ESSENTIALS OF ABNORMAL PSYCHOLOGY

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About this book

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CHAPTER OVERVIEW

1: What is Abnormal Psychology

Learning Objectives

- Explain what it means to display abnormal behavior.
- Identify types of mental health professionals
- Clarify the manner in which mental health professionals classify mental disorders.
- Describe the effect of stigma on those afflicted with mental illness.
- Outline the history of mental illness.

1.1: Chapter Overview

This is the first chapter in the main body of the text. You can change the text, rename the chapter, add new chapters, and add new parts.

1.2: Understanding Abnormal Behavior

Section Learning Objectives

- Define abnormal psychology, psychopathology, and psychological disorders.
- Explain the concept of dysfunction as it relates to mental illness.
- Explain the concept of distress as it relates to mental illness.
- Explain the concept of deviance as it relates to mental illness.
- Explain the concept of dangerousness as it relates to mental illness.
- Define culture and social norms.
- Know the cost of mental illness to society.
- Identify and describe the various types of mental health professionals.

1.2.1: Definition of Abnormal Psychology and Psychopathology

The term **abnormal psychology** refers to the scientific study of people who are atypical or unusual, with the intent to be able to reliably predict, explain, diagnose, identify the causes of, and treat maladaptive behavior. A more sensitive and less stigmatizing term that is used to refer to the scientific study of psychological disorders is **psychopathology.** These definitions beg the questions of, what is considered abnormal and what is a psychological or mental disorder?

1.2.2: Defining Psychological Disorders

It may be surprising to you, but the concept of mental or psychological disorders has proven very difficult to define and even the American Psychiatric Association (APA, 2013), in its publication, the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5 for short), states that though "no definition can capture all aspects of all disorders in the range contained in the DSM-5" certain aspects are required. While the concept of mental or psychological disorders is difficult to define, and no definition will ever be perfect, it is recognized as an extremely important concept and therefore **psychological disorders** (aka **mental disorders**) have been defined as a psychological dysfunction which causes distress or impaired functioning and deviates from typical or expected behavior according to societal or cultural standards. This definition includes three components (3 Ds). Let's break these down now:

- **Dysfunction** – includes "clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning" (pg. 20). In other words, dysfunction refers to a breakdown in ***cognition***, ***emotion***, and/or***behavior***. For instance, an individual experiencing delusions that he is an omnipotent deity would have a breakdown in cognition because his thought processes are not consistent with reality. An individual who is unable to experience pleasure would have a breakdown in emotion. Finally, an individual who is unable to leave her home and attend work due to fear of having a panic attack would be exhibiting a breakdown in behavior. Abnormal behavior has the capacity to make our well-being difficult to obtain and can be assessed by looking at an individual's current performance and comparing it to what is expected in general or how the person has performed in the past.
- **Distress or Impairment** – ***Distress*** can take the form of psychological or physical pain, or both concurrently. Simply put, distress refers to suffering. Alone though, distress is not sufficient enough to describe behavior as abnormal. Why is that? The loss of a loved one would cause even the most "normally" functioning individual pain and suffering. An athlete who experiences a career-ending injury would display distress as well. Suffering is part of life and cannot be avoided. And some people who display abnormal behavior are generally positive while doing so. Typically, if distress is absent then impairment must be present to deem behavior abnormal. ***Impairment*** refers to when the person experiences a disabling condition "in social, occupational, or other important activities" (pg. 20). In other words, impairment refers to when a person loses the capacity to function normally in daily life (e.g., can no longer maintain minimum standards of hygiene, pay bills, attend social functions, or go to work). Once again typically distress and/or impairment in functioning are required to consider behavior abnormal and to diagnose a psychological disorder.
- **Deviance** – A closer examination of the word abnormal shows that it indicates a move away from what is normal, typical, or average. Our **culture** – or the totality of socially transmitted behaviors, customs, values, technology, attitudes, beliefs, art, and other products that are particular to a group – determines what is normal and so a person is said to be deviant when he or she fails to follow the stated and unstated rules of society, called **social norms**. What is considered "normal" by society can change over time due to shifts in accepted values and expectations. For instance, just a few decades ago homosexuality was considered taboo in the U.S. and it was included as a mental disorder in the first edition of the DSM; but today, it is generally accepted. Likewise, PDAs, or public displays of affection, do not cause a second look by most people unlike the past when these outward expressions of love were restricted to the privacy of one's own house or bedroom. In the U.S., crying is generally seen as a weakness for males but if the behavior occurs in the context of a tragedy such as the Vegas mass shooting on October 1, 2017, in which 58 people were killed and about 500 were wounded, then it is appropriate and understandable. Finally, consider that statistically deviant behavior is not necessarily negative. Genius is an example of behavior that is not the norm.

Though not part of the DSM 5's conceptualization of what abnormal behavior is, many clinicians add a 4th D – dangerousness to this list. **Dangerousness** refers to when behavior represents a threat to the safety of the person or others. Individuals expressing suicidal intent, those experiencing acute paranoid ideation combined with aggressive impulses (e.g., wanting to harm people who are perceived as "being out to get them"), and many individuals with antisocial personality disorder may be considered dangerous. Mental health professionals (and many other professionals including researchers) have a duty to report to law enforcement when an individual expresses an intent to harm themselves or others. Nevertheless, individuals with depression, anxiety, and obsessive-compulsive disorder are typically no more a threat to others than individuals without these disorders. As such, it is important to note that having a mental disorder does not automatically deem one to be dangerous and most dangerous individuals are not mentally ill. Indeed, a review of the literature (Matthias & Angermeyer, 2002) found that only a small proportion of crimes are committed by individuals with severe mental disorders, that strangers are at a lower risk of being attacked by a person with a severe mental disorder than by someone who is mentally healthy, and that elevated risks to behave violently are limited to a small number of symptom constellations. Similarly, Hiday and Burns (2010) showed that dangerousness is more the exception than the rule.

1.2.3: What is the Cost of Mental Illness to Society?

This leads us to consider the cost of mental illness to society. The National Alliance on Mental Illness (NAMI) indicates that depression is the number one cause of disability across the world "and is a major contributor to the global burden of disease." Serious mental illness costs the United States an estimated $193 billion in lost earnings each year. They also point out that suicide is the 10th leading cause of death in the U.S. and 90% of those who die from suicide have an underlying mental illness. In relation to children and teens, 37% of students with a mental disorder age 14 and older drop out of school which is the highest dropout rate of any disability group, and 70% of youth in state and local juvenile justice systems have at least one mental disorder. Source: https://www.nami.org/Learn-More/Mental-Health-By-the-Numbers. In terms of worldwide impact, the World Economic Forum used 2010 data to estimate $2.5 trillion in global costs in 2010 and projected costs of $6 trillion by 2030. The costs for mental illness are greater than the combined costs of cancer, diabetes, and respiratory disorders (Whiteford et al., 2013). And finally, "The Social Security Administration reports that in 2012, 2.6 and 2.7 million people under age 65 with mental illness-related disability received SSI and SSDI payments, respectively, which represents 43 and 27 percent of the total number of people receiving such support, respectively" (Source: https://www.nimh.nih.gov/about/directors/thomas-insel/blog/2015/mental-health-awareness-month-by-the-numbers.shtml). So as you can see the cost of mental illness is quite staggering for the United States and other countries.

Check this out: Seven Facts about America's Mental Health-Care System

https://www.washingtonpost.com/news/...=.12de8bc56941

In conclusion, though there is no one behavior that we can use to classify people as abnormal, most clinical practitioners agree that any behavior that strays from what is considered the norm or is unexpected within the confines of one's culture, that causes dysfunction in cognition, emotion, and/or behavior, and that causes distress and/or impairment in functioning, is abnormal behavior. Armed with this understanding, let's discuss what mental disorders are.

1.2.4: Types of Mental Health Professionals

There are many types of mental health professionals that people may seek out for assistance. They include:

Table 1: Types of Mental Health Professionals

Name	Degree Required	Function/Training	Can they prescribe medications?
Clinical Psychologist	Ph.D.	Trained to make diagnoses and can provide individual and group therapy	Only in select states
School Psychologist	Masters or Ph.D.	Trained to make diagnoses and can provide individual and group therapy but also works with school staff	No
Counseling Psychologist	Ph.D.	Deals with adjustment issues primarily and less with mental illness	No
Clinical Social Worker	M.S.W. or Ph.D.	Trained to make diagnoses and can provide individual and group therapy and is involved in advocacy and case management. Usually in hospital settings.	No
Psychiatrist	M.D.	Has specialized training in the diagnosis and treatment of mental disorders	Yes
Psychiatric Nurse Practitioner	M.R.N.	Has specialized training in the care and treatment of psychiatric patients	Yes
Occupational Therapist	M.S.	Has specialized training with individuals with physical or psychological conditions and helps them acquire needed resources	No
Drug Abuse and/or Alcohol Counselor	B.S. or higher	Trained in alcohol and drug abuse and can make diagnoses and can provide individual and group therapy	No
Child/Adolescent Psychiatrist	M.D. or Ph.D.	Specialized training in the diagnosis and treatment of mental illness in children	Yes
Marital and Family Therapist	Masters	Specialized training in marital and family therapy; Can make diagnoses and can provide individual and group therapy	No

1.2.4.1: Prescription Rights for Psychologists

To reduce inappropriate and over-prescribing it has been proposed to allow appropriately trained psychologists the right to prescribe. Psychologists are more likely to choose between therapy and medications, and so can make the best choice for their patient. The right has already been granted in New Mexico, Louisiana, Guam, the military, the Indian Health Services, and the U.S. Public Health Services. Measures in other states "have been opposed by the American Medical Association and American Psychiatric Association over concerns that inadequate training of psychologists could jeopardize patient safety. Supporters of prescriptive authority for psychologists are quick to point out that there is no evidence to support these concerns (Smith, 2012)."

For more information on types of mental health professionals, please visit:

http://www.mentalhealthamerica.net/types-mental-health-professionals

1.3: Chapter 1- What is Abnormal Psychology?

Chapter Overview

Cassie is an 18-year-old female from suburban Seattle, WA. She was a successful student in high school, graduating valedictorian, and she obtained a National Merit Scholarship for her performance on the PSAT during her junior year. She was accepted to a university on the far eastern side of the state where she received additional scholarships which together, gives her a free ride for her full four years of undergraduate education. Excited to start this new chapter in her life, Cassie's parents begin the 5 hour commute to Pullman where they will leave their only daughter for the first time in her life. The semester begins as it always does in late August. Cassie meets the challenge head-on and does well in all of her classes for the first few weeks of the semester, as expected. Sometime around Week 6, her friends notice she is despondent, detached, and falling behind in her work. After being asked about her condition she replies that she is "just a bit homesick." Her friends accept the answer as this is a typical response to leaving home and starting college for many students. A month later her condition has not improved but actually worsens. She now regularly shirks her responsibilities around her apartment, in her classes, and on her job. Cassie does not hang out with friends like she did when she first arrived at college and stays in bed most of the day. Concerned, her friends contact Health and Wellness for help.

Cassie's story, though hypothetical, is true of many Freshman leaving home for the first time to earn a higher education, whether in rural Washington state or urban areas such as Chicago and Dallas. Most students recover from episodes of depression and go on to be functional members of their collegiate environment and accomplished scholars. Some learn to cope on their own while others seek assistance from their university's health and wellness center or from friends who have already been through similar ordeals. This is a normal reaction. But in Cassie's case and that of other students, the path to recovery is not as clear and instead of learning how to cope, their depression increases until it reaches clinical levels and becomes an impediment to success in multiple domains of life such as home, work, school, and social circles.

In Chapter 1, we will explore what it means to display abnormal behavior, how mental disorders are classified and how society views them both today and has throughout history.

Chapter Outline

- 1.1. Understanding Abnormal Behavior
- 1.2. Classifying Mental Disorders
- 1.3. The History of Mental Illness

Chapter Learning Outcomes

- Explain what it means to display abnormal behavior.
- Identify types of mental health professionals
- Clarify the manner in which mental health professionals classify mental disorders.
- Describe the effect of stigma on those afflicted with mental illness.
- Outline the history of mental illness.

1.4: Classifying Mental Disorders

Section Learning Objectives

- Define and exemplify classification.
- Define nomenclature.
- Define epidemiology.
- Define presenting problem and clinical description.
- Differentiate prevalence and incidence and subtypes of prevalence.
- Define comorbidity.
- Define etiology.
- Define course.
- Define prognosis.
- Define treatment.
- Explain the concept of stigma and its three forms.
- Define courtesy stigma.
- Describe what the literature shows about stigma.

1.4.1: Classification and Definitions

1.4.1.1: Classification

Classification is not a foreign concept and as a student, you have likely taken at least one biology class that discussed the taxonomic classification system of Kingdom, Phylum, Class, Order, Family, Genus, and Species revolutionized by Swedish botanist, Carl Linnaeus. You probably even learned a witty mnemonic such as 'King Phillip, Come Out For Goodness Sake' to keep the order straight. The Library of Congress uses classification to organize and arrange their book collections and includes such categories as B – Philosophy, Psychology, and Religion; H – Social Sciences; N – Fine Arts; Q – Science; R – Medicine; and T – Technology.

Simply, **classification** is the way in which we organize or categorize things. The second author's wife has been known to color code her DVD collection by genre, movie title, and at times release date. It is useful for us to do the same with abnormal behavior and classification provides us with a **nomenclature**, or naming system, to structure our understanding of mental disorders in a meaningful way. Of course, we want to learn as much as we can about a given disorder so we can understand its cause, predict its future occurrence, and develop ways to treat it.

1.4.1.2: Definitions

Epidemiology is the scientific study of the frequency and causes of diseases and other health-related states in specific populations such as a school, neighborhood, a city, country, or the entire world. **Psychiatric or mental health epidemiology** refers to the study of the frequency of occurrence of mental disorders in a population. In mental health facilities, we say that a patient presents with a specific problem, or the **presenting problem**, and we give a **clinical description** of it which includes information about the thoughts, feelings, and behaviors that constitute that mental disorder. We also seek to gain information about the occurrence of the disorder, its cause, course, and treatment possibilities.

Occurrence can be investigated in several ways. First, **prevalence** is the percentage of people in a population that has a mental disorder. It can also be conceptualized as the number of cases of the disorder per some number of people (usually 100). For instance, if 1 person out of 100 has schizophrenia, then the prevalence rate is 1% (or 1 in 100). Prevalence can be measured in several ways:

- **Point prevalence** indicates the percentage of a population that has the disorder at a specific point in time. In other words, it is the number of active cases at a given point in time.
- **Period prevalence** indicates the percentage of a population that has the disorder at any point during a given period of time, typically the past year (Note: when it is the past year it may also be referred to as the one-year prevalence).
- **Lifetime prevalence** indicates the percentage of a population that has had the disorder at any time during their lives.

According to the National Survey on Drug Use and Health (NSDUH), in 2015 there was an estimated 9.8 million U.S. adults aged 18 years or older with a *serious* mental illness, or 4% of all U.S. adults, and 43.4 million adults aged 18 years or older with any

mental illness, or 17.9% of all U.S. adults.

Source: https://www.nimh.nih.gov/health/statistics/prevalence/index.shtml

Incidence indicates the number of *new* cases in a population over a specific period of time. This measure is usually lower since it does not include existing cases as prevalence does. If you wish to know the number of new cases of social phobia during the past year (going from say Aug 21, 2015 to Aug 20, 2016), you would only count cases that began during this time and ignore cases that emerged before the start date, even if people are currently afflicted with the mental disorder. Incidence is often studied by medical and public health officials so that causes can be identified and future cases prevented.

Comorbidity describes when two or more mental disorders are occurring at the same time and in the same person. The National Comorbidity Survey Replication (NCS-R) study conducted by the National Institute of Mental Health (NIMH) and published in the June 6, 2005 issue of the Archives of General Psychiatry, sought to discover trends in prevalence, impairment, and service use during the 1990s. It revealed that 45% of those with one mental disorder met the diagnostic criteria for two or more disorders. The authors also found that the severity of mental illness, in regards to disability, is strongly related to comorbidity and that substance use disorders often result from disorders such as anxiety and bipolar mood disorders. The implications of this are substantial as services to treat substance abuse and mental disorders are often separate, despite their appearing together.

The **etiology** is the cause of the disorder. As you will see later in this textbook, there is no single cause of any mental disorder. Rather, there are multiple factors that contribute to increase a person's susceptibility to developing a mental disorder. These factors include social, biological, or psychological explanations which need to be understood to identify the appropriate treatment. Likewise, the effectiveness of a treatment may give some hint at the cause of the mental disorder. More on this later.

The **course** of the disorder is its particular pattern. A disorder may be chronic, meaning it lasts a long period of time, *episodic*, meaning the disorder comes and goes (i.e., individuals tend to recover only to have later reoccurrences). Disorders can also be classified as *time-limited*, meaning that recovery will occur in a short period of time regardless of whether any treatment occurs.

Prognosis is the anticipated course the mental disorder will take. A key factor in determining the course is age, with some disorders presenting differently in childhood than adulthood.

Finally, we will discuss several treatment strategies in this book in relation to specific disorders, and in a general fashion in Module 3. **Treatment** is any procedure intended to modify abnormal behavior into normal behavior. The person with the mental disorder seeks the assistance of a trained professional to provide some degree of relief over a series of therapy sessions. The trained mental health professional may utilize psychotherapy and/or medication may be prescribed to bring about this change. Treatment may be sought from the primary care provider (e.g., medical doctor), in an outpatient fashion with a clinical psychologist or psychiatrist, or through inpatient care or hospitalization with at a mental hospital or psychiatric unit of a general hospital.

1.4.2: The Stigma of Mental Disorders

In the previous section, we indicated that care can be sought out in a variety of ways. The problem is that many people who need care never seek it out. Why is that? We already know that society dictates what is considered abnormal behavior through culture and social norms, and you can likely think of a few implications of that. But to fully understand society's role in why people do not seek care, we need to consider the stigma that is often attached to the label mental disorder.

Stigma refers to when negative stereotyping, labeling, rejection, and loss of status occur. Stigma often takes on three forms as described below:

- *Public stigma* – when members of a society endorse negative stereotypes of people with a mental disorder and discriminate against them. They might avoid them altogether resulting in social isolation. An example is when an employer intentionally does not hire a person because their mental illness is discovered.
- *Label avoidance* – In order to avoid being labeled as "crazy" people needing care may avoid seeking it all together or stop care once started. Due to these labels, funding for mental health services could be restricted and instead, physical health services funded.
- *Self-stigma* – When people with mental illnesses internalize the negative stereotypes and prejudice, and in turn, discriminate against themselves. They may experience shame, reduced self-esteem, hopelessness, low self-efficacy, and a reduction in coping mechanisms. An obvious consequence of these potential outcomes is the *why try* effect, or the person saying, 'Why should I try and get that job? I am not unworthy of it' (Corrigan, Larson, & Rusch, 2009; Corrigan, et al., 2016).

Another form of stigma that is worth noting is that of **courtesy stigma** or when stigma affects people associated with the person with a mental disorder. Karnieli-Miller et. al. (2013) found that families of the afflicted were often blamed, rejected, or devalued when others learned that one of their family members had a serious mental illness. Due to this, they felt hurt and betrayed and an important source of social support during the difficult time had been removed, resulting in greater levels of stress. To cope, they had decided to conceal their relative's illness and some parents struggled to decide whether it was their place to disclose information about their child's mental illness or their child's place to do so. Others fought with the issue of confronting the stigma through attempts at education or to just ignore it due to not having enough energy or a desire to maintain personal boundaries. There was also a need to understand responses of others and to attribute those responses to a lack of knowledge, experience, and/or media coverage. In some cases, the reappraisal allowed family members to feel compassion for others rather than feeling put down or blamed. The authors concluded that each family "develops its own coping strategies which vary according to its personal experiences, values, and extent of other commitments" and that the "coping strategies families employ change over-time."

Other effects of stigma include experiencing work-related discrimination resulting in higher levels of self-stigma and stress (Rusch et al., 2014), higher rates of suicide especially when treatment is not available (Rusch, Zlati, Black, and Thornicroft, 2014; Rihmer & Kiss, 2002), and a decreased likelihood of future help-seeking (Lally et al., 2013). The results of the latter study also showed that personal contact with someone with a history of mental illness led to a decreased likelihood of seeking help. This is important because 48% of the sample stated that they needed help for an emotional or mental health issue during the past year but did not seek help. Similar results have been reported in other studies (Eisenberg, Downs, Golberstein, & Zivin, 2009). It is important to also point out that social distance, a result of stigma, has also been shown to increase throughout the lifespan, suggesting that anti-stigma campaigns should focus primarily on older people (Schomerus, et al., 2015).

One potentially disturbing trend is that mental health professionals have been shown to hold negative attitudes toward the people they serve. Hansson et al. (2013) found that staff members at an outpatient clinic in the southern part of Sweden held the most negative attitudes about whether an employer would accept an applicant for work, willingness to date a person who had been hospitalized, and hiring a patient to care for children. Attitudes were stronger when staff treated patients with psychosis or in inpatient settings. In a similar study, Martensson, Jacobsson, and Engstrom (2014) found that staff had more positive attitudes towards persons with mental illness if their knowledge of such disorders is less stigmatized, their workplaces were in the county council – as they were more likely to encounter patients who recover and return to normal life in society compared to municipalities where patients have long-term and recurrent mental illness -, and they have or had one close friend with mental health issues.

To help deal with stigma in the mental health community, Papish et al. (2013) investigated the effect of a one-time contact-based educational intervention compared to a four-week mandatory psychiatry course on the stigma of mental illness among medical students at the University of Calgary. The course included two methods involving contact with people who had been diagnosed with a mental disorder – patient presentations or two, one-hour oral presentations in which patients shared their story of having a mental illness; and "clinical correlations" in which students are mentored by a psychiatrist while they directly interacted with patients with a mental illness in either inpatient or outpatient settings. Results showed that medical students did hold stigmatizing attitudes towards mental illness and that comprehensive medical education can reduce this stigma. As the authors stated, "These results suggest that it is possible to create an environment in which medical student attitudes towards mental illness can be shifted in a positive direction." That said, the level of stigma was still higher for mental illness than it was for a stigmatized physical illness, type 2 diabetes mellitus.

What might happen if mental illness is presented as a treatable condition? McGinty, Goldman, Pescosolido, and Barry (2015) found that portraying schizophrenia, depression, and heroin addiction as untreated and symptomatic increased negative public attitudes towards people with these conditions but when the same people were portrayed as successfully treated, the desire for social distance was reduced, there was less willingness to discriminate against them, and belief in treatment's effectiveness increased.

Self-stigma has also been shown to affect self-esteem, which then affects hope, which then affects the quality of life of people with serious mental illnesses. As such, hope should play a central role in recovery (Mashiach-Eizenberg et al., 2013). Narrative Enhancement and Cognitive Therapy (NECT) is an intervention designed to reduce internalized stigma and targets both hope and self-esteem (Yanos et al., 2011). The intervention replaces stigmatizing myths with facts about the illness and recovery which leads to hope in clients and greater levels of self-esteem. This may then reduce susceptibility to internalized stigma.

Stigma has been shown to lead to health inequities (Hatzenbuehler, Phelan, & Link, 2013) prompting calls for change in stigma. Targeting stigma leads to two different agendas. The *services agenda* attempts to remove stigma so the person can seek mental health services while the *rights agenda* tries to replace discrimination that "robs people of rightful opportunities with affirming

attitudes and behavior" (Corrigan, 2016). The former is successful when there is evidence that people with mental illness are seeking services more or becoming better engaged, while the latter is successful when there is an increase in the number of people with mental illnesses in the workforce and receiving reasonable accommodations. The federal government has tackled this issue with landmark legislation such as the Patient Protection and Affordable Care Act of 2010, Mental Health Parity and Addiction Equity Act of 2008, and the Americans with Disabilities Act of 1990. However, protections are not uniform across all subgroups due to "1) explicit language about inclusion and exclusion criteria in the statute or implementation rule, 2) vague statutory language that yields variation in the interpretation about which groups qualify for protection, and 3) incentives created by the legislation that affect specific groups differently" (Cummings, Lucas, and Druss, 2013).

1.5: The History of Mental Illness

Section Learning Objectives

- Describe prehistoric and ancient beliefs about mental illness.
- Describe Greco-Roman thought on mental illness.
- Describe thoughts on mental illness during the Middle Ages.
- Describe thoughts on mental illness during the Renaissance.
- Describe thoughts on mental illness during the 18th and 19th centuries.
- Describe thoughts on mental illness during the 20th and 21st centuries.
- Outline the use of psychoactive drugs throughout time and their impact.
- Outline Freud's theories and approaches to mental illness.
- Describe and provide examples of the various defense mechanisms

As we have seen so far, what is considered abnormal behavior is often dictated by the culture/society a person lives in, and unfortunately, the past has not treated the afflicted very well. In this section, we will examine how past societies viewed and dealt with mental illness.

1.5.1: **Prehistoric and Ancient Beliefs**

Prehistoric cultures often held a supernatural view of abnormal behavior and saw it as the work of evil spirits, demons, gods, or witches who took control of the person. This form of demonic possession was believed to occur when the person engaged in behavior contrary to the religious teachings of the time. Treatment by cave dwellers included a technique called **trephination**, in which a stone instrument known as a *trephine* was used to remove part of the skull, creating an opening. They believed that evil spirits could escape through the hole in the skull, thereby ending the person's mental affliction and returning them to normal behavior. Early Greek, Hebrew, Egyptian, and Chinese cultures used a treatment method called **exorcism** in which evil spirits were cast out through prayer, magic, flogging, starvation, noise-making, or having the person ingest horrible tasting drinks.

1.5.2: **Greco-Roman Thought**

Rejecting the idea of demonic possession, Greek physician, Hippocrates (460-377 B.C.), said that mental disorders were akin to physical disorders and had natural causes. Specifically, he suggested that they arose from *brain pathology*, or head trauma/brain dysfunction or disease, and were also affected by heredity. Hippocrates classified mental disorders into three main categories – melancholia, mania, and phrenitis (brain fever) and gave detailed clinical descriptions of each. He also described four main fluids or **humors** that directed normal functioning and personality – *blood* which arose in the heart, *black bile* arising in the spleen, *yellow bile* or *choler* from the liver, and *phlegm* from the brain. Mental disorders occurred when the humors were in a state of imbalance such as an excess of yellow bile causing frenzy/mania and too much black bile causing melancholia/depression. Hippocrates believed mental illnesses could be treated as any other disorder and focused on the underlying pathology.

Also important was Greek philosopher, Plato (429-347 B.C.), who said that the mentally ill were not responsible for their own actions and so should not be punished. He emphasized the role of social environment and early learning in the development of mental disorders and believed it was the responsibility of the community and their families to care for them in a humane manner using rational discussions. Greek physician, Galen (A.D. 129-199) said mental disorders had either physical or mental causes that included fear, shock, alcoholism, head injuries, adolescence, and changes in menstruation.

In Rome, physician Asclepiades (124-40 BC) and philosopher Cicero (106-43 BC) rejected Hippocrates' idea of the four humors and instead stated that melancholy arises from grief, fear, and rage; not excess black bile. Roman physicians treated mental disorders with massage and warm baths, with the hope that their patients be as comfortable as possible. They practiced the concept of "*contrariis contrarius*", meaning opposite by opposite, and introduced contrasting stimuli to bring about balance in the physical and mental domains. An example would be consuming a cold drink while in a warm bath.

1.5.3: **The Middle Ages – 500 AD to 1500 AD**

The progress made during the time of the Greeks and Romans was quickly reversed during the Middle Ages with the increase in power of the Church and the fall of the Roman Empire. Mental illness was yet again explained as possession by the Devil and methods such as exorcism, flogging, prayer, the touching of relics, chanting, visiting holy sites, and holy water were used to rid the

person of the Devil's influence. In extreme cases, the afflicted were confined, beat, and even executed. Scientific and medical explanations, such as those proposed by Hippocrates, were discarded at this time.

Group hysteria, or **mass madness**, was also seen in which large numbers of people displayed similar symptoms and false beliefs. This included the belief that one was possessed by wolves or other animals and imitated their behavior, called **lycanthropy**, and a mania in which large numbers of people had an uncontrollable desire to dance and jump, called **tarantism**. The latter was believed to have been caused by the bite of the wolf spider, now called the tarantula, and spread quickly from Italy to Germany and other parts of Europe where it was called **Saint Vitus's dance**.

Perhaps the return to supernatural explanations during the Middle Ages makes sense given events of the time. The Black Death or Bubonic Plague had killed up to a third, and according to other estimates almost half, of the population. Famine, war, social oppression, and pestilence were also factors. Death was ever present which led to an epidemic of depression and fear. Nevertheless, near the end of the Middle Ages, mystical explanations for mental illness began to lose favor and government officials regained some of their lost power over nonreligious activities. Science and medicine were once again called upon to explain mental disorders.

1.5.4: The Renaissance – 14th to 16th Centuries

The most noteworthy development in the realm of philosophy during the Renaissance was the rise of **humanism**, or the worldview that emphasizes human welfare and the uniqueness of the individual. This helped continue the decline of supernatural views of mental illness. In the mid to late 1500s, Johann Weyer (1515-1588), a German physician, published his book, *On the Deceits of the Demons*, that rebutted the Church's witch-hunting handbook, the *Malleus Maleficarum*, and argued that many accused of being witches and subsequently imprisoned, tortured, hung, and/or burned at the stake, were mentally disturbed and not possessed by demons or the Devil himself. He believed that like the body, the mind was susceptible to illness. Not surprisingly, the book was met with vehement protest and even banned from the church. It should be noted that these types of acts occurred not only in Europe but also in the United States. The most famous example was the Salem Witch Trials of 1692 in which more than 200 people were accused of practicing witchcraft and 20 were killed.

The number of **asylums**, or places of refuge for the mentally ill where they could receive care, began to rise during the 16th century as the government realized there were far too many people afflicted with mental illness to be left in private homes. Hospitals and monasteries were converted into asylums. Though the intent was benign in the beginning, as they began to overflow patients came to be treated more like animals than people. In 1547, the Bethlem Hospital opened in London with the sole purpose of confining those with mental disorders. Patients were chained up, placed on public display, and often heard crying out in pain. The asylum became a tourist attraction, with sightseers paying a penny to view the more violent patients, and soon was called "Bedlam" by local people; a term that today means "a state of uproar and confusion" (https://www.merriam-webster.com/dictionary/bedlam).

1.5.5: Reform Movement – 18th to 19th Centuries

The rise of the **moral treatment movement** occurred in Europe in the late 18th century and then in the United States in the early 19th century. Its earliest proponent was Phillipe Pinel (1745-1826) who was assigned as the superintendent of la Bicetre, a hospital for mentally ill men in Paris. He emphasized the importance of affording the mentally ill respect, moral guidance, and humane treatment, all while considering their individual, social, and occupational needs. Arguing that the mentally ill were sick people, Pinel ordered that chains be removed, outside exercise be allowed, sunny and well-ventilated rooms replace dungeons, and patients be extended kindness and support. This approach led to considerable improvement for many of the patients, so much so, that several were released.

Following Pinel's lead in England, William Tuke (1732-1822), a Quaker tea merchant, established a pleasant rural estate called the York Retreat. The Quakers believed that all people should be accepted for who they were and treated kindly. At the retreat, patients could work, rest, talk out their problems, and pray (Raad & Makari, 2010). The work of Tuke and others led to the passage of the County Asylums Act of 1845 which required that every county in England and Wales provide asylum to the mentally ill. This was even extended to English colonies such as Canada, India, Australia, and the West Indies as word of the maltreatment of patients at a facility in Kingston, Jamaica spread, leading to an audit of colonial facilities and their policies.

Reform in the United States started with the figure largely considered to be the father of American psychiatry, Benjamin Rush (1745-1813). Rush advocated for the humane treatment of the mentally ill, showing them respect, and even giving them small gifts from time to time. Despite this, his practice included treatments such as bloodletting and purgatives, the invention of the "tranquilizing chair," and a reliance on astrology, showing that even he could not escape from the beliefs of the time.

