoms and improved functioning.

Case Study: Kelli



A 15-year-old girl, Kelli, is referred to a neurologist due to unexplained symptoms of involuntary, uncontrollable behavior that includes eye-blinking, shoulder shrugging, frequent throat clearing, and randomly moving her arm around in circles. These symptoms have been present since she was in preschool and have increased in intensity. She told the doctor that she had difficulty reading because she was constantly blinking and had to re-read sentences over and over. She tried to control the behaviors at school because kids in her class called her “weird” and “freakshow,” but by the time she got home she was exhausted, experienced more involuntary movement, and had difficulty completing homework. When asked how she felt about her symptoms and experience, she began crying and asked if there was medication to “just make everything stop” so she could be “normal.”

Treatment

Kelli may benefit from a course of comprehensive behavioral intervention for her tics in addition to psychotherapy to treat any comorbid depression she experiences from isolation and bullying at school. Psychoeducation and approaches to reduce stigma will also likely be very helpful for both her and her family, as well as bringing awareness to her school and those involved in her education.

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1. Kolaitis, G., Bouwkamp, C.G., Papakonstantinou, A. et al. A boy with conduct disorder (CD), attention deficit hyperactivity disorder (ADHD), borderline intellectual disability, and 47,XXY syndrome in combination with a 7q11.23 duplication, 11p15.5 deletion, and 20q13.33 deletion. *Child Adolesc Psychiatry Ment Health* 10, 33 (2016). https://doi.org/10.1186/s13034-016-0121-8↵

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13.18: Putting It Together- Disorders of Childhood and Adolescence

In this chapter, you covered childhood neurodevelopmental disorders such as communication disorders, autism, and conduct disorders, including oppositional defiant disorder. By this point, you should have a basic, yet firm grasp on understanding how each is unique in its presentation of symptoms, how they affect children, and common treatment methods. Take a look at some of the popular treatment methods. What might a treatment plan look like if you were to develop one based on a certain psychological perspective? Or how might one's theoretical background or way of thinking about a disorder, prompt the research of a helpful method for treating it? How might you respond if you were a therapist treating someone with symptoms similar to those outlined in this module? How would you respond to the idea that some disabilities may simply be a matter of social construct or the perspective of neurodiversity?



As you reflect on what you've gleaned from this module, spend a moment reviewing the disorders discussed in this module:

- **Down syndrome**, also known as trisomy 21, is a genetic disorder caused by the presence of all or part of a third copy of chromosome 21.
- **Fetal alcohol syndrome** is a disorder caused by environmental factors due to ethanol exposure while developing in the womb.
- **Fragile X syndrome** is a genetic disorder resulting from a single gene mutation in FMR1.
- **Communication disorder:** any disorder that affects someone's ability to comprehend, detect, or apply language and speech to engage in discourse effectively with others. The following are types of communication disorders:
  - **Speech sound disorder**—previously called phonological disorder, for those with problems with pronunciation and articulation of their native language.
  - **Childhood-onset fluency disorder (stuttering)**—standard fluency and rhythm of speech is interrupted, often causing the repetition of whole words and syllables. May also include the prolongation of words and syllables, pauses within a word, and/or the avoidance of pronouncing difficult words and replacing them with easier words that the individual is better able to pronounce. This disorder causes many communication problems for the individual and may interfere with social communication and performance in work and/or school settings where communication is essential.
  - **Social (pragmatic) communication disorder**—this diagnosis described difficulties in the social uses of verbal and nonverbal communication in naturalistic contexts that affects the development of social relationships and discourse comprehension. The difference between this diagnosis and autism spectrum disorder is that in the latter there is also a restricted or repetitive pattern of behavior.
  - **Unspecified communication disorder**—for those who have symptoms of a communication disorder but who do not meet all criteria, and whose symptoms cause distress or impairment.
  - **Articulation disorders** (also called phonetic disorders)
  - **Phonemic disorders** (also called phonological disorders)
- **Autism spectrum disorder** is a childhood disorder characterized by deficits in social interaction and communication, and repetitive patterns of behavior or interests.
- **Neurodevelopmental disorder** is one of the disorders that are first diagnosed in childhood and involve developmental problems in academic, intellectual, social functioning.
- Attention-deficit/hyperactivity disorder is childhood disorder characterized by inattentiveness and/or hyperactive, impulsive behavior
- **Conduct disorder** is a mental disorder diagnosed in childhood or adolescence that presents itself through a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate norms are violated.

- **Oppositional defiant disorder (ODD)** is a disorder in the DSM-5 under disruptive, impulse-control, and conduct disorders, defined as “a pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness” in children and adolescents.
- **Motor disorders** are malfunctions of the nervous system that cause involuntary or uncontrollable movements or actions of the body. These disorders can cause a lack of intended movement or an excess of involuntary movement.
  - Developmental coordination disorder, also known as developmental motor coordination disorder, developmental dyspraxia, or simply, dyspraxia, is a chronic neurological disorder beginning in childhood. It is also known to affect planning of movements and coordination as a result of brain messages not being accurately transmitted to the body.
  - **Stereotypic movement disorder** is motor disorder with onset in childhood involving repetitive, nonfunctional motor behavior (e.g., hand waving or head banging), that markedly interferes with normal activities or results in bodily injury.
  - **Tic disorders** is a motor disorder that can involve motor tics (e.g., blinking or shrugging the shoulders) or vocal tics (e.g., humming, clearing the throat, or yelling out a word or phrase), although they might not always happen at the same time.
- Enuresis is defined as voiding of urine into bed/clothing in children who are at least five years of age.
- Encopresis is defined as either voluntary or involuntary voiding of the bowels (fecal incontinence) in inappropriate places in children who are at least four years of age.

Link to Learning

For an interesting perspective on working with challenging children, watch this TedX video.

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CHAPTER OVERVIEW

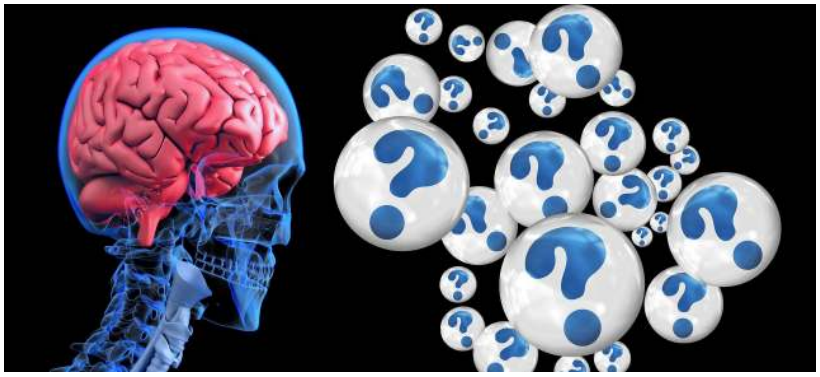
14: Neurocognitive Disorders

14.1: Why It Matters- Neurocognitive and Other Disorders
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14.1: Why It Matters- Neurocognitive and Other Disorders

Why learn about neurocognitive disorders?



Cognitive disorders (CDs), also known as **neurocognitive disorders (NCDs)**, are a category of mental health disorders that primarily affect cognitive abilities including learning, memory, perception, and problem-solving. Neurocognitive disorders include delirium and mild and major neurocognitive disorder (previously known as dementia). Cognitive disorders (CDs) are defined by deficits in cognitive ability that are acquired (as opposed to developmental), typically represent decline, and may have an underlying brain pathology. The DSM-5 defines six key domains of cognitive function: executive function, learning and memory, perceptual-motor function, language, complex attention, and social cognition.

Although Alzheimer's disease accounts for most cases of neurocognitive disorders, there are various medical conditions that affect mental functions such as memory, thinking, and the ability to reason, including frontotemporal degeneration, Huntington's disease, Lewy body disease, traumatic brain injury (TBI), Parkinson's disease, prion disease, and dementia/neurocognitive issues due to HIV infection. Neurocognitive disorders are diagnosed as mild and major based on the severity of their symptoms. Additionally, developmental disorders such as autism spectrum disorders are typically developed at birth or early in life as opposed to the acquired nature of neurocognitive disorders.

Causes vary between the different types of disorders, but most include damage to the memory portions of the brain. Treatments depend on how the disorder is caused. Medication and therapies are the most common treatments; however, for some types of disorders such as certain types of amnesia, treatments can suppress the symptoms but there is currently no cure.

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14.2: Introduction to Neurocognitive Disorders

What you'll learn to do: examine and explain major and mild neurocognitive disorders caused by Alzheimer's, Lewy bodies, and Parkinson's disease



Neurocognitive disorders are described as those with "a significant impairment of cognition or memory that represents a marked deterioration from a previous level of function." The subsections of neurocognitive disorders include delirium and mild and major neurocognitive disorder, which have multiple types and causes that we'll begin to examine in this section, including the well-known diseases of Alzheimer's and Parkinson's disease.

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14.3: Major and Mild Neurocognitive Disorders

Learning Objectives

- Describe and differentiate between major and mild neurocognitive disorders

Abnormal Loss of Cognitive Functioning during Late Adulthood

Neurocognitive disorders (NCDs), previously collectively referred to as *dementia*, are those that involve impairments in cognitive abilities such as memory, problem-solving, and perception. **Dementia** is the umbrella category used to describe the general long-term and often gradual decrease in the ability to think and remember that affects a person's daily functioning. The DSM-5 does not use the term *dementia*, but instead classifies the decline in cognitive functioning as either a type of major neurocognitive disorder if symptoms are severe, with milder symptoms classified as mild cognitive impairment.

Common symptoms of neurocognitive disorders (NCDs) include emotional problems, difficulties with language, and a decrease in motivation. A person's consciousness is usually not affected. About 10% of people develop the disorder at some point in their lives, and it becomes more common with age. About 3% of people between the ages of 65 and 74 have a neurocognitive disorder, 19% between 75 and 84, and nearly half of those over 85 years of age. In 2015, neurocognitive disorders (NCDs) resulted in about 1.9 million deaths, up from 0.8 million in 1990. As more people are living longer, neurocognitive disorders are becoming more common in the population as a whole. The DSM-5 has separate classifications for major neurocognitive disorders and mild neurocognitive disorders.



Figure **14.3.1**: Neurocognitive disorders include changes in cognitive abilities, usually including memory loss.

Neurocognitive disorders generally refer to severely impaired judgment, memory, or problem-solving ability. Neurocognitive disorders can occur before old age and is not an inevitable development even among the very old. Dementia can be caused by numerous diseases and circumstances, all of which result in similar general symptoms of impaired judgment, memory, and problem-solving ability. Alzheimer's disease is the most common form of neurocognitive disorder and is incurable, but there are also nonorganic causes of neurocognitive disorders that can be prevented. Malnutrition, alcoholism, depression, and mixing medications can also result in symptoms of dementia. If symptoms of dementia causes are properly identified, they can be treated. Cerebral vascular disease can also reduce cognitive functioning.



Figure **14.3.2**: Many people erroneously believe that all elderly people experience dementia, but that is not the case. Neurocognitive disorders caused by conditions such as Alzheimer's increase significantly with age, but half to two-thirds of the population above age 85 live life without experiencing a neurocognitive disorder.

Major neurocognitive disorder is a syndrome that progresses with significant deterioration of cognitive domains as compared to previous levels of cognitive performance in memory, speech, reasoning, intellectual function, and/or spatiotemporal perception, and may also be associated with changes in emotional behavior and difficulties at the functional level. The cognitive decline is initially noticed by the individual, the family, or the general practitioner/doctor who is usually responsible for the early diagnosis.[1] The

cognitive deficits interfere with daily life and require assistance from others to maintain everyday activities. The decline in functioning are not caused by delirium or another mental disorder.

Neurocognitive disorders can have numerous causes: genetics, brain trauma, stroke, and heart issues. The main causes are neurodegenerative diseases such as Alzheimer's disease, Parkinson's Disease, and Huntington's disease because they affect or deteriorate brain functions. Other diseases and conditions that cause NDCs include vascular dementia, frontotemporal degeneration, Lewy body disease, prion disease, normal pressure hydrocephalus, and dementia/neurocognitive issues due to HIV infection. These other conditions may also include dementia due to substance abuse or exposure to toxins.

Neurocognitive disorder may also be caused by brain trauma, including concussion and traumatic brain injuries, as well as post-traumatic stress and alcoholism. These examples are referred to as amnesia, and is characterized by damage to major memory encoding parts of the brain such as the hippocampus. Difficulty creating recent term memories is called anterograde amnesia and is caused by damage to the hippocampus part of the brain, which is a major part of the memory process. Retrograde amnesia is also caused by damage to the hippocampus, but the memories that were encoded or in the process of being encoded in long-term memory are erased.

Medications as well have an impact on amnesia, especially anti-anxiety drugs. Anterograde amnesia is also associated with anti-anxiety medications, such as lorazepam. Among benzodiazepines, lorazepam has relatively strong amnesic effects, but people soon develop tolerance to this medication with regular use. To avoid amnesia (or excess sedation) being a problem, the initial total daily lorazepam dose should not exceed two mg. Potential side effects of amnesia also apply to use for night sedation. For example, in a sleep study, five participants were prescribed lorazepam four mg at night, and the next evening, three subjects unexpectedly reported memory gaps for parts of that day. After two to three days of treatment, the amnesia effect subsided completely. Amnesic effects cannot be estimated from the degree of sedation present since the two effects are unrelated.

Mild neurocognitive disorder is similar to major neurocognitive disorder, but is characterized by a less extreme cognitive decline in one or more areas, such as attention, memory, language, social cognition, etc. Generally, people with mild neurocognitive disorder can still live independently, but still struggle in some cognitive areas.

In the DSM-5, both major and mild neurocognitive disorders are further specified as to whether it is due to the following causes:

- Alzheimer's disease
- frontotemporal lobar degeneration
- Lewy body disease
- vascular disease
- traumatic brain injury
- substance/medication use
- HIV infection
- prion disease
- Parkinson's disease
- Huntington's disease
- another medical condition
- multiple etiologies
- unspecified

Try It

Glossary

dementia: an umbrella category for neurocognitive disorders, characterized by progressive and gradual cognitive deficits due to severe cerebral atrophy

major neurocognitive disorder: deficits in cognitive ability that are acquired (as opposed to developmental), typically represent decline, and may have an underlying brain pathology

mild neurocognitive disorder: neurological disorders involving cognitive impairments beyond those expected based on an individual's age and education, but which are not significant enough to interfere with instrumental activities of daily living

neurocognitive disorders (NCDs): a category of mental health disorders that primarily affect cognitive abilities including learning, memory, perception, and problem-solving

1. Sousa S, Teixeira L and Paúl C (2020) Assessment of Major Neurocognitive Disorders in Primary Health Care: Predictors of Individual Risk Factors. Front. Psychol. 11:1413. doi: 10.3389/fpsyg.2020.01413 ↵

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14.4: Delirium

Learning Objectives

- Explain delirium and its etiology

Delirium, also known as **acute confusional state**, is an organically caused decline from a previous baseline mental functioning that develops over a short period of time, typically hours to days. Delirium is a syndrome encompassing disturbances in attention, consciousness, and cognition. Delirium may also involve other neurological deficits, such as psychomotor disturbances (e.g., hyperactive, hypoactive, or mixed), impaired sleep-wake cycle, emotional disturbances, and perceptual disturbances (e.g., hallucinations and delusions), although these features are not required for diagnosis.