Due to the rise of the moral treatment movement in both Europe and the United States, asylums became habitable places where those afflicted with mental illness could recover. However, it is often said that the moral treatment movement was a victim of its own success. The number of mental hospitals greatly increased leading to staffing shortages and a lack of funds to support them. Though treating patients humanely was a noble endeavor, it did not work for some and other treatments were needed, though they had not been developed yet. It was also recognized that the approach worked best when the facility had 200 or fewer patients. However, waves of immigrants arriving in the U.S. after the Civil War were overwhelming the facilities, with patient counts soaring to 1,000 or more. Prejudice against the new arrivals led to discriminatory practices in which immigrants were not afforded moral treatments provided to native citizens, even when the resources were available to treat them.

Another leader in the moral treatment movement was Dorothea Dix (1802-1887), a New Englander who observed the deplorable conditions suffered by the mentally ill while teaching Sunday school to female prisoners. She instigated the **mental hygiene movement**, which focused on the physical well-being of patients. Over the span of 40 years, from 1841 to 1881, she motivated people and state legislators to do something about this injustice and raised millions of dollars to build over 30 more appropriate mental hospitals and improve others. Her efforts even extended beyond the U.S. to Canada and Scotland.

Finally, in 1908 Clifford Beers (1876-1943) published his book, *A Mind that Found Itself*, in which he described his personal struggle with bipolar disorder and the "cruel and inhumane treatment people with mental illnesses received. He witnessed and experienced horrific abuse at the hands of his caretakers. At one point during his institutionalization, he was placed in a straightjacket for 21 consecutive nights." (http://www.mentalhealthamerica.net/our-history). His story aroused sympathy in the public and led him to found the National Committee for Mental Hygiene, known today as Mental Health America, which provides education about mental illness and the need to treat these people with dignity. Today, MHA has over 200 affiliates in 41 states and employs 6,500 affiliate staff and over 10,000 volunteers.

For more information on MHA, please visit: http://www.mentalhealthamerica.net/

1.5.6: 20th – 21st Centuries

The decline of the moral treatment approach in the late 19th century led to the rise of two competing perspectives – the biological or somatogenic perspective and the psychological or psychogenic perspective.

1.5.6.1: Biological or Somatogenic Perspective

Recall that Greek physicians Hippocrates and Galen said that mental disorders were akin to physical disorders and had natural causes. Though the idea fell into oblivion for several centuries it re-emerged in the late 19th century for two reasons. First, German psychiatrist, Emil Kraepelin (1856-1926), discovered that symptoms occurred regularly in clusters which he called **syndromes**. These syndromes represented a unique mental disorder with its own cause, course, and prognosis. In 1883 he published his textbook, *Compendium der Psychiatrie* (Textboook of Psychiatry), and described a system for classifying mental disorders that became the basis of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders (DSM)* that is currently in its 5th edition (published in 2013).

Secondly, in 1825, the behavioral and cognitive symptoms of advanced syphilis were identified to include delusions (e.g., false beliefs that everyone is plotting against you or that you are God), and were termed *general paresis* by French physician A.L.J. Bayle. In 1897, Viennese psychiatrist Richard von Krafft-Ebbing injected patients with general paresis with matter from syphilis spores and noted that none of the patients developed symptoms of syphilis, indicating they must have been previously exposed and were now immune. This led to the conclusion that syphilis (a bacterial infection) was the cause of the general paresis. In 1906, August von Wassermann developed a blood test for syphilis and in 1917 a cure was stumbled upon. Julius von Wagner-Jauregg noticed that patients with general paresis who contracted malaria recovered from their symptoms. To test this hypothesis, he injected nine patients with blood from a soldier afflicted with malaria. Three of patients fully recovered while three others showed great improvement in their paretic symptoms. The high fever caused by malaria burned out the syphilis bacteria. Hospitals in the United States began incorporating this new cure for paresis into their treatment approach by 1925.

Also noteworthy was the work of American psychiatrist John P. Grey. Appointed as superintendent of the Utica State Hospital in New York, Grey asserted that insanity always had a physical cause. As such, the mentally ill should be seen as physically ill and treated with rest, proper room temperature and ventilation, and a proper diet.

The 1930s also saw the use of electric shock as a treatment method, which was stumbled upon accidentally by Benjamin Franklin while experimenting with electricity in the early 18th century. He noticed that after experiencing a severe shock his memories had changed and in published work, suggested physicians study electric shock as a treatment for melancholia.

Beginning in the 1950s, psychiatric or psychotropic drugs were used for the treatment of mental illness and made an immediate impact. Though drugs alone cannot cure mental illness, they can improve symptoms. Classes of psychiatric drugs include antidepressants used to treat depression and anxiety, mood-stabilizing medications to treat bipolar disorder, antipsychotic drugs to treat schizophrenia and other psychotic disorders, and anti-anxiety drugs used to treat generalized anxiety disorder or panic disorder

(Source: https://www.nimh.nih.gov/health/topics/mental-health-edications/index.shtml).

Frank (2006) found that by 1996, psychotropic drugs were used in 77% of mental health cases and spending on these drugs to treat mental disorders grew from $2.8 billion in 1987 to about $18 billion in 2001 (Coffey et al., 2000; Mark et al., 2005), representing a greater than sixfold increase. The largest classes of psychotropic drugs are anti-psychotic and anti-depressant medications followed closely by anti-anxiety medications. Frank, Conti, and Goldman (2005) point out, “The expansion of insurance coverage for prescription drugs, the introduction and diffusion of managed behavioral health care techniques, and the conduct of the pharmaceutical industry in promoting their products all have influenced how psychotropic drugs are used and how much is spent on them.” Is it possible then that we are overprescribing these mediations? Davey (2014) provides ten reasons why this may be so to include individuals believing that recovery is out of their hands but instead in the hands of their doctors, increased risk of relapse, drug companies causing the “medicalization of perfectly normal emotional processes, such as bereavement” to ensure their own survival, side effects, and a failure to change the way the way the person thinks or the socioeconomic environments that may be the cause of the disorder. For more on this article, please see: https://www.psychologytoday.com/blog/why-we-worry/201401/overprescribing-drugs-treat-mental-health-problems. Smith (2012) echoed similar sentiments in an article on inappropriate prescribing and cites the approval of Prozac by the Food and Drug Administration (FDA) in 1987 as when the issue began and the overmedication/overdiagnosis of children with ADHD as a more recent example.

A result of the use of psychiatric drugs was **deinstitutionalization** or the release of patients from mental health facilities. This shifted resources from inpatient to outpatient care and placed the spotlight back on the biological or somatogenic perspective. Today, when people with severe mental illness do need inpatient care, it is typically in the form of short-term hospitalization.

1.5.6.2: Psychological or Psychogenic Perspective

The **psychological or psychogenic perspective** states that emotional or psychological factors are the cause of mental disorders and represented a challenge to the biological perspective.

1.5.6.2.1: The History of Hypnosis

This perspective had a long history but did not gain favor until the work of Viennese physician Franz Anton Mesmer (1734-1815). Influenced heavily by Newton’s theory of gravity, he believed that the planets also affected the human body through the force of animal magnetism and that all people had a universal magnetic fluid that determined how healthy they were. He demonstrated the usefulness of his approach when he cured Franzl Oesterline, a 27-year old woman experiencing what he described as a convulsive malady. Mesmer used a magnet to disrupt the gravitational tides that were affecting his patient and produced a sensation of the magnetic fluid draining from her body. This removed the illness from her body and produced a near instantaneous recovery. In reality, the patient was placed in a trancelike state which made her highly suggestible. With other patients, Mesmer would have them sit in a darkened room filled with soothing music, into which he would enter dressed in a colorful robe and passed from person to person touching the afflicted area of their body with his hand or a special rod/wand. He successfully cured deafness, paralysis, loss of bodily feeling, convulsions, menstrual difficulties, and blindness.

His approach gained him celebrity status as he demonstrated it at the courts of English nobility. The medical community was hardly impressed. A royal commission was formed to investigate his technique but could not find any proof for his theory of animal magnetism. Though he was able to cure patients when they touched his “magnetized” tree, the result was the same when “non-magnetized” trees were touched. As such, Mesmer was deemed a charlatan and forced to leave Paris. His technique was called **mesmerism,** and today we know it as an early form of hypnosis.

The psychological perspective gained popularity after two physicians practicing in the city of Nancy in France discovered that they could induce the symptoms of hysteria in perfectly healthy patients through hypnosis and then remove the symptoms in the same way. The work of Hippolyte-Marie Bernheim (1840-1919) and Ambroise-Auguste Liebault (1823-1904) came to be part of what was called the Nancy School and showed that hysteria was nothing more than a form of self-hypnosis. In Paris, this view was challenged by Jean Charcot (1825-1893) who stated that hysteria was caused by degenerative brain changes, reflecting the biological perspective. He was proven wrong and eventually turned to their way of thinking.

The use of hypnosis to treat hysteria was also carried out by fellow Frenchman Pierre Janet (1859-1947), and student of Charcot, who believed that hysteria had psychological, not biological causes. Namely, these included unconscious forces, fixed ideas, and

memory impairments. In Vienna, Josef Breuer (1842-1925) induced hypnosis and had patients speak freely about past events that upset them. Upon waking, he discovered that patients sometimes were free of their symptoms of hysteria. Success was even greater when patients not only recalled forgotten memories but also relieved them emotionally. He called this the **cathartic method** and our use of the word *catharsis* today indicates a purging or release, in this case, of pent-up emotion. Sigmund Freud's development of psychoanalysis followed on the heels of the work of Breuer, and others who came before him.

1.5.6.2.2: Psychodynamic Theory

In 1895, the book, *Studies on Hysteria*, was published by Josef Breuer (1842-1925) and Sigmund Freud (1856-1939), and marked the birth of psychoanalysis, though Freud did not use this actual term until a year later. The book published several case studies, including that of Anna O., born February 27, 1859, in Vienna to Jewish parents Siegmund and Recha Pappenheim, strict Orthodox adherents considered millionaires at the time. Bertha, known in published case studies as Anna O., was expected to complete the formal education of a girl in the upper middle class which included foreign language, religion, horseback riding, needlepoint, and piano. She felt confined and suffocated in this life and took to a fantasy world she called her "private theater." Anna also developed hysteria which included symptoms of memory loss, paralysis, disturbed eye movements, reduced speech, nausea, and mental deterioration. Her symptoms appeared as she cared for her dying father and her mother called on Breuer to diagnosis her condition (note that Freud never actually treated her). Hypnosis was used at first and relieved her symptoms, as it had done for many patients (See Chapter 1). Breuer made daily visits and allowed her to share stories from her private theater which she came to call "talking cure" or "chimney sweeping." Many of the stories she shared were actually thoughts or events she found troubling and reliving them helped to relieve or eliminate the symptoms. Breuer's wife, Mathilde, became jealous of her husband's relationship with the young girl, leading Breuer to terminate treatment in the June of 1882 before Anna had fully recovered. She relapsed and was admitted to Bellevue Sanatorium on July 1, eventually being released in October of the same year. With time, Anna O. did recover from her hysteria and went on to become a prominent member of the Jewish Community, involving herself in social work, volunteering at soup kitchens, and becoming 'House Mother' at an orphanage for Jewish girls in 1895. Bertha (Anna O.) became involved in the German Feminist movement, and in 1904 founded the League of Jewish Women. She published many short stories; a play called *Women's Rights*, in which she criticized the economic and sexual exploitation of women, and wrote a book in 1900 called *The Jewish Problem in Galicia*, in which she blamed the poverty of the Jews of Eastern Europe on their lack of education. In 1935 she was diagnosed with a tumor and was summoned by the Gestapo in 1936 to explain anti-Hitler statements she had allegedly made. She died shortly after this interrogation on May 28, 1936. Freud considered the talking cure of Anna O. to be the origin of psychoanalytic therapy and what would come to be called the cathartic method.

For more on Anna O., please see:

https://www.psychologytoday.com/blog/freuds-patients-serial/201201/bertha-pappenheim-1859-1936

1.5.6.2.3: The Structure of Personality.

Freud's psychoanalysis was unique in the history of psychology because it did not arise within universities as most of the major school of thought in our history did, but from medicine and psychiatry, it dealt with psychopathology and examined the unconscious. Freud believed that consciousness had three levels – 1) **consciousness** which was the seat of our awareness, 2) **preconscious** that included all of our sensations, thoughts, memories, and feelings, and 3) the **unconscious** which was not available to us. The contents of the unconscious could move from the unconscious to preconscious, but to do so, it had to pass a Gate Keeper. Content that was turned away was said to be repressed by Freud.

According to Freud, our personality has three parts – the id, superego, and ego, and from these, our behavior arises. First, the **id** is the impulsive part that expresses our sexual and aggressive instincts. It is present at birth, completely unconscious, and operates on the *pleasure principle*, resulting in our selfishly seeking immediate gratification of our needs no matter what the cost. The second part of personality emerges after birth with early formative experiences and is called the **ego**. The ego attempts to mediate the desires of the id against the demands of reality, and eventually the moral limitations or guidelines of the superego. It operates on the *reality principle*, or an awareness of the need to adjust behavior to meet the demands of our environment. The last part of personality to develop is the **superego** which represents society's expectations, moral standards, rules, and represents our conscience. It leads us to adopt our parent's values as we come to realize that many of the id's impulses are unacceptable. Still, we violate these values at times which lead to feelings of guilt. The superego is partly conscious but mostly unconscious. The three parts of personality generally work together well and compromise, leading to a healthy personality, but if conflicts among these components are not resolved, intrapsychic conflicts can arise and lead to mental disorders.

1.5.6.2.4: The Development of Personality.

Freud also proposed that personality develops over the course of five distinct stages (oral, anal, phallic, latency, genital), in which the libido is focused on different parts of the body. First, **libido** is the psychic energy that drives a person to pleasurable thoughts and behaviors. Our life instincts, or **Eros**, are manifested through it and are the creative forces that sustain life. They include hunger, thirst, self-preservation, and sex. In contrast, **Thanatos**, or our death instinct, is either directed inward as in the case of suicide and masochism or outward via hatred and aggression. Both types of instincts are sources of stimulation in the body and create a state of tension which is unpleasant, thereby motivating us to reduce them. Consider hunger, and the associated rumbling of our stomach, fatigue, lack of energy, etc., that motivates us to find and eat food. If we are angry at someone we may engage in physical or relational aggression to alleviate this stimulation.

Freud's psychosexual stages of personality development are listed below. Freud proposed that a person may become **fixated** at any stage, meaning they become stuck, thereby affecting later development and possibly leading to abnormal functioning, or psychopathology.

1. **Oral Stage** – Beginning at birth and lasting to 24 months, the libido is focused on the mouth and sexual tension is relieved by sucking and swallowing at first, and then later by chewing and biting as baby teeth come in. Fixation is linked to a lack of confidence, argumentativeness, and sarcasm.
2. **Anal Stage** – Lasting from 2-3 years, the libido is focused on the anus as toilet training occurs. If parents are too lenient children may become messy or unorganized. If parents are too strict, children may become obstinate, stingy, or orderly.
3. **Phallic Stage** – Occurring from about age 3 to 5-6 years, the libido is focused on the genitals. The Oedipus complex develops in boys and results in the son falling in love with his mother while fearing that his father will find out and castrate him. Meanwhile, girls fall in love with the father and fear that their mother will find out, called the Electra complex. A fixation at this stage may result in low self-esteem, feelings of worthlessness, and shyness.
4. **Latency Stage** – From 6-12 years of age, children lose interest in sexual behavior and boys play with boys and girls with girls. Neither sex pays much attention to the opposite sex.
5. **Genital Stage** – Beginning at puberty, sexual impulses reawaken and unfulfilled desires from infancy and childhood can be satisfied with sex.

1.5.6.2.5: Defense Mechanisms.

The ego has a challenging job to fulfill, balancing both the will of the id and the superego, and the overwhelming anxiety and panic this creates. **Defense mechanisms** are in place to protect us from this pain but are considered maladaptive if they are misused and become our primary way of dealing with stress. They protect us from anxiety and operate unconsciously, also distorting reality. Defense mechanisms include the following:

- **Repression** – when unacceptable ideas, wishes, desires, or memories are blocked from consciousness such as forgetting a horrific car accident that you caused. Eventually, though, it must be dealt with or else the repressed memory can cause problems later in life.
- **Reaction formation** – When an impulse is repressed and then expressed by its opposite. As an example, if we are angry with our boss but cannot lash out at him/her, we may be overly friendly instead. Another example is having lustful thoughts about a coworker that you cannot express because you are married, and so you are mean to this person.
- **Displacement** – When we satisfy an impulse with a different object because focusing on the primary object may get us in trouble. A classic example is taking out your frustration with your boss on your wife and/or kids when you get home. If we lash out at our boss we could be fired. The substitute target is less dangerous than the primary target.
- **Projection** – When we attribute threatening desires or unacceptable motives to others. An example is when we do not have the skills necessary to complete a task but we blame the other members of our group for being incompetent and unreliable. Another example is projecting your feelings of love toward your therapist onto your therapist, believing he/she is in love with you.
- **Sublimation** – When we find a socially acceptable way to express a desire. If we are stressed out or upset, we may go to the gym and box or lift weights. A person who desires to cut things may become a surgeon.
- **Denial** – Sometimes life is so hard all we can do is deny how bad it is. An example is denying a diagnosis of lung cancer given by your doctor.
- **Identification** – this is when we find someone who has found a socially acceptable way to satisfy their unconscious wishes and desires and we model that behavior.
- **Regression** – When we move from a mature behavior to one that is infantile in nature. If your significant other is nagging you, you might regress and point your hands over your ears and say, "La la la la la la la…"

- **Rationalization** – When we offer well thought out reasons for why we did what we did but in reality, these are not the real reason. Students sometimes rationalize not doing well in a class by stating that they really are not interested in the subject or saying the instructor writes impossible to pass tests when in reality they are not putting enough effort into learning the material.
- **Intellectualization**– When we avoid emotion by focusing on intellectual aspects of a situation such as ignoring the sadness we are feeling after the death of our mother by focusing on planning the funeral.

For more on defense mechanisms, please visit:

https://www.psychologytoday.com/blog/fulfillment-any-age/201110/the-essential-guide-defense-mechanisms

1.5.6.2.6: **Psychodynamic Techniques.**

Freud used three primary assessment techniques as part of **psychoanalysis**, or psychoanalytic therapy, to understand the personalities of his patients and to expose repressed material, which included free association, transference, and dream analysis. First, **free association** involves the patient describing whatever comes to mind during the session. The patient continues but always reaches a point when he/she cannot or will not proceed any further. The patient might change the subject, stop talking, or lose his/her train of thought. Freud said this was **resistance** and revealed where issues were.

Second, **transference** is the process through which patients transfer to the therapist attitudes he/she held during childhood. They may be positive and include friendly, affectionate feelings, or negative, and include hostile and angry feelings. The goal of therapy is to wean patients from their childlike dependency on the therapist.

Finally, Freud used **dream analysis** to understand a person's innermost wishes. The content of dreams include the person's actual retelling of the dreams called **manifest content**, and the hidden or symbolic meaning called **latent content**. In terms of the latter, some symbols are linked to the person specifically while others are common to all people.

1.5.6.2.7: **Evaluating Psychodynamic Theory.**

Freud's psychodynamic theory has made a lasting impact on the field of psychology but also has been criticized heavily. First, most of Freud's observations were made in an unsystematic, uncontrolled way and he relied on the case study method. Second, the participants in his studies were not representative of the larger body of people whom he tried to generalize to and he really based his theory on a few patients. Third, he relied solely on the reports of his patients and sought out no observer reports. Fourth, it is difficult to empirically study psychodynamic principles since most operate unconsciously. This begs the question of how can we really know that they exist. Finally, psychoanalytic treatment is expensive and time-consuming and since Freud's time, drug therapies have become more popular and successful. Still, the work of Sigmund Freud raised awareness about the role the unconscious plays in both normal and abnormal behavior and he developed useful therapeutic tools for clinicians.

By the end of the 19th century, it had become evident that mental disorders were caused by a combination of biological and psychological factors and the investigation of how they develop began. Today, rather than arguing for a purely biological or psychological approach to understanding mental disorders we focus on a more integrative multidimensional approach. This contemporary approach is the focus of Chapter 2.

Chapter Recap

In Chapter 1, we undertook a fairly lengthy discussion of what abnormal behavior is by first looking at what normal behavior is. What emerged was a general set of guidelines focused on mental disorders as causing dysfunction, distress, deviance, and at times, being dangerous for the afflicted and others around him/her. We acknowledged that mental illness is stigmatized in our society and provided a basis for why this occurs and what to do about it. We introduced the various members of the mental health team and defined several key terms including occurrence, cause, course, prognosis, and treatment. We concluded with a lengthy discussion of the history of mental illness. It is with this foundation in mind that we move to examine contemporary models of mental disorders in Chapter 2.

CHAPTER OVERVIEW

2: Contemporary Models of Abnormal Psychology

Learning Objectives

- Differentiate uni- and multi-dimensional models of abnormality.
- Describe how the biological model explains mental illness.
- Describe how psychological perspectives explain mental illness.
- Describe how the sociocultural model explains mental illness.

In Chapter 2, we will discuss three models of abnormal behavior to include the biological, psychological, and sociocultural models. Each is unique in its own right and no one model can account for all aspects of abnormality. Hence, a multi-dimensional and not a uni-dimensional model will be advocated for.

2.1: Uni- vs. Multi-Dimensional Models

Section Learning Objectives

- Define the uni-dimensional model.
- Explain the need for a multi-dimensional model of abnormality.
- Define model.
- List and describe the three models of abnormality.

2.1.1: Uni-Dimensional Models

In order to effectively treat a mental disorder, it is helpful to understand its cause. This could be a single factor such as a chemical imbalance in the brain, relationship with a parent, socioeconomic status (SES), a fearful event encountered during middle childhood, or the way in which the individual copes with life's stressors. This single factor explanation is called a **uni-dimensional model**. The problem with this approach is that mental disorders are not typically caused by a solitary factor, but instead, they are caused by multiple factors. Admittedly, single factors do emerge during the course of the person's life, but as they arise they become part of the individual and in time, the cause of the person's disorder is due to all of these individual factors.

2.1.2: **Multi-Dimensional**

So, in reality, it is better to subscribe to a **multi-dimensional model** that integrates multiple causes of psychopathology and affirms that each cause comes to affect other causes over time. Uni-dimensional models alone are too simplistic to fully understand the etiology of something as complex as mental disorders.

Before introducing the main models subscribed to today, it is important to understand what a model is. In a general sense, a **model** is defined as a representation or imitation of an object (dictionary.com). Models help mental health professionals understand mental illness since disorders such as depression cannot be touched or experienced firsthand. To be considered distinct from other conditions, a mental illness must have its own set of symptoms. But as you will see, the individual does not have to present with the entire range of symptoms to be diagnosed with major depressive disorder, schizophrenia, avoidant personality disorder, or illness anxiety disorder. Five out of nine symptoms may be enough to diagnose a disorder, for example. There will be some variability in terms of what symptoms the afflicted displays, but in general all people with a specific mental disorder have symptoms from that group. We can also ask the patient probing questions, seek information from family members, examine medical records, and in time, organize and process all of this information to better understand the person's condition and potential causes. Models aid us with doing all of this but we must be cautious to remember that the model is a starting point for the researcher, and due to this, determine what causes might be investigated, at the exclusion of other causes. Often times, proponents of a given model find themselves in disagreement with proponents of other models. All forget that there is no one model that completely explains human behavior, or in this case, abnormal behavior and so each model contributes in its own way. So what are the models we will examine in this chapter?

- **Biological** – Includes genetics, chemical imbalances in the brain, the functioning of the nervous system, etc.
- **Psychological** – includes learning, personality, stress, cognition, self-efficacy, and early life experiences. We will examine several perspectives that make up the psychological model to include psychodynamic, behavioral, cognitive, and humanistic-existential.
- **Sociocultural** – includes factors such as one's gender, religious orientation, race, ethnicity, and culture, for example.

2.2: The Biological Model

Section Learning Objectives

- Describe how communication in the nervous system occurs.
- List the parts of the nervous system.
- Describe the structure of the neuron and all key parts.
- Outline how neural transmission occurs.
- Identify and define important neurotransmitters.
- List the major structures of the brain.
- Clarify how specific areas of the brain are involved in mental illness.
- Describe the role of genes in mental illness.
- Describe the role of hormonal imbalances in mental illness.
- Describe commonly used treatments for mental illness.
- Evaluate the usefulness of the biological model.

Proponents of the biological model view mental illness as being a result of a malfunction in the body to include issues with brain anatomy or chemistry. As such, we will need to establish a foundation for how communication in the nervous system occurs, what the parts of the nervous system are, what a neuron is and its structure, how neural transmission occurs, and what the parts of the brain are. While doing this, we will identify areas of concern for psychologists focused on the treatment of mental disorders.

2.2.0.1: **Brain Structure and Chemistry**

2.2.0.1: **Communication in the Nervous System**

To really understand brain structure and chemistry, it is a good idea to understand how communication occurs within the nervous system. Simply:

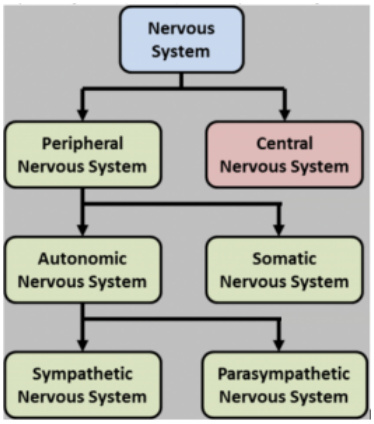
1. Receptor cells in each of the five sensory systems detect energy.
2. This information is passed to the nervous system due to the process of transduction and through sensory or afferent neurons, which are part of the peripheral nervous system.
3. The information is received by brain structures (central nervous system) and perception occurs.
4. Once the information has been interpreted, commands are sent out, telling the body how to respond, also via the peripheral nervous system.

Please note that we will not cover this process in full, but just the parts relevant to our topic of psychopathology.

2.2.0.1: **The Nervous System**

The nervous system consists of two main parts – the central and peripheral nervous systems. The **central nervous system(CNS)** is the control center for the nervous system which receives, processes, interprets, and stores incoming sensory information. It consists of the brain and spinal cord. The **peripheral nervous system** consists of everything outside the brain and spinal cord. It handles the CNS's input and output and divides into the somatic and autonomic nervous systems. The **somatic nervous system** allows for voluntary movement by controlling the skeletal muscles and it carries sensory information to the CNS. The **autonomic nervous system** regulates the functioning of blood vessels, glands, and internal organs such as the bladder, stomach, and heart. It consists of sympathetic and parasympathetic nervous systems. The **sympathetic nervous system** is involved when a person is intensely aroused. It provides the strength to fight back or to flee (fight-or-flight response). Eventually, the response brought about by the sympathetic nervous system must end so the **parasympathetic nervous system** kicks in to calm the body.

Figure 2.1. The Structure of the Nervous System



2.2.0.1: The Neuron

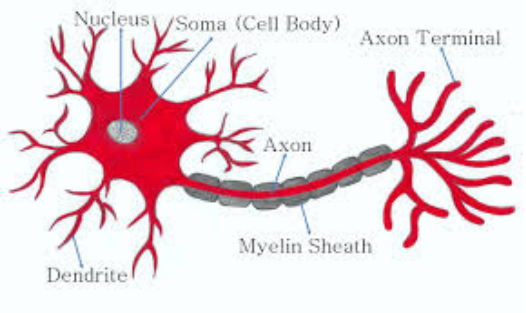
The fundamental unit of the nervous system is the neuron, or nerve cell (See Figure 2.3). It has several structures in common with all cells in the body. The **nucleus** is the control center of the body and the **soma** is the cell body. In terms of structures that make it different, these focus on the ability of a neuron to send and receive information. The **axon** sends signals/information through the neuron while the **dendrites** receive information from neighboring neurons and look like little trees. Notice the *s* on the end of dendrite and that axon has no such letter. In other words, there are lots of dendrites but only one axon. Also of importance to the neuron is the **myelin sheath** or the white, fatty covering which: 1) provides insulation so that signals from adjacent neurons do not affect one another and, 2) increases the speed at which signals are transmitted. The **axon terminals** are the end of the axon where the electrical impulse becomes a chemical message and is released into the **synaptic cleft** which is the space between neurons.

Though not neurons, **glial cells** play an important part in helping the nervous system to be the efficient machine that it is. Glial cells are support cells in the nervous system that serve five main functions.

1. They act as a glue and hold the neuron in place.
2. They form the myelin sheath.
3. They provide nourishment for the cell.
4. They remove waste products.
5. They protect the neuron from harmful substances.

Finally, **nerves** are a group of axons bundled together like wires in an electrical cable.

Figure 2.2. The Structure of the Neuron



2.2.0.1: Neural Transmission

Transducers or receptor cells in the major organs of our five sensory systems – vision (the eyes), hearing (the ears), smell (the nose), touch (the skin), and taste (the tongue) – convert the physical energy that they detect or sense, and send it to the brain via the neural impulse. How so? We will cover this process in three parts.

Part 1. The Neural Impulse

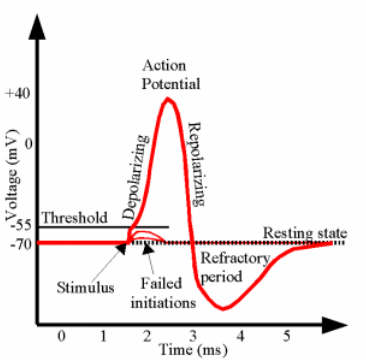
- Step 1 – Neurons waiting to fire are said to be in **resting potential** and to be **polarized** (meaning they have a negative charge inside the neuron and a positive charge outside).

- Step 2 – If adequately stimulated, the neuron experiences an **action potential** and becomes **depolarized**. When this occurs, ion gated channels open allowing positively charged Sodium (Na) ions to enter. This shifts the polarity to positive on the inside and negative outside.
- Step 3 – Once the action potential passes from one segment of the axon to the next, the previous segment begins to **repolarize**. This occurs because the Na channels close and Potassium (K) channels open. K has a positive charge and so the neuron becomes negative again on the inside and positive on the outside.
- Step 4 – After the neuron fires, it will not fire again no matter how much stimulation it receives. This is called the **absolute refractory period**.
- Step 5 – After a short period of time, the neuron can fire again, but needs greater than normal levels of stimulation to do so. This is called the **relative refractory period**.
- Step 6 – Please note that the process is cyclical. Once the relative refractory period has passed the neuron returns to its resting potential.

Part 2. The Action Potential

Let's look at the electrical portion of the process in another way and add some detail.

Figure 2.3. The Action Potential



- Recall that a neuron is normally at resting potential and polarized. The charge inside is -70mV at rest.
- If it receives sufficient stimulation meaning that the polarity inside the neuron rises from -70 mV to -55mV defined as the **threshold of excitation**, the neuron will **fire** or send an electrical impulse down the length of the axon (the action potential or depolarization). It should be noted that it either hits -55mV and fires or it does not. This is the **all-or-nothing principle**. The threshold must be reached.
- Once the electrical impulse has passed from one segment of the axon to the next, the neuron begins the process of resetting called repolarization.
- During repolarization, the neuron will not fire no matter how much stimulation it receives. This is called absolute refractory period.
- The neuron next moves into relative refractory period meaning it can fire, but needs greater than normal levels of stimulation. Notice how the line has dropped below -70mV. Hence, to reach -55mV and fire, it will need more than the normal gain of +15mV (-70 to -55 mV).
- And then it returns to resting potential, as you saw in Figure 2.3

Ions are charged particles found both inside and outside the neuron. It is positively charged Sodium (Na) ions that cause the neuron to depolarize and fire and positively charged Potassium (K) ions that exit and return the neuron to a polarized state.