Figure **14.4.1**: Delirium is an acute disturbance of mental abilities

Delirium is caused by an acute organic process, which is a physically identifiable structural, functional, or chemical problem in the brain that may arise from a disease process *outside* the brain that nonetheless affects the brain. Delirium may result from an underlying disease process (e.g., infection or hypoxia), a side effect of a medication, withdrawal from drugs, over-consumption of alcohol, usage of hallucinogenic deliriants, or from any number of factors affecting one's overall health (e.g., malnutrition, pain, etc.). In contrast, fluctuations in mental status/function due to changes in primarily psychiatric processes or diseases (e.g., schizophrenia or bipolar disorder) do not, by definition, meet the criteria for delirium.

Delirium may be difficult to diagnose without the proper establishment of a person's usual mental function. Without careful assessment and history, delirium can easily be confused with a number of psychiatric disorders or chronic organic brain syndromes because of many overlapping signs and symptoms in common with dementia, depression, psychosis, etc. Delirium may manifest from a baseline of existing mental illness, baseline intellectual disability, or dementia, without being due to any of these problems. Delirium is distinguished from dementia (chronic organic brain syndrome), which describes an acquired (non-congenital) and usually irreversible cognitive and psychosocial decline in function.

? The difference between delirium and similar psychiatric illness

Delirium is a disorder that makes situational awareness and processing new information very difficult for those diagnosed. Delirium usually has a high rate of onset ranging from minutes to hours and sometimes days, but it does not last for very long, only a few hours to weeks. Delirium can also be accompanied by a shift in attention, mood swings, violent or unordinary behaviors, and hallucinations. Delirium may be caused by a preexisting medical condition. Delirium is noted by sudden changes in cognition. Alzheimer's disease, depression, and some psychotic disorders tend to be more of a gradual cognition decline.

Table 1. Characteristics of delirium, Alzheimer's disease, depression, and psychotic disorders

Attributes	Delirium	Alzheimer's disease	Depression	Psychotic Disorders
Onset	Sudden/acute/subacute	Gradual	Gradual	Acute or gradual
Progression	Shifts in severity, likely to resolve in days to weeks.	Worsens over a period of time	Acute or chronic with acute exacerbation	Chronic with acute exacerbation
Hallucinations	May be present, mostly visual	Mostly absent (exceptions: Lewi body dementia, etc.)	May be present if associated with psychotic features	Present
Delusions	Fleeting	Mostly not present	May be present	Present
Psychomotor activity	Increased or decreased, may shift from increased to decreased states.	May or may not change	Change	Change
Attention	Poor attention span and impaired short term memory.	Progressive worsening short-term memory. Attention span is likely to be affected in severe cases	May be altered	May be altered
Consciousness	Altered, rapidly shifts	Mostly intact until severe stages	Normal	Normal
Attention	Altered, rapidly shifts	Mostly intact until severe stages	May be altered	May be altered
Orientation	Altered, rapidly shifts	Mostly intact until severe stages	Not altered	Not altered
Speech	Not coherent	Errors	Slow	Normal or pressured
Thought	Disorganized	Impoverished	Normal	Disorganized
Perceptions	Altered, rapidly shifts	Mostly intact until severe stages	Normal	May be altered
EEG	Moderate to severe background slowing	Normal or mild diffuse slowing	Normal	Normal
Reversibility	Mostly	Very rarely	Yes	Rarely

Delirium exists as a stage of consciousness somewhere in the spectrum between normal awake/alert state and coma. While requiring an acute disturbance in consciousness/attention and cognition, delirium is a syndrome encompassing an array of neuropsychiatric symptoms. Delirium arises through the interaction of a number of predisposing and precipitating factors.

The most important predisposing factors are

- older age (> 65 years old)
- male sex
- cognitive impairment / dementia
- physical comorbidity (biventricular failure, cancer, and cerebrovascular disease)
- psychiatric comorbidity (e.g., depression)
- sensory impairment (vision, hearing)
- functional dependence (e.g., requiring assistance for self-care or mobility)
- dehydration/malnutrition
- drugs and drug-dependence
- alcohol dependence

Individuals with multiple and/or significant predisposing factors are highly at risk for suffering an episode of delirium with a single and/or mild precipitating factor. Conversely, delirium may only result in healthy individuals if they suffer serious or multiple precipitating factors. The factors affecting an individual may change over time, thus an individual's risk of delirium is dynamic.

Some of the most common precipitating factors of delirium are listed below:

- prolonged sleep deprivation
- environmental or physical/psychological stress
  - inadequately controlled pain
  - admission to an intensive care unit
  - immobilization or use of physical restraints
  - urinary retention or use of a bladder catheter
  - emotional stress
  - severe constipation and/or fecal impaction
- medications
  - sedatives (benzodiazepines, opioids), anticholinergics, dopaminergics, corticosteroids, polypharmacy
  - general anesthetic
  - substance intoxication or withdrawal
- primary neurologic diseases
  - severe drop in blood pressure, relative to the patient's normal blood pressure (orthostatic hypotension) resulting in inadequate blood flow to the brain (cerebral hypoperfusion)
  - stroke/Transient ischemic attack (TIA)
  - intracranial bleeding
  - brain inflammation from meningitis or encephalitis

- Concurrent illness
  - Infections – especially respiratory (e.g. pneumonia) and urinary tract infections
  - Latrogenic complications
  - Hypoxia, hypercapnea, anemia
  - Poor nutritional status, dehydration, electrolyte imbalances, hypoglycemia
  - Cardiac shock, heart attacks, heart failure
  - Metabolic derangements (e.g. SIADH, Addison's disease, hyperthyroidism)
  - Chronic or terminal illness (e.g. cancer)
  - Post-traumatic event (e.g. fall, fracture)
- Surgery
  - Cardiac, orthopedic, prolonged cardiopulmonary bypass, thoracic surgeries

The range of ***clinical features*** includes poor attention/vigilance (100%), memory impairment (64–100%), clouding of consciousness (45–100%), disorientation (43–100%), acute onset (93%), disorganized thinking/thought disorder (59–95%), diffuse cognitive impairment (77%), language disorder (41–93%), sleep disturbance (25–96%), mood lability (43–63%), psychomotor changes (e.g. hyperactive, hypoactive, mixed) (38–55%), delusions (18–68%), and perceptual change/hallucinations (17–55%).

These various clinical features of delirium are further described below and can be assessed by several specialized tests, such as The Delirium Rating Scale-Revised-98 (DRS-R-98):



Figure **14.4.2**: Sleep disturbances and deprivation are risk factors for delirium.

- *Inattention*: As a required symptom to diagnose delirium, inattention is characterized by distractibility and an inability to shift and/or sustain attention.
- *Memory impairment* : Memory impairment is linked to inattention, especially reduced formation of new *long-term* memory where higher degrees of attention is more necessary than for short-term memory. Since older memories are retained without the need for concentration, previously formed long-term memories (i.e., those formed before the onset of delirium) are usually preserved in all but the most severe cases of delirium.
- *Disorientation*: As another symptom of confusion, and usually a more severe one, disorientation describes the loss of awareness of the surroundings, environment, and context in which the person exists. An individual may be disoriented to time, place, or self.
- *Disorganized thinking* : Disorganized thinking is usually noticed with speech that makes limited sense with apparent irrelevancies, and can involve poverty of speech, loose associations, perseveration, tangentiality, and other signs of a formal thought disorder.
- *Language disturbances*: Anomic aphasia, paraphasia, impaired comprehension, agraphia, and word-finding difficulties all involve impairment of linguistic information processing.
- *Sleep changes* : Sleep disturbances in delirium reflect disturbed circadian rhythm regulation, typically involving fragmented sleep or even sleep-wake cycle reversal (i.e., active at night and sleeping during the day) and often preceding the onset of a delirium episode
- *Psychotic symptoms*: Symptoms of psychosis include suspiciousness, overvalued ideation, and frank delusions. Delusions are typically poorly formed and less stereotyped than in schizophrenia or Alzheimer's disease. The delusions usually relate to persecutory themes of impending danger or threat in the immediate environment (e.g., being poisoned by nurses).
- *Mood lability* : Distortions to perceived or communicated emotional states as well as fluctuating emotional states can manifest in a delirious person (e.g., rapid changes between terror, sadness, and joking).
- *Motor activity changes*: Delirium has been commonly classified into psychomotor subtypes of hypoactive, hyperactive, and mixed, though studies are inconsistent as to the prevalence of these subtypes. Hypoactive cases are prone to non-detection or misdiagnosis as depression. A range of studies suggest that motor subtypes differ regarding underlying pathophysiology, treatment needs, and prognosis for function and mortality though inconsistent subtype definitions and poorer detection of hypoactive subtypes impact the interpretation of these findings.

Treatment of delirium requires identifying and managing the underlying causes, managing delirium symptoms, and reducing the risk of complications. In some cases, temporary or symptomatic treatments are used to comfort the person or to facilitate other care (e.g., preventing people from pulling out a breathing tube). Antipsychotics are not supported for the treatment or prevention of delirium among those who are in hospital. When delirium is caused by alcohol or sedative-hypnotic withdrawal, benzodiazepines are typically used. Evidence has found that the risk of delirium in hospitalized people can be reduced by systematic good general care. Delirium affects 14–24% of all hospitalized individuals. The overall prevalence for the general population is 1%-2% but this prevalence increases with age, reaching 14% of adults over age 85. Among older adults, delirium occurs in 15–53% of those post-surgery, 70–87% of those in the ICU, up to 60% of those in nursing homes or post-acute care settings. Among individuals requiring critical care, delirium is a risk for death within the next year.

Prevention

There is substantial evidence that delirium results in long-term poor outcomes in older persons admitted to hospital. Recent long-term studies showed that many patients still meet the criteria for delirium for a prolonged period after hospital discharge, with up to 21% of patients showing persistent delirium at six months post-discharge.

Using a tailored multifaceted approach clinicians can decrease rates of delirium by 27% among the elderly. At least 30–40% of all cases of delirium could be prevented, and high rates of delirium reflect negatively on the quality of care. Episodes of delirium can be prevented by identifying hospitalized people at risk of the condition: those over age 65, those with a known cognitive impairment, those with hip fracture, those with severe illness.

Delirium may be prevented and treated by using non-pharmacologic approaches focused on risk factors, such as constipation, dehydration, low oxygen levels, immobility, visual or hearing impairment, sleep deprivation, functional decline, and removing or minimizing problematic medications. Ensuring a therapeutic environment (e.g., individualized care;

clear communication; adequate reorientation and lighting during daytime; promoting uninterrupted sleep hygiene with minimal noise and light at night; minimizing bed relocation; having familiar objects like family pictures; providing earplugs; and providing adequate nutrition, pain control, and assistance toward early mobilization) can also yield benefit toward preventing delirium. Research into pharmacologic prevention and treatment is weak and insufficient to make proper recommendations.

Watch It

This video provides an overview of the causes, symptoms, diagnosis, and treatment of delirium.



You can view the transcript for "Delirium – causes, symptoms, diagnosis, treatment & pathology" here (opens in new window).

Glossary

delirium: an organically caused decline from a previous baseline mental functioning that develops over a short period of time, typically hours to days

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14.5: Neurocognitive Disorder Due to Alzheimer's Disease

Learning Objectives

- Describe the characteristics of Alzheimer's disease and its causes

Alzheimer's Disease

Alzheimer's disease (AD), also referred to simply as Alzheimer's, is the most common cause of a neurocognitive disorder, accounting for 60–70% of its cases. Alzheimer's is a progressive disease causing problems with memory, thinking, and behavior. Symptoms of Alzheimer's (AD) usually develop slowly and get worse over time and eventually become severe enough to interfere with daily life functioning and tasks. [1]

The most common early symptom is difficulty in remembering recent events. As the disease advances, symptoms can include problems with language, disorientation (including easily getting lost), mood swings, loss of motivation, not managing self-care, and behavioral issues. In the early stages of Alzheimer's disease (AD), memory loss is mild but in people with late-stage Alzheimer's, individuals will lose the ability to carry on conversations and respond to their environment.

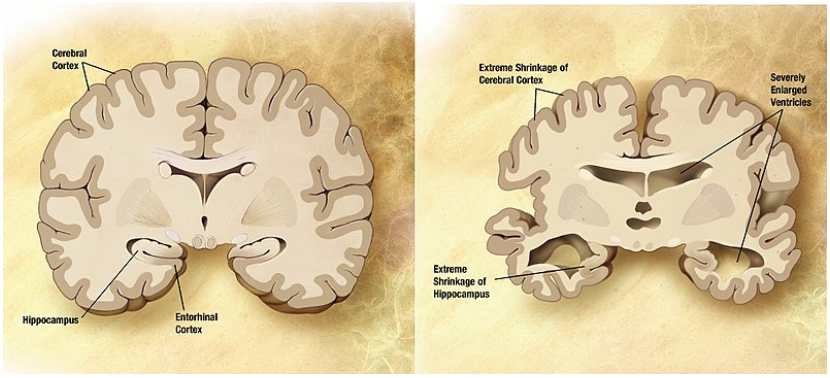


Figure **14.5.1**: Alzheimer's disease is not simply part of the aging process. It is a disease with physiological symptoms and decay in the brain.

Alzheimer's is the sixth-leading cause of death in the United States. On average, a person with Alzheimer's will live for four to eight years after diagnosis, but can live as long as 20 years, depending on other health factors. It is important to note that increasing age is the greatest known risk factor, but Alzheimer's is not a normal part of aging. The majority of people with Alzheimer's are 65 and older, though approximately 200,000 Americans under the age of 65 have younger-onset Alzheimer's disease (also known as early onset Alzheimer's). (What is Alzheimer's? Alzheimer's Association. Retrieved from https://www.alz.org/alzheimers-dementia/what-is-alzheimers.)

Link to Learning

Samuel Cohen researches Alzheimer's disease and other neurodegenerative disorders. Listen to Cohen's TED Talk on Alzheimer's disease to learn more.

Etiology

The cause of Alzheimer's disease is poorly understood. About 70% of the risk is believed to be inherited from a person's parents with many genes usually involved. Other risk factors include a history of head injuries, depression, and hypertension. The disease process is associated with plaques and neurofibrillary tangles in the brain.

The genetic heritability of Alzheimer's disease (and memory components thereof), based on reviews of twin and family studies, ranges from 49% to 79%. Around 0.1% of the cases are familial forms of autosomal (not sex-linked) dominant inheritance, which have an onset before age 65. This form of the disease is known as early onset familial Alzheimer's disease. Most autosomal dominant familial AD can be attributed to mutations in one of three genes: those encoding amyloid precursor protein (APP) and presenilins 1 and 2.

A probable diagnosis is based on the history of the illness and cognitive testing with medical imaging and blood tests to rule out other possible causes. Initial symptoms are often mistaken for normal aging, but an examination of brain tissue, specifically of structures called plaques and tangles, is needed for a definite diagnosis. Though qualified physicians can be up to 90% certain of a correct diagnosis of Alzheimer's, currently, the only way to make a 100% definitive diagnosis is by performing an autopsy of the person and examining the brain tissue. In 2015, there were approximately 29.8 million people worldwide with AD. In developed countries, AD is one of the most financially costly diseases.

Epidemiology

Two main measures are used in epidemiological studies: incidence and prevalence. Incidence is the number of new cases per unit of person at risk (usually the number of new cases divided by the persons at risk at a given time). For example, a study could look at the number of new cases of neurocognitive disorder over a five-year period, then divide that by the total time each individual was observed to find the incidence, or person-year rate. Prevalence is the total number of cases of the disease in the population at any given time.

Regarding incidence, cohort longitudinal studies (studies where a disease-free population is followed over the years) provide rates between 10 and 15 per thousand person-years for all dementias and five to eight per thousand for Alzheimer's Disease, which means that half of the new dementia cases each year are AD. Advancing age is a primary risk factor for the disease and incidence rates are not equal for all ages: every five years after the age of 65, the risk of acquiring the disease approximately doubles, increasing from three to as much as 69 per thousand person-years. There are also sex differences in the incidence rates, women having a higher risk of developing AD particularly in the population older than 85. In the United States, the risk of dying from Alzheimer's disease is 26% higher among the non-Hispanic White population than among the non-Hispanic Black population, whereas the Hispanic population has a 30% lower risk than the non-Hispanic White population.

Learning Objectives

Alzheimer's disease (AD): an irreversible, progressive brain disorder that slowly destroys memory and thinking skills and eventually the ability to carry out the simplest tasks

delirium: an abrupt change in the brain that causes mental confusion and emotional disruption, making it difficult to think, remember, sleep, pay attention, and more

dementia: an umbrella category for neurocognitive disorders, characterized by progressive and gradual cognitive deficits due to severe cerebral atrophy

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1. What is Alzheimer's? *Alzheimer's Association*. Retrieved from https://www.alz.org/alzheimers-dementia/what-is-alzheimers↵

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14.6: Neurocognitive Disorder with Lewy Bodies or Due to Parkinson's Disease

Learning Objectives

- Describe the characteristics of neurocognitive disorder with Lewy bodies
- Describe the characteristics of neurocognitive disorder due to Parkinson's disease

Neurocognitive Disorder with Lewy Bodies

Lewy bodies are abnormal aggregations of protein that develop inside nerve cells, contributing to Parkinson's disease (PD), the Lewy body dementias (Parkinson's disease dementia and dementia with Lewy bodies), and some other disorders. Lewy bodies are also seen in cases of multiple system atrophy, particularly the parkinsonian variant (MSA-P).

Lewy bodies can be identified under the microscope during histology tests on brain tissue. Lewy bodies appear as spherical masses that displace other cell components. Lewy bodies may be found in the brainstem (within the substantia nigra) or within the cortex. A classical Lewy body is an eosinophilic cytoplasmic inclusion consisting of a dense core surrounded by a halo of 10-nm-wide radiating fibrils, the primary structural component of which is alpha-synuclein. Cortical Lewy bodies are also composed of alpha-synuclein fibrils, but are less defined and lack halos. In histopathology, cortical Lewy bodies are a distinguishing feature for dementia with Lewy bodies (DLB), but may occasionally be seen in ballooned neurons characteristic of Pick's disease and corticobasal degeneration, as well as in patients with other tauopathies.

Dementia with Lewy bodies (DLB) is the second most common cause of dementia following Alzheimer's disease. About 0.4% of those over the age 65 are affected with dementia with Lewy bodies (DLB), and between one and four per 1,000 people develop the condition each year. Symptoms usually appear between the ages of 50 and 80, (median 76) and it is not uncommon for it to be diagnosed before the age of 65. An estimated 10–15% of diagnosed dementias are Lewy body type, but estimates range as high as 23% for those in clinical studies.

Dementia with Lewy bodies usually affects patients aged older than 50 years. The hallmark of DLB is the presence of Lewy bodies, which are intracytoplasmic inclusion bodies that contain synuclein proteins located in the cortical and subcortical neurons. However, these findings are not specific to DLB and may similarly be present in other neurodegenerative diseases. Although the majority of DLB cases are due to sporadic development, DLB may be familial, suggesting a genetic predisposition. People with the disease have a life expectancy of about eight years after diagnosis.

Clinically, DLB is characterized by the development of a triad of features, namely cognitive, neurological, and psychiatric symptoms. The diagnosis of DLB is difficult, given the absence of distinguishing features on imaging and neuropathology. The diagnosis of the DLB is usually made clinically based on history-taking and findings on physical examination in the absence of metabolic, vascular, and degenerative disorders. Unfortunately, the clinical features of DLB frequently overlap with other neurodegenerative diseases, namely Alzheimer's disease (AD) and Parkinson's disease, and some experts consider DLB to be part of the Alzheimer's/Parkinsonism spectrum. Clinicians should rely exclusively on the presence of core and suggestive clinical features that may distinguish DLB from other neurodegenerative disorders that cause dementia and symptoms of parkinsonism, such as Alzheimer's disease and Parkinson's disease with dementia (PDD).

Distinguishing features of DLB include the presence of dementia followed by early Parkinsonism symptoms within less than one year of symptoms onset, vivid and detailed visual hallucinations, depressive symptoms, REM sleep disorders, neuroleptic sensitivity, early onset postural instability and falls, and prominent visuospatial and verbal learning impairment. There is currently no cure for DLB, but pharmacologic and non-pharmacologic interventions can help manage DLB and improve the quality of life.

? What are Lewy Bodies?

Lewy bodies are abnormal aggregates of protein that develop inside nerve cells. Lewy bodies are identified under the microscope when histology is performed on brain tissue.

Lewy bodies appear as spherical masses that displace other cell components. There are two morphological types: classical (brain stem) Lewy bodies and cortical Lewy bodies. A classical Lewy body is an eosinophilic cytoplasmic inclusion that consists of a dense core surrounded by a halo of 10-nm wide radiating fibrils, the primary structural component of which is

alpha-synucleinL. In contrast, a cortical Lewy body is less well defined and lacks the halo. Nonetheless, it is still made up of alpha-synuclein fibrils.

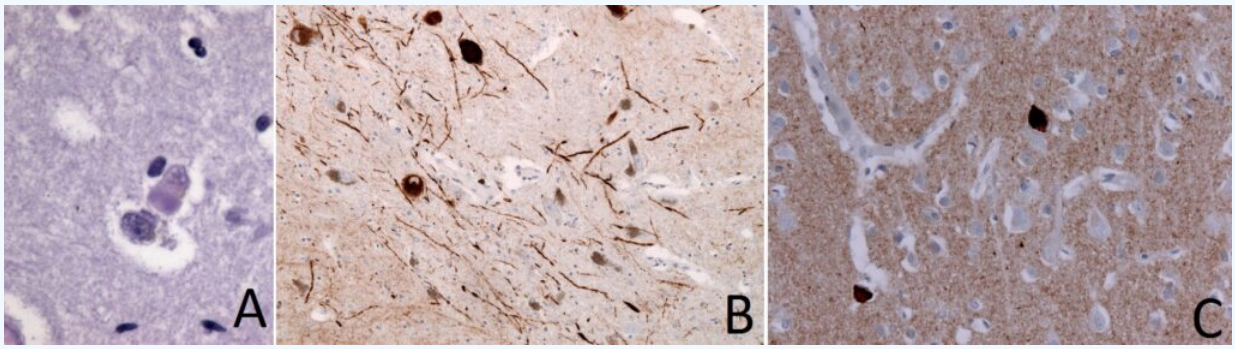


Figure 14.6.1: Figure: A. Lewy bodies (synuclein-positive, eosinophilic, neuronal inclusions) in brain tissue of patient with dementia with Lewy bodies (DLB); B. Immunohistochemical staining of Lewy Neurites; C. Immunhistochemical staining of frontal cortex in DLB demonstrates alpha-Synuclein positive inclusions

Classically, Lewy bodies in DLB are initially present in the amygdala. As the disease advances, these Lewy body cells spread to the limbic cortex and then to the neocortex. These findings may explain the predominance of dementia in patients with early DLB and the consequent development of parkinsonism symptoms. The clinical features of DLB are directly associated with the severity of Lewy-related pathology. In turn, the severity is measured by the pattern of regional involvement of Lewy bodies rather than the number of Lewy bodies.

DSM-V Diagnostic Criteria for Major or Mild Neurocognitive Disorder with Lewy Bodies

- The criteria are met for major or mild neurocognitive disorder AND
- The disorder has an insidious onset and gradual progression AND
- The disorder meets a combination of core diagnostic features and suggestive diagnostic features for either probable or possible neurocognitive disorder with Lewy bodies.

For *probable* major or mild neurocognitive disorder with Lewy bodies, the individual has two core features or one suggestive feature with one or more core features.

For *possible* major or mild neurocognitive disorder with Lewy bodies, the individual has only one core feature or one or more suggestive features.

Core diagnostic features of neurocognitive disorder with Lewy bodies include the following:

1. fluctuating cognition with pronounced variations in attention and alertness
2. recurrent visual hallucinations that are well-formed and detailed
3. spontaneous features of parkinsonism, with onset subsequent to the development of cognitive decline
4. suggestive diagnostic features:
    1. meets criteria for rapid eye movement sleep behavior disorder
    2. severe neuroleptic sensitivity
5. disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder

? Dementia with Lewy Bodies and Robin Williams

On August 11, 2014, at his home in Paradise Cay, California, renowned actor Robin Williams committed suicide. An examination of his brain tissue suggested Williams suffered from “diffuse Lewy body dementia.” Describing the disease as “the terrorist inside my husband’s brain,” his widow Susan Schneider Williams said that, “However you look at it—the presence of Lewy bodies took his life,” referring to his previous diagnosis of Parkinson’s. She revealed that in the year before his death Williams had experienced a sudden and prolonged spike in fear and anxiety, depression, and insomnia, which worsened in severity to include memory loss, paranoia, and delusions.



Figure 14.6.2: Robin Williams had been suffering from dementia with Lewy bodies prior to his 2014 suicide.

The Lewy Body Dementia Association (LBDA) clarified the distinction between the term used in the autopsy report, "diffuse Lewy body dementia"—which is more commonly called *diffuse Lewy body disease* and refers to the underlying disease process—and the umbrella term *Lewy body dementia*—which encompasses both Parkinson's disease dementia (PDD) and dementia with Lewy bodies (DLB). According to LBDA spokesperson Dennis Dickson, "The report confirms he experienced depression, anxiety, and paranoia, which may occur in either Parkinson's disease or dementia with Lewy bodies. . . . In early PDD, Lewy bodies are generally limited in distribution, but in DLB, the Lewy bodies are spread widely throughout the brain, as was the case with Robin Williams." Ian G. McKeith, professor, and researcher of Lewy body dementias, commented that Williams' symptoms and autopsy findings were explained by DLB.



You can view the transcript for "Lewy body dementia – causes, symptoms, diagnosis, treatment, pathology" here (opens in new window).

? Neurocognitive Disorder Due to Parkinson's Disease

Defining Parkinson's Disease

Parkinson's disease is a degenerative disorder of the central nervous system mainly affecting the motor system. Early in the course of the disease, the most obvious symptoms are movement related, including shaking, rigidity, slowness of movement, and difficulty with walking and gait. Later, thinking and behavioral problems may arise, with dementia commonly occurring in the advanced stages of the disease, whereas depression is the most common psychiatric symptom. Other symptoms include sensory, sleep, and emotional problems. Parkinson's disease is more common in older people, with most cases occurring after the age of 50; when it is seen in young adults, it is called early onset Parkinson's disease.

Diagnosis of typical cases is mainly based on symptoms, with tests such as neuroimaging used to rule out other diseases. Parkinson's disease typically occurs in people over the age of 60, of whom about 1% are affected. Males are more often affected than females at a ratio of around three males to every one female. When it is seen in people before the age of 50, it is called early onset Parkinson's disease. In 2015, Parkinson's disease affected 6.2 million people and resulted in about 117,400 deaths globally. On average following diagnosis, people live between seven and 15 years.

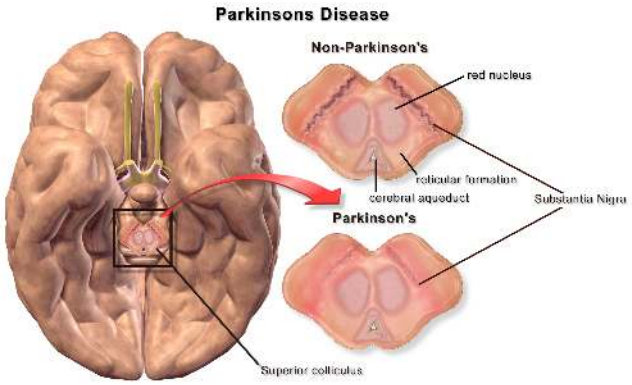


Figure 14.6.2: The main pathological characteristics of Parkinson's disease are cell death in the brain's basal ganglia and the presence of Lewy bodies in many of the remaining neurons. This loss of neurons is accompanied by the death of astrocytes (star-shaped glial cells) and a significant increase in the number of microglia (another type of glial cell) in the substantia nigra, as shown here.

A physician will diagnose Parkinson's disease from medical history and a neurological examination. There is no lab test that will clearly identify the disease, but brain scans are sometimes used to rule out disorders that could give rise to similar symptoms.

DSM-5 Diagnostic Criteria

The essential feature of neurocognitive disorder due to Parkinson's disease is the presence of a dementia that is judged to be the direct pathophysiological consequence of Parkinson's disease. Dementia has been reported to occur in approximately 20–60% of individuals with Parkinson's disease and is more likely to be present in older individuals or those with more severe or advanced disease.

The dementia associated with Parkinson's disease is characterized by cognitive and motoric slowing, executive dysfunction, and impairment in memory retrieval. Declining cognitive performance in individuals with Parkinson's disease is frequently exacerbated by depression. Findings on physical examination include the characteristic abnormal motor signs of resting tremor, evidence of slowness and poverty of movement (such as micrographia), or muscular rigidity and loss of associated movements.

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. The disturbance occurs in the setting of established Parkinson's disease.
C. There is insidious onset and gradual progression of impairment.
D. The neurocognitive disorder is not attributable to another medical condition and is not better explained by another mental disorder.

Major or mild neurocognitive disorder probably due to Parkinson's disease should be diagnosed if A and B are both met. Major or mild neurocognitive disorder possibly due to Parkinson's disease should be diagnosed if A or B is met:

- There is no evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease or another neurological, mental, or systemic disease or condition likely contributing to cognitive decline).
- The Parkinson's disease clearly preceded the onset of the neurocognitive disorder.

Differential Diagnosis

- Major or mild neurocognitive disorder (NCD) with Lewy bodies
  - This distinction is based substantially on the timing and sequence of motor and cognitive symptoms. For neurocognitive disorder (NCD) to be attributed to Parkinson's disease, the motor and other symptoms of Parkinson's disease must be present well before (by convention, at least one year prior) cognitive decline has reached the level of major neurocognitive disorder (NCD) whereas in major or mild NCD with Lewy bodies, cognitive symptoms begin shortly before, or concurrent with, motor symptoms. For mild NCD, the timing is harder to establish because the diagnosis itself is less clear and the two disorders exist on a continuum. Unless Parkinson's disease has been established for some time prior to the onset of cognitive

decline, or typical features of major or mild NCD with Lewy bodies are present, it is preferable to diagnose unspecified mild NCD.

- Major or mild neurocognitive disorder due to Alzheimer's disease
  - The motor features are the key to distinguished major or mild NCD due to Parkinson's disease from major or mild NCD due to Alzheimer's disease. However, the two disorders can co-occur.

Etiology

Parkinson's disease in most people is idiopathic (having no specific known cause). However, a small proportion of cases can be attributed to known genetic factors. Other factors have been associated with the risk of developing Parkinson's disease, but no causal relationships have been proven. A number of environmental factors have been associated with an increased risk of Parkinson's, including pesticide exposure, head injuries, and exposure to heavy metals.

Those with a family member affected are more likely to get the disease themselves. People exposed to certain pesticides or who have prior head injuries have an increased risk for Parkinson's disease. In contrast, there is a reduced risk for people who smoke tobacco and drink coffee or tea. The motor symptoms of Parkinson's disease result from the death of cells in the substantia nigra, a region of the midbrain, leading to a dopamine deficit in the brain. The cause of this cell death is poorly understood, but involves the build-up of proteins into Lewy bodies in neurons.