Part 3. The Synapse

The electrical portion of the neural impulse is just the start. The actual code passes from one neuron to another in a chemical form called a **neurotransmitter**. The point where this occurs is called the **synapse**. The synapse consists of three parts – the axon *terminals* of the sending neuron (presynaptic neuron); the *space* in between called the **synaptic cleft, space**, or **gap**; and the *dendrite* of the receiving neuron (postsynaptic neuron). Once the electrical impulse reaches the end of the axon, called the **axon terminal**, it stimulates synaptic vesicles or neurotransmitter sacs to release the neurotransmitter. Neurotransmitters will only bind to their specific **receptor sites**, much like a key will only fit into the lock it was designed for. You might say neurotransmitters are part of a lock-and-key system. What happens to the neurotransmitters that do not bind to a receptor site? They might go through

reuptake which is a process in which the presynaptic neuron takes back excess neurotransmitters in the synaptic space for future use or **enzymatic degradation** when enzymes destroy excess neurotransmitters in the synaptic space.

2.2.0.1: Neurotransmitters

What exactly are some of the neurotransmitters which are so critical for neural transmission, and are important to our discussion of psychopathology?

- **Dopamine** – controls voluntary movements and is associated with the reward mechanism in the brain
- **Serotonin** – controls pain, sleep cycle, and digestion; leads to a stable mood and so low levels leads to depression
- **Norepinephrine** – increases the heart rate and blood pressure and regulates mood
- **GABA** – an inhibitory neurotransmitter responsible for blocking the signals of excitatory neurotransmitters responsible for anxiety and panic.
- **Glutamate** – an excitatory neurotransmitter associated with learning and memory

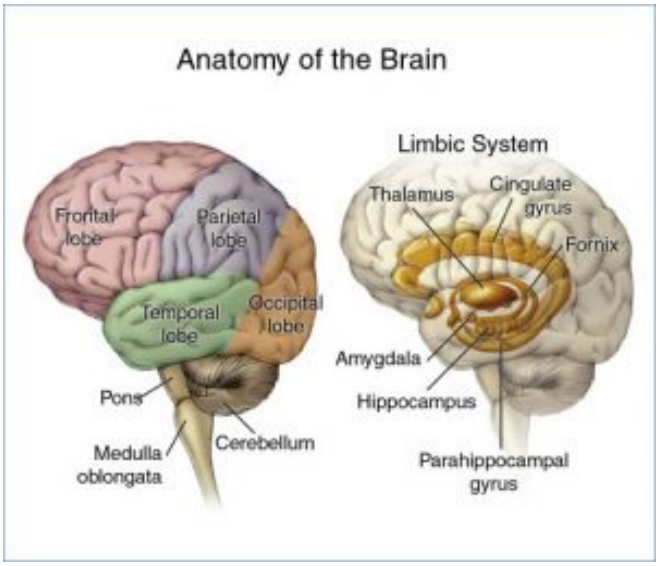
The critical thing to understand here is that there is a belief in the realm of mental health that chemical imbalances are responsible for many mental disorders. Chief among these are neurotransmitter imbalances. For instance, people with Seasonal Affective Disorder (SAD) have difficulty regulating serotonin. More on this throughout the book as we discuss each disorder.

2.2.1: The Brain

The central nervous system consists of the brain and spinal cord; the former we will discuss briefly and in terms of key structures which include:

- **Medulla** – regulates breathing, heart rate, and blood pressure
- **Pons** – acts as a bridge connecting the cerebellum and medulla and helps to transfer messages between different parts of the brain and spinal cord.
- **Reticular formation** – responsible for alertness and attention
- **Cerebellum** – involved in our sense of balance and for coordinating the body's muscles so that movement is smooth and precise. Involved in the learning of certain kinds of simple responses and acquired reflexes.
- **Thalamus** – major sensory relay center for all senses except smell.
- **Hypothalamus** – involved in drives associated with the survival of both the individual and the species. It regulates temperature by triggering sweating or shivering and controls the complex operations of the autonomic nervous system
- **Amygdala –** responsible for evaluating sensory information and quickly determining its emotional importance
- **Hippocampus** – our "gateway" to memory. Allows us to form spatial memories so that we can accurately navigate through our environment and helps us to form new memories (involved in memory consolidation)
- The **cerebrum** has four distinct regions in each cerebral hemisphere. First, the **frontal lobe** contains the motor cortex which issues orders to the muscles of the body that produce voluntary movement. The frontal lobe is also involved in emotion and in the ability to make plans, think creatively, and take initiative. The **parietal lobe** contains the somatosensory cortex and receives information about pressure, pain, touch, and temperature from sense receptors in the skin, muscles, joints, internal organs, and taste buds. The occipital lobe contains the **visual** cortex and receives and processes visual information. Finally, the temporal lobe is involved in memory, perception, and emotion. It contains the **auditory** cortex which processes sound.

Figure 2.4. Anatomy of the Brain



Of course, this is not an exhaustive list of structures found in the brain but gives you a pretty good idea of function and which structures help to support those functions. What is important to mental health professionals is that for some disorders, specific areas of the brain are involved. For instance, individuals with borderline personality disorder have been shown to have structural and functional changes in brain areas associated with impulse control and emotional regulation while imaging studies reveal differences in the frontal cortex and subcortical structures of individuals with OCD.

Check out the following from Harvard Health for more on depression and the brain as a cause:
https://www.health.harvard.edu/mind-and-mood/what-causes-depression

2.2.2: Genes, Hormonal Imbalances, and Viral Infections

2.2.2.1: Genetic Issues and Explanations

DNA, or deoxyribonucleic acid, is our heredity material and is found in the nucleus of each cell packaged in threadlike structures known as *chromosomes*. Most of us have 23 pairs of chromosomes or 46 total. Twenty-two of these pairs are the same in both sexes, but the 23rd pair is called the sex chromosome and differs between males and females. Males have X and Y chromosomes while females have two Xs. According to the Genetics Home Reference website as part of NIH's National Library of Medicine, a *gene* is "the basic physical and functional unit of heredity" (https://ghr.nlm.nih.gov/primer/basics/gene). They act as the instructions to make proteins and it is estimated by the Human Genome Project that we have between 20,000 and 25,000 genes. We all have two copies of each gene and one is inherited from our mother and one from our father.

Recent research has discovered that autism, ADHD, bipolar disorder, major depression, and schizophrenia all share genetic roots. They "were more likely to have suspect genetic variation at the same four chromosomal sites. These included risk versions of two genes that regulate the flow of calcium into cells." For more on this development, please check out the article at: https://www.nimh.nih.gov/news/science-news/2013/five-major-mental-disorders-share-genetic-roots.shtml. Likewise, twin and family studies have shown that people with first-degree relatives with OCD are at higher risk of developing the disorder themselves. The same is true of most mental disorders. Indeed, it is presently believed that genetic factors contribute to all mental disorders but typically account for less than half of the explanation. Moreover, most mental disorders are linked to abnormalities in many genes, rather than just one; that is, most are **polygenetic**.

Moreover, there are important gene-environment interactions that are unique for every person (even twins) which help to explain why some people with a genetic predisposition toward a certain disorder develop that disorder and others do not (e.g., why one identical twin may develop schizophrenia but the other does not). The **diathesis-stress model** posits that people can inherit tendencies or vulnerabilities to express certain traits, behaviors, or disorders, which may then be activated under certain environmental conditions like stress (e.g., abuse, traumatic events). However, it is also important to note that certain protective factors (like being raised in a consistent, loving, supportive environment) may modify the response to stress and thereby help to protect individuals against mental disorders.

For more on the role of genes in the development of mental illness, check out this article from Psychology Today:

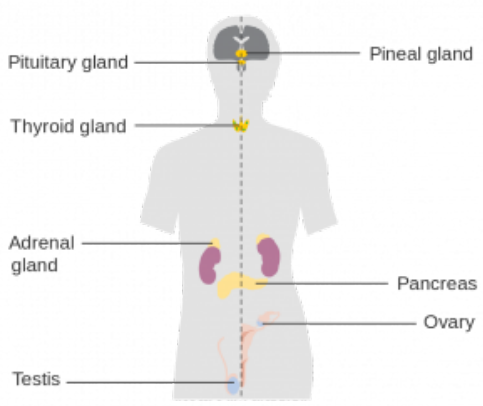
https://www.psychologytoday.com/blog/saving-normal/201604/what-you-need-know-about-the-genetics-mental-disorders

2.2.2.2: Hormonal Imbalances

The body has two coordinating and integrating systems in the body. The nervous system is one and the endocrine system is the second. The main difference between these two systems is in terms of the speed with which they act. The nervous system moves quickly with nerve impulses moving in a few hundredths of a second. The endocrine system moves slowly with hormones, released by endocrine glands, taking seconds, or even minutes, to reach their target. Hormones are important to psychologists because they organize the nervous system and body tissues at certain stages of development and activate behaviors such as alertness or sleepiness, sexual behavior, concentration, aggressiveness, reaction to stress, a desire for companionship.

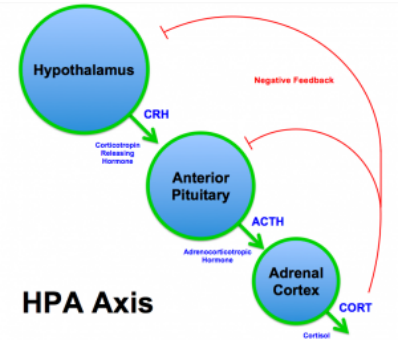
The **pituitary gland** is the "master gland" which regulates other endocrine glands. It influences blood pressure, thirst, contractions of the uterus during childbirth, milk production, sexual behavior and interest, body growth, the amount of water in the body's cells, and other functions as well. The **pineal gland** produces melatonin which helps regulate the sleep-wake cycle and other circadian rhythms. Overproduction of the hormone melatonin can lead to Seasonal Affective Disorder (a specific type of Major Depressive Disorder). The **thyroid gland** produces thyroxin which facilitates energy, metabolism, and growth. Hypothyroidism is a condition in which the thyroid glands become underactive and this condition can produce symptoms of depression. In contrast, hyperthyroidism is a condition in which the thyroid glands becomes overactive and this condition can produce symptoms of mania. Therefore it is important for individuals experiencing these symptoms to have their thyroid checked, because conventional treatments for depression and mania will not correct the problem with the thyroid, and will therefore not resolve the symptoms. Rather, individuals with these conditions need to be treated with thyroid medications. Also of key importance to mental health professionals are the **adrenal glands** which are located on top of the kidneys, and release *cortisol* which helps the body deal with stress. However, chronically, elevated levels of cortisol can lead to increased weight gain, interfere with learning and memory, decrease the immune response, reduce bone density, increase cholesterol, and increase the risk of depression.

Figure 2.5. Hormone Systems



The **Hypothalamic-Pituitary-Adrenal-Cortical Axis (HPA Axis)** is the connection between the hypothalamus, pituitary glands, and adrenal glands. Specifically, the hypothalamus releases corticotropin-releasing factor (CRF) which stimulates the anterior pituitary to release adrenocorticotrophic hormone (ACTH), which in turn stimulates the adrenal cortex to release cortisol (see Figure 2.4). Malfunctioning of this system is implicated in a wide range of mental disorders including, depression, anxiety, and post-traumatic stress disorder. Exposure to chronic, unpredictable stress during early development can sensitive this system, making it over-responsive to stress (meaning it activates too readily and does not shut down appropriately). Sensitization of the HPA axis leads to an overproduction of cortisol which once again can damage the body and brain when it remains at chronically high levels.

Figure 2.6. The HPA Axis



For more on the link between cortisol and depression, check out this article:

https://www.psychologytoday.com/blog/the-athletes-way/201301/cortisol-why-the-stress-hormone-is-public-enemy-no-1

2.2.2.3: **Viral Infections**

Infections can cause brain damage and lead to the development of mental illness or an exacerbation of symptoms. For example, evidence suggests that contracting strep infection can lead to the development of OCD, Tourette's syndrome, and tic disorder in children (Mell, Davis, & Owens, 2005; Giedd et al., 2000; Allen et al., 1995; https://www.psychologytoday.com/blog/the-perfectionists-handbook/201202/can-infections-result-in-mental-illness). Influenza epidemics have also been linked to schizophrenia (Brown et al., 2004; McGrath and Castle, 1995; McGrath et al., 1994; O'Callaghan et al., 1991) though more recent research suggests this evidence is weak at best (Selten & Termorshuizen, 2017; Ebert & Kotler, 2005).

2.2.3: **Treatments**

2.2.3.1: **Psychopharmacology and Psychotropic Drugs**

One option to treat severe mental illness is psychotropic medications. These medications fall into five major categories.

Antidepressants are used to treat depression, but also anxiety, insomnia, or pain. The most common types of antidepressants are selective serotonin reuptake inhibitors (SSRIs) and include Citalopram (Celexa), Paroxetine, and Fluoxetine (Prozac). They can often take 2-6 weeks to take effect. Possible side effects include weight gain, sleepiness, nausea and vomiting, panic attacks, or thoughts about suicide or dying.

Anti-anxiety medications help with the symptoms of anxiety and include the benzodiazepines such as Diazepam (Valium), Alprazolam (Xanax), and Lorazepam (Ativan). These medications are effective in reducing anxiety in the short-term and take less time to take effect than antidepressants which are also commonly prescribed for anxiety. However, benzodiazepines are rather addictive. As such, tolerance to these drugs can develop quickly and individuals may experience withdrawal symptoms (e.g., anxiety, panic, insomnia) when they cease taking the drugs. For this reason, benzodiazepines should not be used in the long-term. Side effects include drowsiness, dizziness, nausea, difficulty urinating, and irregular heartbeat, to name a few.

Stimulants increase one's alertness and attention and are frequently used to treat ADHD. They include Lisdexamfetamine, the combination of dextroamphetamine and amphetamine, and Methylphenidate (Ritalin). Stimulants are generally effective and produce a calming effect. Possible side effects include loss of appetite, headache, motor tics or verbal tics, and personality changes such as appearing emotionless.

Antipsychotics are used to treat psychosis (i.e., hallucinations and delusions). They can also be used to treat eating disorders, severe depression, PTSD, OCD, ADHD, and Generalized Anxiety Disorder. Common antipsychotics include Chlorpromazine, Perphenazine, Quetiapine, and Lurasidone. Side effects include nausea, vomiting, blurred vision, weight gain, restlessness, tremors, and rigidity.

Mood stabilizers are used to treat bipolar disorder and at times depression, schizoaffective disorder, and disorders of impulse control. A common example is Lithium and side effects include loss of coordination, hallucinations, seizures, and frequent urination.

For more information on psychotropic medications, please visit:

https://www.nimh.nih.gov/health/topics/mental-health-medications/index.shtml

The use of these drugs has been generally beneficial to patients. Most report that their symptoms decline, leading them to feel better and improve their functioning. Also, long-term hospitalizations are less likely to occur as a result, though the medications do not benefit the individual in terms of improved living skills.

2.2.3.2: Electroconvulsive Therapy

According to Mental Health America, “Electroconvulsive therapy (ECT) is a procedure in which a brief application of electric stimulus is used to produce a generalized seizure.” Patients are placed on a padded bed and administered a muscle relaxant to avoid injury during the seizures. Annually, approximately 100,000 are treated using ECT for conditions including severe depression, acute mania, and suicidality. The procedure is still the most controversial available to mental health professionals due to “its effectiveness vs. the side effects, the objectivity of ECT experts, and the recent increase in ECT as a quick and easy solution, instead of long-term psychotherapy or hospitalization” (http://www.mentalhealthamerica.net/ect). Its popularity has declined since the 1940s and 1950s.

2.2.3.3: Psychosurgery

Another option to treat mental disorders is to perform brain surgeries. In the past, we have conducted trephining and lobotomies, neither of which are used today. Today’s techniques are much more sophisticated and have been used to treat schizophrenia, depression, and obsessive-compulsive disorder, though critics cite obvious ethical issues with conducting such surgeries as well as scientific issues. Due to these issues, psychosurgery is only used as a radical last resort when all other treatment options have failed to resolve a serious mental illness.

For more on psychosurgery, check out this article from Psychology Today:

https://www.psychologytoday.com/articles/199203/psychosurgery

2.2.4: Evaluation of the Model

The biological model is generally well respected today but suffers a few key issues. First, consider the list of side effects given for the psychotropic medications. You might make the case that some of the side effects are worse than the condition they are treating. Second, the viewpoint that all human behavior is explainable in biological terms, and therefore, when issues arise they can be treated using biological methods, overlooks factors that are not biological in nature. More on that over the next two sections.

2.3: Psychological Models

Section Learning Objectives

- Describe learning.
- Outline classical conditioning and the work of Pavlov and Watson.
- Outline operant conditioning and the work of Thorndike and Skinner.
- Outline observational learning/social-learning theory and the work of Bandura.
- Evaluate the usefulness of the behavioral model.
- Define the cognitive model.
- Exemplify the effect of maladaptive cognitions on creating abnormal behavior.
- List and describe cognitive therapies.
- Evaluate the usefulness of the cognitive model.
- Describe the humanistic perspective.
- Describe the existential perspective.
- Evaluate the usefulness of the humanistic and existential perspectives.

2.3.1: **The Behavioral Model**

2.3.1.1: **What is Learning?**

The behavioral model concerns the cognitive process of learning. Simply, **learning** is any relatively permanent change in behavior due to experience and practice and has two main forms – associative learning and observational learning. First, associative learning is the linking together of information sensed from our environment. **Conditioning**, a type of associative learning, occurs which two events are linked and has two forms – classical conditioning, or linking together two types of stimuli, and operant conditioning, or linking together a response with its consequence. Second, **observational learning** occurs when we learn by observing the world around us.

We should also note the existence of non-associative learning or when there is no linking of information or observing the actions of others around you. Types include **habituation**, or when we simply *stop responding* to repetitive and harmless stimuli in our environment such as a fan running in your laptop as you work on a paper, and **sensitization**, or when our reactions are *increased* due to a strong stimulus, such as an individual who experienced a mugging and now experiences panic when someone walks up behind him/her on the street.

Behaviorism is the school of thought associated with learning that began in 1913 with the publication of John B. Watson's article, "Psychology as the Behaviorist Views It," in the journal, *Psychological Review* (Watson, 1913). It was Watson's belief that the subject matter of psychology was to be observable behavior and to that end said that psychology should focus on the prediction and control of behavior. Behaviorism was dominant from 1913 to 1990 before being absorbed into mainstream psychology. It went through three major stages – behaviorism proper under Watson and lasting from 1913-1930 (discussed as respondent conditioning), neobehaviorism under Skinner and lasting from 1930-1960 (discussed as operant conditioning), and sociobehaviorism under Bandura and Rotter and lasting from 1960-1990 (discussed as social learning theory).

2.3.1.2: **Classical Conditioning**

You have likely heard about Pavlov and his dogs but what you may not know is that this was a discovery made accidentally. Ivan Petrovich Pavlov (1906, 1927, 1928), a Russian physiologist, was interested in studying digestive processes in dogs in response to being fed meat powder. What he discovered was the dogs would salivate even *before* the meat powder was presented. They would salivate at the sound of a bell, footsteps in the hall, a tuning fork, or the presence of a lab assistant. Pavlov realized there were some stimuli that automatically elicited responses (such as salivating to meat powder) and those that had to be paired with these automatic associations for the animal or person to respond to it (such as salivating to a bell). Armed with this stunning revelation, Pavlov spent the rest of his career investigating this learning phenomenon.

The important thing to understand is that not all behaviors occur due to reinforcement and punishment as operant conditioning says. In the case of classical conditioning, stimuli exert complete and automatic control over some behaviors. We see this in the case of reflexes. When a doctor strikes your knee with that little hammer it extends out automatically. You do not have to do anything but watch. Babies will root for a food source if the mother's breast is placed near their mouth. If a nipple is placed in their mouth, they

will also automatically suck, as per the sucking reflex. Humans have several of these reflexes though not as many as other animals due to our more complicated nervous system.

Classical conditioning (also called response or Pavlovian conditioning) occurs when we link a previously neutral stimulus with a stimulus that is unlearned or inborn, called an unconditioned stimulus. In respondent conditioning, learning occurs in three phases: preconditioning, conditioning, and postconditioning. See Figure 2.1 for an overview of Pavlov's classic experiment.

Preconditioning. This stage of learning signifies is that some learning is already present. There is no need to learn it again as in the case of primary reinforcers and punishers in operant conditioning. In Panel A, food makes a dog salivate. This does not need to be learned and is the relationship of an unconditioned stimulus (UCS) yielding an unconditioned response (UCR). Unconditioned means unlearned. In Figure 2.1, we also see that a neutral stimulus (NS) yields nothing. Dogs do not enter the world knowing to respond to the ringing of a bell (which it hears).

Conditioning. Conditioning is when learning occurs. Through the pairing of a neutral stimulus and unconditioned stimulus (bell and food, respectively) the dog will learn that the bell ringing (NS) signals food coming (UCS) and salivate (UCR). The pairing must occur more than once so that needless pairings are not learned such as someone farting right before your food comes out and now you salivate whenever someone farts (…at least for a while. Eventually the fact that no food comes will extinguish this reaction but still, it will be weird for a bit).

Postconditioning. Postconditioning, or *after* learning has occurred, establishes a *new* and not naturally occurring relationship of a conditioned stimulus (CS; previously the NS) and conditioned response (CR; the same response). So the dog now reliably salivates at the sound of the bell because he expects that food will follow, and it does.

Figure 2.1. Pavlov's Classic Experiment

One of the most famous studies in psychology was conducted by Watson and Rayner (1920). Essentially, they wanted to explore the possibility of conditioning emotional responses. The researchers ran a 9-month-old child, known as Little Albert, through a series of trials in which he was exposed to a white rat. At first, he showed no response except curiosity. Then the researchers began to make a loud sound (UCS) whenever the rat was presented. Little Albert exhibited the normal fear response to this sound. After several conditioning trials like these, Albert responded with fear to the mere presence of the white rat.

As fears can be learned, so too they can be unlearned. Considered the follow-up to Watson and Rayner (1920), Jones (1924) wanted to see if a child (named Peter) who learned to be afraid of white rabbits could be conditioned to become unafraid of them. Simply, she placed Peter in one end of a room and then brought in the rabbit. The rabbit was far enough away so as to not cause distress. Then, Jones gave Peter some pleasant food (i.e., something sweet such as cookies; remember the response to the food is unlearned). She continued this procedure with the rabbit being brought in a bit closer each time until eventually, Peter did not respond with distress to the rabbit. This process is called **counterconditioning** or **extinction**, or the reversal of previous learning.

Another way to unlearn a fear is called **flooding** or exposing the person to the maximum level of stimulus and as nothing aversive occurs, the link between CS and UCS producing the CR of fear should break, leaving the person unafraid. This type of treatment is rather extreme and is not typically practiced by psychologists.

2.3.1.3: Operant Conditioning

Influential on the development of Skinner's operant conditioning, Thorndike proposed the **law of effect** (Thorndike, 1905) or the idea that if our behavior produces a favorable consequence, in the future when the same stimulus is present, we will be more likely to make the response again, expecting the same favorable consequence. Likewise, if our action leads to dissatisfaction, then we will

not repeat the same behavior in the future. Thorndike developed the law of effect thanks to his work with the Puzzle Box. Cats were food deprived the night before the experimental procedure was to occur. The next morning, they were placed in the puzzle box and a small amount of food was placed outside the box close enough to be smelled, but the cat could not reach the food. To get out, a series of switches, buttons, levers, etc. had to be manipulated and once done, the cat could escape the box and eat some of the food. But just some. The cat was then promptly placed back in the box to figure out how to get out again, the food being its reward for doing so. With each subsequent escape and re-insertion into the box, the cat became faster until he/she knew exactly what had to be done to escape. This is called **trial and error learning,** or making a response repeatedly if it leads to success. Thorndike also said that stimulus and responses were connected by the organism and this lead to learning. This approach to learning was called **connectionism**.

Operant conditioning is a type of associate learning which focuses on consequences that follow a response or behavior that we make (anything we do, say, or think/feel) and whether it makes a behavior more or less likely to occur. This should sound much like what you just read about in terms of Thorndike's work. Skinner talked about **contingencies** or when one thing occurs due to another. Think of it as an If-Then statement. If I do X then Y will happen. For operant conditioning, this means that if I make a behavior, then a specific consequence will follow. The events (response and consequence) are linked in time.

What form do these consequences take? There are two main ways they can present themselves.

- **Reinforcement** – Due to the consequence, a behavior/response is more likely to occur in the future. It is strengthened.
- **Punishment** – Due to the consequence, a behavior/response is less likely to occur in the future. It is weakened.

Reinforcement and punishment can occur as two types – positive and negative. These words have no affective connotation to them meaning they do not imply good or bad. *Positive* means that you are giving something – good or bad. *Negative* means that something is being taken away – good or bad. Check out the figure below for how these contingencies are arranged.

Figure 2.2. Contingencies in Operant Conditioning

	Some "**Bad**" Thing	Some "**Good**" Thing
Giving	Positive Punishment	Positive Reinforcement
Taking Away	Negative Reinforcement	Negative Punishment

Let's go through each:

- **Positive Punishment (PP)** – If something bad or aversive is given or added, then the behavior is less likely to occur in the future. If you talk back to your mother and she slaps your mouth, this is a PP. Your response of talking back led to the consequence of the aversive slap being delivered or given to your face. Ouch!!!
- **Positive Reinforcement (PR)** – If something good is given or added, then the behavior is more likely to occur in the future. If you study hard and earn an A on your exam, you will be more likely to study hard in the future. Similarly, your parents may give you money for your stellar performance. Cha Ching!!!
- **Negative Reinforcement (NR)** – This is a tough one for students to comprehend because the terms don't seem to go together and are counterintuitive. But it is really simple and you experience NR all the time. This is when you are more likely to engage in a behavior that has resulted in the removal of something aversive in the past. For instance, what do you do if you have a headache? You likely answered take Tylenol. If you do this and the headache goes away, you will take Tylenol in the future when you have a headache. Another example is continually smoking marijuana because it temporarily decreases feelings of anxiety. The behavior of smoking marijuana is being reinforced because it reduces a negative state.
- **Negative Punishment (NP)** – This is when something good is taken away or subtracted making a behavior less likely in the future. If you are late to class and your professor deducts 5 points from your final grade (the points are something good and the loss is negative), you will hopefully be on time in all subsequent classes. Another example is taking away a child's allowance when he misbehaves.

2.3.1.4: Observational Learning

There are times when we learn by simply watching others. This is called **observational learning** and is contrasted with **enactive learning**, which is learning by doing. There is no firsthand experience by the learner in observational learning. You can learn desirable behaviors such as exercising because your mother engaged in exercise every day and you can learn undesirable ones too. If your parents resort to alcohol consumption to deal with the stressors life presents, then you too might do the same. What is critical is what happens to the model in all of these cases. If my mother seems genuinely happy and pleased with herself after exercising, then I will be more likely to adopt this behavior. If my mother or father consumes alcohol to feel better when things are tough, and it works, then I might do the same. On the other hand, if we see a sibling constantly getting in trouble with the law then we may not model this behavior due to the negative consequences.

Albert Bandura conducted pivotal research on observational learning and you likely already know all about it from previous psychology courses. In Bandura's experiment, children were first brought into a room to watch a video of an adult model playing nicely or aggressively with a Bobo doll. Next, the children were placed in a room with toys and a Bobo doll. Children who watched the aggressive model behaved aggressively with the Bobo doll while those who saw the nice model, played nice.

Figure 2.3. Bandura's Classic Bobo Doll Experiment

Bandura said if all behaviors are learned by observing others and we model our behaviors on theirs, then undesirable behaviors can be altered or relearned in the same way. **Modeling** techniques are used to change behavior by having clients observe a model in a situation that usually causes them some anxiety. By seeing the model interact calmly with the fear-evoking stimulus, their fear should subside. This form of behavior therapy is widely used in clinical and classroom situations. In the classroom, we might use modeling to demonstrate to a student how to do a math problem. In fact, in many college classrooms, this is exactly what the instructor does.

But keep in mind that we do not model everything we see. Why? First, we cannot pay attention to everything going on around us. We are more likely to model behaviors by someone who commands our attention. Second, we must remember what a model does in order to imitate it. If a behavior is not memorable, it will not be imitated. Finally, we must try to convert what we see into action. If we are not motivated to perform an observed behavior, we probably will not show what we have learned.

2.3.1.5: Evaluating the Behavioral Model

Within the context of abnormal behavior or psychopathology, the behavioral perspective is useful because it suggests that maladaptive behavior occurs when learning goes awry. The good thing is that what is learned can be unlearned or relearned using **behavior modification** which refers to the process of changing behavior. To begin, an applied behavior analyst will identify a target behavior, or behavior to be changed, define it, work with the client to develop goals, conduct a functional assessment to understand what the undesirable behavior is, what causes it, and what maintains it. Armed with this knowledge, a plan is developed and consists of numerous strategies to act on one or all of these elements – antecedent, behavior, and/or consequence.

The greatest strength or appeal of the behavioral model is that its tenets are easily tested in the laboratory unlike those of the psychodynamic model. Also, a large number of treatment techniques have been developed and proven to be effective over the years. For example, desensitization (Wolpe, 1997) teaches clients to respond calmly to fear-producing stimuli. It begins with the individual learning a relaxation technique such as diaphragmatic breathing. Next, a fear hierarchy, or list of feared objects and situations, is constructed in which the individual moves from least to most feared. Finally, the individual either imagines

(systematic) or experiences in real life (in-vivo) each object or scenario from the hierarchy and uses the relaxation technique while doing so. This represents individual pairings of feared object or situation and relaxation and so if there are 10 objects/situations in the list, the client will experience ten such pairings and eventually be able to face each without fear. Outside of phobias, desensitization has been shown to be effective in the treatment of Obsessive Compulsive Disorder symptoms (Hakimian and D'Souza, 2016) and limitedly with the treatment of depression that is co-morbid with OCD (Masoumeh and Lancy, 2016).

Critics of the behavioral perspective point out that it oversimplifies behavior and often ignores inner determinants of behavior. Behaviorism has also been accused of being mechanistic and seeing people as machines. Watson and Skinner defined behavior as what we do or say, but later, behaviorists added what we think or feel. In terms of the latter, cognitive behavior modification procedures arose after the 1960s along with the rise of cognitive psychology. This lead to a cognitive-behavioral perspective which combines concepts from the behavioral and cognitive models, the latter is discussed in the next section.

2.3.2: The Cognitive Model

2.3.3: 2.3.2.1. What is It?

As noted earlier, the idea of people being machines was a key feature of behaviorism and other schools of thought in psychology until about the 1960s or 1970s. In fact, behaviorism said psychology was to be the study of observable behavior. Any reference to cognitive processes was dismissed as this was not overt, but covert according to Watson and later Skinner. Of course, removing cognition from the study of psychology ignored an important part of what makes us human and separates us from the rest of the animal kingdom. Fortunately, the work of George Miller, Albert Ellis, Aaron Beck, and Ulrich Neisser demonstrated the importance of cognitive abilities in understanding thoughts, behaviors, and emotions, and in the case of psychopathology, they helped to show that people can create their own problems by how they come to interpret events experienced in the world around them. How so?

2.3.3.1: Maladaptive Cognitions

Irrational or dysfunctional thought patterns can be the basis of psychopathology. Throughout this book, we will discuss several treatment strategies that are used to change unwanted, maladaptive cognitions, whether they are present as an *excess* such as with paranoia, suicidal ideation, or feelings of worthlessness; or as a *deficit* such as with self-confidence and self-efficacy. More specifically, cognitive distortions/maladaptive cognitions can take the following forms:

- Overgeneralizing – You see a larger pattern of negatives based on one event.
- What if? – Asking yourself what if something happens without being satisfied by any of the answers.
- Blaming – Focusing on someone else as the source of your negative feelings and not taking any responsibility for changing yourself.
- Personalizing – Blaming yourself for negative events rather than seeing the role that others play.
- Inability to disconfirm – Ignoring any evidence that may contradict your maladaptive cognition.
- Regret orientation – Focusing on what you could have done better in the past rather than on making an improvement now.
- Dichotomous thinking – Viewing people or events in all-or-nothing terms.