Treatment

There is no cure for Parkinson's disease, but medications, surgery, and multidisciplinary management can provide relief from the symptoms. There is some evidence that speech or mobility problems can improve with rehabilitation, although studies are scarce and of low quality. Regular physical exercise with or without physiotherapy can be beneficial to maintain and improve a patient's mobility, flexibility, strength, gait speed, and quality of life. However, when an exercise program is performed under the supervision of a physiotherapist, there are more improvements in motor symptoms, mental and emotional functions, daily living activities, and quality of life compared to a self-supervised exercise program at home.

Watch It

Watch this video for an overview of Parkinson's disease.



You can view the transcript for "Understanding Parkinson's disease" here (opens in new window).

Glossary

Lewy body dementia: an umbrella term that encompasses two similar dementias, both of which are characterized by abnormal deposits of the protein alpha-synuclein in the brain

Parkinson's disease: a long-term, degenerative disorder of the central nervous system that mainly affects the motor system

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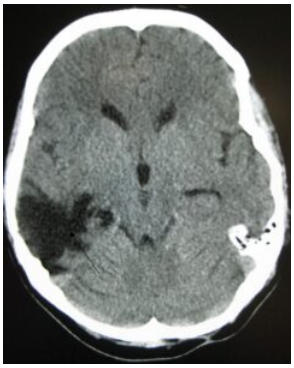
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14.7: Introduction to Other Neurocognitive Disorders

What you'll learn to do: explain other neurocognitive disorders caused by diseases, illnesses, or medications



In this section, we will learn about more major and mild neurocognitive disorders that may result from frontotemporal damage, vascular disease, Prion's disease, Huntington's disease, HIV, traumatic brain injury, substance abuse, or medications.

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14.8: Neurocognitive Disorder Due to Other Neurological Disorders

Learning Objectives

- Describe the characteristics of neurocognitive disorder due to frontotemporal neurocognitive disorder
- Describe the characteristics of vascular neurocognitive disorder
- Describe the characteristics of neurocognitive disorder due to prion disease
- Describe the characteristics of neurocognitive disorder due to Huntington's disease

Frontotemporal Neurocognitive Disorder

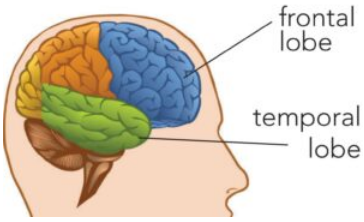


Figure **14.8.1**: FTLD is associated with the shrinking of the frontal and temporal lobes of the brain and shares some characteristics of Alzheimer's disease, including misfolded proteins and loss of brain cells.

Damage to the brain's frontal and temporal lobes causes forms of neurocognitive disorders (dementia) called **frontotemporal lobar degeneration (FTLD)** or frontotemporal disorders.

Frontotemporal disorders (FTLD) are the result of damage to neurons (nerve cells) in parts of the brain called the frontal and temporal lobes. As neurons die in the frontal and temporal regions, these lobes atrophy, or shrink. Gradually, the damage causes difficulties in thinking and behaviors normally controlled by these parts of the brain. Many possible symptoms can result, including unusual behaviors, emotional problems, trouble communicating, difficulty with work, or difficulty with walking.

Frontotemporal disorders (FTLD) are forms of dementia caused by a family of brain diseases known as frontotemporal lobar degeneration. Dementia is defined as a severe loss of thinking abilities that interferes with a person's ability to perform daily activities such as working, driving, and preparing meals. Other brain diseases that can cause dementia include Alzheimer's disease and multiple strokes. Scientists think that FTLD is the most common cause of dementia in people younger than age 60. Roughly 60% of people with FTLD are 45–64 years old. People with FTLD have changes in the brain due to another form of tau or the protein TDP43 and the range of symptoms can include unusual behaviors, emotional problems, communication difficulties, motor coordination (i.e., difficulty walking), and executive functions (i.e., difficulty with employment). In comparison, Alzheimer's is primarily associated with amyloid and tau proteins and is characterized predominately by memory problems.

People can live with frontotemporal disorders for up to 10 years, sometimes longer, but it is difficult to predict the time course for an individual patient. The frontotemporal disorders are progressive. In the early stages of FTLD, people may exhibit only one symptom. As FTLD progresses, patients will begin to experience other types of symptoms as more parts of the brain are affected.

Currently, there is no cure or treatments that slow or stop the progression of frontotemporal disorders. However, as awareness and understanding of FTLD expand, research has increased development to include advancements on better diagnosis, improved care, and, eventually, new treatments.

? What Do the Terms FTD and FLTD Mean?

One of the challenges shared by patients, families, clinicians, and researchers is confusion about how to classify and label frontotemporal disorders. A diagnosis by one doctor may be called something else by a second, and the same condition or syndrome may be referred to by another name by a pathologist who examines the brain after death.

For many years, scientists and physicians used the term frontotemporal dementia (FTD) to describe this group of illnesses. After further research, FTD is now understood to be just one of several possible variations and is more precisely called behavioral variant frontotemporal dementia, or bvFTD.

Frontotemporal disorders affect the frontal and temporal lobes of the brain. Frontotemporal disorder can begin in the frontal lobe, the temporal lobe, or both. Initially, frontotemporal disorders leave other brain regions untouched, including those that control short-term memory.

The frontal lobes, situated above the eyes and behind the forehead on the right and left sides of the brain, direct executive functioning. Direct executive functioning includes planning and sequencing (thinking through which steps come first, second, third, and so on), prioritizing (doing more important activities first and less important activities last), multitasking (shifting from one activity to another as needed), and monitoring and correcting errors. Symptoms are determined by which part of the lobe is affected first. The frontal lobe is responsible for decision-making, so the first symptom might be trouble managing finances.

? Case Study

Brian, an attorney, began having trouble organizing his cases. In time, his law firm assigned him to do paperwork only. Brian's wife thought he was depressed because his father had died two years earlier. At the age of 56, Brian was treated for depression, but his symptoms got worse. He became more disorganized and began making inappropriate sexual comments to his wife's female friends. Even more unsettling, he neither understood nor cared that his behavior disturbed his family and friends. As time went on, Brian had trouble paying bills and was less affectionate toward his wife and young son. Three years after Brian's symptoms began, his counselor recommended a neurological evaluation. Brian was diagnosed with behavioral variant frontotemporal dementia (bvFTD).

When the frontal lobes are functioning well, the frontal lobes also help manage emotional responses. The frontal lobes enable people to avoid inappropriate social behaviors, such as shouting loudly in a library or at a funeral. The frontal lobes also help people make decisions that make sense for a given situation. When the frontal lobes are damaged, people may focus on insignificant details and ignore important aspects of a situation or engage in purposeless activities. The frontal lobes are also involved in language, particularly linking words to form sentences, and in motor functions, such as moving the arms, legs, and mouth.

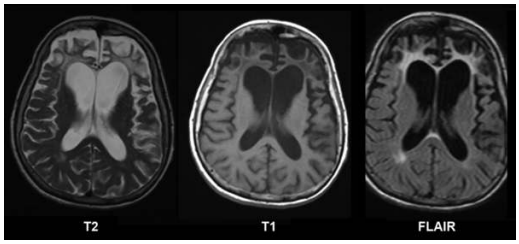


Figure 14.8.2: Brain MRI of a female, 65-year-old patient with Pick's disease. Cortex and white matter atrophy of the frontal lobes is clearly visible.

The temporal lobes, located below and to the side of each frontal lobe on the right and left sides of the brain, contain essential areas for memory but also play a major role in language and emotions. The temporal lobes help people understand words, speak, read, write, and connect words with their meanings. The temporal lobes also allow people to recognize objects and to relate appropriate emotions to objects and events. When the temporal lobes are dysfunctional, people may have difficulty recognizing emotions and responding appropriately to them. Issues in the part of the temporal lobe that connects emotions to objects may show an inability to recognize potentially dangerous objects. Examples would be a person reaching for a snake or plunging a hand into boiling water.

It was first described by Arnold Pick in 1892 and was originally called *Pick's disease*, a term now reserved for Pick disease, one specific type of frontotemporal dementia. A defining characteristic of the disease is a buildup of tau proteins in neurons, accumulating into silver-staining, spherical aggregations known as *Pick bodies*. Common early symptoms noticed are changes in personality and emotion and deterioration of language.

Vascular Disease

Another possible cause of neurocognitive disorder is a cardiovascular disease affecting the supply of blood to the brain. This condition, called **vascular neurocognitive disorder (VaD)** is highly prevalent, considered the second most common form of dementia after Alzheimer's disease (AD) in older adults. One study found that in the United States, the prevalence of vascular

dementia in all people over the age of 71 is 2.43%, and another found that the prevalence of the vascular neurocognitive disorder (VaD) doubles with every 5.1 years of age.[1]

Vascular neurocognitive disorder (VaD) is caused by problems in the supply of blood to the brain, typically caused by a series of minor strokes, leading to worsening cognitive decline that occurs step by step. The term refers to a syndrome consisting of a complex interaction of cerebrovascular disease and risk factors that lead to changes in the brain structures due to strokes and lesions, and resulting changes in cognition. The temporal relationship between stroke and cognitive effects is needed to make the diagnosis.



Figure **14.8.3**: Vascular neurocognitive disorder affects the supply of blood to the brain.

People with vascular neurocognitive disorder present with progressive cognitive impairment, acutely or subacutely as in mild cognitive impairment, frequently step-wise, after multiple cerebrovascular events (strokes). Some people may appear to improve between events and decline after further silent strokes. A rapidly deteriorating condition may lead to death from a stroke, heart disease, or infection.

Signs and symptoms are cognitive, motor, behavioral, and a significant proportion of patients also show affective symptoms. These signs and symptoms typically occur over a period of five to 10 years. Signs of vascular neurocognitive disorder are typically the same as other dementias, but primarily include cognitive decline and memory impairment of sufficient severity that these impairments interfere with a person's ability to complete activities of daily living.

In order to diagnose, clinicians will look for the presence of focal neurological signs and evidence of features consistent with a cerebrovascular disease on brain imaging (CT or MRI). The observable neurologic signs localizing to certain areas of the brain include hemiparesis, bradykinesia, hyperreflexia, extensor plantar reflexes, ataxia, pseudobulbar palsy, gait problems, and swallowing difficulties. People with vascular neurocognitive disorder may have deficits in terms of cognitive testing, though individuals tend to have better free recall and fewer recall intrusions when compared to patients with Alzheimer's disease. In the more severely affected patients or patients affected by infections in Wernicke's or Broca's areas, specific problems with speaking called dysarthrias and aphasia may be present.

In small vessel disease, such as vascular disease, the frontal lobes are often affected. Consequently, patients with vascular dementia tend to perform worse than their Alzheimer's disease counterparts in frontal lobe tasks, such as verbal fluency, and may present with frontal lobe problems: apathy, abulia (lack of will or initiative), problems with attention, orientation, and urinary incontinence. VaD patients may also present with a general slowing of processing ability, difficulty shifting sets, and impairment in abstract thinking. Apathy early in the disease is more suggestive of vascular dementia.

Many studies have been conducted to determine the average survival of patients with dementia. The studies were frequently small and limited, which caused contradictory results in the connection of mortality to the type of dementia and the patient's gender. A very large study conducted in the Netherlands in 2015 found that the one-year mortality was three to four times higher in patients after their first referral to a day clinic for dementia when compared to the general population. If the patient was hospitalized for dementia, the mortality was even higher than in patients hospitalized for cardiovascular disease. Vascular dementia was found to have either comparable or worse survival rates when compared to Alzheimer's Disease; another very large 2014 Swedish study found that the prognosis for VaD patients was worse for male and older patients. Unlike Alzheimer's Disease, which weakens the patient, causing them to succumb to bacterial infections like pneumonia, vascular dementia can be a direct cause of death due to the possibility of a fatal interruption in the brain's blood supply.

Currently, there are no medications that have been approved specifically for the prevention or treatment of vascular dementia. Smoking cessation and the Mediterranean diet have not been found to help patients with cognitive impairment; physical activity was consistently the most effective method of preventing cognitive decline.

Neurocognitive Disorder Due to Prion Disease

Neurocognitive disorder due to **prion disease** is a rare neurological disorder that researchers believe is caused by an infectious agent that results in abnormal protein accumulations in the brain, known as prions. Prions are misfolded proteins with the ability to transmit their misfolded shape onto normal variants of the same protein. Prions characterize several fatal and transmissible neurodegenerative diseases in humans and many other animals. Prion variants of the prion protein (PrP), whose specific function is uncertain, are hypothesized as the cause of transmissible spongiform encephalopathies (meaning that large holes develop in brain tissue), including scrapie in sheep, chronic wasting disease (CWD) in deer, bovine spongiform encephalopathy (BSE) in cattle (commonly known as *mad cow disease*) and **Creutzfeldt–Jakob disease** (CJD) in humans. All known prion diseases in mammals affect the structure of the brain or other neural tissue; all are progressive, have no known effective treatment, and are always fatal.



Figure **14.8.3**: Features of CJD can be accompanied by physical problems like balance and coordination dysfunction.

In humans, prions are believed to be the cause of Creutzfeldt–Jakob disease (CJD), CJD variant (vCJD), Gerstmann–Sträussler–Scheinker syndrome (GSS), fatal familial insomnia (FFI), and kuru (a rare, incurable, and fatal neurodegenerative disorder that was formerly common among the Fore people of Papua New Guinea). The most common type is Creutzfeldt-Jakob disease (CJD). Variant CJD is much rarer and is associated with bovine spongiform encephalopathy (BSE) or “mad cow disease,” and is transmitted through infected meat.

The first symptom of CJD is usually rapidly progressive dementia, leading to memory loss, personality changes, and hallucinations. Myoclonus (jerky movements) typically occurs in 90% of cases but may be absent at initial onset. Other frequently occurring features include anxiety, depression, paranoia, obsessive-compulsive symptoms, and psychosis. The occurring features are accompanied by physical problems such as speech impairment, balance, and coordination dysfunction (ataxia), changes in gait, and rigid posture. In most people with CJD, these symptoms are accompanied by involuntary movements.

Managing Creutzfeldt-Jakob disease

The duration of the disease varies greatly, but sporadic (non-inherited) CJD can be fatal within months or even weeks. Most victims die six months after initial symptoms appear, often of pneumonia due to impaired coughing reflexes. About 15% of people with CJD survive for two or more years, though there is currently no cure. Some of the symptoms like twitching can be managed, but otherwise, treatment is palliative care. Psychiatric symptoms like anxiety and depression can be treated with sedatives and antidepressants. Myoclonic jerks can be handled with clonazepam or sodium valproate. Opiates can help in pain. Seizures are very uncommon and can be treated with antiepileptic drugs.

Although CJD is the most common human prion disease, it is still believed to be rare, estimated to occur in about one out of every one million people every year. However, an autopsy study published in 1989 and others suggest that between three and 13% of people diagnosed with Alzheimer’s were actually misdiagnosed and instead had CJD. Presumably, those afflicted have become infected through prion-contaminated beef from cattle with subclinical atypical BSE (bovine spongiform encephalopathy), which has a very long incubation period. CJD usually affects people aged 45–75, most commonly appearing in people between the ages of 60–65. The exception to this is the more recently recognized variant CJD (vCJD), which occurs in younger people.

Neurocognitive Disorder Due to Huntington’s Disease

What Is Huntington’s Disease?

Huntington’s disease (HD) is an inherited disorder that causes brain cells, called neurons, to die in various areas of the brain, including those that help to control voluntary (intentional) movement. Symptoms of the disease, which gets progressively worse, include uncontrolled movements (called chorea); abnormal body postures; and changes in behavior, emotion, judgment, and

cognition. People with Huntington's disease (HD) also develop impaired coordination, slurred speech, and difficulty feeding and swallowing. Huntington's disease (HD) typically begins between ages 30 and 50. An earlier onset form called *juvenile HD* occurs under age 20. HD's symptoms differ somewhat from adult-onset HD and include rigidity, slowness, difficulty at school, rapid involuntary muscle jerks called myoclonus, and seizures. More than 30,000 Americans have HD.

Huntington's disease is caused by a mutation in the gene for a protein called huntingtin. The defect causes the cytosine, adenine, and guanine (CAG) building blocks of DNA to repeat many more times than is normal. Each child of a parent with HD has a 50-50 chance of inheriting the HD gene. A child who does not inherit the HD gene will not develop the disease and generally cannot pass it to subsequent generations. A person who inherits the HD gene will eventually develop the disease. HD is generally diagnosed based on a genetic test, medical history, brain imaging, and neurological and laboratory tests.

Link to Learning

This Osmosis video explains more about the etiology of Huntinton's disease.

The essential feature of neurocognitive disorder due to Huntington's disease (HD) is the presence of dementia that is judged to be the direct pathophysiological consequence of Huntington's disease.



Figure **14.8.4**: Woody Guthrie, a famous American folk singer (he wrote "This Land Is Your Land" among many others). He died of HD at the age of 55. His mother and two of his children also passed away from the disease. Increasingly unable to control his muscles, Guthrie was hospitalized during the last eleven years of his life at psychiatric facilities. Little was known about the disorder at the time, and Guthrie eventually lost his ability to speak and was known to experience emotional extremes during his final years.

Diagnostic Criteria

- The criteria are met for major or mild neurocognitive disorder.
- There is insidious onset and gradual progression.
- There is a clinically established Huntington's disease, or risk for HD based on family history or genetic testing.
- The neurocognitive disorder is not attributable to another medical condition and is not better explained by another mental disorder.

Differential Diagnosis

Early symptoms of Huntington's disease may include instability of mood, irritability, or compulsive behaviors that may suggest another mental disorder. However, genetic testing or the development of motor symptoms will distinguish the presence of Huntington's disease.

The early symptoms of Huntington's disease, particularly symptoms of executive dysfunction and impaired psychomotor speed, may resemble other neurocognitive disorders (NCDs), such as major or mild vascular NCD.

Prognosis

Huntington's disease causes disability that gets worse over time. Currently, no treatment is available to slow, stop, or reverse the course of HD. The length of the trinucleotide repeat accounts for 60% of the variation in the age symptoms appear and the rate they progress. A longer repeat results in an earlier age of onset and a faster progression of symptoms. Individuals with more than 60 repeats often develop the disease before age 20, while those with fewer than 40 repeats may not ever develop noticeable symptoms. The remaining variation is due to environmental factors and other genes that influence the mechanism of the disease.

Life expectancy in HD is generally around 20 years following the onset of visible symptoms. Most life-threatening complications result from muscle coordination and, to a lesser extent, behavioral changes induced by declining cognitive function. The largest risk is pneumonia, which causes death in one-third of those with HD.

Treatment

There is no treatment that can stop or reverse the course of HD. Tetrabenazine and deuterabenazine can treat chorea associated with HD. Antipsychotic drugs may ease chorea and help to control hallucinations, delusions, and violent outbursts. Drugs may be prescribed to treat depression and anxiety. Side effects of drugs used to treat the symptoms of HD may include fatigue, sedation, decreased concentration, restlessness, or hyperexcitability, and should be only used when symptoms create problems for the individual. Although there have been relatively few studies of exercises and therapies that help rehabilitate cognitive symptoms of HD, there is some evidence for the usefulness of physical therapy, occupational therapy, and speech therapy. An association between caffeine intake and earlier age of onset in Huntington's disease has been found, but, since this finding was based on retrospective questionnaire data rather than a blinded, randomized trial or case-control study, this association is a poor basis for guiding lifestyle decisions.

? Key Takeaways: Other Neurocognitive Disorders

Table 1. Neurocognitive Disorders

Disorder	DSM-5 Criteria	Epidemiology/Prevalence/Treatment
NCD due to FTLD	The criteria are met for a major or mild NCD and there are marked changes in either behavior (disinhibition, apathy, loss of sympathy, a decline in social cognition and executive abilities, etc.), or language decline. There must also be evidence of a causative frontotemporal neurocognitive disorder genetic mutation, from either family history or genetic testing or evidence of disproportionate frontal and/or temporal lobe involvement from neuroimaging.	Currently, there is no cure for FTD, but some treatments are available to manage the behavioral symptoms. Disinhibition and compulsive behaviors can be controlled by selective serotonin reuptake inhibitors.(SSRI) share certain symptoms, they cannot be treated with the same pharmacological agents because the cholinergic systems are not affected in FTD. Because FTD often occurs in younger people (i.e., in their 40s or 50s), it can severely affect families.
VaD	The establishment of the presence of a major or mild neurocognitive disorder caused by vascular disease. VaD can be caused by ischemic or hemorrhagic infarcts affecting multiple brain areas, including the anterior cerebral artery territory, the parietal lobes, or the cingulate gyrus. On rare occasion, infarcts in the hippocampus or thalamus are the cause of dementia. A history of stroke increases the risk of developing dementia by around 70%, and recent stroke increases the risk by around 120%.	Currently, there are no medications that have been approved specifically for prevention or treatment of VaD. The use of medications for treatment of Alzheimer's dementia, such as cholinesterase inhibitors and memantine, has shown small improvement of cognition in VaD. Multiple studies found a small benefit in VaD treatment with: memantine, a non-competitive N-methyl-D-aspartate (NMDA) receptor antagonist; cholinesterase inhibitorsgalantamine, donepezil, revastigmine; and ginkgo biloba extract.
Neurocognitive disorder due to Prion Disease	The criteria are met for major or mild NCD that can be attributed to prion disease. Prions cause neurodegenerative disease by aggregating extracellularly within the central nervous system to form plaques known as amyloids, which disrupt the normal tissue structure. This disruption is characterized by "holes" in the tissue with resultant spongy architecture due to the vacuole formation in the neurons.	The most common prion disease is Creutzfeldt-Jakob disease (CJD). CJD occurs worldwide at a rate of about 1 case per million population per year. There are no effective treatments for prion diseases.
NCD due to Huntington's Disease	The criteria are met for major or mild neurocognitive disorder that can be explained by Huntington's disease. HD is typically inherited, although up to 10% of cases are due to a new mutation. The earliest symptoms are often subtle problems with mood or mental abilities. A general lack of coordination and an unsteady gait often follow. As the disease advances, uncoordinated, involuntary body movements known as chorea become more apparent. Physical abilities	There is no cure for HD, but there are treatments available to reduce the severity of some of its symptoms, including physical therapy, occupational therapy, and speech therapy.

Disorder	DSM-5 Criteria	Epidemiology/Prevalence/Treatment
	gradually worsen until coordinated movement becomes difficult and the person is unable to talk.	

Glossary

frontotemporal neurocognitive disorder: term to describe several types of dementia involving the frontal and temporal lobes, including Pick's disease

vascular neurocognitive disorder: dementia caused by problems in the supply of blood to the brain, typically a series of minor strokes, leading to worsening cognitive decline that occurs step by step

prion's disease: a type of disease of structurally abnormal proteins; includes Creutzfeldt–Jakob disease

Huntington's disease: mostly an inherited neurodegenerative disease that results in the death of brain cells

1. Plassman BL, Langa KM, Fisher GG, Heeringa SG, Weir DR, Ofstedal MB, Burke JR, Hurd MD, Potter GG, Rodgers WL, Steffens DC, Willis RJ, Wallace RB (2007). "Prevalence of dementia in the United States: the aging, demographics, and memory study". Neuroepidemiology. 29 (1–2): 125–32. doi:10.1159/000109998. PMC 2705925. PMID 17975326. ↵

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14.9: Neurocognitive Disorder Due to Other Medical Conditions, Substances, or Medications

Learning Objectives

- Describe the characteristics of neurocognitive disorder due to HIV
- Describe the characteristics of neurocognitive disorder due to substance/medication
- Describe the characteristics of neurocognitive disorder due to TBI

As we have seen, neurocognitive disorders may be caused by several medical conditions, such as Alzheimer's disease, Parkinson's disease, and Huntington's disease because they affect or deteriorate brain functions. There are many other diseases and conditions that cause NDCs, including vascular dementia, frontotemporal degeneration, Lewy body disease, and prion disease. In addition, dementia/neurocognitive issues can arise due to infection (e.g., HIV), substance abuse (e.g., substance abuse and addiction), mental health (e.g., post-traumatic stress syndrome), exposure (e.g., toxins). and brain trauma (e.g., concussions and traumatic brain injuries).

HIV

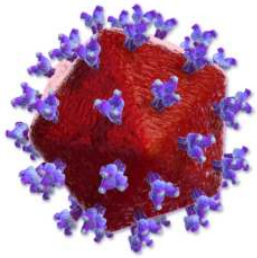


Figure **14.9.1**: The HIV virus, known for weakening the immune system, may also lead to cognitive impairment. Though once thought to have a prevalence of nearly 50% rates have gone down significantly since the advent of highly active antiretroviral therapy.

Patients with human immunodeficiency virus infection (HIV infection) have been noted to have various forms of neuropsychiatric illnesses. Notably, neuropsychiatric illnesses include symptoms of both cognitive disorders as well as mood and anxiety symptoms. The essential features of AIDS dementia complex include, but are not limited to, disabling cognitive impairment accompanied by motor dysfunction, speech problems, and behavioral change. Cognitive impairment is characterized by mental slowness, trouble with memory, and poor concentration. Motor symptoms include a loss of fine motor control leading to clumsiness, poor balance, and tremors. Behavioral changes may present as apathy, lethargy, diminished emotional responses, and spontaneity.

In the past, before the advent of antiretroviral treatment, several neuropsychiatric disorders remained untreated, resulting in a large mortality rate. Most of these disorders stemmed from untreated infectious diseases secondary to AIDS (acquired immunodeficiency syndrome) like toxoplasmosis and encephalitis. The decline in the number of neurocognitive disorders notably came hand in hand in 1996 with the advent of highly active antiretroviral therapy (HAART). Since this combination treatment for HIV, HIV-related neuropsychiatric disorders started to show a decline.

? Substance Abuse

Research over the last two decades has demonstrated that abuse of psychostimulants results in several cognitive deficits such as impulsivity (i.e., inability to inhibit disadvantageous rapid behavioral responses), risky and/or poor decision-making, impaired cognitive flexibility (i.e., impaired ability to alter behavioral responses based on changing environmental contingencies), deficits in learning and memory, and/or hyperattentiveness. Interestingly, individuals with pre-existing deficits in cognition and/or suffering from psychiatric disease states that are associated with impaired cognitive function (e.g., schizophrenia and depression) are more vulnerable to abusing illicit and licit stimulants. Importantly, recovering addicts with significant cognitive deficits are more

vulnerable to relapse. Cognitive deficits in recovering drug addicts irrespective of whether they were pre-existing or drug-induced need to be adequately treated to promote abstinence among drug addicts.[2]

Alcohol-related dementia (ARD) is a broad term currently preferred among medical professionals. Many experts use the terms alcohol (or alcoholic) dementia to describe a specific form of ARD, characterized by impaired executive function (planning, thinking, and judgment). Another form of ARD is known as wet brain (Wernick-Korsakoff syndrome), which is characterized by short-term memory loss and a thiamine (vitamin B1) deficiency. ARD patients often have symptoms of both types of ARD that include impaired ability to plan, apathy, and memory loss. ARD may occur with other forms of dementia (mixed dementia). The diagnosis of ARD is widely recognized but rarely applied, due to a lack of specific diagnostic criteria.

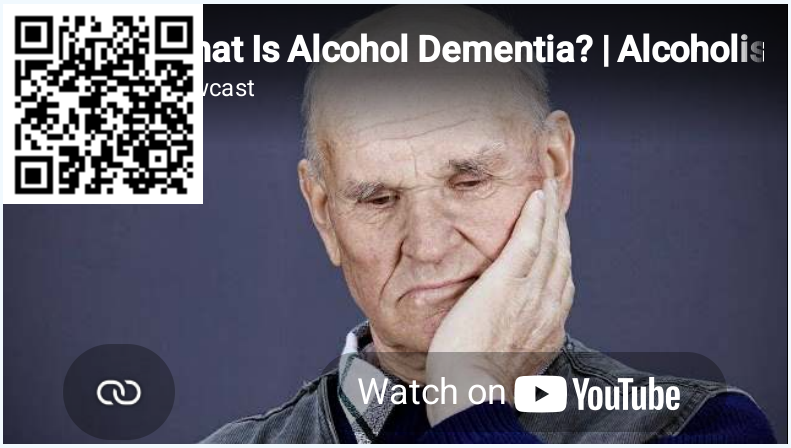
Alcohol-related dementia presents as a global deterioration in intellectual function not specific to memory but it may also occur with other forms of dementia therefore resulting in a wide range of symptoms. Certain individuals with alcohol-related dementia present with damage to the frontal lobes of their brain causing disinhibition, loss of planning and executive functions, and a disregard for the consequences of their behavior. Other types of alcohol-related dementia such as Korsakoff's syndrome cause the destruction of certain areas of the brain, where changes in memory, primarily a loss of short-term memory, are the main symptom. Most presentations of alcohol dementia are somewhere along the spectrum between global dementia and Korsakoff's psychosis and may include symptoms of both.

Individuals affected by alcohol-related dementia may develop memory problems, language impairment, and an inability to perform complex motor tasks such as getting dressed. People with heavy alcohol abuse may develop cerebellar ataxia, which consists of damages the nerves in arms and legs, i.e., peripheral neuropathy as well as the cerebellum. These patients frequently have problems with sensation in their extremities and may demonstrate unsteadiness on their feet.

Alcohol-related dementia can produce a variety of psychiatric problems including psychosis (disconnection from reality), depression, anxiety, and personality changes. Patients with alcoholic dementia often develop apathy, related to frontal lobe damage, which may mimic depression. People with alcoholism are more likely to become depressed than people without alcoholism, and it may be difficult to differentiate between depression and alcohol dementia. Epidemiological studies show an association between long-term alcohol intoxication and dementia. Alcohol can damage the brain directly as a neurotoxin, or it can damage it indirectly by causing malnutrition, primarily a loss of thiamine (vitamin B1). Approximately 10% of all dementia cases are related to alcohol consumption, making it the second leading cause of dementia.[3]

Watch It

In this video, Dr. Rinaldi explains alcohol-related dementia. He discusses the brain changes that happen over the long-term and the resulting characteristics of alcohol dementia.



You can view the transcript for "What Is Alcohol Dementia? | Alcoholism" here (opens in new window).

Traumatic Brain Injury (TBI)

Traumatic brain injury (TBI) occurs when an individual experiences a significant trauma or injury to the head. Neurocognitive disorder due to traumatic brain injury (TBI) is diagnosed when persistent cognitive impairment is observed immediately following the head injury, along with one or more of the following symptoms: loss of consciousness, posttraumatic amnesia, disorientation, and confusion or neurological impairment (APA, 2013).

Each year, traumatic brain injury (TBI) is responsible for more than 80,000 emergency department visits for those above age 65, approximately three-quarters of which result in hospitalization. The leading causes of TBI include fallsand motor vehicle accidents. [4] TBI is present in 85% of traumatically injured children, either alone or with other injuries. The greatest number of TBIs occur in people aged 15–24. The age groups most at risk for TBI are children ages five to nine and adults over age 80, and the highest rates of death and hospitalization due to TBI are in people over age 65. The incidence of fall-related TBI in first-world countries is increasing as the population ages; thus the median age of people with head injuries has increased.

Regardless of age, TBI rates are higher in males. Men suffer twice as many TBIs as women do and have a fourfold risk of fatal head injury, and males account for two-thirds of childhood and adolescent head trauma. However, when matched for severity of injury, women appear to fare more poorly than men.