For more on cognitive distortions, check out this website: http://www.goodtherapy.org/blog/20-cognitive-distortions-and-how-they-affect-your-life-0407154

2.3.3.2: Cognitive Therapies

According to the National Alliance on Mental Illness (NAMI), **cognitive behavioral therapy** (CBT) "focuses on exploring relationships among a person's thoughts, feelings and behaviors. During CBT a therapist will actively work with a person to uncover unhealthy patterns of thought and how they may be causing self-destructive behaviors and beliefs." CBT attempts to identifying negative or false beliefs and restructure them. They add, "Oftentimes someone being treated with CBT will have homework in between sessions where they practice replacing negative thoughts with more realistic thoughts based on prior experiences or record their negative thoughts in a journal." For more on CBT, visit: https://www.nami.org/Learn-More/Treatment/Psychotherapy. Some commonly used strategies include cognitive restructuring, cognitive coping skills training, and acceptance techniques.

First, **cognitive restructuring (**also called rational restructuring) involves replacing maladaptive cognitions with more adaptive ones. To do this, the client must be aware of the distressing thoughts, when they occur, and their effect on them. Next, the therapist works to help the client stop thinking these thoughts and to replace them with more rational ones. It's a simple strategy, but an

important one. Psychology Today published a great article on January 21, 2013 which described 4 ways to change your thinking through cognitive restructuring. Briefly, these included:

1. Notice when you are having a maladaptive cognition such as making "negative predictions." They suggest you figure out what is the worst thing that could happen and what other outcomes are possible.
2. Track the accuracy of the thought. For instance, if you believe ruminating on a problem generates a solution then write down each time you ruminate and then the result. You can generate a percentage of times you ruminated to the number of successful problem-solving strategies you generated.
3. Behaviorally test your thought. As an example, if you think you don't have time to go to the gym then figure out if you really do not have time. Record what you do each day and then look at open times of the day. Explore if you can make some minor, or major, adjustments to your schedule to free up an hour to exercise.
4. Examine the evidence both for and against your thought. If you do not believe you do anything right, list evidence of when you did not do something right and then evidence of when you did. Then write a few balanced statements such as the one the article suggests, "I've made some mistakes that I feel embarrassed about but a lot of the time, I make good choices."

The article also suggested a few non-cognitive restructuring techniques to include mindfulness meditation and self-compassion. For more on these visit: https://www.psychologytoday.com/blog/in-practice/201301/cognitive-restructuring

A second major strategy is to use what is called **cognitive coping skills training**. This strategy involves teaching social skills, communication, and assertiveness through direct instruction, role-playing, and modeling. For social skills, therapists identify appropriate social behavior such as making eye contact, saying no to a request, or starting up a conversation with a stranger and examine whether the client is inhibited from engaging in the behavior due to anxiety. For communication, the therapist can help determine if the problem is with speaking, listening, or both and then develop a plan the client can use in various interpersonal situations. Finally, assertiveness training aids the client protect their rights and obtain what they want from others. Treatment starts with determining situations in which assertiveness is lacking and generating a hierarchy of assertiveness opportunities. Least difficult situations are handled first, followed by more difficult situations, all while rehearsing and mastering all the situations present in the hierarchy. For more on these techniques, visit http://cogbtherapy.com/cognitive-behavioral-therapy-exercises/.

Finally, **acceptance techniques** can be used to reduce a client's worry and anxiety. Life involves a degree of uncertainty and at times we need to just accept this uncertainty. However, many clients, especially those with anxiety, have difficulty tolerating uncertainty. Acceptance techniques might include weighing the pros of fighting uncertainty against the cons of doing so. The cons should outweigh the pros and help the client to end the struggle and accept what is unknown. Chances are the client is already accepting the unknown in some areas of life and identifying those can help them to see why it is helpful to accept uncertainty which may help them to do so in more difficult areas. Finally, the therapist may help the client to question whether uncertainty necessarily leads to a negative end. The client may think so, but reviewing the evidence for and against this statement will show them that uncertainty does not always lead to negative outcomes which can help to reduce how threatening uncertainty seems.

2.3.3.3: Evaluating the Cognitive Model

The cognitive model made up for an obvious deficit in the behavioral model – overlooking the importance of our thoughts and the role cognitive processes play in our feelings and behaviors. Right before his death, Skinner (1990) reminded psychologists that the only thing we can truly know and study is observable behavior. Cognitive processes cannot be empirically and reliably measured and so should be ignored. Is there merit to this view? **Social desirability** states that sometimes people do not tell us the truth about what they are thinking, feeling or doing (or have done) because they do not want us to think less of them or to judge them harshly if they are outside the social norm. In other words, they present themselves in a favorable light. If this is true, how can we really know what they are thinking? The person's true intentions or thoughts and feelings are not readily available to us or are covert, and so do not make for good empirical data. Still, cognitive-behavioral therapies have proven their efficacy for the treatment of OCD (McKay et al., 2015); perinatal depression (Sockol, 2015); insomnia (de Bruin et al., 2015), bulimia nervosa (Poulsen et al., 2014), hypochondriasis (Olatunji et al., 2014), and social anxiety disorder (Leichsenring et al., 2014) to name a few. Other examples will be discussed throughout this book.

2.3.4: The Humanistic and Existential Perspectives

2.3.4.1: The Humanistic Perspective

The humanistic perspective, or third force psychology (psychoanalysis and behaviorism being the other two forces), emerged in the 1960s and 1970s as an alternative viewpoint to the largely deterministic view of personality espoused by psychoanalysis and the

view of humans as machines advocated by behaviorism. Key features of the perspective include a belief in human perfectibility, personal fulfillment, valuing self-disclosure, placing feelings over intellect, an emphasis on the present, and hedonism. Its key figures were Abraham Maslow who proposed the hierarchy of needs and Carl Rogers who we will focus on here.

Rogers said that all people want to have *positive regard* from significant others in their life. When the individual is accepted as they are they receive *unconditional positive regard* and become a *fully functioning person*. They are open to experience, live every moment to the fullest, are creative, accept responsibility for their decisions, do not derive their sense of self from others, strive to maximize their potential, and are self-actualized. Their family and friends may disapprove of some of their actions but overall, respect and love them. They then realize their worth as a person but also that they are not perfect. Of course most people do not experience this but instead are made to feel that they can only be loved and respected if they meet certain standards, called *conditions of worth*. Hence, they experience *conditional positive regard*. According to Rogers, their self-concept is now seen as having worth only when these significant others approve and so becomes distorted, leading to a disharmonious state and psychopathology. Individuals in this situation are unsure what they feel, value, or need leading to dysfunction and the need for therapy. Rogers stated that the humanistic therapist should be warm, understanding, supportive, respectful, and accepting of his/her clients. This approach came to be called **client-centered therapy**.

2.3.4.2: The Existential Perspective

This existential perspective stresses the need for people to continually re-create themselves and be self-aware, acknowledges that anxiety is a normal part of life, focuses on free will and self-determination, emphasizes that each person has a unique identity known only through relationships and the search for meaning, and finally, that we develop to our maximum potential. Abnormal behavior arises when we avoid making choices, do not take responsibility, and fail to actualize our full potential. Existential therapy is used to treat a myriad of disorders and problems including substance abuse, excessive anxiety, apathy, avoidance, despair, depression, guilt, anger, and rage. It also focuses on life-enhancing experiences such as love, caring, commitment, courage, creativity, spirituality, and acceptance, to name a few (For more information, please visit: https://www.psychologytoday.com/therapy-types/existential-therapy).

2.3.4.3: Evaluating the Humanistic and Existential Perspectives

The biggest criticism of these models is that the concepts are abstract and fuzzy and as such are very difficult to research. The exception to this was Rogers who did try to scientifically investigate his propositions, though most other humanistic-existential psychologists rejected the use of the scientific method. They also have not developed much in the way of theory and their perspectives tend to work best with people who have adjustment issues and not as well with severe mental illness. The perspectives do offer hope to people who have experienced tragedy by asserting that we control our own destiny and can make our own choices.

2.4: The Sociocultural Model

Section Learning Objectives

- Describe the sociocultural model.
- Clarify how socioeconomic factors affect mental illness.
- Clarify how gender factors affect mental illness.
- Clarify how environmental factors affect mental illness.
- Clarify how multicultural factors affect mental illness.
- Evaluate the sociocultural model.

Outside of biological and psychological factors on mental illness, race, ethnicity, gender, religious orientation, socioeconomic status, sexual orientation, etc. also play a role, and this is the basis of the **sociocultural model**. Next, we explore a few of these factors.

2.4.1: **Socioeconomic Factors**

Low socioeconomic status has been linked to higher rates of mental and physical illness (Ng, Muntaner, Chung, & Eaton, 2014) due to persistent concern over unemployment or under-employment, low wages, lack of health insurance, no savings, and the inability to put food on the table, which can then lead to feeling hopeless, helpless, and dependent on others. This situation places considerable stress on an individual and can lead to higher rates of anxiety disorders and depression. Borderline personality disorder has also been found to be higher in people in low-income brackets (Tomko et al., 2014).

2.4.2: **Gender Factors**

Gender plays an important, though at times, unclear role in mental illness. It is important to understand that gender is not the cause of mental illness, though differing demands placed on males and females by society and their culture can influence the development and course of a disorder. Consider the following:

- Rates of eating disorders are higher among women than, men, though both genders are affected. In the case of men, *muscle dysphoria* is of concern and is characterized by extreme concern over not be muscular enough.
- OCD has an earlier age of onset in boys than girls, with most people being diagnosed by age 19.
- Women are at greater risk for developing an anxiety disorder than men.
- ADHD is more common in males than females, though females are more likely to have inattention issues.
- Boys are more likely to be diagnosed with Autism Spectrum Disorder.
- Depression occurs with greater frequency in women than men.
- Women are more likely to develop PTSD compared to men.
- Rates of SAD (Seasonal Affective Disorder) are four times greater in women than men.

Consider this…

In relation to men: “Men and women experience many of the same mental disorders but their willingness to talk about their feelings may be very different. This is one of the reasons that their symptoms may be very different as well. For example, some men with depression or an anxiety disorder hide their emotions and may appear to be angry or aggressive while many women will express sadness. Some men may turn to drugs or alcohol to try to cope with their emotional issues.”

https://www.nimh.nih.gov/health/topics/men-and-mental-health/index.shtml

In relation to women: “Some women may experience symptoms of mental disorders at times of hormone change, such as perinatal depression, premenstrual dysphoric disorder, and perimenopause-related depression. When it comes to other mental disorders such as schizophrenia and bipolar disorder, research has not found differences in rates that men and women experience these illnesses. But, women may experience these illnesses differently – certain symptoms may be more common in women than in men, and the course of the illness can be affected by the sex of the individual.”

https://www.nimh.nih.gov/health/topics/women-and-mental-health/index.shtml

2.4.3: Environmental Factors

Environmental factors also play a role in the development of mental illness. How so?

- In the case of borderline personality disorder, many people report experiencing traumatic life events such as abandonment, abuse, unstable relationships or hostility, and adversity during childhood.
- Cigarette smoking, alcohol use, and drug use during pregnancy are risk factors for ADHD.
- Divorce or the death of a spouse can increase the risk of developing an anxiety disorder.
- Trauma, stress, and other extreme stressors are predictive of depression.
- Malnutrition before birth, exposure to viruses, and other psychosocial factors are believed to contribute to the risk of developing schizophrenia.
- Seasonal Affective Disorder (SAD) occurs with greater frequency for those living far north or south of the equator (Melrose, 2015). Horowitz (2008) found that rates of SAD are just 1% for those living in Florida while 9% of Alaskans are diagnosed with the disorder. This is due to differences in exposure to sunlight in these regions.

Source: https://www.nimh.nih.gov/health/topics/index.shtml

2.4.4: Multicultural Factors

Racial, ethnic, and cultural factors are also relevant to understanding the development and course of mental disorders. Multicultural psychologists assert that both normal behavior and abnormal behavior need to be understood in relation to the individual's unique culture and the group's value system. Racial and ethnic minorities must contend with prejudice, discrimination, racism, economic hardships, etc. as part of their daily life and these stressors can increase vulnerability to a mental disorder (Lo & Cheng, 2014; Jones, Cross, & DeFour, 2007; Satcher, 2001), though some research suggests that ethnic identity can buffer against these stressors and protect mental health (Mossakowski, 2003). To address this unique factor, **culture-sensitive therapies** have been developed and include increasing the therapist's awareness of cultural values, hardships, stressors, and/or prejudices faced by their client; the identification of suppressed anger and pain; and raising the client's self-worth (Prochaska & Norcross, 2013).

2.4.4.1: Evaluation of the Model

The sociocultural model has contributed greatly to our understanding of the nuances of diagnosis, prognosis, course, and treatment of mental disorders for other races, cultures, genders, ethnicities. In Chapter 3 we will discuss diagnosing and classifying abnormal behavior from the perspective of the DSM 5 (Diagnostic and Statistical Manual of Mental Disorders, 5th edition). Important here is that specific culture- and gender-related diagnostic issues are discussed for each disorder, demonstrating increased awareness of the impact of these factors. Still, the sociocultural model suffers from issues with the findings being difficult to interpret and not allowing for the establishment of causal relationships due to a reliance on more qualitative data gathered from case studies and ethnographic analyses (one such example is Zafra, 2016).

Chapter Recap

In Chapter 2, we first distinguished uni- and multi-dimensional models of abnormality and made a case that the latter was better to subscribe to. We then discussed biological, psychological, and sociocultural models of abnormality. In terms of the biological model, neurotransmitters, brain structures, hormones, genes, and viral infections were discussed as potential causes of mental disorders and several treatment options were described. In terms of psychological perspectives, behavioral, cognitive, humanistic and existential perspectives were discussed. Finally, the sociocultural model indicated the roles that socioeconomic status, gender, environmental, and multicultural factors can play in abnormal behavior.

CHAPTER OVERVIEW

3: Clinical Assessment, Diagnosis, and Treatment

Learning Objectives

- Describe clinical assessment and methods used in it.
- Clarify how mental health professionals diagnosis mental disorders in a standardized way.
- Discuss reasons to seek treatment and the importance of psychotherapy

Chapter 3 covers the issues of clinical assessment, diagnosis, and treatment. We will define assessment and then describe key issues such as reliability, validity, standardization, and specific methods that are used. In terms of clinical diagnosis, we will discuss the two main classification systems used around the world – the DSM-5 and ICD-10. Finally, we discuss reasons why people may seek treatment and what to expect when doing so.

3.1: Clinical Assessment

Section Learning Objectives

- Define clinical assessment.
- Clarify why clinical assessment is an ongoing process.
- Define and exemplify reliability.
- Define and exemplify validity.
- Define standardization.
- List and describe six methods of assessment.

3.1.1: **What is Clinical Assessment?**

In order for a mental health professional to be able to effectively treat a client and know that the selected treatment actually worked (or is working), he/she first must engage in the clinical assessment of the client. **Clinical assessment** refers to collecting information and drawing conclusions through the use of observation, psychological tests, neurological tests, and interviews to determine what the person's problem is and what symptoms he/she is presenting with. This collection of information involves learning about the client's skills, abilities, personality characteristics, cognitive and emotional functioning, social context (e.g., environmental stressors), and cultural factors particular to them such as their language or ethnicity. Clinical assessment is not just conducted at the beginning of the process of seeking help but all throughout the process. Why is that?

Consider this. First, we need to determine if a treatment is even needed. By having a clear accounting of the person's symptoms and how they affect daily functioning we can determine to what extent the individual is adversely affected. Assuming treatment is needed, our second reason to engage in clinical assessment is to determine what treatment will work best. As you will see later in this chapter, there are numerous approaches to treatment. These include Behavior Therapy, Cognitive Therapy, Cognitive-Behavioral Therapy (CBT), Humanistic-Experiential Therapies, Psychodynamic Therapies, Couples and Family Therapy, and biological treatments (e.g., psychopharmacology). Of course, for any mental disorder, some of the aforementioned therapies will have greater efficacy than others. Even if several can work well, it does not mean a particular therapy will work well for that specific client. Assessment can help the clinician figure this out. Finally, we need to know if the treatment worked. This will involve measuring symptoms and behavior before any treatment is used and then measuring symptoms and behavior while the treatment is in place. We will even want to measure symptoms and behavior after the treatment ends to make sure symptoms do not return. Knowing what the person's baselines are for different aspects of psychological functioning will help us to see when improvement occurs. In recap, obtaining the baselines happens in the beginning, implementing the treatment plan happens more so in the middle, and then making sure the treatment produces the desired outcome occurs at the end. It should be clear from this discussion that clinical assessment is an *ongoing* process.

3.1.2: **Key Concepts in Assessment**

Important to the assessment process are three critical concepts – reliability, validity, and standardization. Actually, these three are important to science in general. First, we want assessment to be **reliable** or consistent. Outside of clinical assessment, when our car has an issue and we take it to the mechanic, we want to make sure that what one mechanic says is wrong with our car is the same as what another says or even two others. If not, the measurement tools they use to assess cars are flawed. The same is true of a patient who is experiencing a mental disorder. If one mental health professional says the person has major depressive disorder and another says the issue is borderline personality disorder, then there is an issue with the assessment tool being used. Ensuring that two different raters (e.g., mechanics, mental health professionals) are consistent in their assessments is called *interrater reliability*. Another type of reliability occurs when a person takes a test one day, and then the same test on another day. We would expect the person's answers to be consistent with one another, which is called *test-retest reliability*. An example is if the person takes the Minnesota Multiphasic Personality Inventory (MMPI) on Tuesday and then the same test on Friday, then unless something miraculous or tragic happened over the two days in between tests, the scores on the MMPI should be nearly identical to one another. In other words, the two scores (test and retest) should be correlated with one another. If the test is reliable, the correlation should be very high (remember, a correlation goes from -1.00 to +1.00 and positive means as one score goes up, so does the other, so the correlation for the two tests should be high on the positive side).

In addition to reliability, we want to make sure the test measures what it says it measures. This is called **validity**. Let's say a new test is developed to measure symptoms of depression. It is compared against an existing, and proven test, such as the Beck

Depression Inventory (BDI). If the new test measures depression, then the scores on it should be highly correlated with the ones obtained by the BDI. This is called *concurrent* or *descriptive validity*. We might even ask if an assessment tool looks valid. If we answer yes, then it has *face* validity, though it should be noted that this is not based on any statistical or evidence-based method of assessing validity. An example would be a personality test that asks about how people behave in certain situations. It, therefore, seems to measure personality or we have an overall feeling that it measures what we expect it to measure.

A tool should also be able to accurately predict what will happen in the future, called *predictive validity*. Let's say we want to tell if a high school student will do well in college. We might create a national exam to test needed skills and call it something like the Scholastic Aptitude Test (SAT). We would have high school students take it by their senior year and then wait until they are in college for a few years and see how they are doing. If they did well on the SAT, we would expect that at that point, they should be doing well in college. If so, then the SAT accurately predicts college success. The same would be true of a test such as the Graduate Record Exam (GRE) and its ability to predict graduate school performance.

Finally, we want to make sure that the experience one patient has when taking a test or being assessed is the same as another patient taking the test the same day or on a different day, and with either the same tester or another tester. This is accomplished with the use of clearly laid out rules, norms, and/or procedures, and is called **standardization**. Equally important is that mental health professionals interpret the results of the testing in the same way or otherwise it will be unclear what the meaning of a specific score is.

3.1.3: Methods of Assessment

So how do we assess patients in our care? We will discuss psychological tests, neurological tests, the clinical interview, behavioral assessment, and a few others in this section.

3.1.3.1: The Clinical Interview

A clinical interview is 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. 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.

A **mental status examination** is used to organize the information collected during the interview and to systematically evaluate the client through a series of observations and questions assessing appearance and behavior (e.g., grooming and body language), thought processes and content (e.g., disorganized speech or thought and false beliefs), mood and affect (e.g., hopelessness or elation), intellectual functioning (e.g., speech and memory), and awareness of surroundings (e.g., does the client know where he/she is, when it is, and who he/she is?). The exam covers areas not normally part of the interview and allows the mental health professional to determine which areas need to be examined further. The limitation of the interview is that it lacks reliability, especially in the case of the unstructured interview.

3.1.3.2: Psychological Tests and Inventories

Psychological tests are used to assess the client's personality, social skills, cognitive abilities, emotions, behavioral responses, or interests and can be administered either individually or to groups. **Projective tests** consist of simple ambiguous stimuli that can elicit an unlimited number of responses. They include the Rorschach test or inkblot test and the **Thematic Apperception Test** which requires the individual to write a complete story about each of 20 cards shown to them and give details about what led up to the scene depicted, what the characters are thinking, what they are doing, and what the outcome will be. From these responses, the clinician gains perspective on the patient's worries, needs, emotions, conflicts. Another projective test is the *sentence completion test* and asks individuals to finish an incomplete sentence. Examples include 'My mother' …. or 'I hope.'

Personality inventories ask clients to state whether each item in a long list of statements applies to them, and could ask about feelings, behaviors, or beliefs. Examples include the MMPI or Minnesota Multiphasic Personality Inventory and the NEO-PI-R which is a concise measure of the five major domains of personality – Neuroticism, Extroversion, Openness, Agreeableness, and Conscientiousness. Six facets define each of the five domains and the measure assess emotional, interpersonal, experimental, attitudinal, and motivational styles (Costa & McCrae, 1992). These inventories have the advantage of being easy to administer by either a professional or the individual taking it, are standardized, objectively scored, and are completed either on the computer or through paper and pencil. That said, personality cannot be directly assessed and so you can never completely know the individual on the basis of these inventories.

3.1.3.3: Neurological Tests

Neurological tests are also used to diagnose cognitive impairments caused by brain damage due to tumors, infections, or head injury; or changes in brain activity. *Positron Emission Tomography or PET* is used to study the brain's functioning and begins by injecting the patient with a radionuclide which collects in the brain. Patients then lie on a scanning table while a ring-shaped machine is positioned over their head. Images are produced that yield information about the functioning of the brain. *Magnetic Resonance Imaging or MRI* produces 3D images of the brain or other body structures using magnetic fields and computers. They are used to detect structural abnormalities such as brain and spinal cord tumors or nervous system disorders such as multiple sclerosis. Finally, *computed tomography* or the *CT scan* involves taking X-rays of the brain at different angles that are then combined. They are used to detect structural abnormalities such as brain tumors and brain damage caused by head injuries.

3.1.3.4: Physical Examination

Many mental health professionals recommend the patient see their family physician for a physical examination which is much like a check-up. Why is that? Some organic conditions, such as hyperthyroidism or hormonal irregularities, manifest behavioral symptoms that are similar to mental disorders and so ruling such conditions out can save costly therapy or surgery.

3.1.3.5: Behavioral Assessment

Within the realm of behavior modification and applied behavior analysis, is **behavioral assessment** which is simply the measurement of a target behavior. The **target behavior** is whatever behavior we want to change and it can be in excess (needing to be reduced), or in a deficit state (needing to be increased). During behavioral assessment we assess the ABCs of behavior:

- **Antecedents** are the environmental events or stimuli that trigger a behavior
- **Behaviors** are what the person does, says, thinks/feels; and
- **Consequences** are the outcome of a behavior that either encourages it to be made again in the future or discourages its future occurrence.

Though we might try to change another person's behavior using behavior modification, we can also change our own behavior using **self-monitoring** which refers to measuring and recording one's own ABCs. In the context of psychopathology, behavior modification can be useful in treating phobias, reducing habit disorders, and ridding the person of maladaptive cognitions.

A limitation of this method is that the process of observing and/or recording a behavior can cause the behavior to change, called **reactivity.** Have you ever noticed someone staring at you while you sat and ate your lunch? If you have, what did you do? Did you change your behavior? Did you become self-conscious? Likely yes and this is an example of reactivity. Another issue is that the behavior that is made in one situation may not be made in other situations, such as your significant other only acting out at their favorite team's football game and not at home. This form of validity is called **cross-sectional validity.**

3.1.3.6: Intelligence Tests

Intelligence testing is occasionally used to determine the client's level of cognitive functioning. Intelligence testing consists of a series of tasks asking the patient to use both verbal and nonverbal skills. An example is the *Stanford-Binet Intelligence test* which is used to assess fluid reasoning, knowledge, quantitative reasoning, visual-spatial processing and working memory. These tests are rather time-consuming and require specialized training to administer. As such, they are typically only used in cases where there is a suspected cognitive disorder or intellectual disability. Intelligence tests have been criticized for not predicting future behaviors such as achievement and reflecting social or cultural factors/biases and not actual intelligence.

3.2: Diagnosing and Classifying Abnormal Behavior

Section Learning Objectives

- Explain what it means to make a clinical diagnosis.
- Define syndrome.
- Clarify and exemplify what a classification system does.
- Identify the two most used classification systems.
- Outline the history of the DSM.
- Identify and explain the elements of a diagnosis.
- Outline the major disorder categories of the DSM-5.
- Describe the ICD-10.
- Clarify why the DSM-5 and ICD-11 need to be harmonized.

3.2.1: Clinical Diagnosis and Classification Systems

To begin any type of treatment, the client/patient must be clearly diagnosed with a mental disorder. **Clinical diagnosis** is the process of using assessment data to determine if the pattern of symptoms the person presents with is consistent with the diagnostic criteria for a specific mental disorder set forth in an established classification system such as the DSM-5 or ICD-10 (both will be described shortly). Any diagnosis should have clinical utility, meaning it aids the mental health professional in determining the prognosis, the treatment plan, and possible outcomes of treatment (APA, 2013). Receiving a diagnosis does not necessarily mean the person requires treatment. This decision is made based upon how severe the symptoms are, the level of distress caused by the symptoms, symptom salience such as expressing suicidal ideation, risks and benefits of treatment, disability, and other factors (APA, 2013). Likewise, a patient may not meet full criteria for a diagnosis but require treatment nonetheless.

Symptoms that cluster together on a regular basis are called a **syndrome**. If they also follow the same, predictable course, we say that they are characteristic of a *specific disorder*. **Classification systems** for mental disorders provide mental health professionals with an agreed upon list of disorders falling in distinct categories for which there are clear descriptions and criteria for making a diagnosis. Distinct is the key word here. People experiencing delusions, hallucinations, disorganized speech, catatonia, and/or negative symptoms are different from people presenting with a primary clinical deficit in cognitive functioning that is not developmental in nature but has been acquired (i.e. they have shown a decline in cognitive functioning over time). The former would likely be diagnosed with a schizophrenia spectrum disorder while the latter likely has a neurocognitive disorder (NCD). The latter can be further distinguished from neurodevelopmental disorders which manifest early in development and involve developmental deficits that cause impairments in social, academic, or occupational functioning (APA, 2013). These three disorder groups or categories can be clearly distinguished from one another. Classification systems also permit the gathering of statistics for the purpose of determining incidence and prevalence rates, they facilitate research on the etiology and treatment of disorders, and they conform to the requirements of insurance companies for the payment of claims.

The most widely used classification system in the United States is the *Diagnostic and Statistical Manual of Mental Disorders* currently in its 5th edition and produced by the American Psychiatric Association (APA, 2013). Alternatively, the World Health Organization (WHO) produces the *International Statistical Classification of Diseases and Related Health Problems (ICD)* currently in its 10th edition with an 11th edition expected to be published in 2018. We will begin by discussing the DSM and then move to the ICD.

3.2.2: The DSM Classification System

3.2.2.1: A Brief History of the DSM

The DSM 5 was published in 2013 and took the place of the DSM IV-TR (TR means Text Revision; published in 2000) but the history of the DSM goes back to 1844 when the American Psychiatric Association published a predecessor of the DSM which was a "statistical classification of institutionalized mental patients" and "…was designed to improve communication about the types of patients cared for in these hospitals" (APA, 2013, p. 6). However, the first official version of the DSM was not published until 1952. The DSM evolved through four subsequent editions after World War II into a diagnostic classification system to be used by psychiatrists and physicians, but also other mental health professionals. The Herculean task of revising the DSM IV-TR began in 1999 when the APA embarked upon an evaluation of the strengths and weaknesses of the DSM in coordination with the World Health Organization (WHO) Division of Mental Health, the World Psychiatric Association, and the National Institute of Mental Health (NIMH). This resulted in the publication of a monograph in 2002 called, *A Research Agenda for DSM-V*. From 2003 to 2008, the APA, WHO, NIMH, the National Institute on Drug Abuse (NIDA), and the National Institute on Alcoholism and Alcohol Abuse (NIAAA) convened 13 international DSM-5 research planning conferences, "to review the world literature in specific diagnostic areas to prepare for revisions in developing both DSM-5 and the International Classification of Disease, 11th Revision (ICD-11)" (APA, 2013).

After the naming of a DSM-5 Task Force Chair and Vice-Chair in 2006, task force members were selected and approved by 2007 and workgroup members were approved in 2008. What resulted from this was an intensive process of "conducting literature reviews and secondary analyses, publishing research reports in scientific journals, developing draft diagnostic criteria, posting preliminary drafts on the DSM-5 Web site for public comment, presenting preliminary findings at professional meetings, performing field trials, and revisiting criteria and text"(APA, 2013).

What resulted was a "common language for communication between clinicians about the diagnosis of disorders" along with a realization that the criteria and disorders contained within were based on current research and may undergo modification with new evidence gathered (APA, 2013). Additionally, some disorders were not included within the main body of the document because they did not have the scientific evidence to support their widespread clinical use, but were included in Section III under "Conditions for Further Study" to "highlight the evolution and direction of scientific advances in these areas to stimulate further research" (APA, 2013).

3.2.2.2: Elements of a Diagnosis

The DSM 5 states that the following make up the key elements of a diagnosis (APA, 2013):

- **Diagnostic Criteria and Descriptors** – Diagnostic criteria are the guidelines for making a diagnosis. When the full criteria are met, mental health professionals can add severity and course specifiers to indicate the patient's current presentation. If the full criteria are not met, designators such as "other specified" or "unspecified" can be used. If applicable, an indication of severity (mild, moderate, severe, or extreme), descriptive features, and course (type of remission – partial or full – or recurrent) can be provided with the diagnosis. The final diagnosis is based on the clinical interview, text descriptions, criteria, and clinical judgment.
- **Subtypes and Specifiers** – Since the same disorder can be manifested in different ways in different individuals the DSM uses subtypes and specifiers to better characterize an individual's disorder. *Subtypes* denote "mutually exclusive and jointly exhaustive phenomenological subgroupings within a diagnosis" (APA, 2013).

For example, non-rapid eye movement sleep arousal disorders can have either a sleepwalking or sleep terror type. Enuresis is nocturnal only, diurnal only, or both. *Specifiers* are not mutually exclusive or jointly exhaustive and so more than one specifier can be given. For instance, binge eating disorder has remission and severity specifiers. Major depressive disorder has a wide range of specifiers that can be used to characterize the severity, course, or symptom clusters. Again the fundamental distinction between subtypes and specifiers is that there can be only one subtype but multiple specifiers.

- **Principle Diagnosis** – A *principal diagnosis* is used when more than one diagnosis is given for an individual (when an individual has *comorbid disorders).* The principal diagnosis is the reason for the admission in an inpatient setting or the reason for a visit resulting in ambulatory care medical services in outpatient settings. The principal diagnosis is generally the main focus of treatment.
- **Provisional Diagnosis** – If not enough information is available for a mental health professional to make a definitive diagnosis, but there is a strong presumption that the full criteria will be met with additional information or time, then the *provisional* specifier can be used.