DSM-5 Criteria for Neurocognitive Disorder Due to TBI

- The criteria are met for major or mild neurocognitive disorder.
- There is evidence of a traumatic brain injury—that is, an impact to the head or other mechanisms of rapid movement or displacement of the brain within the skull, with one or more of the following:
  - loss of consciousness.
  - post-traumatic amnesia.
  - disorientation and confusion.
  - neurological signs (e.g., neuroimaging demonstrating injury, new onset of seizures, a marked worsening of a preexisting seizure disorder, visual field cuts, anosmia, or hemiparesis).
- The neurocognitive disorder presents immediately after the occurrence of the TBI or immediately after recovery of consciousness and persists past the acute post-injury period.

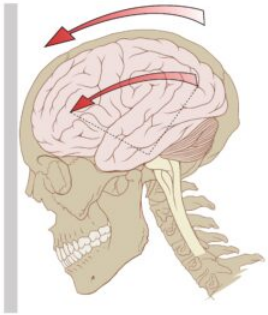


Figure **14.9.1**: A concussion may lead to neurocognitive disorder due to TBI.

The presentation of symptoms varies among individuals and depends largely on the location of the injury and the intensity of the trauma. Furthermore, the effects of TBI can be temporary or permeant. Symptoms generally range from headaches, disorientation, confusion, irritability, fatigue, and poor concentration as well as emotional and behavioral changes. More severe injuries can result in more significant neurological symptoms such as seizures, paralysis, and visual disturbances (APA, 2013).

The most common type of TBI is a concussion. A **concussion** occurs when there is a significant blow to the head, followed by changes in brain functioning. It often causes immediate disorientation or loss of consciousness, along with headaches, dizziness, nausea, and sensitivity to light (Alla, Sullivan, & McCrory, 2012). While symptoms of a concussion are usually temporary, there can be more permanent damage due to repeated concussions, particularly if they are within close time periods. The media has brought considerable attention to this with the recent discussions of **chronic traumatic encephalopathy (CTE)** which is a progressive, degenerative condition due to repeated head trauma. Chronic traumatic encephalopathy (CTE) is a distinctive tau-

protein associated neurodegenerative disease. There has been a rise of chronic traumatic encephalopathy (CTE) diagnosis in athletes, especially American football players, as well as in military veterans in combat settings. Although chronic traumatic encephalopathy (CTE) has been publicly recognized relatively recently, it was first described as “punch drunk” syndrome in a classic article by Martland et al. The report was focused on a number of boxers who had suffered repetitive head blows throughout their careers and were presented with both psychiatric symptoms as well as severe memory and neurocognitive deficits that were analogous to typical dementia patients. In addition to the neurological symptoms, psychological symptoms such as depression and poor impulse control have been observed in individuals with CTE. Individuals with CTE also appear to be at greater risk for development of dementia (McKee et al., 2013).

Prevention is another key strategy that needs to be implemented in various sports and military settings. Providing education for safe practice techniques, such as safe tackling and hitting, and providing ready access to full neuropsychiatric assessment by team physicians could have measurable benefits. The combination of advanced research techniques including neuroimaging, as well as increasing public awareness of CTE, offers promising vistas for research advancement.

Example of CTE

Read this article describing how concussions have affected NHL player Marc Savard.

Glossary

chronic traumatic encephalopathy (CTE): a progressive, degenerative condition due to repeated head trauma

concussion: occurs when there is a significant blow to the head, followed by changes in brain functioning

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14.10: Introduction to Perspectives and Treatments Related to Neurocognitive Disorders

What you'll learn to do: examine psychological approaches and treatments related to neurocognitive disorders



In this section, we will look at treatment methods and examples of case studies related to neurocognitive disorders.

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14.11: Treatments for Neurocognitive Disorders

Learning Objectives

- Describe psychological perspectives and treatments for neurocognitive disorders

In this course, we have learned to assess and analyze disorders from multiple psychological perspectives, such as the psychodynamic, biological, humanistic, behavioral, and cognitive perspectives. Neurocognitive disorders, by definition, mostly relate to the cognitive perspective, as the cognitive perspective views psychological disorders as originating from an interruption, whether short or long, in our basic cognitive functions, i.e., memory processing, perception, problem-solving, and language. All the neurocognitive disorders have biological causes, as explained in the previous readings, because they are attributed to biological changes caused by Alzheimer's, Parkinson's, Huntington's disease, HIV, traumatic brain injury (TBI), or other illnesses. The focus in understanding and treating neurocognitive disorders lies then in understanding the biological changes and what measures can be taken to slow cognitive decline. There is evidence that some measures are effective in preventing or slowing the progression of cognitive symptoms.

Recall that major neurocognitive disorder (MND) is a syndrome that progresses with significant deterioration of cognitive domains as compared to previous levels of cognitive performance in memory, speech, reasoning, intellectual function, and/or spatiotemporal perception, and may also be associated with changes in emotional behavior and difficulties at the functional level. The decline is initially noticed by the individual, the family, or the general practitioner (GP) who is usually responsible for the early diagnosis (American Psychiatric Association [APA], 2014).

MND may result from brain disorders, classified as primary (degenerative), or be a consequence of other conditions (secondary) (Emre, 2009). The most common types of MND are Alzheimer's disease, vascular dementia (VaD), Lewy body dementia, and frontotemporal dementia (FD). In secondary MND (e.g., alcoholic dementia and infectious diseases), the symptoms may be treated and/or prevented. Therefore, a correct diagnosis is crucial for proper disease management and treatment. This is supported by a detailed collection of the person's clinical history, neurological and neuropsychological examination, and the comprehensive use of laboratory and imaging tests. In primary MND, early diagnosis is equally crucial either to delay the progression of cognitive symptoms and to control/stabilize psychiatric manifestations (Ribeira et al., 2004).

Predictive Factors of MND

MND is likely to develop in a continuous process (Brooks and Loewenstein, 2010). Individual factors affect the likelihood of developing MND and these factors predicting the development of the disease should be known. If known, preventive interventions may prevent or protect people at risk of MND.

Previous studies have identified predictive factors of MND, which can be grouped into the following:

- sociodemographic factors (e.g., sex, age, and years spent in education and social isolation)
- health factors (e.g., hearing loss, cardiovascular diseases, hypertension, diabetes, handgrip strength, and nutritional status)
- bio-behavioral factors (e.g., smoke, alcohol, and physical activity) (Helzner et al., 2009; Nagai et al., 2010; Polidori et al., 2012; Baumgart et al., 2015; Santana et al., 2015; Schwarzinger et al., 2018)

Given that most of these factors are all potentially modifiable (e.g., diabetes, cholesterol, depression, or malnutrition; Chen et al., 2017), the individual can play an active role in the prevention or management of MND, which creates the opportunity to allow for more efficient intervention. Primary prevention in the primary health care context is important for the course of MND and should focus on the identification of situations that increase the likelihood of occurrence or worsening of symptoms. However, few studies identify predictive factors associated with the severe stage of MND (Eshkoor et al., 2016).

A study by Sousa, Laetitia Teixeira, and Constança Paúl identified four predictors of MND including age, years spent in education, physical activity, and hand strength. Physical activity, hand strength, and education play a protective role ("the more the better"). On the other hand and as expected, while age increases, the risk of MND also increases.[1]

There is extensive and persuasive evidence from mechanistic and well-designed prospective cohort studies that reducing the exposure to high blood pressure and hypertension in mid-life and to diabetes in mid-and late life, reducing tobacco use, and

increasing the education level of populations can effectively reduce the dementia risk for populations (Prince et al, 2014).

Treating Neurocognitive Disorders

There is no cure for neurocognitive disorders or the diseases that cause them. Pharmacological approaches combined with behavioral and environmental interventions are most successful in treating neurocognitive disorders.



Figure **14.11.1**: High blood pressure is a vascular risk factor for developing a neurocognitive disorder.

Antidepressants, antipsychotics, and other medications that treat memory loss and behavioral symptoms are also available and may help to treat the diseases. Ongoing psychotherapy and psychosocial support for patients and families are usually necessary for clear understanding and proper management of the disorder and to help maintain a better quality of life for caregivers and patients. In addition, speech therapy has been shown to help neurocognitive dementia patients with language impairment.

Studies suggest that diets with high Omega 3 content and low in saturated fats and sugars, along with regular exercise can increase the level of brain plasticity. Other studies have shown that mental exercise such as newly developed computerized brain training programs can also help build and maintain targeted specific areas of the brain. These studies have been very successful for those diagnosed with schizophrenia and can improve fluid intelligence and the ability to adapt and deal with new problems or challenges the first time encountered; in young people, it can still be effective in later life.

A person with amnesia may slowly be able to recall their memories or work with an occupational therapist to learn new information to replace what was lost or to use intact memories as a basis for taking in new information. If amnesia is caused by an underlying cause such as Alzheimer's disease or infections, the cause may be treated but the amnesia may not be.

Pharmacological Interventions

Targets for pharmacological treatment include

- cognitive impairment (e.g., memory loss, disorientation, and decrease in attention and problem-solving).
- behavioral symptoms (e.g., agitation and aggression).
- psychological symptoms (e.g., depression, anxiety, and psychosis).

There is a large body of evidence for the efficacy of cholinesterase inhibitors (ChEIs), such as donepezil, rivastigmine, and galantamine, in the treatment of mild to moderate Alzheimer's disease (Institute for Quality and Efficiency in Healthcare, 2014). The use of each of these medications is associated with modest and short-term comparable improvements in cognitive function, global clinical state, and activities of daily living. However, the evidence base for cholinesterase inhibitors (ChEIs) in low- and middle-income countries is limited. Moreover, the efficacy of this class of drugs in severe dementia is unclear, although behavioral symptom improvement was identified for galantamine (Institute for Quality and Efficiency in Healthcare, 2014). A fourth drug for the treatment of cognitive impairment, memantine, has a different mode of action and is well tolerated, but evidence for its efficacy is limited to people with moderate to severe dementia. Cholinesterase inhibitors (ChEIs) and memantine are less efficacious in vascular dementia than other forms. ChEIs and memantine's efficacy in the treatment of behavioral disturbances is not established; manufacturer-sponsored licensing trials and post hoc analyses indicate small improvements.



Figure **14.11.2**: Haloperidol is an atypical antipsychotic medication that can be administered through injection.

Use of haloperidol and atypical antipsychotic medications for the treatment of agitation and behavioral and psychological symptoms of dementia (BPSD) indicate small treatment effects, most evident for aggression, although these must be weighed against the associated mortality risk (Kales et al, 2012). Atypical antipsychotic drugs have been widely prescribed for psychosis in dementia, but a meta-analysis of their efficacy indicated that only aripiprazole and risperidone had a statistically and clinically significant effect on psychiatric symptoms (Tan et al, 2015). An important caveat to the use of these medications in dementia is the associated increased risk of death and cerebrovascular adverse events. The literature on antipsychotic treatment in older people with dementia reveals that although improvement in the behavioral disturbance was minimal after six to 12 weeks, there was a significant increase in absolute mortality risk of approximately 1% (Banerjee et al, 2009). The literature suggests that prescribing antipsychotics in dementia beyond six to 12 weeks is likely to be substantially harmful in continued antipsychotic treatment in dementia. Therefore, many recommend nonpharmacological treatments, such as psychological and training interventions, to reduce BPSD rather than antipsychotic management (Duedon et al, 2009). A meta-analysis of the efficacy of antidepressants in people with dementia was inconclusive (Leong, 2014). Antidepressants have been proposed for the treatment of BPSD with encouraging results (Henry et al, 2011).

Nonpharmacological Interventions

A well-conducted randomized control trial of cognitive stimulation (reality orientation, games, and discussions based on information processing rather than knowledge) conducted in the United Kingdom as a group intervention, and a small pilot trial from Brazil, suggest that cognitive benefits from this intervention are similar to those for ChEIs (Aguirre et al, 2013). More specific cognitive training produced no benefits. Cognitive rehabilitation, an individualized therapy designed to enhance residual cognitive skills and the ability to cope with deficits, showed promise in uncontrolled case series in hospital incident command systems. A meta-analysis of four trials of reminiscence therapy (the discussion of past activities, events, and experiences) provides evidence for short-term improvement in cognition, mood, and caregiver strain, but the quality of these trials was poor (Bahar-Fuchs, 2013; Woods et al, 2005; Woods et al, 2012).

In terms of nonpharmacologic therapies, **cognitive stimulation therapy** has been shown to be cost-effective for people with mild-to-moderate dementia when delivered biweekly over seven weeks, though was found to have modest effects when continued for longer when added to administration of acetylcholinesterase inhibitors (D'Amico et al, 2015). An exercise intervention was found to have the potential to be cost effective when considering behavioral and psychological symptoms, but did not appear cost effective when considering quality-adjusted life year gains. The START (Strategies for Relatives) study, a randomized controlled trial to determine the clinical effectiveness and cost-effectiveness of a manual-based coping strategy program in promoting the mental health of carers of people with dementia, found the intervention to be cost effective with respect to the caregiver and patient outcomes, and National Institute for Health and Care Excellence (NICE) thresholds (Livingston et al, 2014). In a health economic analysis of resource costs and costs of formal care on a psychosocial intervention for family caregivers of persons with dementia, those in the intervention group reported a higher quality of life while their spouse was living at home (Dahlrup et al, 2014).

Deep brain stimulation (DBS) is a neurosurgical procedure involving the placement of a medical device called a neurostimulator (sometimes referred to as a *brain pacemaker*), which sends electrical impulses, through implanted electrodes, to specific targets in the brain (brain nuclei) for the treatment of movement and some neurocognitive disorders, including Parkinson's disease and epilepsy. Deep brain stimulation (DBS) is used to manage some of the symptoms of Parkinson's disease that cannot be adequately controlled with medications. Deep brain stimulation (DBS) is recommended for people who have Parkinson's disease with motor fluctuations and tremor inadequately controlled by medication, or to those who are intolerant to medication, as long as they do not have severe neuropsychiatric problems. Four areas of the brain have been treated with neural stimulators in Parkinson's disease.

These are the globus pallidus internus, thalamus, subthalamic nucleus and the pedunculopontine nucleus. However, most DBS surgeries in routine practice target either the globus pallidus internus, or the subthalamic nucleus. Generally DBS is associated with 30–60% improvement in motor score evaluations.[2]

Glossary

deep brain stimulation: a neurosurgical procedure involving the placement of a medical device called a neurostimulator that sends electrical impulses, through implanted electrodes, to specific targets in the brain

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14.12: Case Studies- Neurocognitive Disorders

Learning Objectives

- Identify neurological disorders in case studies

Case Study: Sarah

Sarah is a 78-year-old female and is very outspoken. Sarah has been an actor in off Broadway shows as well as working as a consultant in the education industry for 30 years prior to retirement. Sarah and her family members over the past year have noticed that she is not remembering things as well. Sarah even admitted to her husband that she is more and more having forgetful moments over the past two years. Sarah's mother and aunt were diagnosed with a neurocognitive disorder several years before they passed away. Sarah agreed to go to the doctor and was a bit worried about the biological impact of her mother's disorder, but kept an open mind. Sarah went to the doctor and discussed what was going on, and the doctor referred Sarah to specialists who focus on memory. Sarah was asked if she partook in any substances, which she said she occasionally has wine to drink to unwind some evenings, but nothing problematic. Sarah was administered several memory tasks and the doctor said the results were OK, as she remembered two out of four items and said that he wanted to see her again in three months.

Sarah went back in three months and there was no change, but at six months she was only able to remember one out of four items on a memory task and he suggested starting treatment. Sarah was administered an acetylcholine agent that could help limit memory loss for a period of time. Over the next three years, Sarah remained with mild cognitive loss, but after three years on the medication, the effectiveness was not showing, unfortunately. Sarah was then told that she needed permanent care as her memory was progressing at a negative rate. Sarah was at home for another three years, but then was unable take care of herself and was put in a nursing home facility.



Figure **14.12.1**: Gina has seen a decline in her desire to participate in her usual activities alongside a decline in cognitive abilities.

Case Study: Gina

Gina is 76 years old and went to her doctor for a regular physical as she did each year. Gina told the physician that she recently has socially isolated herself and has not felt comfortable visiting and spending time with family. Gina also was having hallucinations and found that she had symptoms that were consistent with Parkinson's disease, but was not sure. Gina also told the physician that she seems to forget things a great deal more than she used to and wanted to find out why. Gina also discussed with her doctor that her alertness and attention varied quite heavily. Gina said her family members have said that they noticed a 20–25% decrease in her cognition over the past six months. Gina discussed as well that she is not as active as she was six months ago, and sometimes she does not have the energy to go outside and go for a walk as she has done in the past. Gina took part of a memory task at the physician and was only unable to recall one out of four items that the doctor presented to remember. Gina's doctor suggested that she receive an opinion from a specialist and referred her to them.

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14.13: Introduction to Other Mental Disorders

What you'll learn to do: examine other conditions related to understanding and treating mental illness



While the DSM-5 covers an extensive list of mental disorders, there are other sections in the DSM-5 that discuss other medical conditions and influences related to mental disorders. We will take a look at a few of these in this section.

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14.14: Medication-Induced Movement Disorders

Learning Objectives

- Describe complications connected to medications used to treat mental disorders

Other Mental Disorders

As we have learned in this course, in the United States, the DSM serves as the principal authority for psychiatric diagnoses. Treatment recommendations, as well as payment by health care providers, are often determined by DSM classifications. The categories of other mental disorders are as follows:

- neurodevelopmental disorders
- schizophrenia spectrum and other psychotic disorders
- bipolar and related disorders
- depressive disorders
- anxiety disorders
- obsessive-compulsive and related disorders
- trauma and stressor-related disorders
- dissociative disorders
- somatic symptom and related disorders
- feeding and eating disorders
- elimination disorders
- sleep-wake disorders
- sexual dysfunctions
- gender dysphoria
- disruptive, impulse control, and conduct-related disorders
- substance-related and addictive disorders
- neurocognitive disorders
- personality disorders
- paraphilic disorders

There are a few lesser-known areas of the DSM-5 that aid in classifying disorders related to other medical conditions or medications. Some disorders coincide with other medical conditions that cause distress or impairment of a mental disorder, but that do not meet the full criteria for another mental disorder; these include other specified mental disorder due to another medical condition or unspecified mental disorder due to another medical condition. Other mental disorders may also be labeled *other specified mental disorder* if there is significant distress or impairment in functioning, but the full criteria are not met for a mental disorder or unspecified mental disorder if there is likely a mental disorder present but insufficient information for the diagnosis.

Medication-Induced Movement Disorders

The DSM-5 also contains a section on medication-induced movement disorders and other adverse effects of medication that are *not* mental disorders, but complications or problems connected to the medication used to manage mental disorders.

Movement disorders are clinical syndromes with either an excess of movement or a paucity of voluntary and involuntary movements, unrelated to weakness or spasticity. Movement disorders are synonymous with basal ganglia or extrapyramidal diseases. Movement disorders are conventionally divided into two major categories—hyperkinetic and hypokinetic. **Hyperkinetic** movement disorders refer to dyskinesia, or excessive, often repetitive, involuntary movements that intrude upon the normal flow of motor activity. **Hypokinetic** movement disorders refer to akinesia (lack of movement), hypokinesia (reduced amplitude of movements), bradykinesia (slow movement), and rigidity. In primary movement disorders, abnormal movement is the primary manifestation of the disorder. In secondary movement disorders, abnormal movement is a manifestation of another systemic or neurological disorder.



Figure 14.14.1: An artist's rendition of akathisia. They describe suffering from akathisia as having burning pain and involuntary movements.

Types of disorders mentioned in the DSM-5 include the following:

- **neuroleptic-induced parkinsonism**: Parkinson's-like tremors, rigidity, or changes in movement following a change in medication
- **neuroleptic malignant syndrome**: hyperthermia (high body temperature) caused by antipsychotic medications, specifically a dopamine antagonist
- **medication-induced acute dystonia**: abnormal muscles contractions following a change in medication
- **medication-induced acute akathisia**: restlessness and fidgeting following a change in medication
- **tardive dyskinesia**: involuntary, repetitive body movements, which may include grimacing, sticking out the tongue, or smacking the lips
- **tardive dystonia**: permanent sustained or repetitive muscle contractions result in twisting and repetitive movements or abnormal fixed postures
- **tardive akathisia**: involves painful feelings of inner tension and anxiety and a compulsive drive to move the body
- **medication-induced postural tremor** and **other medication-induced movement disorder**: involuntary muscle contractions or twitching
- **antidepressant discontinuation syndrome**: flu-like symptoms, trouble sleeping, nausea, poor balance, sensory changes, and anxiety

Tardive Dyskinesia

Tardive dyskinesia is a disorder that results in involuntary, repetitive body movements, which may include grimacing, sticking out the tongue, or smacking the lips. Additionally, symptoms may be rapid jerking movements or slow writhing movements. In about 20% of people with tardive dyskinesia, the disorder interferes with daily functioning.

Tardive dyskinesia occurs in some people as a result of long-term use of dopamine-receptor blocking medications such as antipsychotics and metoclopramide. These specific medications are usually used for mental illness, but may also be given for gastrointestinal or neurological problems. The condition typically develops only after months to years of use. A diagnosis is based on the symptoms after ruling out other potential causes.

Antidepressants are sometimes associated with movement disorders. Among antidepressants, mirtazapine, vortioxetine, amoxapine, phenelzine, tryptophan, and fluvoxamine were associated with the highest level of movement disorders and citalopram, paroxetine, duloxetine, and mirtazapine were the most frequently associated with movement disorders. A potential harmful association was found between movement disorders and the use of the antidepressants mirtazapine, vortioxetine, amoxapine, phenelzine, tryptophan, fluvoxamine, citalopram, paroxetine, duloxetine, bupropion, clomipramine, escitalopram, fluoxetine, mianserin, sertraline, venlafaxine, and vilazodone.

Efforts to prevent the condition include either using the lowest possible dose or discontinuing the use of neuroleptics. Treatment includes stopping the neuroleptic medication if possible or switching to clozapine. Other medications such as valbenzaline, tetrabenazine, or botulinum toxin may be used to lessen the symptoms. With treatment, some see a resolution of symptoms while others do not. Rates in those on atypical antipsychotics are about 20%, while those on typical antipsychotics have rates of about 30%. The risk of acquiring the condition is greater in older people. The term *tardive dyskinesia* first came into use in 1964.

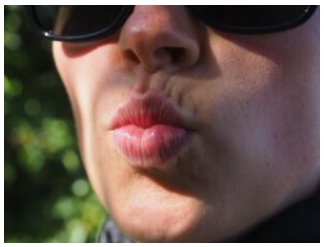


Figure **14.14.2**: Involuntary puckering or pursing of the lips can be associated with tardive dyskinesia.

Tardive dyskinesia is characterized by repetitive, involuntary movements. Some examples of these types of involuntary movements include

- grimacing.
- tongue movements.
- lip-smacking.
- lip puckering.
- pursing of the lips.
- excessive eye blinking.
- rapid, involuntary movements of the limbs, torso, and fingers.

Prevention of tardive dyskinesia is achieved by using the lowest effective dose of a neuroleptic for the shortest time. However, with diseases of chronic psychosis such as schizophrenia, this strategy must be balanced with the fact that increased dosages of neuroleptics are more beneficial in preventing the recurrence of psychosis. If tardive dyskinesia is diagnosed, the causative drug should be discontinued.

Antidepressant Associated Movement Disorder

A study on antidepressants using the WHO Pharmacovigilance database was assessed among adverse drug reactions related to any antidepressant, from January 1967 to February 2017, through a case/non-case design. The association between nine subtypes of movement disorders (akathisia, bruxism, dystonia, myoclonus, parkinsonism, restless legs syndrome, tardive dyskinesia, tics, and tremor) and antidepressants was estimated through the calculation first of crude Reporting Odds Ratio (ROR), then adjusted ROR on four potential confounding factors: age, sex, drugs described as able to induce movement disorders, and drugs used to treat movement disorders.

Antidepressants are one of the most frequently prescribed drug classes in Western countries. They have broad therapeutic indications, from depression to anxiety or obsessive-compulsive disorders, but also enuresis, chronic pain, or eating disorders. The most important classes of antidepressants are selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), tricyclic antidepressants (TCAs), and monoamine oxidase inhibitors (MAOIs). Antidepressants act mainly through the monoamine neurotransmitters, serotonin, and noradrenaline. They can induce several adverse drug reactions, including not only digestive disorders, sexual dysfunction, fatigue, or sleepiness, but also hyponatremia, hepatitis, or bleeding.

Movement disorders are clinical syndromes with either an excess or a paucity of voluntary and involuntary movements, unrelated to weakness or spasticity. They include extrapyramidal symptoms (akathisia, tardive dyskinesia, dystonia, and parkinsonism) but also a wide range of disorders, from tremor to tics and bruxism, to name a few. Although not the most frequent adverse drug reactions of antidepressants, antidepressant-induced movement disorders have been described and can lead to severe and disabling conditions. Reports of extrapyramidal symptoms associated with antidepressants have been documented for SSRIs, SNRIs, and other antidepressants.

When comparing each of the classes of antidepressants with the others, a significant association was observed for all subtypes of movement disorders except restless legs syndrome with SSRIs only. Among antidepressants, mirtazapine, vortioxetine, amoxapine, phenelzine, tryptophan, and fluvoxamine were associated with the highest level of movement disorders and citalopram, paroxetine, duloxetine, and mirtazapine were the most frequently associated with movement disorders. A potential harmful association was found between movement disorders and the use of the antidepressants mirtazapine, vortioxetine, amoxapine, phenelzine, tryptophan, fluvoxamine, citalopram, paroxetine, duloxetine, bupropion, clomipramine, escitalopram, fluoxetine, mianserin, sertraline, venlafaxine, and vilazodone.

Glossary

- **dyskinesia:** a movement disorder that results in involuntary, repetitive body movements, which may include grimacing, sticking out the tongue, or smacking the lips

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14.15: Other Conditions Related to Mental Disorders

Learning Objectives

- Explain how relational, emotional, situational, and environmental problems are connected with mental health

The DSM-5 also includes content about issues that are not mental disorders, but that may be a focus of clinical attention. These conditions are connected to mental disorders and may be the reason that a person first seeks help from a clinician. These conditions include the following:

- relational problems
  - abuse and neglect
  - child physical abuse, sexual abuse, neglect, and psychological abuse
  - adult or maltreatment and neglect problems (physical abuse, sexual abuse, neglect, and psychological abuse)
- emotional and occupational problems
- housing and economic problems
- other problems related to the social environment
- problems related to crime or interaction with the legal system
- other health service encounters for counseling and medical advice
- problems related to other psychosocial, personal, and environmental circumstances
- other circumstances of personal history

Relational Problems

Neglect is a form of abuse where the perpetrator, who is responsible for caring for someone who is unable to care for themselves, fails to do so. Neglect can be a result of carelessness, indifference, or unwillingness and abuse. Studies of neglected children show heightened levels of depression and hopelessness and higher incidents of suicide attempts. Also, too little parental availability can result in difficulties in problem-solving, coping with stressful situations, and social relationships.



Figure **14.15.1**: Being neglected as a child can have negative long-term effects on a person's life.

Neglect may include the failure to provide sufficient supervision, nourishment, or medical care, or the failure to fulfill other needs for which the victim cannot provide themselves. Neglect can carry on in a child's life falling into many long-term side effects such as: physical injuries, developmental trauma disorder, low self-esteem, attention disorders, violent behavior, and can even cause death.

There are many different types of neglect but they all have consequences, whether it be physical or mental. Neglect can affect the body physically by affecting a child's development and health, sometimes leading to chronic medical problems. Children experiencing neglect often suffer from malnutrition, which causes abnormal patterns for development. Not being given the proper nutrients at certain growth periods can result in stunted growth, and inadequate bone and muscle growth. Brain functioning and information processing may also be affected by neglect and may lead to difficulty in understanding directions, poor understanding of social relationships, or the inability to complete academic tasks without assistance. Neglected children or adults can have physical injuries like fractures or severe burns that go untreated, or infections, lice and other signs of lack of care. There are many physical effects neglect can have on a person.

Not only is neglect associated with physical problems, it also has an effect on a person mentally, ranging from poor peer relationships to violent behavior. Not only is behavior affected, but the way a person looks at themselves, which can lead to low

self-esteem and the feeling of being unwanted. Neglect is more severe in younger children when it comes to psychological consequences.

Child abuse or child maltreatment is physical, sexual, and/or psychological maltreatment or neglect of a child or children, especially by a parent or a caregiver. Child abuse may include any act or failure to act by a parent or a caregiver that results in actual or potential harm to a child and can occur in a child's home or in the organizations, schools, or communities the child interacts with.

The terms *child abuse* and *child maltreatment* are often used interchangeably, although some researchers make a distinction between them, treating *child maltreatment* as an umbrella term to cover neglect, exploitation, and trafficking.

Child abuse can result in immediate adverse physical effects but it is also strongly associated with developmental problems and with many chronic physical and psychological effects, including subsequent ill-health, including higher rates of chronic conditions, high-risk health behaviors, and shortened lifespan. Children who have a history of neglect or physical abuse are at risk of developing psychiatric problems or a disorganized attachment style. In addition, children who experience child abuse or neglect are 59% more likely to be arrested as juveniles, 28% more likely to be arrested as adults, and 30% more likely to commit a violent crime. Disorganized attachment is associated with a number of developmental problems, including dissociative symptoms, as well as anxiety, depressive, and acting out symptoms. A study by Dante Cicchetti found that 80% of abused and maltreated infants exhibited symptoms of disorganized attachment. When some of these children become parents, especially if they suffer from PTSD, dissociative symptoms, and other sequelae of child abuse, they may encounter difficulty when faced with their infant and young children's needs and normative distress, which may in turn lead to adverse consequences for their child's social-emotional development.

Housing and Poverty Problems

Housing inequality is a disparity in the quality of housing in a society that is a form of economic inequality. The right to housing is recognized by many national constitutions, and the lack of adequate housing can have adverse consequences for an individual or a family. The term may apply regionally (across a geographic area), temporally (between one generation and the next) or culturally (between groups with different racial or social backgrounds). Housing inequality is directly related to racial, social, income, and wealth inequality. It is often the result of market forces, discrimination, and segregation.

Housing inequality and poverty may lead to anxiety and depression, among other psychological disorders, due to constant stressors. Housing inequality is also a cause and an effect of poverty. The results of one study found that people who thrive with financial stability or fall under low SES, tend to perform worse cognitively due to external pressure imposed upon them. The research found that stressors such as low income, inadequate health care, discrimination, and exposure to criminal activities all contribute to mental disorders. This study also found that it slows cognitive thinking in children when they are exposed to poverty-stricken environments.[1] In kids, it is seen that kids perform better under the care and nourishment from their parents, and found that children tend to adopt speaking language at a younger age. Since being in poverty from childhood is especially more harmful than it is for an adult, it is seen that children in poor households tend to fall behind in certain cognitive abilities compared to other average families.