3.2.3: DSM-5 Disorder Categories

The DSM-5 includes the following categories of disorders:

Table 3.1. DSM-5 Classification System of Mental Disorders

Disorder Category	Short Description
Neurodevelopmental Disorders	A group of conditions that arise in the developmental period and include intellectual disability, communication disorders, autism spectrum disorder, motor disorders, and ADHD
Schizophrenia Spectrum and Other Psychotic Disorders	Disorders characterized by one or more of the following: delusions, hallucinations, disorganized thinking and speech, disorganized motor behavior, and negative symptoms
Bipolar and Related Disorders	Characterized by mania or hypomania and possibly depressed mood; includes Bipolar I and II, cyclothymic disorder
Depressive Disorders	Characterized by sad, empty, or irritable mood, as well as somatic and cognitive changes that affect functioning; includes major depressive and persistent depressive disorders
Anxiety Disorders	Characterized by excessive fear and anxiety and related behavioral disturbances; Includes phobias, separation anxiety, panic attack, generalized anxiety disorder
Obsessive-Compulsive and Related Disorders	Characterized by obsessions and compulsions and includes OCD, hoarding, and body dysmorphic disorders
Trauma- and Stressor-Related Disorders	Characterized by exposure to a traumatic or stressful event; PTSD, acute stress disorder, and adjustment disorders
Dissociative Disorders	Characterized by a disruption or disturbance in memory, identity, emotion, perception, or behavior; dissociative identity disorder, dissociative amnesia, and depersonalization/derealization disorder
Somatic Symptom and Related Disorders	Characterized by prominent somatic symptoms to include illness anxiety disorder somatic symptom disorder, and conversion disorder
Feeding and Eating Disorders	Characterized by a persistent disturbance of eating or eating-related behavior to include bingeing and purging
Elimination Disorders	Characterized by the inappropriate elimination of urine or feces; usually first diagnosed in childhood or adolescence
Sleep-Wake Disorders	Characterized by sleep-wake complaints about the quality, timing, and amount of sleep; includes insomnia, sleep terrors, narcolepsy, and sleep apnea
Sexual Dysfunctions	Characterized by sexual difficulties and include premature ejaculation, female orgasmic disorder, and erectile disorder
Gender Dysphoria	Characterized by distress associated with the incongruity between one's experienced or expressed gender and the gender assigned at birth
Disruptive, Impulse-Control, and Conduct Disorders	Characterized by problems in self-control of emotions and behavior and involve the violation of the rights of others and cause the individual to be in violation of societal norms; Includes oppositional defiant disorder, antisocial personality disorder, kleptomania, etc.
Substance-Related and Addictive Disorders	Characterized by the continued use of a substance despite significant problems related to its use
Neurocognitive Disorders	Characterized by a decline in cognitive functioning over time and the NCD has not been present since birth or early in life
Personality Disorders	Characterized by a pattern of stable traits which are inflexible, pervasive, and leads to distress or impairment
Paraphilic Disorders	Characterized by recurrent and intense sexual fantasies that can cause harm to the individual or others; includes exhibitionism, voyeurism, and sexual sadism

3.2.4: The ICD-10

In 1893, the International Statistical Institute adopted the International List of Causes of Death which was the first edition of the ICD. The World Health Organization was entrusted with the development of the ICD in 1948 and published the 6th version (ICD-6), which was the first version to include mental disorders. The ICD-10 was

endorsed in May 1990 by the 43rd World Health Assembly. The WHO states:

ICD is the foundation for the identification of health trends and statistics globally, and the international standard for reporting diseases and health conditions. It is the diagnostic classification standard for all clinical and research purposes. ICD defines the universe of diseases, disorders, injuries and other related health conditions, listed in a comprehensive, hierarchical fashion that allows for:

- easy storage, retrieval and analysis of health information for evidence-based decision-making;
- sharing and comparing health information between hospitals, regions, settings, and countries;
- and data comparisons in the same location across different time periods.

Source: *http://www.who.int/classifications/icd/en/*

The *ICD* lists many types of diseases and disorders and includes Chapter V: Mental and Behavioral Disorders. The list of mental disorders is broken down as follows:

- Organic, including symptomatic, mental disorders
- Mental and behavioral disorders due to psychoactive substance use
- Schizophrenia, schizotypal and delusional disorders
- Mood (affective) disorders
- Neurotic, stress-related and somatoform disorders
- Behavioral syndromes associated with physiological disturbances and physical factors
- Disorders of adult personality and behavior
- Mental retardation
- Disorders of psychological development
- Behavioral and emotional disorders with onset usually occurring in childhood and adolescence
- Unspecified mental disorder

3.2.5: Harmonization of DSM-5 and ICD-11

As noted earlier, the ICD-11 is currently in development with an expected publication date in 2018. According to the DSM-5, there is an effort to harmonize the two classification systems so that there can be a more accurate collection of national health statistics and design of clinical trials, increased ability to replicate scientific findings across national boundaries and to rectify the lack of agreement between the DSM-IV and ICD-10 diagnoses. (APA, 2013).

3.3: Treatment of Mental Disorders – An Overview

Section Learning Objectives

- Clarify reasons why an individual may need to seek treatment.
- Critique myths about psychotherapy.

3.3.1: **Seeking Treatment**

3.3.1.1: **Who Seeks Treatment?**

Would you describe the people who seek treatment as being on the brink, crazy, or desperate? Or can the ordinary Joe in need of advice seek out mental health counseling? The answer is that anyone can. David Sack, M.D. (2013) writes in an article entitled, *5 Signs Its Time to Seek Therapy*, published in Psychology Today, that "most people can benefit from therapy at some point in their lives" and that though the signs one needs to seek help are obvious at times, people often try "to sustain [their] busy life until it sets in that life has become unmanageable." So when should we seek help? First, if we feel sad, angry, or not like ourselves. We might be withdrawing from friends and families or sleeping more or less than we usually do. Second, if we are abusing drugs, alcohol, food, or sex to deal with life's problems. In this case, our coping skills may need some work. Third, in instances when we have lost a loved one or something else important to us, whether due to a death or divorce, the grief may be too much to process. Fourth, a traumatic event may have occurred such as abuse, a crime, an accident, chronic illness, or rape. Finally, if we have stopped doing the things we enjoy the most. Sack (2013) says, "If you decide that therapy is worth a try, it doesn't mean you're in for a lifetime of "head shrinking." In fact, a 2001 study in the Journal of Counseling Psychology found that most people feel better within seven to 10 visits. In another study, published in 2006 in the Journal of Consulting and Clinical Psychology, 88 percent of therapy-goers reported improvements after just one session."

For more on this article, please visit:

https://www.psychologytoday.com/blog...e-seek-therapy

3.3.1.2: **When Friends, Family, and Self-Healing are Not Enough**

If you are experiencing any of the aforementioned issues, you should seek help. Instead of facing the potential stigma of talking to a mental health professional, many people think that talking through their problems with friends or family is just as good. Though you will ultimately need these people to see you through your recovery, they do not have the training and years of experience that a psychologist or similar professional has. "Psychologists can recognize behavior or thought patterns objectively, more so than those closest to you who may have stopped noticing — or maybe never noticed. A psychologist might offer remarks or observations similar to those in your existing relationships, but their help may be more effective due to their timing, focus or your trust in their neutral stance" (http://www.apa.org/helpcenter/psychotherapy-myths.aspx). You also should not wait to recover on your own. It is not a failure to admit you need help and there could be a biological issue that makes it almost impossible to heal yourself.

3.3.1.3: **Prevention**

As a society, we often to wait for a mental or physical health issue to emerge and then we scramble to treat it. More recently, medicine and science have taken a **prevention** stance which involves identifying the factors that cause specific mental health issues and implementing interventions to stop them from happening, or at least minimize their deleterious effects. Our focus has shifted from individuals to the population. Mental health promotion programs have been instituted with success in schools (Shoshani & Steinmetz, 2014; Weare & Nind, 2011; Berkowitz & Bier, 2007), in the workplace (Czabała, Charzyńska, & Mroziak, B., 2011), with undergraduate and graduate students (Conley et al., 2017; Bettis et al., 2017), in relation to bullying (Bradshaw, 2015), and with the elderly (Forsman et al., 2011). Many researchers believe the time is ripe to move from knowledge to action and to expand public mental health initiatives (Wahlbeck, 2015).

3.3.1.4: **So What Exactly is Psychotherapy?**

APA states that in **psychotherapy**, "psychologists apply scientifically validated procedures to help people develop healthier, more effective habits." Several different approaches can be utilized to include behavior, cognitive and cognitive-behavior, humanistic-experiential, psychodynamic, couples and family, and biological therapies/treatments. (article quoted can be found at: http://www.apa.org/helpcenter/understanding-psychotherapy.aspx)

3.3.1.5: The Client-Therapist Relationship

What is key is the client-therapist relationship. APA says, “Psychotherapy is a collaborative treatment based on the relationship between an individual and a psychologist. Grounded in dialogue, it provides a supportive environment that allows you to talk openly with someone who’s objective, neutral and nonjudgmental. You and your psychologist will work together to identify and change the thought and behavior patterns that are keeping you from feeling your best.” It’s not just about solving the problem you saw the therapist for, but also about learning new skills to better help you cope in the future when faced with the same or similar environmental stressors.

So how do you find a psychotherapist? Several strategies may prove fruitful. You could ask family and friends, your primary care physician (PCP), look online, consult an area community mental health center, your local university’s psychology department, state psychological association, or use APA’s Psychologist Locator Service (https://locator.apa.org/?_ga=2.160567293.1305482682.1516057794-1001575750.1501611950). Once you find a list of psychologists or other practitioners, choose the right one for you by determining if you plan on attending alone or with family, what you wish to get out of your time with a psychotherapist, how much your insurance company pays for (and if you have to pay out of pocket how much you can afford), when you can attend sessions, and how far you are willing to travel. Once you have done this, make your first appointment.

But what should you bring? APA suggests, “To make the most of your time, make a list of the points you want to cover in your first session and what you want to work on in psychotherapy. Be prepared to share information about what’s bringing you to the psychologist. Even a vague idea of what you want to accomplish can help you and your psychologist proceed efficiently and effectively.” Additionally, they suggest taking report cards, a list of medications, information on the reasons for a referral, a notebook, a calendar to schedule future visits if needed, and a form of payment.

What should you expect? Your therapist and you will work to develop a full history which could take several visits. From this, a treatment plan will be developed. “This collaborative goal-setting is important, because both of you need to be invested in achieving your goals. Your psychologist may write down the goals and read them back to you, so you’re both clear about what you’ll be working on. Some psychologists even create a treatment contract that lays out the purpose of treatment, its expected duration and goals, with both the individual’s and psychologist’s responsibilities outlined.”

After the initial visit, the mental health professional may conduct tests to further understand your condition but will definitely continue talking through the issue. He/she may even suggest involving others especially in cases of relationship issues. Resilience is a skill that will be taught so that you can better handle future situations.

3.3.1.6: Does it Work?

APA writes, “Reviews of these studies show that about 75 percent of people who enter psychotherapy show some benefit. Other reviews have found that the average person who engages in psychotherapy is better off by the end of treatment than 80 percent of those who don’t receive treatment at all.” Treatment works due to finding an evidence-based treatment that is specific for the person’s problem; the expertise of the therapist; and the characteristics, values, culture, preferences, and personality of the client.

3.3.1.7: How Do You Know You are Finished?

“How long psychotherapy takes depends on several factors: the type of problem or disorder, the patient’s characteristics and history, the patient’s goals, what’s going on in the patient’s life outside psychotherapy and how fast the patient is able to make progress.” It is important to note that psychotherapy is not a lifelong commitment and it is a joint decision of client and therapist as to when it ends. Once over, expect to have a periodic check-up with your therapist. This might be weeks or even months after your last session. If you need to see him/her sooner, schedule an appointment. APA calls this a “mental health tune up” or a “booster session.”

For more on psychotherapy, please see the very interesting APA article on this matter:

http://www.apa.org/helpcenter/unders...hotherapy.aspx

Chapter Recap

With the conclusion of Chapter 3, you now have the necessary foundation to understand each of the groups of disorders we discuss in the remaining chapters. In Chapter 3 we discussed clinical assessment, diagnosis, and treatment. In terms of assessment, we covered key concepts such as reliability, validity, and standardization; and discussed methods of assessment such as the clinical

interview, psychological tests, personality inventories, neurological tests, the physical examination, behavioral assessment, and intelligence tests. In terms of diagnosis, we discussed the classification systems of the DSM-5 and ICD-10. For treatment, we discussed reasons why someone may seek treatment, self-treatment, psychotherapy, the client-therapist relationship, and evidence for the success of psychotherapy. We discussed some of the specific therapies in Chapter 3 but will cover others throughout this book and in terms of the disorders they are used to treat.

CHAPTER OVERVIEW

4: Anxiety Disorders

Learning Objectives

- Describe the various anxiety disorders and their symptoms.
- Describe the epidemiology of anxiety disorders.
- Describe comorbidity in relation to anxiety disorders.
- Describe treatment options for anxiety disorders.
- Describe the etiology of anxiety disorders.

In Chapter 4, we will discuss matters related to anxiety disorders including their clinical presentation, epidemiology, comorbidity, treatment options, and etiology. Our discussion will include Panic Disorder, Generalized Anxiety Disorder, Specific Phobias, Social Anxiety Disorder, and Agoraphobia. Be sure you refer to Chapters 1-3 for explanations of key terms (Chapter 1), an overview of the various models to explain psychopathology (Chapter 2), and descriptions of the various therapies (Chapter 3).

4.1: Panic Disorder

Section Learning Objectives

- Describe how panic disorder presents itself.
- Describe the epidemiology of panic disorder.
- Indicate which disorders are commonly comorbid with panic disorder.
- Describe the treatment options for panic disorder.

4.1.1: Clinical Description

Panic disorder consists of a series of recurrent, unexpected panic attacks coupled with the fear of future panic attacks. A panic attack is defined as a sudden or abrupt surge or fear or impending doom along with at least four physical or cognitive symptoms (listed below). The symptoms generally peak within a few minutes, although it seems much longer for the individual experiencing the panic attack.

There are two key components to panic disorder—the attacks are *unexpected* meaning there is nothing that triggers them, and they are *recurrent* meaning they occur multiple times. Because these panic attacks occur frequently and essentially "out of the blue," they cause significant worry or anxiety in the individual as they are unsure of when the next attack will occur. In some individuals, significant behavioral changes such as fear of leaving their home or attending large events occur as the individual is fearful an attack will happen in one of these situations, causing embarrassment. Additionally, individuals report worry that other's will think they are "going crazy" or losing control if they were to observe an individual experiencing a panic attack. Occasionally, an additional diagnosis of agoraphobia is given to an individual with panic disorder *if* their behaviors meet diagnostic criteria for this disorder as well (see more below).

The frequency and intensity of these panic attacks vary widely among individuals. Some people report panic attacks occurring once a week for months on end, others report more frequent attacks multiple times a day, but then experience weeks or months without any attacks. The intensity of symptoms also varies among individuals, with some individuals reporting experiencing nearly all 14 symptoms and others only reporting the minimum 4 required for the diagnosis. Furthermore, individuals report variability within their own panic attack symptoms, with some panic attacks presenting with more symptoms than others. It should be noted that at this time, there is no identifying information (i.e. demographic information) to suggest why some individuals experience panic attacks more frequently or more severe than others.

4.1.2: Epidemiology

Prevalence rates for panic disorder are estimated at around 2-3% in adults and adolescents. Higher rates of panic disorder are found in American Indians and non-Latino whites. Females are more commonly diagnosed than males with a 2:1 diagnosis rate—this gender discrepancy is seen throughout the lifespan. Although panic disorder can occur in young children, it is generally not observed in individuals younger than 14 years of age.

4.1.3: Comorbidity

Panic disorder rarely occurs in isolation, as many individuals also report symptoms of other anxiety disorders, major depression, and substance abuse. There is mixed evidence as to whether panic disorder precedes other comorbid psychological disorders—estimates suggest that 1/3 of individuals with panic disorder will experience depressive symptoms prior to panic symptoms whereas the remaining 2/3 will experience depressive symptoms concurrently or after the onset of panic disorder (APA, 2013).

Unlike some of the other anxiety disorders, there is a high comorbid diagnosis with general medical symptoms. More specifically, individuals with panic disorder are more likely to report somatic symptoms such as dizziness, cardiac arrhythmias, asthma, irritable bowel syndrome, and hyperthyroidism (APA, 2013). The relationship between panic symptoms and somatic symptoms is unclear; however, there does not appear to be a direct medical cause between the two.

4.1.4: **Treatment**

4.1.4.1: **Cognitive Behavioral Therapy (CBT)**

CBT is the most effective treatment option for individuals with panic disorder as the focus is on correcting misinterpretations of bodily sensations (Craske & Barlow, 2014). Nearly 80 percent of people with panic disorder report complete remission of

symptoms after mastering the following five components of CBT for panic disorder (Craske & Barlow, 2014).

1. **Psychoeducation.** Treatment begins by educating the client on the nature of panic disorder, the underlying causes of panic disorder, as well as the mechanisms that maintain the disorder such as the physical, cognitive, and behavioral response systems (Craske & Barlow, 2014). This part of treatment is fundamental in correcting any myths or misconceptions about panic symptoms, as they often contribute to the exacerbation of panic symptoms.
2. **Self-monitoring**. Self-monitoring, or the awareness of self-observation, is essential to the CBT treatment process for panic disorder. In this part of treatment, the individual is taught to identify the physiological cues immediately leading up to and during a panic attack. The client is then encouraged to identify and document/record the thoughts and behaviors associated with these physiological symptoms. By bringing awareness to the symptoms, as well as the relationship between physical arousal and cognitive/behavioral responses, the client is learning the fundamental processes in which they can manage their panic symptoms (Craske & Barlow, 2014).
3. **Relaxation training**. Prior to engaging in exposure training, the individual must learn a relaxation technique to apply during the onset of panic attacks. While breathing training was once included as the relaxation training technique of choice for panic disorder, due to the high report of hyperventilation during panic attacks more recent research has failed to support this technique as effective in the use of panic disorder (Schmidt et al., 2000). Findings suggest that breathing retraining is more commonly misused as a means for avoiding physical symptoms as opposed to an effective physiological response to stress (Craske & Barlow, 2014). To replace the breathing retraining, Craske & Barlow (2014) suggest **progressive muscle relaxation** (PMR). In PMR, the client learns to tense and relax various large muscle groups throughout the body. Generally speaking, the client is encouraged to start at either the head or the feet, and gradually work their way up through the entire body, holding the tension for roughly 10 seconds before relaxing. The theory behind PMR is that in tensing the muscles for a prolonged period of time, the individual exhausts those muscles, forcing them (and eventually) the entire body to engage in relaxation (McCallie, Blum, & Hood, 2006).
4. **Cognitive restructuring**. Cognitive restructuring, or the ability to recognize cognitive errors and replace them with alternate, more appropriate thoughts, is likely the most powerful part of CBT treatment for panic disorder, aside from the exposure part. Cognitive restructuring involves identifying the role of thoughts in generating and maintaining emotions. The clinician encourages the individual to view these thoughts as “hypotheses” as opposed to facts, which allows the thoughts to be questioned and challenged. This is where the detailed recordings in the self-monitoring section of treatment are helpful. By discussing specifically what the client has recorded for the relationship between physiological arousal and thoughts/behaviors, the clinician is able to help the individual restructure the maladaptive thought processes to more positive thought processes which in return, helps to reduce fear and anxiety.
5. **Exposure**. Next, the client is encouraged to engage in a variety of exposure techniques such as in vivo exposure and *interoceptive exposure*, while also incorporating the cognitive restructuring and relaxation techniques previously learned in efforts to reduce and eliminate ongoing distress. **Interoceptive exposure** involves inducing panic specific symptoms to the individual repeatedly, for a prolonged time period, so that maladaptive thoughts about the sensations can be disconfirmed and conditional anxiety responses are extinguished (Craske & Barlow, 2014). Some examples of these exposure techniques are spinning a client repeatedly in a chair to induce dizziness and breathing in a paper bag to induce hyperventilation. These treatment approaches can be presented in a gradual manner; however, the client must endure the physiological sensations for at least 30 seconds to 1 minute to ensure adequate time for applying cognitive strategies to misappraisal of cognitive symptoms (Craske & Barlow, 2014). Interoceptive exposure is continued both in and outside of treatment until panic symptoms remit. Over time, the habituation of fear within an exposure session will ultimately lead to habituation across treatment, which leads to long-term remission of panic symptoms (Foa & McNally, 1996). Occasionally, panic symptoms will return in individuals who report complete remission of panic disorder. Follow-up booster sessions reviewing the steps above is generally effective in eliminating symptoms again.

4.1.4.2: Pharmacological Interventions

According to Craske & Barlow (2014), nearly half of people with panic disorder present to psychotherapy already on medication, likely prescribed by their primary care physician. Some researchers argue that anti-anxiety medications impede the progress of CBT treatment as the individual is not able to fully experience the physiological sensations during exposure sessions, thus limiting their ability to modify maladaptive thoughts maintaining the panic symptoms. Results from large clinical trials suggest *no advantage* during or immediately after treatment of combining CBT and medication (Craske & Barlow, 2014). Additionally, when medications were discontinued post-treatment, the CBT+ medication groups fared worse than the CBT treatment alone groups, thus

supporting the theory that immersion in interoceptive exposure is limited by the use of medication. Therefore, it is suggested that medications are reserved for those who do not respond to CBT therapy alone (Kampman, Keijers, Hoogduin & Hendriks, 2002).

4.2: Generalized Anxiety Disorder

Section Learning Objectives

- Describe how generalized anxiety disorder presents itself.
- Describe the epidemiology of generalized anxiety disorder.
- Indicate which disorders are commonly comorbid with generalized anxiety disorder.
- Describe the treatment options for generalized anxiety disorder.

4.2.1: **Clinical Description**

Generalized anxiety disorder, commonly referred to as GAD, is a disorder characterized by an underlying excessive worry related to a wide range of events or activities. While many individuals experience some levels of worry throughout the day, individuals with GAD experience worry of a greater intensity and for longer periods of times than the average person. Additionally, they are often unable to control their worry through various coping strategies, which directly interferes with their ability to engage in daily social and occupational tasks. There are six characteristic symptoms of generalized anxiety disorder and in order to be diagnosed with the disorder, individuals must experience at least three of them. These symptoms are: feeling restless, being easily fatigued, having difficulty concentrating, feeling irritable, having muscle tension, experiencing problems with sleep.

4.2.2: Epidemiology

The prevalence rate for generalized anxiety disorder is estimated to be 3% of the general population, with nearly 6% of individuals experiencing GAD sometime during their lives. While it can present at any age, it generally appears first in childhood or adolescence. Similar to most anxiety-related disorders, females are twice as likely to be diagnosed with GAD as males (APA, 2013).

4.2.3: Comorbidity

There is a high comorbidity between generalized anxiety disorder and the other anxiety-related disorders, as well as major depressive disorder, suggesting they all share common vulnerabilities, both biological and psychological.

4.2.4: Treatment

4.2.4.1: **Psychopharmacology**

Benzodiazepines, a class of sedative-hypnotic drugs, originally replaced barbiturates as the leading anti-anxiety medication due to their less addictive nature, yet equally effective ability to calm individuals at low dosages. Unfortunately, as more research was conducted on benzodiazepines, serious side effects, as well as physical dependence have routinely been documented (NIMH, 2013). Due to these negative effects, selective serotonin-reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) are generally considered to be first-line medication options for those with GAD. Findings indicate a 30-50% positive response rate to these psychopharmacological interventions (Reinhold & Rickels, 2015). Unfortunately, none of these medications continue to provide any benefit once they are stopped; therefore, other more effective treatment options such as CBT, relaxation training, and biofeedback are often encouraged before the use of pharmacological interventions.

4.2.4.2: **Rational-Emotive Therapy**

Rational emotive therapy was developed by Albert Ellis in the mid-1950s as one of the first forms of cognitive-behavioral therapy. Ellis proposed that individuals were not aware of the effect their negative thoughts had on their behaviors and various relationships and thus, identified a treatment aimed to address these thoughts in an effort to provide relief to those experiencing anxiety and depression. The goal of rational emotive therapy is to identify irrational, self-defeating assumptions, challenge the rationality of those assumptions, and to replace them with new more productive thoughts and feelings. It is proposed that through identifying and replacing these assumptions that one will experience relief of GAD symptoms (Ellis, 2014).

4.2.4.3: **Cognitive Behavioral Therapy (CBT)**

CBT is among the most effective treatment options for a variety of anxiety disorders, including GAD. In fact, findings suggest 60% of individuals report a significant reduction/elimination in anxious thoughts one-year post-treatment (Hanrahan, Field, Jones, & Davy, 2013). The fundamental goal of CBT is a combination of cognitive and behavioral strategies aimed to identify and restructure maladaptive thoughts while also providing opportunities to utilize these more effective thought patterns through

exposure based experiences. Through repetition, the individual will be able to identify and replace anxious thoughts outside of therapy sessions, ultimately reducing their overall anxiety levels (Borkovec, & Ruscio, 2001).

4.2.4.4: **Biofeedback**

Biofeedback provides a visual representation of a clients's physiological arousal. To achieve this feedback, a client is connected to a computer that provides continuous information on their physiological states. There are several ways a client can be connected to the computer. Among the most common is **electromyography (EMG)**. EMG measures the amount of muscle activity currently experienced by the individual. An electrode is placed on a individuals's skin just above a major muscle group- commonly the forearm or the forehead. Other common types of measurement are **electroencephalography** (EEG) which measures the neurofeedback or brain activity; **heart rate variability** (HRV) which measures autonomic activity such as heart rate or blood pressure; and **galvanic skin response** (GSR) which measures sweat.

Once the client is connected to the biofeedback machine, the clinician is able to walk the client through a series of relaxation scripts or techniques as the computer simultaneously measures the changes in muscle tension. The theory behind biofeedback is that in providing a client with a visual representation of changes in their physiological state, they become more skilled at voluntarily reducing their physiological arousal, and thus, their overall sense of anxiety or stress. While research has identified only a modest effect of biofeedback on anxiety levels, clients do report a positive experience with the treatment due to the visual feedback of their physiological arousal (Brambrink, 2004).

4.3: Specific Phobia

Section Learning Objectives

- Describe how specific phobia presents itself.
- Describe the epidemiology of specific phobia.
- Indicate which disorders are commonly comorbid with specific phobia.
- Describe the treatment options for specific phobia.

4.3.1: Clinical Description

Specific phobia is distinguished by an individual's fear or anxiety specific to an object or a situation. While the amount of fear or anxiety related to the specific object or situation varies among individuals, it also varies related to the proximity of the object/situation. When individuals are face-to-face with their specific phobia, immediate fear is present. It should also be noted that these fears are more excessive and more persistent than a "normal" fear, often severely impacting one's daily functioning (APA, 2013).

Individuals can experience multiple specific phobias at one time. In fact, nearly 75% of individuals with a specific phobia report fear in more than one object (APA, 2013). When making a diagnosis of specific phobia, it is important to identify the specific phobic stimulus. Among the most commonly diagnosed specific phobias are animals, natural environments (height, storms, water), blood-injection-injury (needles, invasive medical procedures), or situational (airplanes, elevators, enclosed places; APA, 2013). Given the high percentage of individuals who experience more than one specific phobia, all specific phobias should be listed as a diagnosis in efforts to identify an appropriate treatment plan.

4.3.2: Epidemiology

The prevalence rate for specific phobias is 7-9% within the united states. While young children have a prevalence rate of approximately 5%, teens have nearly a double prevalence rate than that of the general public at 16%. There is a 2:1 ratio of females to males diagnosed with specific phobia; however, this rate changes depending on the different phobic stimuli. More specifically, animal, natural environment, and situational specific phobias are more commonly diagnosed in females, whereas blood-injection-injury phobia is reportedly diagnosed equally between genders.

4.3.3: Comorbidity

Seeing as the onset of specific phobias occurs at a younger age than most other anxiety disorders, it is generally the primary diagnosis with generalized anxiety disorder as an occasional comorbid diagnosis. It should be noted that children/teens diagnosed with a specific phobia are at an increased risk for additional psychopathology later in life. More specifically, other anxiety disorders, depressive disorders, substance-related disorders and somatic symptom disorders.

4.3.4: Treatment

4.3.4.1: **Exposure Treatments**

While there are many treatment options for specific phobias, research routinely supports the behavioral techniques as the most effective treatment strategies. Seeing as the behavioral theory suggests phobias are developed via classical conditioning, the treatment approach revolves around breaking the maladaptive association developed between the object and fear. This is generally accomplished through **exposure treatments**. As the name implies, the individual is *exposed* to their feared stimuli. This can be done using several different approaches: *systematic desensitization, flooding, and modeling.*

Systematic desensitization is an exposure technique that utilizes relaxation strategies to help calm the individual as they are presented with the fearful object. The notion behind this technique is that both fear and relaxation cannot exist at the same time; therefore, the individual is taught how to replace their fearful reaction with a calm, relaxing reaction. To begin, the client, with assistance from the clinician, will identify a *fear hierarchy*, or a list of feared objects/situations ordered from least fearful to most fearful. After learning intensive relaxation techniques, the clinician will present items from the fear hierarchy- starting from the least fearful object/subject- while the patient practices using the learned relaxation techniques. The presentation of the feared object/situation can be in person (**in vivo exposure)** or it can be imagined (**imaginal exposure).** Imaginal exposure tends to be less intensive than in vivo exposure; however, it is less effective than in vivo exposure in eliminating the phobia. Depending on the phobia, in vivo exposure may not be an option, such as with a fear of a tornado. Once the patient is able to effectively employ

relaxation techniques to reduce their fear/anxiety to a manageable level, the clinician will slowly move up the fear hierarchy until the individual does not experience excessive fear of any objects on the list.

Another exposure technique is **flooding.** In flooding, the clinician does not utilize a fear hierarchy, but rather repeatedly exposes the individual to their most feared object/subject. Similar to systematic desensitization, flooding can be done in either in vivo or imaginal exposure. Clearly, this technique is more intensive than the systematic or gradual exposure to feared objects. Because of this, patients are at a greater likelihood of dropping out of treatment, thus not successfully overcoming their phobias.

Finally, **modeling** is a common technique that is used to treat specific phobias (Kelly, Barker, Field, Wilson, & Reynolds, 2010). In this technique, the clinician approaches the feared object/subject while the patient observes. Like the name implies, the clinician models appropriate behaviors when exposed to the feared stimulus, implying that the phobia is irrational. After modeling several times, the clinician encourages the patient to confront the feared stimulus with the clinician, and then ultimately, without the clinician.

4.4: Social Anxiety Disorder

Section Learning Objectives

- Describe how social anxiety disorder presents itself.
- Describe the epidemiology of social anxiety disorder.
- Indicate which disorders are commonly comorbid with social anxiety disorder.
- Describe the treatment options for social anxiety disorder.

4.4.1: Clinical Description

For social anxiety disorder (formerly known as social phobia), the anxiety is directed toward the fear of social situations, particularly those in which an individual can be evaluated by others. More specifically, the individual is worried that they will be judged negatively and viewed as stupid, anxious, crazy, unlikeable, or boring to name a few. Some individuals report feeling concerned that their anxiety symptoms will be obvious to others via blushing, stuttering, sweating, trembling, etc. These fears severely limit an individual's behavior in social settings. For example, an individual may avoid holding drinks or plates if they know they will tremble in fear of dropping or spilling food/water. Additionally, if one is known to sweat a lot in social situations, they may limit physical contact with others, refusing to shake hands.

Unfortunately, for those with social anxiety disorder, all or nearly all social situations provoke this intense fear. Some individuals even report significant anticipatory fear days or weeks before a social event is to occur. This anticipatory fear often leads to avoidance of social events in some individuals; others will attend social events with a marked fear of possible threats. Because of these fears, there is a significant impact on one's social and occupational functioning.

It is important to note that the cognitive interpretation of these social events is often excessive and out of proportion to the actual risk of being negatively evaluated. There are instances where one may experience anxiety toward a real threat such as bullying or ostracizing. In this instance, social anxiety disorder would not be diagnosed as the negative evaluation and threat are real.

4.4.2: Epidemiology

The overall prevalence rate of social anxiety disorder is significantly higher in the United States than in other countries worldwide, with an estimated 7% of the US population diagnosed with social anxiety disorder. Within the US, the prevalence rate remains the same among children through adults; however, there appears to a significant decrease in the diagnosis of social anxiety disorder among older individuals. With regards to gender, there is a higher diagnosis rate in females than males. This gender discrepancy appears to be larger in children/adolescents than adults.