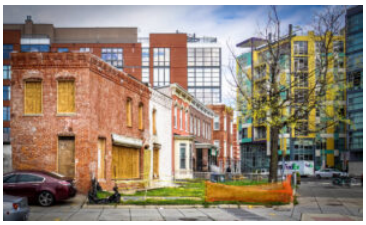


Figure **14.15.2**: A direct effect of housing inequality is the lack or inequality of neighborhood amenities.

Disparities in housing explain variations in the conversion of income into human capabilities in different social climates. Income does not always translate into desirable outcomes such as health care, education, and housing quality. According to economist and philosopher Amartya Sen, an individual's freedoms (or capabilities) are significant indicators of the kind of life they value or have a reason to value. As economic equality varies by the economic system, historical period, and society, so does housing inequality.

Economic inequality is a primary contributing factor to housing inequality. The distribution of wealth in a region affects who has access to housing and at what level.

Social inequality is the difference in access to social goods brought about by social constructs such as power, religion, kinship, prestige, race, ethnicity, gender, age, and class. Social goods include the labor market, a source of income, healthcare, freedom of speech, education, and political representation and participation. Social inequality may be a factor of housing inequality, as when individuals are denied access to housing based on social characteristics. Conversely, social inequality can be a result of housing inequality (such as social stigma associated with living in a certain neighborhood or type of home).

The most direct effect of housing inequality is an inequality of neighborhood amenities, which include the condition of surrounding houses, the availability of social networks, the amount of air pollution, the crime rate, and the quality of local schools. A neighborhood with a certain quality of amenities typically includes individual residences of corresponding quality. Those with lower incomes usually live in areas with poor amenities to win the spatial competition for housing. A neighborhood amenity includes satisfaction derived from living in a nice area, and many studies suggest that growing up in a high-poverty neighborhood affects social and economic outcomes later in life.

Another way the poor compete for housing is by renting homes rather than owning them, which furthers the negative effects of housing inequality by restricting access to household wealth. Although the focus of housing inequality has changed with time, contemporary international analyses tend to center on urbanization and the move to metropolitan areas. International housing inequality is largely characterized by urban disparities.

In developing countries, housing inequality is increasingly caused by rural-to-urban migration, increasing urban poverty and inequality, insecure tenure, and globalization. All these factors contribute to the creation and continuation of slums in poorer areas of the world. One proposed solution is slum upgrading.

These market forces are subject to other socio-economic factors; no one cause can explain housing inequality. In the United States, Thomas Shapiro and Jessica Kenty-Drane point to the wealth gaps between African Americans and other groups as likely causes of the housing disparity between African Americans and the rest of the country. According to Shapiro and Kenty-Drane, historical and social obstacles (slavery and racial segregation) have prevented African Americans from securing and accumulating assets including quality housing. Yinger also suggests that racial discrimination still plays a role in housing; Black and Latino households must pay higher search costs, accept lower-quality housing ,and live in lower-quality neighborhoods due to discrimination. One study found that 20% of potential moves made by African-American households and 17% of potential moves made by Latino households were discouraged by discrimination in the search process.

Social Environment Problems

The **social environment**, or social context, refers to the immediate physical and social setting in which people live or in which something happens or develops. The social environment includes the culture that the individual was educated or lives in and the people and institutions with whom they interact. The interaction may be in person or through communication media, even anonymous or one way, and may not imply equality of social status. The social environment is a broader concept than that of social class or social circle. People with the same social environment often develop a sense of social solidarity; people often tend to trust and help one another, and to congregate in social groups. People will often think in similar styles and patterns, even though the conclusions which they reach may differ.

In sociology and social psychology, racial identity and the acquisition of that identity is often used as a variable in racism studies. Racial ideologies and racial identity affect individuals' perception of race and discrimination. Cazenave and Maddern (1999) define racism as "a highly organized system of 'race'-based group privilege that operates at every level of society and is held together by a sophisticated ideology of color/'race' supremacy. Racial centrality (the extent to which a culture recognizes individuals' racial identity) appears to affect the degree of discrimination African American young adults perceive whereas racial ideology may buffer the detrimental emotional effects of that discrimination." Sellers and Shelton (2003) found that a relationship between racial discrimination and emotional distress was moderated by racial ideology and social beliefs.

Some sociologists also argue that, particularly in the West, where racism is often negatively sanctioned in society, racism has changed from being a blatant to a more covert expression of racial prejudice. The "newer" (more hidden and less easily detectable) forms of racism—which can be considered embedded in social processes and structures—are more difficult to explore as well as

challenge. It has been suggested that, while in many countries overt or explicit racism has become increasingly taboo, even among those who display egalitarian explicit attitudes, an implicit or aversive racism is still maintained subconsciously.

This process has been studied extensively in social psychology as implicit associations and implicit attitudes, a component of implicit cognition. Implicit attitudes are evaluations that occur without conscious awareness towards an attitude object or the self. These evaluations are generally either favorable or unfavorable. They come about from various influences in the individual experience. Implicit attitudes are not consciously identified (or they are inaccurately identified) traces of past experience that mediate favorable or unfavorable feelings, thoughts, or actions towards social objects. These feelings, thoughts, or actions have an influence on behavior of which the individual may not be aware.

Cultural Syndromes

Some mental disorders are unique to specific cultures; while they may share similarities with others mentioned in the DSM-5, these disorders are only considered recognizable within their respective cultures. In the DSM-4, these were called culture-bound syndromes, but are now identified in the DSM-5 as cultural concepts of distress. Some of these cultural concepts of distress are described below:[2]

Syndrome	Description	Populations
Ataque de nervios	Commonly reported symptoms include uncontrollable shouting, attacks of crying, trembling, heat in the chest rising into the head, and verbal or physical aggression. Dissociative experiences, seizure-like or fainting episodes, and suicidal gestures are prominent in some attacks but absent in others. A general feature of an ataque de nervios is a sense of being out of control. Ataques de nervios frequently occur as a direct result of a stressful event relating to the family (e.g., death of a close relative, separation or divorce from a spouse, conflict with spouse or children, or witnessing an accident involving a family member). People can experience amnesia for what occurred during the ataque de nervios, but they otherwise return rapidly to their usual level of functioning. Although descriptions of some ataques de nervios most closely fit with the DSM-4 description of panic attacks, the association of most ataques with a precipitating event and the frequent absence of the hallmark symptoms of acute fear or apprehension distinguish them from panic disorder. Ataques range from normal expressions of distress not associated with a mental disorder to symptom presentations associated with anxiety, mood dissociative, or somatoform disorders.	Caribbean, Latin American, Latin Mediterranean
Dhat *(jiryan* in India, *skra prameha* in Sri Lanka, *shen-k'uei* in China)	A folk diagnosis for severe anxiety and hypochondriacal concerns associated with the discharge of semen, whitish discoloration of the urine, weakness, and exhaustion.	Asian Indian
Nervios	Refers both to a general state of vulnerability to stress and to a syndrome evoked by difficult life circumstances. *Nervios* includes a wide range of symptoms of emotional distress, somatic disturbance, and inability to function. Common symptoms include headaches and "brain aches," irritability, stomach disturbances, sleep difficulties, nervousness, tearfulness, inability to concentrate, trembling, tingling sensations, and *mareos* (dizziness with	Latin American

Syndrome	Description	Populations
	occasional vertigo-like exacerbations). *Nervios* tends to be an ongoing problem, although it is variable in the degree of disability manifested. *Nervios* is a broad syndrome that ranges from cases free of a mental disorder to presentations resembling adjustment, anxiety, depressive, dissociative, somatoform, or psychotic disorders. Differential diagnosis depends on the constellation of symptoms, the kind of social events associated with onset and progress, and the level of disability experienced.	
Shenjing shuairuo	A condition characterized by physical and mental fatigue, headaches, difficulty concentrating, dizziness, sleep disturbance, and memory loss. Other symptoms include gastrointestinal problems, sexual dysfunction, irritability, excitability, and autonomic nervous system disturbances.	Chinese
Susto *(espanto, pasmo, tripa ida, perdida del alma,* or *chibih)*	An illness attributed to a frightening event that causes the soul to leave the body and results in unhappiness and sickness. Individuals with *susto* also experience significant strains in key social roles. Symptoms can appear days or years after the fright is experienced. In extreme cases, *susto* can result in death. Typical symptoms include appetite disturbances, inadequate or excessive sleep, troubled sleep or dreams, sadness, lack of motivation, and feelings of low self-worth or dirtiness. Somatic symptoms accompanying *susto* include muscle aches and pains, headache, stomachache, and diarrhea. Ritual healings focus on calling the soul back to the body and cleansing the person to restore bodily and spiritual balance. *Susto* can be related to major depressive disorder, posttraumatic stress disorder, and somatoform disorders. Similar etiological beliefs and symptom configurations are found in many parts of the world.	Latino American, Mexican, Central and South American
Taijin kyofusho	This syndrome refers to an individual's intense fear that his or her body, its parts, or its functions displease, embarrass, or	Japanese

Syndrome	Description	Populations
	are offensive to other people in appearance, odor, facial expressions, or movement. This syndrome is included in the official Japanese diagnostic system for mental disorders.	

Glossary

abuse: the improper usage or treatment of a person, often to unfairly or improperly gain benefit

child abuse: physical, sexual, and/or psychological maltreatment or neglect of a child or children, especially by a parent or a caregiver

housing inequality: a disparity in the quality of housing in a society; a form of economic inequality

neglect: a form of abuse where the perpetrator, who is responsible for caring for someone who is unable to care for themselves, fails to do so

social environment: the immediate physical and social setting in which people live or in which something happens or develops

1. Sleek, Scott (31 August 2015). "How Poverty Affects the Brain and Behavior". APS Observer. 28 (7). ↵
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14.16: Putting It Together- Neurocognitive Disorders

Considering all that you have learned during this module, you now are more aware of the different, neurological, neurocognitive, and medication-induced movement disorders. For a quick wrap-up, the main differences between the three are these:



Figure **14.16.1**: Paralysis as a result of a neurological disorder can make everyday tasks difficult.

A *neurological disorder* is any disorder of the nervous system. Structural, biochemical, or electrical abnormalities in the brain, spinal cord, or other nerves can result in a range of symptoms. They may be caused by damage to the brain. Examples of symptoms include paralysis, muscle weakness, poor coordination, loss of sensation, seizures, confusion, pain, and altered levels of consciousness.

Neurocognitive disorders (NCDs) are a category of mental health disorders that primarily affect cognitive abilities including learning, memory, perception, and problem-solving. Neurocognitive disorders include delirium and mild and major neurocognitive disorder (previously known as dementia). They are defined by deficits in cognitive ability that are acquired (as opposed to developmental), typically represent decline, and may have an underlying brain pathology. These are the main types of disorders we discussed in this module, although the *neuro* part of the definition correctly implies that there is a neurological cause for the disorder, so the terms have some overlapping.

Movement disorders are clinical syndromes with either an excess of movement or a paucity of voluntary and involuntary movements, unrelated to weakness or spasticity. They can be caused by medications used to treat mental disorders. Movement disorders are synonymous with basal ganglia or extrapyramidal diseases. Movement disorders are conventionally divided into two major categories: hyperkinetic and hypokinetic.

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GLOSSARY

Acute Stress Disorder | A trauma-related condition characterized by severe anxiety and dissociation occurring within a month of a traumatic event.

Adjustment Disorders | Emotional or behavioral symptoms in response to a specific stressor, disproportionate to the stressor's intensity.

Anorexia Nervosa | An eating disorder characterized by intense fear of gaining weight and self-starvation leading to extreme weight loss.

Antisocial Personality Disorder | A Cluster B personality disorder marked by disregard for societal norms, impulsivity, and lack of empathy.

Assessment | The process of gathering information to understand and diagnose mental health conditions.

Behavior Therapy | A treatment focused on modifying harmful behaviors through conditioning techniques.

Bipolar Disorder | A mood disorder characterized by alternating periods of mania and depression.

Body Dysmorphic Disorder | A preoccupation with perceived flaws in physical appearance that are often unnoticed by others.

Case Study | An in-depth investigation of an individual or group, used in descriptive research.

Cognitive Behavioral Therapy | A structured psychotherapy approach combining cognitive and behavioral techniques to modify unhelpful thought patterns and behaviors.

Conversion Disorder | A somatic symptom disorder involving neurological symptoms that lack a medical explanation, such as paralysis or blindness.

Cyclothymic Disorder | A mood disorder involving chronic, fluctuating moods that are less severe than bipolar disorder.

Delirium | A sudden disturbance in attention, awareness, and cognition often caused by medical conditions.

Dissociative Identity Disorder | A dissociative disorder characterized by the presence of two or more distinct personality states.

DSM-5 | A classification system for mental disorders used in the U.S.

Epidemiology | The study of the prevalence, causes, and effects of health conditions within populations.

Etiology | The study of causes and factors that contribute to the development of mental disorders.

Excoriation Disorder | A compulsive skin-picking disorder leading to lesions.

Functional Neurological Symptom Disorder | A condition where neurological symptoms (e.g., seizures, paralysis) are present without an identifiable medical cause.

Generalized Anxiety Disorder | A chronic anxiety disorder characterized by excessive worry across various situations.

Hallucinogen-Related Disorders | Disorders involving the misuse of hallucinogens, leading to perceptual and cognitive distortions.

Hoarding Disorder | Persistent difficulty discarding possessions, leading to clutter that disrupts living spaces.

Intelligence Testing | Assessments designed to measure intellectual capabilities and cognitive functioning.

International Classification of Diseases | A global diagnostic tool maintained by the WHO for classifying diseases and health conditions, including mental disorders.

Labeling | The process of assigning a diagnostic category to an individual, which can lead to stigma.

Major Depressive Disorder | A mood disorder characterized by persistent feelings of sadness, hopelessness, and a lack of interest in activities.

Mental Status Examination | A clinical assessment tool used to evaluate cognitive, emotional, and psychological functioning.

Multidimensional Model | A framework for understanding mental disorders by integrating biological, psychological, and sociocultural factors.

Narcolepsy | A sleep-wake disorder involving uncontrollable episodes of falling asleep.

Neurocognitive Disorders | Disorders involving a decline in cognitive functioning, often caused by neurological or medical conditions.

Neurodevelopmental Disorders | Disorders that emerge in childhood, affecting development, such as autism spectrum disorder or ADHD.

Obsessive-Compulsive Disorder | A disorder involving intrusive thoughts (obsessions) and repetitive behaviors (compulsions) performed to alleviate distress.

Oppositional Defiant Disorder | A childhood disorder characterized by defiant, hostile, and disobedient behavior toward authority figures.

Paraphilic Disorders | Disorders involving intense and persistent sexual interests outside of typical norms, leading to distress or impairment.

Personality Disorders | A group of mental disorders involving rigid, unhealthy patterns of thinking, functioning, and behavior.

Post-Traumatic Stress Disorder | A disorder triggered by experiencing or witnessing traumatic events, leading to intrusive memories and heightened arousal.

Reactive Attachment Disorder | A disorder in children characterized by difficulty forming emotional bonds with caregivers due to early neglect or abuse.

Schizophrenia | A psychotic disorder involving disturbances in thought, perception, and behavior, including hallucinations and delusions.

Social Anxiety Disorder | An anxiety disorder marked by intense fear or avoidance of social situations.

Somatic Symptom Disorder | A condition where individuals experience excessive concern about physical symptoms, which may not have a medical cause.

Stigma | Negative attitudes or discrimination toward individuals with mental health conditions.

Treatment Modalities | Various therapeutic approaches and methods used to treat mental disorders, such as individual therapy, group therapy, or medication.

Trichotillomania | A compulsive hair-pulling disorder leading to noticeable hair loss.

Why It Matters | Sections in the textbook designed to highlight the relevance and importance of understanding specific disorders or research areas.

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