4.4.3: Comorbidity

Among the most common comorbid diagnoses with social anxiety disorder are other anxiety-related disorders, major depressive disorder, and substance-related disorders. Generally speaking, social anxiety disorders will precede that of other mental health disorders, with the exception of separation anxiety disorder and specific phobia, seeing as these two disorders are more commonly diagnosed in childhood (APA, 2013). The high comorbidity rate among anxiety-related disorders and substance-related disorders is likely related to the efforts of self-medicating. For example, an individual with social anxiety disorder may consume larger amounts of alcohol in social settings in efforts to alleviate the anxiety of the social situation.

4.4.4: Treatment

4.4.4.1: **Exposure**

A hallmark treatment approach for all anxiety disorders is exposure. Specific to social anxiety disorder, the individual is encouraged to engage in social situations where they are likely to experience increased anxiety. Initially, the clinician will engage in role-playing of various social situations with the client so that he/she can practice social interactions in a safe, controlled environment (Rodebaugh, Holaway, & Heimberg, 2004). As the client becomes habituated to the int4.5: Agoraphobiaeraction with the clinician, the clinician and client may venture outside of the treatment room and engage in social settings with random strangers at various locations such as fast food restaurants, local stores, libraries, etc. The client is encouraged to continue with these exposure based social interactions outside of treatment to help reduce anxiety related to social situations.

4.4.4.2: Social Skills Training

This treatment is specific to social anxiety disorder as it focuses on skill deficits or inadequate social interactions displayed by the client that contributes to the negative social experiences and anxiety. The clinician may use a combination of skills such as modeling, corrective feedback, and positive reinforcement to provide feedback and encouragement to the client regarding his/her behavioral interactions (Rodebaugh, Holaway, & Heimberg, 2004). By incorporating the clinician's feedback into their social repertoire, the client can engage in positive social behaviors outside of the treatment room in hopes to improve overall social interactions and reduce ongoing social anxiety.

4.4.4.3: Cognitive Restructuring

While exposure and social skills training are helpful treatment options, research routinely supports the need to incorporate cognitive restructuring as an additive component in treatment to provide substantial symptom reduction. Here the client will work with the therapist to identify negative, automatic thoughts that contribute to the distress in social situations. The clinician can then help the client establish new, positive thoughts to replace these negative thoughts. Research indicates that implementing cognitive restructuring techniques before, during, and after exposure sessions enhances the overall effects of treatment of social anxiety disorder (Heimberg & Becker, 2002).

4.5: Agoraphobia

Section Learning Objectives

- Describe how agoraphobia presents itself.
- Describe the epidemiology of agoraphobia.
- Indicate which disorders are commonly comorbid with agoraphobia.
- Describe the treatment options for agoraphobia.

4.5.1: **Clinical Description**

Similar to GAD, agoraphobia is defined as an intense fear triggered by a wide range of situations; however, unlike GAD, agoraphobia's fears are related to situations in which the individual is in public situations where escape may be difficult. In order to receive a diagnosis of agoraphobia, there must be a presence of fear in at least two of the following situations: using public transportation such as planes, trains, ships, buses; being in large, open spaces such as parking lots or on bridges; being in enclosed spaces like stores or movie theaters; being in a large crowd similar to those at a concert; or being outside of the home in general (APA, 2013). When an individual is in one (or more) of these situations, they experience significant fear, often reporting panic-like symptoms (see Panic Disorder). It should be noted that fear and anxiety related symptoms are present *every time* the individual is presented with these situations. Should symptoms only occur occasionally, a diagnosis of agoraphobia is not warranted.

Due to the intense fear and somatic symptoms, individuals will go to great lengths to avoid these situations, often preferring to remain within their home where they feel safe, thus causing significant impairment in one's daily functioning. They may also engage in active avoidance, where the individual will intentionally avoid agoraphobic situations. These avoidance behaviors may be behavioral, including having food delivery to avoid going to grocery store or only taking a job that does *not* require the use of public transportation, or cognitive, by using distraction and various other cognitive techniques to successfully get through the agoraphobic situation.

4.5.2: Epidemiology

The yearly prevalence rate for agoraphobia across the lifespan is roughly 1.7%. Females are twice as likely as males to be diagnosed with agoraphobia (notice the trend…). While it can occur in childhood, agoraphobia typically does not develop until late adolescence/early adulthood and typically tapers off in later adulthood.

4.5.3: Comorbidity

Similar to the other anxiety disorders, comorbid diagnoses include other anxiety disorders, depressive disorders, and substance use disorders, all of which typically occur after the onset of agoraphobia (APA, 2013). Additionally, there is also a high comorbidity between agoraphobia and PTSD. While agoraphobia can be a symptom of PTSD, an additional diagnosis of agoraphobia is made when all symptoms of agoraphobia are met in addition to the PTSD symptoms.

4.5.4: Treatment

Similar to the treatment approaches for specific phobias, exposure-based treatment techniques are among the most effective treatment options for individuals with agoraphobia; however, unlike the high success rate in specific phobias, exposure-based treatment for agoraphobia has been less effective in providing complete relief of the disorder. The success rate may be impacted by the high comorbidity rate of agoraphobia and panic disorder. Because of the additional presentation of panic symptoms, exposure-based treatments alone are not the most effective in eliminating symptoms as residual panic symptoms often remain (Craske & Barlow, 2014). Therefore, the best treatment approach for those with agoraphobia and panic disorder is a combination of exposure and CBT techniques (see panic disorder treatment).

For individuals with agoraphobia *without* panic symptoms, the use of group therapy in combination with individual exposure-based therapy has been identified as a successful treatment option. The group therapy format allows the individual to engage in exposure-based field trips to various community locations, while also maintaining a sense of support and security from a group of individuals whom they know. Research indicates that this exposure based type of treatment provides improvement for nearly 60% to 80% of patients with agoraphobia; however, there is a relatively high rate of partial relapse suggesting that long-term treatment or booster sessions at a minimum should be continued for several years (Craske & Barlow, 2014).

4.6: Anxiety Disorders Etiology

Section Learning Objectives

- Describe the biological causes of anxiety disorders.
- Describe the psychological causes of anxiety disorders.
- Describe the sociocultural causes of anxiety disorders.

4.6.1: **Biological**

4.6.1.1: **Genetic Influences**

While genetics have been known to contribute to the presentation of anxiety symptoms, the interaction between genetics and stressful environmental influences accounts for more of anxiety disorders than genetics alone (Bienvenu, Davydow, & Kendler, 2011). The quest to identify specific genes that may **predispose** individuals to develop anxiety disorders has lead researchers to the serotonin transporter gene (5-HTTLPR). Mutation of the 5-HTTLPR gene has been found to be related to a reduction in serotonin activity and an increase in anxiety-related personality traits (Munafo, Brown, & Hairiri, 2008).

4.6.1.2: **Neurobiological Structures**

Researchers have identified several brain structures and pathways that are likely responsible for anxiety responses. Among those structures is the **amygdala,** the area of the brain that is responsible for storing memories related to emotional events (Gorman, Kent, Sullivan, & Coplan, 2000). When presented with a fearful situation, the amygdala initiates a reaction in efforts to prepare the body for a response. First, the amygdala triggers the hypothalamic-pituitary-adrenal (HPA) axis to prepare for immediate action—either to fight or flight. The second pathway is activated by the feared stimulus itself, by sending a sensory signal to the **hippocampus** and **prefrontal cortex**, to determine if the threat is real or imagined. If it is determined that no threat is present, the amygdala sends a calming response to the HPA axis, thus reducing the level of fear. If there is a threat present, the amygdala is activated, producing a fear response.

Specific to *panic disorder* is the implication of the **locus coeruleus**, the brain structure that serves as an "on-off" switch for norepinephrine neurotransmitters. It is believed that increased activation of the locus coeruleus results in panic like symptoms; therefore, individuals with panic disorder may have a hyperactive locus coeruleus, leaving them more susceptible to experience more intense and frequent physiological arousal than the general public (Gorman, Kent, Sullivan, & Coplan, 2000). This theory is supported by studies in which individuals experienced increased panic symptoms following injection of norepinephrine (Bourin, Malinge, & Guitton, 1995). Unfortunately, norepinephrine and the locus coeruleus fail to fully explain the development of panic disorder, as treatment would be much easier if *only* norepinephrine was implicated. Therefore, researchers argue that a more complex neuropathway is likely implicated in the development of panic disorder. More specifically, the **corticostriatal-thalamocortical (CSTC) circuit**, also known as the fear-specific circuit, is theorized as a major contributor to panic symptoms (Gutman, Gorman, & Hirsch, 2004). When an individual is presented with a frightening object or situation, the amygdala is activated, sending a fear response to the anterior cingulate cortex and the orbitofrontal cortex. Additional projection from the amygdala to the hypothalamus activates endocrinologic responses to fear- releasing adrenaline and cortisol to help prepare the body to fight or flight (Gutman, Gorman, & Hirsch, 2004). This complex pathway supports the theory that panic disorder is mediated by several neuroanatomical structures and their associated neurotransmitters.

4.6.2: **Psychological**

4.6.2.1: **Cognitive**

The cognitive perspective on the development of anxiety disorders centers around dysfunctional thought patterns. **Maladaptive assumptions** are routinely observed in individuals with anxiety disorders, as they often interpret events as dangerous and overreact to potentially stressful events, which contributes to a heightened overall anxiety level. These **negative appraisals**, in combination with a biological predisposition to anxiety likely contribute to the development of anxiety symptoms (Gallagher et al., 2013).

Sensitivity to physiological arousal not only contributes to anxiety disorders in general, but also for panic disorder where individuals experience various physiological sensations and misinterpret them as catastrophic. One explanation for this theory is that individuals with panic disorder are actually more susceptible to more frequent and intensive physiological symptoms than the general public (Nillni, Rohan, & Zvolensky, 2012). Others argue that these individuals have had more trauma-related experiences

in the past, and therefore, are quick to misevaluate their physical symptoms as a potential threat. This misevaluation of symptoms as impending disaster likely maintain symptoms as the cognitive misinterpretations to physiological arousal creates a negative feedback loop, leading to more physiological changes.

Social anxiety is also largely explained by cognitive theorists. Individuals with social anxiety disorder tend to hold unattainable or extremely high social beliefs and expectations. Furthermore, they often engage in preconceived maladaptive assumptions that they will behave incompetently in social situations and that their behaviors will lead to terrible consequences. Because of these beliefs, they anticipate social disasters will occur and therefore, avoid social encounters (or limit them to close friends/family members) in efforts to prevent the disaster (Moscovitch et al., 2013). Unfortunately, these cognitive appraisals are not only isolated before and during the event. Individuals with social anxiety disorder will also evaluate the social event after it has taken place, often obsessively reviewing the details (i.e., ruminating over social events). This over-evaluation of social performance negatively reinforces future avoidance of social situations.

4.6.2.2: **Behavioral**

The behavioral explanation for the development of anxiety disorders is largely reserved for phobias- both specific and social phobia. More specifically, behavioral theorists focus on **classical conditioning** – when two events that occur close together become strongly associated with one another, despite their lack of causal relationship. Watson and Rayner's (1920) infamous Little Albert experiment is an example of how classical conditioning can be used to induce fear through associations. In this study, Little Albert developed a fear of white rats by pairing a white rate with a loud sound. This experiment, although lacking ethical standards, was groundbreaking in the development of learned behaviors. Over time, researchers have been able to replicate these findings (in more ethically sound ways) to provide further evidence of the role of classical conditioning in the development of phobias.

4.6.2.3: **Modeling**

Modeling is another behavioral explanation of the development of specific and social phobias. In modeling, an individual acquires a far through observation and imitation (Bandura & Rosenthal, 1966). For example, when a young child observes their parent display irrational fears of an animal, the child may then begin to display similar behaviors. Similarly, observing another individual being ridiculed in a social setting may increase the chances of the development of social anxiety, as the individual may become fearful that they would experience a similar situation in the future. It is speculated that the maintenance of these phobias is due to the *avoidance* of the feared item or social setting, thus preventing the individual from learning that the item/social situation is not something that should be feared.

While modeling and classical conditioning largely explain the development of phobias, there is some speculation that the accumulation of a large number of these learned fears will develop into GAD. Through **stimulus generalization,** or the tendency for the conditioned stimulus to evoke similar responses to other conditions, a fear of one item (such as the dog) may become generalized to other items (such as all animals). As these fears begin to grow, a more generalized anxiety may present, as opposed to a specific phobia.

4.6.2.4: **Sociocultural**

Finally, we will review the social constructs that contribute to and maintain anxiety disorders. While characteristics such as living in poverty, experiencing significant daily stressors, and increased exposure to traumatic events are all identified as major contributors to anxiety disorders, additional sociocultural influences such as gender and discrimination have also received a great deal of attention.

Gender has largely been researched within anxiety disorders due to the consistent discrepancy in diagnosis rate between men and women. As previously discussed, women are routinely diagnosed with anxiety disorders more often than men, a trend that is observed throughout the entire lifespan. One potential explanation for this discrepancy is the influence of social pressures on women. Women are more susceptible to experience traumatic experiences throughout their life, which may contribute to anxious appraisals of future events. Furthermore, women are more likely to use **emotion-focused coping**, which is less effective in reducing distress than **problem-focused coping** (McLean & Anderson, 2009). These factors may increase levels of stress hormones (e.g., cortisol) within women that leave them susceptible to develop symptoms of anxiety. Therefore, it appears a combination of genetic, environmental, and social factors may explain why women tend to be diagnosed with anxiety disorders more often than men.

Exposure to discrimination and prejudice, particularly relevant to ethnic minority and other marginalized groups, can also impact an individual's anxiety level. Discrimination and prejudice contribute to negative interactions, which is directly related to negative

affect and an overall decline in mental health (Gibbons et al., 2014). The repeated exposure to discrimination and prejudice over time can lead to fear responses in individuals, along with subsequent avoidance of social situations in efforts to protect themselves emotionally.

Chapter Recap

Chapter 4 covered the topic of anxiety disorders. This discussion included Generalized Anxiety Disorder, Specific Phobias, Agoraphobia, Social Anxiety Disorder, and Panic Disorder. As with other chapters in this book, we discussed the clinical presentation, epidemiology, comorbidity, and treatment of the anxiety disorders. Etiology was also discussed in the context of biological, psychological, and sociocultural theories.

CHAPTER OVERVIEW

5: Obsessive-Compulsive and Related Disorders

Learning Objectives

- Describe obsessive-compulsive disorder and body dysmorphic disorder.
- Describe the epidemiology of obsessive-compulsive disorder.
- Describe comorbidities of obsessive-compulsive disorder and body dysmorphic disorder.
- Describe the etiology of these disorders.
- Describe treatment options for these disorders.

In Chapter 5, we will discuss matters related to obsessive-compulsive and related disorders to include their clinical presentation, diagnostic criteria, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include obsessive-compulsive disorder (OCD) and body dysmorphic disorder (BDD). However, it is worth noting that hoarding disorder, trichotillomania (excessive hair pulling), and excoriation disorder (excessive skin picking) were recently added to the new obsessive-compulsive and related disorders section of the DSM 5. Be sure you refer Chapters 1-3 for explanations of key terms (Chapter 1), an overview of the various models to explain psychopathology (Chapter 2), and descriptions of the various therapies (Chapter 3).

5.1: Obsessive-Compulsive Disorder

Section Learning Objectives

- Describe how obsessive-compulsive disorder presents itself.
- Describe the epidemiology of obsessive-compulsive disorder.
- Indicate which disorders are commonly comorbid with obsessive-compulsive disorder.
- Describe the biological, cognitive, and behavioral theories for the etiology of obsessive-compulsive disorder.
- Describe the treatment options for obsessive-compulsive disorder.

5.1.1: Clinical Description

Obsessive-compulsive disorder, more commonly known as OCD, requires the presence of **obsessions** and/or **compulsions.** Obsessions are defined as repetitive and intrusive thoughts, urges, or images. These obsessions are persistent, time-consuming, and unwanted, often causing significant distress and impairment in an individual's daily functioning. Common obsessions are contamination (dirt on self or objects), errors of uncertainty regarding daily behaviors (locking a door, turning off appliances), thoughts of physical harm or violence, and orderliness, to name a few (Cisler, Adams, et. al., 2011; Yadin & Foa, 2009). Often the individual will try to ignore these thoughts, urges, or images. When they are unable to ignore them, the individual will engage in compulsatory behaviors to alleviate the anxiety.

Compulsions are defined as repetitive behaviors or mental acts that an individual typically performs in response to an obsession. Common examples of compulsions are checking (i.e. repeatedly checking if the stove is turned off even though the first four times they checked it was off), counting (i.e. flicking the lights off and on 5 times), hand washing, organizing objects in a symmetrical manner, and repeating specific words. These compulsive behaviors are typically performed in an attempt to alleviate the anxiety associated with the obsessive thoughts. For example, an individual may feel as though his hands are dirty after using utensils at a restaurant. He may obsess over this thought for a period of time, impacting his ability to interact with others or complete a specific task. This obsession will ultimately lead to the individual performing a compulsion where he will wash his hands with extremely hot water to rid all the germs, or even wash his hands a specified number of times if he also has a counting compulsion. At this point, the individual's anxiety may be temporarily relieved.

These obsessions and compulsions are more excessive than the typical "cleanliness" as they consume a large part of the individual's day. Indeed, in order to be considered clinical OCD, the obsessions or compulsions must consume more than 1 hour per day, cause distress, or result in impairment in functioning. Given the example above, an individual with a fear of contamination may refuse to eat out at restaurants or may bring his own utensils with him and insist on using them when he is not eating at home.

5.1.2: Epidemiology

The one-year prevalence rate for OCD is approximately 1.2% both in the US, and worldwide (APA, 2013). OCD has a balanced sex ratio in adults; however, in childhood, boys are diagnosed more frequently than girls (APA, 2013). With respect to gender and symptoms, females are more likely to be diagnosed with cleaning-related obsessions and compulsions, whereas males are more likely to display symptoms related to forbidden thoughts and symmetry (APA, 2013). Additionally, males have an earlier age of onset (5-15 yrs) compared to women (20-24 yrs; Rasmussen & Eisen, 1990). Approximately two-thirds of all individuals with OCD had some symptoms present before the age of 15 (Rasmussen & Eisen, 1990). Overall the average age of onset of OCD iis 19.5 years.

5.1.3: Comorbidity

There is a high comorbidity rate between OCD and other anxiety disorders. Nearly 76% of individuals with OCD will be diagnosed with another anxiety disorder, most commonly panic disorder, social anxiety disorder, generalized anxiety disorder, or a specific phobia (APA, 2013). Additionally, 63% of those with OCD will also be diagnosed with a mood disorder (APA, 2013).

There is a high comorbidity rate between OCD and tic disorder, particularly in males with an onset of OCD in childhood. Children presenting with early-onset OCD typically have a different presentation of symptoms than traditional OCD. Research has also indicated a strong triad of OCD, Tic disorder, and attention-deficit/hyperactivity disorder in children. Due to this triad of psychological disorders, it is believed there is a neurobiological mechanism at fault for the development and maintenance of the disorders.

It should be noted that there are several disorders- schizophrenia, bipolar disorder, eating disorders, and Tourettes – where there is a higher incidence of OCD than the general public (APA, 2013). Therefore, clinicians who have a client diagnosed with one of the disorders above, should also routinely assess him/her for OCD.

5.1.4: Etiology

5.1.4.1: **Biological**

There are a few biological explanations for obsessive-compulsive related disorders including: hereditary transmission, neurotransmitter deficits, and abnormal functioning in brain structures.

Hereditary Transmission

With regards to heritability studies, twin studies routinely support the role of genetics in the development of obsessive-compulsive behaviors, as monozygotic twins have a substantially greater concordance rate (80-87%) than dizygotic twins (47-50%; Carey & Gottesman, 1981; van Grootheest, Cath, Beekman, & Boomsma, 2005). Additionally, first degree relatives of individuals diagnosed with OCD are twice as likely to develop OCD (APA, 2013).

Interestingly, a study conducted by Nestadt and colleagues (2000) exploring the familial role in the development of obsessive-compulsive disorder found that family members of individuals with OCD had higher rates of both obsessions and compulsions than control families; however, obsessions were more specific to the family members than that of the disorder. This suggests that there is a stronger heritability association for obsessions than compulsions. This study also found a relationship between age of onset of OCD symptoms and family heritability. Individuals who experienced an earlier age of onset, particularly before age 17, were found to have more first-degree relatives diagnosed with OCD. In fact, after the age of 17, there was no relationship between family diagnoses, suggesting those who develop OCD at an older age may have a different diagnostic origin (Nestadt, et al., 2000).

Neurotransmitters

Neurotransmitters, particularly serotonin have been identified as a contributing factor to obsessive and compulsive behaviors. This discovery was actually on accident. When individuals with depression and comorbid OCD were given antidepressant medications clomipramine and/or fluoxetine (both of which increase levels of serotonin) to mediate symptoms of depression, not only did they report a significant reduction in their depressive symptoms, but they also experienced significant improvement in their symptoms of OCD (Bokor & Anderson, 2014). Interestingly enough, antidepressant medications that do not affect serotonin levels are *not* effective in managing obsessive and compulsive symptoms, thus offering additional support for deficits of serotonin levels as an explanation of obsessive and compulsive behaviors (Sinopoli, Burton, Kronenberg, & Arnold, 2017; Bokor & Anderson, 2014). More recently, there has been some research implicating the involvement of additional neurotransmitters – glutamate, GABA, and dopamine – in the development and maintenance of OCD, although future studies are still needed to draw definitive conclusions (Marinova, Chuang, & Fineberg, 2017).

Brain Structures

Seeing as neurotransmitters have a direct involvement in the development of obsessive-compulsive behaviors, it's only logical that brain structures that house these neurotransmitters also likely play a role in symptom development. Neuroimaging studies implicate the brain structures and circuits in the frontal lobe, more specifically, the orbitofrontal cortex, which is located just above each eye (Marsh et al., 2014). This brain region is responsible for mediating strong emotional responses and converts them into behavioral responses. Once the orbitofrontal cortex receives sensory/emotional information via sensory inputs, it transmits this information through impulses. These impulses are then passed on to the caudate nuclei which filter through the many impulses received, passing along only the strongest impulses to the thalamus. Once the impulses reach the thalamus, the individual essentially reassesses the emotional response and decides whether or not to act behaviorally (Beucke et al., 2013). It is believed that individuals with obsessive-compulsive behaviors experience overactivity of the orbitofrontal cortex and a lack of filtering in the caudate nuclei, thus causing too many impulses to be transferred to the thalamus (Endrass et al., 2011). Further support for this theory has been shown when individuals with OCD experience brain damage to the orbitofrontal cortex or caudate nuclei and experience remission of OCD symptoms (Hofer et al., 2013).

5.1.4.2: **Cognitive**

Cognitive theorists believe that OCD behaviors occur due to an individual's distorted thinking and negative cognitive biases. More specifically, individuals with OCD are more likely to overestimate the probability of threat and harm, to have an inflated sense of responsibility for preventing harm, to think thoughts are important and need to be controlled, and to be perfectionistic.

Additionally, some research has indicated that those with OCD also experience disconfirmatory bias, which causes the individual to seek out evidence that proves they failed to perform the ritual or compensatory behavior incorrectly (Sue, Sue, Sue, & Sue, 2017). Finally, individuals with OCD often report the inability to trust themselves and their instincts, and therefore, feel the need to repeat the compulsive behavior multiple times to ensure it is done correctly. These cognitive biases are supported throughout research studies that repeatedly find that individuals with OCD experience more intrusive thoughts than those without OCD (Jacob, Larson, & Storch, 2014).

Now that we have identified that individuals with OCD experience cognitive biases and that these biases contribute to the obsessive and compulsive behaviors, we have yet to identify why these cognitive biases occur. Everyone has times when they have repetitive or intrusive thoughts such as: "Did I turn the oven off after cooking dinner?" or "Did I remember to lock the door before I left home?" Fortunately, most individuals are able to either check once or even forgo checking after they confidently talk themselves through their actions, ensuring that the behavior in question was or was not completed. Unfortunately, individuals with OCD are unable to neutralize these thoughts without performing a ritual as a way to put themselves at ease. As you will see in more detail in the behavioral section below, the behaviors (compulsions) used to neutralize the thoughts (obsessions) provide a temporary relief to the individual. As the individual is continually exposed to the obsession and repeatedly engages in the compulsive behaviors to neutralize the anxiety, the behavior is repeatedly reinforced, thus becoming a compulsion. This theory is supported by studies where individuals with OCD report using more neutralizing strategies and report significant reductions in anxiety after employing these neutralizing techniques (Jacob, Larson, & Storch, 2014; Salkovskis, et al., 2003).

5.1.5: **Behavioral**

The behavioral explanation of obsessive-compulsive disorder focuses on the explanation of compulsions rather than obsessions. Behaviorists believe that these compulsions begin with and are maintained by the **classical conditioning**. As you may remember, classical conditioning occurs when an unconditioned stimulus is paired with a conditioned stimulus to produce a conditioned response. How does this help explain OCD? Well, an individual with OCD may experience negative thoughts or anxieties related to an unpleasant event (obsession; unconditioned stimulus). These thoughts/anxieties cause significant distress to the individual, and therefore, they seek out some kind of behavior (compulsion) to alleviate these threats (conditioned stimulus). This provides temporary relief to the individual, thus reinforcing the compulsive behaviors used to alleviate the threat. Over time, the conditioned stimulus (compulsive behaviors) are reinforced due to the repeated exposure of the obsession and the temporary relief that comes with engaging in these compulsive behaviors.

Strong support for this theory is the fact that the behavioral treatment option for OCD – exposure and response prevention – is among the most effective treatments for these disorders. As you will read below, this treatment essentially breaks the classical conditioning associated with the obsessions and compulsions through extinction (by preventing the individual from engaging in the compulsive behavior until anxiety is reduced).

5.1.6: Treatment

5.1.6.1: **Exposure and Response Prevention**

Treatment of OCD has come a long way in recent years. Among the most effective treatment options is exposure and response prevention (March, Frances, Kahn, & Carpenter, 1997). First developed by psychiatrist Victor Meyer (1966), individuals are repeatedly exposed to their obsession, thus causing anxiety/fears, while simultaneously being prevented from engaging in their compulsive behaviors. Exposure sessions are often done *in vivo*, or in real life, via videos, or even imaginary, depending on the type of obsession.

Prior to beginning the exposure and response prevention exercises, the clinician must teach the client relaxation techniques for them to engage to cope with the distress of being exposed to the obsession. Once relaxation techniques are taught, the clinician and client will develop a hierarchy of obsessions. Treatment will start at those with the lowest amount of distress to ensure the client has success with treatment and to reduce the likelihood the client will withdrawal from treatment.

Within the hierarchy of obsessions, the individual is gradually exposed to their obsession. For example, an individual obsessed with germs might first watch a person sneeze on the computer in session. Once anxiety is managed and compulsions are resisted at this level of exposure, the individual would move on to being present in the same room as a sick individual, to eventually shaking hands with someone obviously sick, each time helping the client resist the compulsion to engage in the compulsive behavior. Once this level of the hierarchy is managed, they would move on to the next obsession and so forth until the entire list is complete.

Exposure and response prevention is very effective in treating individuals with OCD. In fact, some studies suggest up to an 86% response rate when treatment is completed (Foa et al., 2005). The largest barrier to treatment with OCD is getting clients to commit to treatment, as the repeated exposures and prevention of compulsive behaviors can be quite distressing to clients.

5.1.6.2: **Psychopharmacology**

There has been minimal support for the treatment of OCD with medication alone. This is likely due to the temporary resolution of symptoms during medication use. Among the most effective medications are those that inhibit the reuptake of serotonin (e.g., clomipramine or SSRI's). Reportedly, up to 60% of people do show improvement in symptoms while taking these medications; however, symptoms are quick to return when medications are discontinued (Dougherty, Rauch, & Jenike, 2002). While there has been some promise in a combined treatment option of exposure and response prevention and SSRIs, these findings were not superior to exposure and response prevention alone, suggesting that the inclusion of medication in treatment does not provide any added benefit (Foa et al., 2005).

5.2: Body Dysmorphic Disorder

Section Learning Objectives

- Describe how body dysmorphic disorder presents itself.
- Describe the epidemiology of body dysmorphic.
- Indicate which disorders are commonly comorbid with body dysmorphic.
- Describe the theories for the etiology of body dysmorphic disorder.
- Describe the treatment for body dysmorphic disorder.

5.2.1: Clinical Description

Body Dysmorphic Disorder (BDD) is another obsessive-compulsive disorder, however, the focus of these obsessions are with a perceived defect or flaw in physical appearance. A key feature of these obsessions with defects or flaws are that they are *not* observable to others. An individual who has a congenital facial defect or a burn victim who is concerned about scars are ***not*** examples of an individual with BDD. The obsessions related to one's appearance can run the spectrum from feeling "unattractive" to "looking hideous." While any part of the body can be a concern for an individual with BDD, the most commonly reported areas are skin (e.g., acne, wrinkles, skin color), hair (e.g., thinning hair or excessive body hair), or nose (e.g., size, shape).

The distressing nature of the obsessions regarding one's body, often drive individuals with BDD to engage in compulsive behaviors that take up a considerable amount of time. For example, an individual may repeatedly compare her body to other people's bodies in the general public; repeatedly look at herself in the mirror; engage in excessive grooming which includes using make-up to modify her appearance. Some individuals with BDD will go as far as having numerous plastic surgeries in attempts to obtain the "perfect" appearance. The problem is plastic surgery does not usually resolve the issue after all the physical defect or flaw is not observable to others. While most of us are guilty of engaging in some of these behaviors, to meet criteria for BDD, one must spend a considerable amount of time preoccupied with his/her appearance (i.e., on average 3-8 hours a day), as well as display significant impairment in social, occupational, or other areas of functioning.

5.2.1.1: **Muscle Dysmorphia.**

While muscle dysmorphia is not a formal diagnosis, it is a common type of BDD, particularly within the male population. Muscle dysmorphia refers to the belief that one's body is too small, or lacks appropriate amount of muscle definition (Ahmed, Cook, Genen & Schwartz, 2014). While severity of BDD between individuals with and without muscle dysmorphia appears to be the same, some studies have found a higher use of substance abuse (i.e. steroid use), poorer quality of life, and an increased reports of suicide attempts in those with muscle dysmorphia (Pope, Pope, Menard, Fay Olivardia, & Philips, 2005).

5.2.2: Epidemiology

The point prevalence rate for BDD among U.S. adults is 2.4% (APA, 2013). Internationally, this rate drops to 1.7% –1.8% (APA, 2013). Despite the difference between the national and international prevalence rates, the symptoms across races and cultures are similar.

Gender-based prevalence rates indicate a fairly balanced sex ratio (2.5% females; 2.2% males; APA, 2013). While the diagnosis rates may be different, general symptoms of BDD appear to be the same across genders with one exception: males tend to report genital preoccupations, while females are more likely to present with a comorbid eating disorder.

5.2.3: Comorbidity

While research on BDD is still in its infancy, initial studies suggest that major depressive disorder is the most common comorbid psychological disorder (APA, 2013). Major depressive disorder typically occurs after the onset of BDD. Additionally, there are some reports of social anxiety, OCD, and substance-related disorders (likely related to muscle enhancement; APA, 2013).

5.2.4: Etiology

Initial studies exploring genetic factors for BDD indicate a hereditary influence as the prevalence of BDD is elevated in first degree relatives of people with BDD. Interestingly, the prevalence of BDD is also heightened in first degree relatives of individuals with OCD (suggesting a shared genetic influence to these disorders).

However, environmental factors appear to play a larger role in the development of BDD than OCD (Ahmed, et al., 2014; Lervolino et al., 2009). Specifically, it is believed that negative life experiences such as teasing in childhood, negative social evaluations about one's body, and even childhood neglect and abuse may contribute to BDD. Cognitive research has further discovered that people with BDD tend to have an attentional bias towards beauty and attractiveness, selectively attending to words related to beauty and attractiveness. Cognitive theories have also proposed that individuals with BDD have dysfunctional beliefs that their worth is inherently tied to their attractiveness and hold attractiveness as one of their primary core values. These beliefs are further reinforced by our society, which overly values and emphasizes beauty.

5.2.5: Treatment

Seeing as though there are strong similarities between OCD and BDD, it should not come as a surprise that the only two effective treatments for BDD are those that are effective in OCD. Exposure and response prevention has been successful in treating symptoms of BDD, as clients are repeatedly exposed to their body imperfections/obsessions and prevented from engaging in compulsions used to reduce their anxiety (Veale, Gournay, et al., 1996; Wilhelm, Otto, Lohr, & Deckersbach, 1999).

The other treatment option, psychopharmacology, has also been shown to reduce symptoms in individuals diagnosed with BDD. Similar to OCD, medications such as clomipramine and other SSRIs are generally prescribed. While these are effective in reducing BDD symptoms, once the medication is discontinued, symptoms resume nearly immediately, suggesting this is not an effective long-term treatment option for those with BDD.

Treatment of BDD appears to be difficult, with one study finding that only 9% of clients had full remission at a 1-year follow-up, and 21% reported partial remission (Phillips, Pagano, Menard & Stout, 2006). A more recent finding reported more promising findings with 76% of participants reporting full remission over an 8-year period (Bjornsson, Dyck, et al., 2011).

5.2.5.1: **Plastic surgery and medical treatments**

It should not come as a surprise that many individuals with BDD seek out plastic surgery to attempt to correct their perceived defects. Phillips and colleagues (2001) evaluated treatments of clients with BDD and found that 76.4% reported some form of plastic surgery or medical treatment, with dermatology treatment the most reported (45%) followed by plastic surgery (23%). The problem with this type of treatment is that the individual is rarely satisfied with the outcome of the procedure, thus leading them to seek out additional surgeries on the same defect (Phillips, et al., 2001). Therefore, it is important that medical professionals thoroughly screen patients for BDD before completing any type of medical treatment.

CHAPTER OVERVIEW

6: Mood Disorders

Learning Objectives

- Describe how depressive disorders present and be able to distinguish between the different types of depressive disorders.
- Describe how bipolar disorders present be able to distinguish between the different types of bipolar disorders.
- Describe the epidemiology of mood disorders.
- Describe comorbidity in relation to mood disorders.
- Describe the etiology of mood disorders.
- Describe treatment options for mood disorders.

In Chapter 6, we will discuss matters related to mood disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include Major Depressive Disorder, Persistent Depressive Disorder (formerly called Dysthymia), Bipolar I Disorder, Bipolar II Disorder, and Cyclothymic Disorder.

6.1: Depressive Disorders

Section Learning Objectives

- Classify the symptoms of depression.
- Identify and describe the two types of depressive disorders.
- Identify the disorders that are commonly comorbid with depressive disorders.
- Describe the epidemiology of depressive disorders.
- Discuss the factors that contribute to depressive disorders.
- Describe treatment options for depressive disorders.

6.1.1: **Clinical Description**

6.1.1.1: **Symptoms of Depressive Disorders**

Symptoms of depression can generally be categorized into four categories to include mood, behavioral, physical and cognitive symptoms.

Mood

While clinical depression can vary in its presentation among individuals, most if not all individuals with depression will report significant mood disturbances such as a depressed mood (e.g., feeling sad, hopeless, discouraged) and/or feelings of **anhedonia**, which is the loss of interest or pleasure in previously interesting activities. These feelings occur transiently in all of us and therefore they must be present most of the day, nearly every day to be considered as symptoms of depression.

Behavioral

Fatigue and/or decreased energy are a symptom of depression that can make even the simplest of tasks (e.g., showering, getting off the couch to get the T.V. remote) seem difficult. Behavioral issues such as decreased physical activity and reduce productivity – both at home and at work – can result from this fatigue and cause disruptions in daily functioning (e.g., difficulty maintaining social interactions and employment responsibilities).

Physical

Changes in sleep patterns are common in those experiencing depression. This can occur at various points throughout the night – either difficulty falling asleep **(initial insomnia)**, waking up in the middle of the night **(middle insomnia)**, or even waking too early and not being able to fall back asleep before having to wake for the day **(terminal insomnia).** Excessive sleeping can also occur **(hypersomnia**)**.**

Additional physical symptoms such as a change in weight or eating behaviors are also a symptom of depression. Some individuals who are experiencing depression report a lack of appetite, often forcing themselves to eat something during the day. On the contrary, others eat excessively, often seeking “comfort foods” such as those high in carbohydrates. Due to these changes in eating behaviors, there may be associated changes to weight. Changes in weight of more than 5% of one’s body weight are considered a symptom of depression.

Finally, **psychomotor agitation**, which is the purposeless physical movement of the body (i.e. pacing around a room, tapping toes, restlessness etc.) and the opposite **psychomotor retardation** (e.g., slowed speech, thinking and movement) are symptoms of depression.

Cognitive

It should not come as a surprise that there are serious disruptions in cognitions as individuals with depressive disorders typically hold a negative view of themselves and the world around them. They are quick to blame themselves when things go wrong, and rarely take credit when they experience positive achievements. Feelings of worthlessness and guilt are common symptoms of depression. These distorted cognitions can create a negative feedback loop and further contribute to feelings of depression. Finally, thoughts of suicide and self-harm do occasionally occur in those with depressive disorders and are considered one of the most severe symptoms of depression.

Individuals with depressive disorders also report difficulty thinking, making decisions, and/or concentrating on tasks. This is supported by research that has found individuals with depression perform worse than those without depression on tasks of memory, attention, and reasoning (Chen et al., 2013).

6.1.1.2: Types of Depressive Disorders

The two most common types of depressive disorders are: **major depressive disorder (MDD)** and **persistent depressive disorder (PDD)**. PDD (previously known as Dysthymia) is thought to be a more chronic, and potentially less severe form depression. More specifically, although symptoms for MDD and PDD are nearly identical, the duration and number of symptoms required to diagnose the two disorders differ substantially. First, while symptoms of MDD must persist for a minimum of two *weeks* to make a diagnosis, symptoms of PDD must persist continuously for a minimum of two *years* for a diagnosis. Second, five symptoms are required to diagnose MDD, while only two are required for a diagnosis of PDD.

As with most of the disorders listed in the DSM 5, diagnoses of both MDD and PDD require that the symptoms cause significant distress or impairment. Moreover, the clinician needs to rule out that the symptoms are caused by something secondary (e.g., a medical condition, a substance, a schizophrenia spectrum disorder) and establish that the individual has *never* have had a manic or hypomanic episode before a diagnosis of MDD or PDD can be made.

It is important to note that these are not the only depressive disorders recognized by the DSM 5. Indeed, the DSM 5 added two new depressive disorders – Disruptive Mood Dysregulation Disorder and Premenstrual Dysphoric Disorder. Since these are new disorders less is known about them and as such we will not consider them in any further detail here.

6.1.2: Epidemiology

According to the DSM-5 (APA, 2013), the prevalence of MDD is approximately 7% within the U.S. (which represents the highest prevalence of depression in the world). The prevalence rate for PDD is much lower, with a 0.5% rate among adults in the U.S. There is a difference among demographics, with individuals in the 18- to 29- year-old age bracket reporting the highest rates of depression than any other age group. Similarly, depression is approximately 1.5 to 3 times higher in females than in males. The estimated lifetime prevalence for MDD in women is 21.3% compared to 12.7% in men (Nolen-Hoeksema, 2001).

Suicidality in depressive disorders is much higher than in the general public. Males and those with a past history of suicide attempts/threats are most at risk for attempting suicide.

6.1.3: Comorbidity

I'm sure it does not come as a surprise that studies exploring depression symptoms among the general population show a substantial pattern of comorbidity with other mental disorders (Kessler, Berglund, et al., 2003). In fact, in a large-scale research study nearly three-fourths of participants with lifetime MDD also met criteria for at least one other DSM disorder (Kessler, Berglund, et al., 2003). Among those that are the most common are anxiety disorders, ADHD, and substance abuse.

Given the extent of comorbidity among individuals with MDD, researchers have tried to identify which disorder precipitated the other. The majority of the studies have identified most cases of depression occur secondarily to another mental health disorder suggesting that the onset of depression is a direct result of the onset of another disorder (Gotlib & Hammen, 2009).

6.1.4: Etiology

6.1.4.1: Biological

Research throughout the years continues to provide evidence that depressive disorders have some biological cause. Most individuals who develop depression have some predisposition to develop a depressive disorder. Among the biological factors are genetic factors, biochemical factors, endocrine factors, and brain structure.

Genetics

As with other disorders, researchers often explore the prevalence rate of depressive disorders among family members, in efforts to determine whether there is some genetic component. If there is a genetic predisposition to developing depressive disorders, one would expect a higher rate of depression within families than that of the general population. Research supports this as there is nearly a 30 percent increase in the risk of depression in relatives of individuals diagnosed with depression, compared to 10 percent of the general population (Levinson & Nichols, 2014).

Another way to study the genetic component of a disorder is via twin studies. One would expect identical twins to have a higher rate of the disorder as opposed to fraternal twins, as identical twins share the same genetic make-up whereas fraternal twins only share that of siblings, roughly 50%. A large-scale study found that there was nearly a 46% chance that if one identical twin was diagnosed with depression, that the other was as well. In contrast, the fraternal twin rate was only 20%. This study provided enough evidence that there is a strong genetic link in the development of depression (McGuffin et al., 1996).

Finally, scientists have more recently been studying depression at a molecular level, exploring possibilities of gene abnormalities underlying depressive disorders. While much of the research is speculative due to sampling issues and low power, there is some evidence that depression may be tied to the 5-HTT gene on chromosome 17, as this is responsible for the activity of serotonin (Jansen et al., 2016).

Biochemical

As you will read in the treatment section, there is strong evidence of a biochemical deficit in depression. More specifically, low activity levels of norepinephrine and serotonin, have long been documented as contributing factors to developing depressive disorders. This was actually discovered accidentally in the 1950's when monoamine oxidase inhibitors (MAOIs) were given to patients with tuberculosis, and miraculously, their depressed moods were also improved. Soon thereafter, medical providers found that medications used to treat high blood pressure, by causing a reduction in norepinephrine, also caused depression in their patients (Ayd, 1956).

While these initial findings were premature in the identification of how neurotransmitters affected the development of depressive symptoms, they did provide insight as to *what* neurotransmitters were involved. Researchers are still trying to determine exact pathways; however, it does appear that *both* norepinephrine and serotonin are involved in the development of symptoms, whether it be between the interaction between them, or their interaction with other neurotransmitters (Ding et al., 2014).

Endocrine System

As described in Chapter 2, the endocrine system is a collection of glands responsible for regulating hormones, metabolism, growth and development, sleep, and mood among other things. Some research has implicated hormones, particularly **cortisol** (a stress hormone), in the development of depression (Owens et al, 2014). Additionally, elevated levels of **melatonin** (a hormone released when it is dark outside to assist with the transition to sleep), may also be related to depressive symptoms, particularly a specific type of depression commonly referred to as seasonal affective disorder which is prominent in northern latitudes where there is less sunlight in the winter.

Brain Anatomy

Seeing as neurotransmitters are involved in depressive disorders, it should not be a surprise that brain anatomy is also involved. While exact anatomy and pathways are yet to be determined, research studies implicate the prefrontal cortex, the hippocampus, and the amygdala. More specifically, drastic changes in blood flow throughout the prefrontal cortex have been linked with depressive symptoms. Similarly, a smaller hippocampus, and consequently, a fewer number of neurons, have also been linked to depressive symptoms (this may also help to account for some of the memory problems commonly reported in depression). Finally, heightened activity and blood flow in the amygdala (the brain area responsible for the fight or flight response), are also consistently found in individuals with depressive symptoms.

6.1.4.2: Cognitive

The cognitive model, arguably the most conclusive model with regards to depressive disorders, focuses on the negative thoughts and perceptions that may contribute to and maintain symptoms of depression. One theory often equated with the cognitive model of depression is **learned helplessness.** The concept of learned helplessness was developed based on Seligman's (1972) laboratory experiment involving dogs. In this study, Seligman restrained dogs in an apparatus and routinely shocked the dogs regardless of their behavior. The following day, the dogs were placed in a similar apparatus; however, this time the dogs were not restrained and there was a small barrier placed between the "shock" floor and the "safe" floor. What Seligman observed was that despite the opportunity to escape the shock, the dogs flurried for a bit, and then ultimately laid down and whimpered while being shocked. Based on this study, Seligman concluded that the animals essentially learned that they were unable to avoid the shock the day prior, and therefore, learned that they were helpless in avoiding the shocks. When they were placed in a similar environment but had the opportunity to escape the shocks, their learned helplessness carried over and they continued to believe they were unable to escape the shocks.

The concept of learned helplessness has been linked to humans through research on **attributional styles** (Nolen-Hoeksema, Girgus & Seligman, 1992). There are two types of attributional styles – positive and negative. A negative attributional style focuses on the *internal*, *stable*, and *global* influences of daily life, whereas a positive attributional style focuses on the *external*, *unstable*, and *specific* influences of the environment. Research has found that individuals with a negative attributional style are more likely to experience depression. This is likely due to their negative interpretation of daily events. For example, if something bad were to happen to them, they would likely conclude that it is *their* fault (internal), bad things *always* happen to them (stable),

and bad things happen *all* the time. Unfortunately, this maladaptive thinking style often takes over their global view of themselves and the world, thus making them more vulnerable to depression.

In addition to attributional style, Aaron Beck also attributed negative thinking as a precursor to depressive disorders (Beck, 2002, 1991, 1967). Often viewed as the grandfather of Cognitive-Behavioral Therapy, Beck went on to coin the terms maladaptive attitudes, cognitive triad, errors in thinking, and automatic negative thoughts – all of which combine to explain the cognitive model of depressive disorders.

Maladaptive attitudes, or negative attitudes about oneself, others, and the world around them are often present in those experiencing depression. These attitudes are inaccurate and often global. For example, "If I fail my exam, the world will know I'm stupid." Will the entire world *really* know you failed your exam? Not likely. Because you fail the exam, are you stupid? No. Individuals with depressive symptoms often develop these maladaptive attitudes regarding everything in their life, indirectly isolating themselves from others. The **cognitive triad** also plays into the maladaptive attitudes in that the individual interprets these negative thoughts about *themselves*, their *experiences*, and their *futures*. An example would be getting dumped and thinking "I am worthless, no one loves me or treats me well, and my future is hopeless," rather than concluding that they were a bad match with the person.

Cognitive distortions, also known as **errors in thinking**, are a key component in Beck's cognitive theory. Beck identified 15 errors in thinking that are most common in individuals with depression. Among the most common are catastrophizing (believing things are far worse than they actually are), jumping to conclusions, and overgeneralization. I always like to use my dad as an example for overgeneralization – whenever we go to the grocery store, he always comments about how *whatever* line he chooses, at *every* store, it is *always* the slowest/takes the longest. Does this happen *every* time he is at the store? I'm doubtful, but his error in thinking makes him perceive this to be true.

Finally, **automatic negative thoughts**, or a constant stream of negative thoughts, also lead to symptoms of depression as individuals regularly think in a pessimistic manner. While some cognitions are manipulated and interpreted in a negative view, Beck stated that there are another set of negative thoughts that occur automatically, such as these. Research studies have continually supported Beck's maladaptive thoughts, attitudes, and errors in thinking as fundamental issues that contribute to and help maintain depressive disorders (Possel & Black, 2014; Lai et al., 2014). Furthermore, as you will see in the treatment section, cognitive strategies are among the most effective forms of treatment for depressive disorders.

6.1.4.3: **Behavioral**

The behavioral model explains depression as a result of a change in the number of rewards and punishments one receives throughout their life. This change can come from work, intimate relationships, family, or even the environment in general. Among the most influential in the field of depression is Peter Lewinsohn. He stated depression occurs in most people due to the reduced positive rewards in their life. Because they are not being positively rewarded, their constructive behaviors occur more infrequently until they stop engaging in the behavior completely (Lewinsohn et al., 1990; 1984). An example of this is a student who continues to receive bad grades on exams despite studying for hours. Over time, the individual will reduce the amount of study time, thus continuing to earn poor grades.

6.1.4.4: **Sociocultural**

In the sociocultural theory, the role of family and one's social environment play a strong role in the development of depressive disorders. These topics will be explored next.

Social Support

Depression is commonly found to be related to a lack of social support. This is supported by research showing that separated and divorced individuals are three times more likely to experience depressive symptoms than those who are married or even widowed (Schultz, 2007). While there are many factors that lead a couple to separate or even end their marriage, some relationships end due to a spouse's mental health issues, particularly depressive symptoms. Depressive symptoms have been positively related to increased interpersonal conflicts, reduced communication, and intimacy issues, all of which are often reported in causal factors leading to a divorce (Najman et al., 2014). The relationship between depression and marital problems appears to be bidirectional with stress and marital discord leading to increased rates of depression in one or both spouses (Nezlek et al., 2000). Further, while some research indicates that having children provides a positive influence in one's life, it can also lead to stress both within the individual, as well as between partners due to the division of work and potential differences in discipline strategies and beliefs.

Research studies have shown that women who had three or more young children who also lacked a close confidante and outside employment, were more likely than other mothers to become depressed (Brown, 2002).

Multi-Cultural Perspective

While depression is experienced across the entire world, one's cultural background may influence *what* symptoms of depression are presented. Common depressive symptoms such as feeling sad, lack of energy, anhedonia, difficulty concentrating and thoughts of suicide are the hallmark in most societies, but other symptoms may be more specific to one's nationality. More specifically, individuals from Asian cultures often focus on the physical symptoms of depression – tiredness, weakness, sleep issues, and there is less of an emphasis on the cognitive symptoms. Individuals from Latino and Mediterranean cultures often experience problems with "nerves" and headaches as primary symptoms of depression (APA, 2013).

Within the United States, many researchers have explored potential differences across ethnic or racial groups in both rates of depression, as well as presenting symptoms of those diagnosed with depression. These studies continually fail to identify any significant differences between ethnic and racial groups; however, one major study has identified a difference in the rate of recurrence of depression in Hispanic and African Americans (Gonzalez et al., 2010). While the exact reason for this is unclear, the researchers propose a lack of treatment opportunities as a possible explanation. According to Gonzalez and colleagues (2010), approximately 54% of depressed Caucasian Americans seek out treatment, compared to the 34% and 40% Hispanic and African Americans, respectively. The fact that there is such a large discrepancy in the use of treatment between Caucasians and minority Americans suggests that minorities are not receiving the effective treatment necessary to resolve the disorder, thus leaving them more vulnerable for repeated depressive episodes.

Gender Differences

As previously discussed, there is a significant difference between rates of depression in men and women, with women being twice as likely to experience an episode of depression than men (Schuch et al., 2014). There are a few speculations of why there is such an imbalance in the rate of depression across genders.

The first theory – *artifact theory* – posits that the difference between genders is due to clinicians or diagnostic systems being more sensitive to diagnosing women with depression than men. While women are often thought to be more "emotional," easily expressing their feelings and more willing to discuss their symptoms with clinicians and physicians, men often withhold their symptoms or present with more traditionally "masculine" symptoms of anger or aggression. While this theory provides a possible explanation for the gender differences in the rate of depression, research has failed to support this theory suggesting that men and women are equally likely to seek out treatment and discuss their depressive symptoms (McSweeney, 2004; Rieker & Bird, 2005).

The second theory – *hormone theory* – suggests that variations in hormone levels trigger depression in women more than in men (Graziottin & Serafini, 2009). While there is biological evidence supporting the changes in hormone levels during various phases of the menstrual cycle and their impact on women's ability to integrate and process emotional information, research has failed to support this theory as the reason for higher rates of depression in women (Whiffen & Demidenko, 2006).

The third theory – *life stress theory* – suggests that women are more likely to experience chronic stressors than men, thus accounting for their higher rate of depression (Astbury, 2010). Women are at an increased risk for facing poverty, lower employment opportunities, discrimination, and poorer quality of housing than men, all of which are strong predictors of depressive symptoms (Garcia-Toro et al., 2013).

The fourth theory – *gender roles theory* – suggests that social and or psychological factors related to traditional gender roles also influence the rate of depression in women. For example, men are often encouraged to develop personal autonomy, seek out activities that interest them, and display achievement-oriented goals, while women are encouraged to empathize and care for others, often fostering an interdependent functioning, which may cause women to value the opinion of others more highly than do their male counterparts.

The final theory – *rumination theory* – suggests that women are more likely than men to ruminate, or intently focus and dwell on their depressive symptoms, thus making them more vulnerable to developing depression at a clinical level (Nolen-Hoeksema, 2012). Several studies have supported this theory and shown that rumination of negative thoughts is positively related to an increase in depression symptoms (Hankin, 2009).

While many theories have been proposed to explain the gender discrepancy in depression, no one single theory has produced enough evidence to fully explain why women experience depression more than men. Due to the lack of evidence, gender

differences in depression remains a highly researched topic, while simultaneously being one of the least understood phenomena in the clinical psychology world.

6.1.5: Treatment

Given that MDD is among the most frequent and debilitating psychiatric disorders, it should not be surprising that the research on the treatment of depression is quite extensive. Among its treatment options, the most efficacious treatments include antidepressant medications, Cognitive-Behavioral Therapy (CBT; Beck et al., 1979), Behavioral Activation (BA; Jacobson et al., 2001), and Interpersonal Therapy (IPT; Klerman et al., 1984). Although CBT is the most widely known and used treatment for depression, there is minimal evidence to support one treatment modality over the other; rather treatment is generally dictated by therapist competence, availability, and client preference (Craighhead & Dunlop, 2014).

6.1.5.1: **Psychopharmacology**

Antidepressant medications are often the most common first-line attempt at treatment for depression for a few reasons. Oftentimes an individual will present with symptoms to their primary caregiver (a medical doctor) who will prescribe some line of antidepressant medication. Medication is often seen as an "easier" treatment for depression as the individual can take the medication at their home, rather than attending weekly therapy sessions. However, this also leaves room for adherence issues as a large percentage of individuals do not take their prescription medication as indicated by their physician. Further, antidepressant medications take 3-6 weeks to begin to take effect.

There are a few different classes or antidepressant medications, each categorized by their structural or functional relationships. It should be noted that no specific antidepressant medication has been proven to be more effective in treating MDD than others (APA, 2010). In fact, many people try several different types of antidepressant medications until they find one that is effective for them, with minimal side effects.

Selective serotonin reuptake inhibitors (SSRIs)

SSRI's are among the most common medications used to treat depression due to their relatively benign side effects. Additionally, the required dose to reach therapeutic levels is low compared to the other medication options. Possible side effects from SSRI's include but are not limited to nausea, insomnia, weight gain, and reduced sex drive. SSRIs improve symptoms of depression by blocking the reuptake of serotonin and/or norepinephrine (SNRIs) in presynaptic neurons, thus allowing more of these neurotransmitters to be available for the postsynaptic neurons. While this is the general mechanism through which all SSRIs work, there are minor biological differences among different types of medications within the SSRI family. These minor differences are actually beneficial to clients in that there are a few treatment options to maximize medication benefits and minimize side effects.

Tricyclic Antidepressants

Although originally developed to treat schizophrenia, tricyclic antidepressants were adapted to treat depression after failing to manage symptoms of schizophrenia (Kuhn, 1958). The term tricyclic came from the molecular shape of the structure which has three rings. Tricyclic antidepressants are similar to SSRIs in that they work by affecting the brain chemistry, altering the availability of neurotransmitters. More specifically, they block the absorption or reuptake of serotonin and norepinephrine, thus increasing their availability for postsynaptic neurons. Tricyclic antidepressants have been shown to be more effective in treating traditionally resistant depression and PDD. While effective, tricyclic antidepressants have been increasingly replaced by SSRIs due to SSRI's reduced side effects. While the majority of side effects of tricyclics are minimal – dry mouth, blurry vision, constipation –others can be serious – sexual dysfunction, weight gain, tachycardia, cognitive and/or memory impairment, to name a few. Tricyclic antidepressants should not be used in cardiac patients as they have been shown to exacerbate cardiac arrhythmias (Roose & Spatz, 1999).

Monoamine Oxidase Inhibitors (MAOIs)

The utility of MAOIs was found serendipitously after producing antidepressant effects in a tuberculosis patient in the early 1950's. Although they are still prescribed, they are not typically the first line medications due to potentially lethal interactions when ingested with common substances like cheese and wine and safety concerns with hypertensive crises. Because of this, individuals on MAOIs have strict diet restrictions in efforts to reduce their risk of hypertensive crises (Shulman, Herrman & Walker, 2013).

How do MAOI's work? In basic terms, monoamine oxidase is released in the brain to remove excess norepinephrine, serotonin, and dopamine. MAOIs essentially prevent the monoamine oxidase (hence the name monoamine oxidase *inhibitors*) from removing these neurotransmitters, thereby leading to an increase in these neurotransmitters (Shulman, Herman & Walker, 2013) associated

with depression. While these drugs are effective, they come with serious side effects. In addition to the hypertensive episodes, they can also cause nausea, headaches, drowsiness, involuntary muscle jerks, reduced sexual desire, and weight gain to name a few (American Psychiatric Association, 2010). Despite these side effects, studies have shown that individual's prescribed MAOI's for depression have a treatment response rate of 50-70% (Krishnan, 2007). Overall, despite their effectiveness, MAOIs are likely the best treatment for later staged, treatment-resistant depression in individuals who have exhausted other treatment options (Krishnan, 2007)

It should be noted that occasionally, antipsychotic medications are used for individuals with MDD; however, these are limited to individuals presenting with psychotic features.

6.1.5.2: Psychotherapy

Cognitive Behavioral Therapy (CBT)

CBT was founded by Aaron Beck in the 1960's and is a widely practiced therapeutic tool used to treat depression. The basics of CBT involve what Beck called the **cognitive triad –** cognitions (thoughts), behaviors, and emotions. Beck believed that these three components are interconnected, and therefore, affect one another. It is believed that CBT can improve emotions in individuals with depression by changing both cognitions and behaviors, which in return will improve mood/emotion. Common cognitive interventions with CBT include monitoring and recording thoughts, identifying cognitive errors, examining the evidence supporting/negating cognitions, and creating rational alternatives to maladaptive thought patterns. Behavioral interventions of CBT include activity planning, pleasant event scheduling, task assignments, and coping skills training.

Cognitive behavioral therapy generally follows three phases of treatment:

- **Phase 1: Increasing pleasurable activities.** Similar to behavioral activation (read below), the clinician encourages the client to identify and engage in activities that are pleasurable to the individual. The clinician is able to help the client identify the activity and plan when they will engage in that activity.
- **Phase 2: Identifying automatic negative thoughts.** During this stage, the clinician provides psychoeducation about the automatic negative thoughts that can maintain symptoms of depression. The client learns to identify these thoughts on their own and maintains a thought journal of these cognitions to review with the clinician in session.
- **Phase 3: Challenging automatic negative thoughts.** Once the individual is consistently able to identify these negative thoughts on a daily basis, the clinician is able to help the client identify *how* these thoughts are maintaining their depressive symptoms. It is at this point that the client begins to have direct insight as to how their cognitions contribute to their disorder. Finally, the client is taught to challenge the negative thoughts and replacing them with positive thoughts.

CBT typically requires 10-20 sessions and it not only assists in recovery from depression but it also assists in preventing relapse. Evidence shows lower relapse rates following CBT (20-35%) compared to controls who received no treatment (70%) and those who were on antidepressant medications and stopped taking them (50%).

Rates of relapse following any treatment for MDD are often associated with individuals whose onset was at a younger age (particularly adolescents), those who have already experienced multiple major depressive episodes, and those with more severe symptomology, especially those presenting with severe suicidal ideation and psychotic features (APA, 2013).

Behavioral Activation (BA)

BA is similar to the behavioral component of CBT in that the goal of treatment is to alleviate depression and prevent future relapse by changing an individual's behavior. Founded by both Ferster (1973) and Lewinsohn and colleagues (Lewinsohn, 1974; Lewinsohn, Biglan, & Zeiss, 1976) the goal of BA is to increase the frequency of behaviors so that individuals have opportunities to experience greater contact with sources of reward in their lives. In order to do this, the clinician assists the client by developing a list of pleasurable activities that s/he can engage in outside of treatment (i.e. going for a walk, going shopping, having dinner with a friend). Additionally, the clinician assists the client in identifying negative behaviors – crying, sleeping too much, avoiding friends – and monitoring them so that they do not impact the outcome of their pleasurable activities. Finally, the clinician works with the client on effective social skills. The thought is that if the negative behaviors are minimized and the pleasurable activities are maximized, the individual will receive more positive rewards or reinforcement from others and their environment, thus improving their overall mood.

Interpersonal Therapy (IPT)

IPT was developed by Klerman, Weissman, and colleagues in the 1970's as a treatment arm for a pharmacotherapy study of depression (Klerman & Weissman, 1994). The treatment was created based on data from post World War II individuals who

expressed a significant impact on their psychosocial life events. Klerman and colleagues noticed a significant relationship between the development of depression and complicated bereavement, role disputes, role transitions, and interpersonal deficits in these individuals (Weissman, 1995). The idea behind IPT therapy is that depression compromises interpersonal functioning, which in return, makes it difficult to manage stressful life events. The basic mechanism of IPT is to establish effective strategies to manage interpersonal issues, which in return, will ameliorate depressive symptoms. There are two main principles of IPT. First, depression is a common, medical illness, with a complex and multi-determined etiology. Since depression is a medical illness, it is also treatable and is *not* the individual's fault. Second, depression is connected to a current or recent life event. The goal of IPT is to identify the interpersonal problem that is connected to the depressive symptoms and resolve this crisis so the individual can improve his/her life situation while relieving depressive symptoms.

6.1.5.3: **Multimodal Treatment**

While both pharmacological and psychological treatment alone is very effective in treating depression, a combination of the two treatments may be helpful for individuals who have not achieved wellness in a single modality.

Multimodal treatments can be offered in three different ways: treatments can be instigated concurrently, treatments can be instigated done sequentially, or stepped treatments can be offered (McGorry et al., 2010). With a stepped treatment, pharmacological therapy is often used initially to treat depressive symptoms. Once the client reports some relief in symptoms, the psychosocial treatment is added to address the remaining symptoms. While all three methods are effective in managing depressive symptoms, matching clients to their treatment preference may produce better outcomes than clinician driven treatment decisions.

6.2: Bipolar Disorders

Section Learning Objectives

- Identify the symptoms of bipolar disorder.
- Identify and distinguish between the three types of bipolar disorders.
- Identify the disorders that are commonly comorbid with bipolar disorders.
- Describe the epidemiology of bipolar disorders.
- Discuss the factors that contribute to bipolar disorders.
- Describe treatment options for bipolar disorders.

6.2.1: **Clinical Description**

There are three bipolar disorders – bipolar I disorder, bipolar II disorder, and cyclothymic disorder.

A diagnosis of **bipolar I disorder** is made when there is at least one **manic episode**. This manic episode may be preceded by or followed by a hypomanic or major depressive episode but neither is required for a diagnosis of bipolar I disorder. In contrast, a diagnosis of **bipolar II disorder** is made when the individual has experienced both a hypomanic episode and a depressive episode. A manic episode (past or present) rules out a diagnosis of bipolar II disorder. In simpler words, if an individual has ever experienced a manic episode, they qualify for a bipolar I diagnosis. If the criteria have been met for both a hypomanic and depressive episode then the individual qualifies for a diagnosis of bipolar II disorder.

So, what defines a **manic episode**? The key feature of a manic episode is an abnormally euphoric or irritable mood that is experienced persistently for at least one week. In order to qualify as a manic episode, the individual must experience at least three other symptoms of a manic episode. These symptoms include inflated self-esteem, decreased need for sleep, pressured speech, racing thoughts, distractibility, psychomotor agitation, and involvement in pleasurable activities that are likely to result in negative consequences (e.g., risky sexual behavior, gambling).

With regards to mood, an individual in a manic episode will appear excessively happy, often engaging haphazardly in sexual or personal interactions. They also display rapid shifts in mood, also known as **mood lability**, ranging from happy, neutral, to irritable. Inflated self-esteem or grandiosity is also commonly present during a manic episode. Occasionally these inflated self-esteem levels can appear delusional. Individuals may believe they are friends with a celebrity, do not need to abide by laws, or even at times think they are God or famous.

Despite their increased activity level, individuals experiencing a manic episode also typically experience a decreased need for sleep, sleeping as little as a few hours a night and still feel rested. In fact, decreased need for sleep may be an indicator that a manic episode is to begin imminently.

It is not uncommon for those in a manic episode to have rapid, pressured speech. It can be difficult to follow their conversation due to the fast nature of their talking, as well as the tangential storytelling (i.e., jumping from topic to topic). Additionally, they can be difficult to interrupt in conversation, often disregarding the reciprocal nature of communication. If the individual is more irritable than expansive, speech can become hostile or even pronounced by angry tirades, particularly if they are interrupted or not allowed to engage in an activity they are seeking out. Based on their speech pattern, it should not be a surprise that manic episodes are also marked by racing thoughts which are commonly referred to as a flight of ideas. Because of these rapid thoughts, speech may become disorganized or incoherent.

Hypomanic episodes are milder versions of manic episodes. While the symptoms of the two are the same, a diagnosis of a hypomanic episode requires only 4 days of symptoms rather than the full week required to diagnosis a manic episode. Moreover, while manic episodes must cause impairment in functioning, significant distress, or require the individual to be hospitalized, hypomanic episodes cannot cause impairment, distress, or the need for hospitalization. If any of these three features are present the episode is considered manic, rather than hypomanic.

It should be noted that there is a subclass of individuals who experience periods of hypomanic symptoms that do not fully meet DSM 5 criteria for a hypomanic episode and depressive symptoms that again do not fully meet DSM 5 criteria for a depressive episode. These individuals are diagnosed with **cyclothymic disorder** (APA, 2013). Cyclothymic disorder is further distinguished from bipolar II disorder by its duration. Specifically, cyclothymic disorder requires a minimum of two years of subthreshold depressive and hypomanic symptoms before a diagnosis can be made.

6.2.2: Epidemiology

Compared to depression, the epidemiological studies on the rates of bipolar disorder suggest a significantly lower prevalence rate for both bipolar I and bipolar II. Within the two disorders, there is a very minimal difference in the prevalence rates with yearly rates reported as 0.6% for bipolar I disorder and 0.8% for bipolar II disorder in the U.S. (APA, 2013). There are no apparent differences in the frequency of men and women diagnosed with bipolar I or bipolar II disorder; however, rapid-cycling episodes (where four or more mood episodes are experienced in a one-year period) are more common in women (Bauer & Pfenning, 2005).

Individuals with bipolar disorder are approximately 15 times greater than the general population to attempt suicide. Prevalence rates of suicide attempts in individuals with bipolar disorder are estimated to be 33%. Furthermore, bipolar disorder may account for one-quarter of all completed suicides (APA, 2013).

While only a small percentage of the population develops cyclothymic disorder (lifetime prevalence estimates range from 0.4 to 1%), it can eventually progress into bipolar I or bipolar II disorder (Zeschel et al., 2015).

As stated previously, bipolar II disorder requires a past or present depressive episode and, while not required, depressive episodes are commonly experienced in bipolar I disorder. The depressive episode can occur before or after the manic/hypomanic episode, and the two types of episodes can alternate or "cycle" throughout one's life.

6.2.3: Comorbidity

The bipolar disorders also have a high comorbidity rate with other mental disorders, particularly anxiety disorders and disruptive/impulse-control disorders such as ADHD and conduct disorder. Substance abuse disorders are also commonly seen in individuals with bipolar disorder. In fact, over half of those with bipolar disorder also meet diagnostic criteria for substance abuse disorder, particularly alcohol abuse. The combination of bipolar disorder and substance abuse disorder places individuals at a greater risk of suicide attempt (APA, 2013). While these comorbidities are high across both bipolar I and bipolar II, bipolar II appears to have more comorbidities, with 60% of individuals with bipolar II disorder meeting criteria for three or more co-occurring mental disorders (APA, 2013).

6.2.4: Etiology

6.2.4.1: Biological

As is typical with most mental disorders there is an elevated prevalence of bipolar disorders among first-degree biological relatives of people with bipolar I or bipolar II disorder. Specifically, first-degree biological relatives of individuals with bipolar I or II disorder have a 10-fold increased risk of developing bipolar disorder. Twin studies within bipolar disorder yield concordance rates for identical twins at as high as 72% and as high as 20% for fraternal twins. Both of these percentages are significantly higher than that of the general population, suggesting a strong genetic component of bipolar disorder (Edvardsen et al., 2008). Indeed, as some of these statistics demonstrate, the genetic contribution to bipolar disorder is believed to be greater than the genetic contribution to depressive disorders. There also seems to be a shared genetic component to the bipolar disorders and major depressive disorder (MDD) as relatives of individuals with bipolar disorder have elevated rates of MDD and MDD is more common in relatives of individuals with cyclothymic disorder.

Due to the close nature of depression and bipolar disorder, researchers initially believed that norepinephrine, serotonin, and dopamine were all implicated in the development of bipolar disorder; however, the idea was that there was a drastic *increase* in serotonin during manic episodes. Unfortunately, research actually supports the opposite. It is believed that manic episodes may, in fact, be explained by low levels of serotonin and high levels of norepinephrine (Soreff & McInnes, 2014). Moreover, following evidence that drugs like cocaine which stimulate dopamine produce manic-like symptoms it is further believed that elevated dopamine may be implicated in bipolar I disorder. Additional research in this area is needed to conclusively determine exactly what is responsible for the manic episodes that characterize bipolar I disorder.

6.2.4.2: Psychological

Stressful life events are believed to trigger early episodes of mania and depression, but as the disorder progresses the cycling from mania to depression can take on a life of its own and become more removed from stressors. Nevertheless, stressful life events can always provoke a relapse. As we saw with the depressive disorders, separated and divorced people have higher rates of bipolar I disorder than do people who are married or who were never married. Once again the direction of this relationship is not clear but it is likely bidirectional with the symptoms of bipolar disorder contributing to marital discord and the termination of a marriage representing a severe psychosocial stressor that can contribute to the onset of a bipolar disorder or trigger a relapse of the disorder.

Finally, a lack of social support is associated with more depressive episodes in bipolar disorder, as it was for the depressive disorders.

6.2.5: Treatment

6.2.5.1: **Psychopharmacology**

Unlike treatment for MDD, there is some controversy over the most effective treatment for bipolar disorder. One suggestion is to treat bipolar disorder aggressively with mood stabilizers such as Lithium or Depakote as these medications do not induce pharmacological mania/hypomania. These mood stabilizers are occasionally combined with antidepressant medications later in treatment *only* if absolutely necessary (Ghaemi, Hsu, Soldani & Goodwin, 2003). Research has shown that mood stabilizers are less powerful in treating depressive symptoms in those with bipolar disorder, and therefore, this combination approach is believed to help reduce the occurrence of both manic and depressive episodes (Nivoli et al., 2011).

The other treatment option is to forgo the mood stabilizer and treat symptoms with newer antidepressants early in treatment. Unfortunately, large-scale research studies have not shown great support for this method (Gijsman, Geddes, Rendell, Nolen, & Goodwin, 2004; Moller, Grunze & Broich, 2006). In fact, antidepressants can trigger a manic or hypomanic episode in people with bipolar disorder. Because of this, the first line treatment option for bipolar disorder is mood stabilizers, particularly Lithium.

Lithium and other mood stabilizers are very effective in managing symptoms of people with bipolar disorder. Unfortunately, adherence to the medication regimen can be problematic. The euphoric highs that are associated with manic and hypomanic episodes are often desired and can lead individuals with bipolar disorder to cease taking their medication. Combination of psychopharmacology and psychotherapy aimed at increasing rate of adherence to medication may be the most effective treatment option for bipolar I and II disorder.

6.2.5.2: **Psychological Treatment**

Although psychopharmacology is the first and most widely used treatment for bipolar disorders, occasionally psychological interventions are also paired with medication as psychotherapy alone is not a sufficient treatment option. The majority of psychological interventions are aimed at medication adherence, as many people with bipolar disorder stop taking their mood stabilizers when they “feel better” (Advokat et al., 2014) or as described above to induce a manic or hypomanic episode. CBT can also be used to help treat and reduce the reoccurrence of depressive episodes. Social skills training and problem-solving skills can also be helpful techniques to address in the therapeutic setting as individuals with bipolar disorder may struggle in these areas. Finally, individuals with bipolar disorder may be advised to stabilize their routines, especially their sleep routines, to reduce the risk of relapse.

Chapter Recap

That concludes our discussion of mood disorders. You should now have a good understanding of the two major types of mood disorders – depressive and bipolar disorders. Be sure you are clear on what makes them different from one another in terms of their clinical presentation, diagnostic criteria, epidemiology, comorbidity, and etiology. Also be sure to understand how the different depressive disorders (MDD and PDD) are distinguished as well as how the various bipolar disorders (bipolar I disorder, bipolar II disorder, and cyclothymic disorder) differ from one another.

CHAPTER OVERVIEW

7: Dissociative Disorders

Learning Objectives

- Describe the dissociative disorders and their symptoms.
- Describe the epidemiology of dissociative disorders.
- Indicate which disorders are commonly comorbid with dissociative disorders.
- Describe the etiology of dissociative disorders.
- Describe treatment options for dissociative disorders.

In Chapter 7, we will discuss dissociative disorders, including their clinical presentation, diagnostic criteria, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include depersonalization/derealization, dissociative amnesia, and dissociative identity disorder. Be sure you refer Chapters 1-3 for explanations of key terms (Chapter 1), an overview of the various models to explain psychopathology (Chapter 2), and descriptions of the various therapies (Chapter 3).

7.1: Chapter Introduction

Chapter Overview

In Chapter 7, we will discuss dissociative disorders, including their clinical presentation, diagnostic criteria, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include depersonalization/derealization, dissociative amnesia, and dissociative identity disorder. Be sure you refer Chapters 1-3 for explanations of key terms (Chapter 1), an overview of the various models to explain psychopathology (Chapter 2), and descriptions of the various therapies (Chapter 3).

Chapter Outline

- 7.2 Depersonalization/derealization disorder
- 7.3 Dissociative amnesia
- 7.4 Dissociative identity disorder

Chapter Learning Outcomes

- Describe the dissociative disorders and their symptoms.
- Describe the epidemiology of dissociative disorders.
- Indicate which disorders are commonly comorbid with dissociative disorders.
- Describe the etiology of dissociative disorders.
- Describe treatment options for dissociative disorders.

Chapter Introduction

Dissociative disorders are a group of disorders categorized by symptoms of disruption in consciousness, memory, identity, emotion, perception, motor control, or behavior (APA, 2013). These symptoms are likely to appear following a significant stressor or years of ongoing stress (i.e. abuse; Maldonadao & Spiegel, 2014). Occasionally, one may experience temporary dissociative symptoms due to lack of sleep or ingestion of a substance, however, these would not qualify as a dissociative disorder due to the lack of impairment in functioning. Furthermore, individuals with acute stress disorder and post-traumatic stress disorder (PTSD) often experience dissociative symptoms, such as amnesia, flashbacks, depersonalization and/or derealization; however, because of the identifiable stressor (and lack of additional symptoms listed below), they meet diagnostic criteria for a stress disorder as opposed to a dissociative disorder.

There are 3 main types of dissociative disorders that will be described in the next three sections: *Depersonalization/Derealization Disorder, Dissociative Amnesia*, and *Dissociative Identity Disorder.*

7.2: Depersonalization/Derealization Disorder

Section Learning Objectives

- Describe how derealization/depersonalization disorder presents itself.
- Describe the epidemiology of derealization/depersonalization disorder .
- Indicate which disorders are commonly comorbid with derealization/depersonalization disorder.
- Describe factors that may contribute to the etiology of derealization/depersonalization disorder.
- Describe the treatment of derealization/depersonalization disorder.

7.2.1: Clinical Description

Depersonalization/Derealization disorder is categorized by recurrent episodes of depersonalization and/or derealization. **Depersonalization** can be defined as a feeling of unreality or detachment from *oneself*. Individuals describe this feeling as an *out-of-bodyexperience* where they are an outside observer of their thoughts, feelings, and physical being. Furthermore, some people report feeling as though they lack speech or motor control, thus feeling at times like a robot. Distortions of one's physical body has also been reported, with various body parts appearing enlarged or shrunken. Individuals may also feel detached from their own feelings.

Symptoms of **derealization** include feelings of unreality or detachment from the *world*—whether it be individuals, objects, or their surroundings. For example, people experiencing derealization may feel as though they are unfamiliar with their surroundings, even though they are in a place they have been to many times before. Feeling emotionally disconnected from close friends or family members whom they have strong feelings for is another common symptom experienced during derealization episodes. Sensory changes have also been reported such as feeling as though the environment is distorted, blurry, or even artificial. Distortions of time, distance, and size/shape of objects may also occur.

These episodes can last anywhere from a few hours to days, weeks, or even months (APA, 2013). The onset is generally sudden, and similar to the other dissociative disorders, is often triggered by a intense stress or trauma. As one can imagine, depersonalization/derealization disorder can cause significant emotional distress, as well as impairment in one's daily functioning (APA, 2013).

7.2.2: Epidemiology

While many individuals experience brief episodes of depersonalization/derealization throughout their life (about 50% of adults have experienced depersonalization/derealization at least once), the estimated number of individuals who experiences these symptoms to the degree of clinical significance is estimated to be 2%, with an equal ratio of men and women experiencing these symptoms (APA, 2013). The mean age of onset is 16 years, with only a minority developing the disorder after the age of 25. About 1/3 of people with the disorder have discrete episodes, 1/3 have continuous symptoms from their onset, and 1/3 have an episodic course that progresses to continuous.

7.2.3: Comorbidity

Depersonalization/derealization disorder has been found to be comorbid with depression and anxiety disorders. With respect to the personality disorders, depersonalization/derealization disorder is most commonly comorbid with avoidant, borderline, and obsessive-compulsive personality disorders. Some evidence indicates that comorbidity with post-traumatic stress disorder is low (APA, 2013).

7.2.4: Epidemiology

The causes of depersonalization/derealization disorder are largely unknown. Very little is understood about the potential genetic underpinnings; however, there is some suggestion that heritability rates for dissociative experiences range from 50-60% (Pieper, Out, Bakermans-Kranenburg, Van Ijzendoorn, 2011). However, as with other psychological disorders, it is suggested that the combination of genetic and environmental factors may play a larger role in the development of dissociative disorders than genetics alone (Pieper, Out, Bakermans-Kranenburg, Van Ijzendoorn, 2011).

There are clear associations between all of the dissociative disorders and childhood trauma and abuse but the association is slightly weaker for depersonalization/derealization disorder than it is for the other dissociative disorders (i.e., dissociative amnesia and

dissociative identity disorder). Emotional abuse, emotional neglect, physical abuse, witnessing domestic violence, being raised by a parent who is seriously impaired and/or mentally ill, and experiencing the unexpected death or suicide of a family member or close friend are early-life stressors that have been identified to be associated with the disorder. The onset of the disorder is commonly triggered by severe stress, depression, anxiety, panic attacks, and drug use (particularly cannabis, hallucinogens, ketamine, ecstasy, and salvia).

7.2.5: Treatment

Depersonalization/derealization disorder symptoms generally occur for an extensive period of time before the individual seeks out treatment. Because of this, there is some evidence to support that the diagnosis alone is effective in reducing symptom intensity, as it also relieves the individual's anxiety surrounding the baffling nature of the symptoms (Medford, Sierra, Baker, & David, 2005).

Due to the high comorbidity of depersonalization/derealization disorder and anxiety/depression, the goal of treatment is often alleviating these secondary mental health symptoms related to the depersonalization/derealization symptoms. While there has been some evidence to suggest treatment with an SSRI is effective in improving mood, the evidence for a combined treatment method of psychopharmacological and psychological treatment is even more compelling (Medford, Sierra, Baker, & David, 2005). The psychological treatment of preference is cognitive-behavioral therapy as it addresses the negative attributions and appraisals contributing to the depersonalization/derealization symptoms (Medford, Sierra, Baker, & David, 2005). By challenging these catastrophic attributions in response to stressful situations, the individual is able to reduce overall anxiety levels, which in return, reduces depersonalization/derealization symptoms.

7.3: Dissociative Amnesia

Section Learning Objectives

- Describe how dissociative amnesia presents itself.
- Describe the epidemiology of dissociative amnesia.
- Indicate which disorders are commonly comorbid with dissociative amnesia.
- Describe factors that may contribute to the etiology of dissociative amnesia.
- Describe the treatment of dissociative amnesia.

7.3.1: Clinical Description

Dissociative amnesia is identified by amnesia for autobiographical information, particularly for traumatic events. This type of amnesia is different from what one would consider a permanent amnesia in that the information was successfully stored in memory, however, the individual cannot retrieve it. Additionally, individuals experiencing permanent amnesia often have a neurobiological cause, whereas dissociative amnesia does not (APA, 2013).

There are a few types of amnesia that people with dissociative amnesia can experience. **Localized amnesia**, the most common type of dissociative amnesia, is the inability to recall events during a specific period of time. The length of time within a localized amnesia episode can vary—it can be as short as the time immediately surrounding a traumatic event, to months or years, should the traumatic event occur that long (as commonly seen in abuse and combat situations). **Selective amnesia** is in a sense, a component of localized amnesia in that the individual can recall some, but not all, of the details during a specific time period. For example, a soldier may experience dissociative amnesia during the time they were deployed, yet still, have some memories of positive experiences such as celebrating Thanksgiving dinner or Christmas dinner with their unit. The onset of localized and selective amnesia may immediately follow the acute stress or be delayed for hours, days, or longer.

Conversely, some individuals experience **generalized amnesia** where they have a complete loss of memory of their entire life history, including their own identity. Individuals who experience this type of amnesia experience deficits in both *semantic* and *procedural* knowledge. This means that individuals have no common knowledge of the world (i.e. cannot identify songs, the current president, or names of colors) nor do they have the ability to engage in learned skills (i.e. typing shoes, driving car). The onset of generalized amnesia is typically acute.

While generalized amnesia is extremely rare, it is also extremely frightening. The onset is acute, and the individual is often found wandering in a state of disorientation. Many times, these individuals are brought into emergency rooms by law enforcement following a dangerous situation such as an individual walking aimlessly on a busy road.

Dissociative fugue is considered to be the most extreme type of dissociative amnesia where not only does an individual forget personal information, but they also flee to a different location (APA, 2013). The degree of the fugue varies among individuals – with some experiencing symptoms for a short time (only hours) to others lasting years, affording individuals to take on new identities, careers, and even relationships. Similar to their sudden onset, dissociative fugues also end abruptly. Post dissociative fugue, the individual generally regains most of their memory. Emotional adjustment after the fugue is dependent on the time the individual spent in the fugue – with those having been in a fugue state longer experiencing more emotional distress than those who experienced a shorter fugue (Kopelman, 2002).

7.3.2: Epidemiology

A large community sample suggested dissociative amnesia occurs in approximately 1.8% of the population with females being about 2.5 times more likely to be diagnosed than males (APA, 2013). Similar to trauma-related disorders, it is believed that more women experience dissociative amnesia due to the increased chances of a woman to experience significant stress/trauma compared to that of men.

7.3.3: Comorbidity

Given that dissociative amnesia is often precipitated by a traumatic experience, many people develop PTSD after the traumatic events are finally recalled. Similarly, a wide range of emotions related to their inability to recall memories during the episode often presents once their memories return (APA, 2013). These emotions often contribute to the development of a depressive episode.

Due to the rarity of these disorders with respect to other mental health disorders, it is often difficult to truly determine comorbid diagnoses. There has been some evidence of comorbid somatic symptom disorder and conversion disorder. Furthermore, dependent, avoidant, and borderline personality disorders have all been suspected as co-occurring disorders among the dissociative disorder family.

7.3.4: Etiology

7.3.0.1: **7.3.4.1**: Biological

As previously indicated, heritability rates for dissociative experiences range from 50-60% (Pieper, Out, Bakermans-Kranenburg, Van Ijzendoorn, 2011). However, it is suggested that the combination of genetic and environmental factors may play a larger role in the development of dissociative disorders than genetics alone (Pieper, Out, Bakermans-Kranenburg, Van Ijzendoorn, 2011).

7.3.0.1: **7.3.4.2**: Cognitive

One proposed cognitive theory of dissociative amnesia proposed by Kopelman (2000) is that the combination of psychological stress and various other biopsychosocial predispositions affects the frontal lobe's executive system's ability to retrieve autobiographical memories (Picard et al., 2013). Neuroimaging studies have supported this theory by showing deficits to several prefrontal regions, which is one area responsible for memory retrieval (Picard et al., 2013). Despite these findings, there is still some debate over which specific brain regions within the executive system are responsible for the retrieval difficulties, as research studies have reported mixed findings.

7.3.0.1: **7.3.4.3**: Environmental/Behavioral

Severe trauma and/or stress commonly precipitate the disorder. The most common precipitating stressors for fugues are marital discord, financial and occupational problems, natural disasters, and combat in war. The likelihood that dissociative amnesia is experienced increases with higher numbers of adverse childhood experiences (e.g., physical and/or sexual abuse), and more severe and frequent interpersonal violence. According to the behavioral perspective, the amnesia is negatively reinforced by avoiding/removing the distressing thoughts and feelings associated with the trauma/stressor.

7.3.0.1: **7.3.4.4: Psychodynamic**

The psychodynamic theory of dissociative amnesia assumes that dissociative disorders are caused by an individual's repressed thoughts and feelings related to an unpleasant or traumatic event (Richardson, 1998). In blocking, or dissociating from, these thoughts and feelings, the individual is subconsciously protecting himself from painful memories.

7.3.5: Treatment

Treatment for dissociative amnesia is limited in part because many individuals recover on their own without any type of intervention. Occasionally treatment is sought out after recovery due to the traumatic nature of memory loss. Further, the rarity of the disorder has offered limited opportunities for research on both the development and effectiveness of treatment methods. While there is no evidence-based treatment for dissociative amnesia, both hypnosis and treatment with barbituates have been shown to produce some positive effects in clients with dissociative amnesia.

Hypnosis. One theory of dissociative amnesia is that it is a form of self-hypnosis and that individuals *hypnotize* themselves to forget information or events that are unpleasant (Dell, 2010). Based on this theory, one type of treatment that has routinely been implemented for individuals with dissociative amnesia is hypnosis. Through hypnosis, the clinician can help the individual contain, modulate, and reduce the intensity of the amnesia symptoms, thus allowing them to process the traumatic or unpleasant events underlying the amnesia episode (Maldonadao & Spiegel, 2014). To do this, the clinician will encourage the client to think of memories just prior to the amnesic episode as though it was the present time. The clinician will then slowly walk them through the events during the amnesic time period in efforts to reorient the individual to experience these events. This technique is essentially a way to encourage a controlled recall of dissociated memories, something that is particularly helpful when the memories include traumatic experiences (Maldonadao & Spiegel, 2014).

Another form of "hypnosis" is the use of barbiturates, also known as "truth serums," to help relax the individual and free their inhibitions. Although not always effective, the theory is that these drugs reduce the anxiety surrounding the unpleasant events enough to allow the individual to recall and process these memories in a safe environment (Ahern et al., 2000).

7.4: Dissociative Identity Disorder

Section Learning Objectives

- Describe how dissociative identity disorder presents itself.
- Describe the epidemiology of dissociative identity disorder.
- Indicate which disorders are commonly comorbid with dissociative identity disorder.
- Describe factors that may contribute to the etiology of dissociative identity disorder.
- Describe the treatment of dissociative identity disorder.

7.4.1: Clinical Description

Dissociative Identity Disorder (DID) is what people commonly refer to as *multiple personality disorder*, as it was labeled as such in the DSM III. The key diagnostic criteria for DID is the presence of two or more distinct personality states or expressions. The identities are distinct in that they often have their own tone of voice, engage in different physical gestures (including different gait), and have their own behaviors – ranging anywhere from cooperative and sweet to defiant and aggressive. Additionally, the identities can be of varying ages and gender.

While personalities can present at any time, there is generally a dominant or *primary* personality that is present majority of the time. From there, an individual may have several **alternate personality states or alters**. Although it is hard to identify how many alters an individual may have at one time, it is believed that there are on average 15 alters for women and 8 for men (APA, 2000).

The presentation of *switching* between alternate personality states varies among individuals and can be as simple as the individual appearing to fall asleep to very dramatic, involving excessive bodily movements. While often sudden and unexpected, switching is generally precipitated by a significant stressor, as the alter is best equipped to handle the current stressor will present. The relationship between alters varies between individuals – with some individuals reporting knowledge of other alters while others have a one-way amnesic relationship with alters, meaning they are not aware of other personality states (Barlow & Chu, 2014). These individuals will experience episodes of “amnesia” when the primary personality is not present.

7.4.2: Epidemiology

Dissociative disorders were once believed to be extremely rare; however, more recent research suggests that they may be more present in the general population than once believed. Estimates for the prevalence rate of DID is 1.5% (APA, 2013), with more women experiencing the disorder. Due to the high comorbidity between childhood abuse and DID, it is believed that symptoms begin in early childhood following the repeated exposure to abuse; however, full onset of the disorder may not be observed (or noticed by others) until adolescence (Sar et al., 2014) or later in life. Over 70% of people with DID have attempted suicide and other self-injurious behaviors are common (APA, 2013).

7.4.3: Comorbidity

People with DID commonly experience a large number of comorbid disorders including PTSD and other trauma and stressor-related disorders, depressive disorders, somatic symptom disorders (e.g., conversion disorder), eating disorders, substance-related disorders, obsessive-compulsive disorder, sleep disturbances, as well as avoidant personality disorder and borderline personality disorder.

7.4.4: Etiology

7.4.0.1: **7.4.4.1**: Biological

Once again, heritability rates for dissociation rage from 50-60% (Pieper, Out, Bakermans-Kranenburg, Van Ijzendoorn, 2011). However, it is suggested that the combination of genetic and environmental factors may play a larger role in the development of dissociative disorders than genetics alone (Pieper, Out, Bakermans-Kranenburg, Van Ijzendoorn, 2011).

7.4.0.1: **7.4.4.2**: Cognitive

Neuroimaging studies have revealed differences in hippocampus activation between alters (Tsai, Condie, Wu & Chang, 1999). As you may recall, the hippocampus is responsible for storing information from short-term to long-term memory. It is hypothesized that this brain region is responsible for the generation of dissociative states and amnesia (Staniloiu & Markowitsch, 2010).

7.4.0.1: 7.4.4.3: Sociocultural

The sociocultural model of dissociative disorders has largely been influenced by Lilienfeld and colleagues (1999) who argue that the influence of mass media and its publications of dissociative disorders, provide a model for individuals to not only learn about dissociative disorders but also engage in similar dissociative behaviors. This theory has been supported by the significant increase in DID cases after the publication of *Sybil*, a documentation of a woman's 16 personalities (Goff & Simms, 1993).

These mass media productions are not just suggestive to patients; mass media also influences the way clinicians gather information regarding dissociative symptoms of patients. For example, therapists may unconsciously use questions or techniques in sessions that evoke dissociative types of problems in their patients following exposure to a media source discussing dissociative disorders.

7.4.0.1: 7.4.4.4: Psychodynamic

Once again, the psychodynamic theory of dissociative disorders assumes that dissociative disorders are caused by an individual's repressed thoughts and feelings related to an unpleasant or traumatic event (Richardson, 1998). In blocking these thoughts and feelings, the individual is subconsciously protecting himself from painful memories.

While dissociative amnesia may be explained by a single repression, psychodynamic theorists believe that DID results from repeated exposure to traumatic experiences, such as severe childhood abuse, neglect, or abandonment (Dalenberg et al., 2012). According to the psychodynamic perspective, children who experience repeated traumatic events such as physical abuse or parental neglect lack the support and resources to cope with these experiences. In efforts to escape from their current situations, children develop different personalities to essentially flee the dangerous situation they are in. While there is limited scientific evidence to support this theory, the nature of severe childhood psychological trauma is consistent with this theory, as individuals with DID have the highest rate of childhood psychological trauma compared to all other psychiatric disorders (Sar, 2011).

7.4.5: Treatment

The ultimate treatment goal for DID is **integration** of alternate personalities to a point of **final fusion** (Chu et al., 2011). Integration refers to the ongoing process of merging alternate personalities into one personality. Psychoeducation is paramount for integration, as the individual must have an understanding of their disorder, as well as acknowledge their alternate personalities. Like mentioned above, many individuals have a one-way amnesic relationship with their alters, meaning they are not aware of one another. Therefore, the clinician must first make the individual aware of the various alters that present across different situations.

Achieving integration requires several steps. First, the clinician needs to build a relationship and strong rapport with the primary personality. From there, the clinician can begin to encourage gradual communication and coordination between the alternate personalities. Making the alternate personalities aware of one another, as well as addressing their conflicts, is an essential component of the integration of these personalities, and the core of DID treatment (Chu et al., 2011).

Once the individual is aware of their personalities, treatment can continue with the goal of **fusion**. Fusion occurs when two or more alternate identities join together (Chu et al., 2011). When this happens, there is a complete loss of separateness. Depending on the number of personalities, this process can take quite a while. Once all alternate personalities are fused together and the individual identifies themselves as one unified self, it is believed the patient has reached **final fusion**.

It should be noted that final fusion is difficult to obtain. As you can imagine, some clients do not find final fusion as a desirable outcome, particularly those with extremely painful histories; chronic, serious stressors; advanced age; and comorbid medical and psychiatric disorders to name a few. For individuals where final fusion is *not* the treatment goal, the clinician may work toward resolution or sufficient integration and coordination of alternate personalities that allows the individual to function independently (Chu et al., 2011). Unfortunately, individuals that do not achieve final fusion are at greater risk for relapse of symptoms, particularly those with whose DID appears to stem from traumatic experiences.

Once an individual reaches final fusion, ongoing treatment is essential to maintain this status. In general, treatment focuses on social and positive coping skills. These skills are particularly helpful for individuals with a history of traumatic events, as it can help them process these events, as well as help to prevent future relapses.

Chapter Recap

In this chapter, we discussed Depersonalization/Derealization Disorder, Dissociative Amnesia, and Dissociative Identity Disorder, in terms of their clinical presentation, diagnostic criteria, epidemiology, comorbidity, etiology, and treatment approaches. This represents the final class of disorders covered in this book.

CHAPTER OVERVIEW

8: Schizophrenia Spectrum and Other Psychotic Disorders

Learning Objectives

- Describe the symptoms of schizophrenia spectrum disorders.
- Distinguish between the various schizophrenia spectrum disorders
- Describe the epidemiology of schizophrenia spectrum disorders.
- Describe comorbidity in relation to schizophrenia spectrum disorders.
- Describe the etiology of schizophrenia spectrum disorders.
- Describe treatment options for schizophrenia spectrum disorders.

In Chapter 8, we will discuss matters related to schizophrenia spectrum disorders to include their clinical presentation, diagnostic criteria, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include schizophrenia, schizophreniform disorder, brief psychotic disorder, schizoaffective disorder, and delusional disorder. We will depart from our usual convention of describing the epidemiology, comorbidity, etiology, and treatment for each disorder separately because there is a great deal of overlap among these for the various schizophrenia spectrum disorders. Be sure you refer Chapters 1-3 for explanations of key terms (Chapter 1), an overview of the various models to explain psychopathology (Chapter 2), and descriptions of the various therapies (Chapter 3).

8.1: Clinical Presentation

Section Learning Objectives

- Identify and describe the five symptoms of schizophrenia spectrum disorders.
- Describe how schizophrenia presents itself.
- Describe how schizophreniform disorder presents itself.
- Describe how brief psychotic disorder presents itself.
- Describe how schizoaffective disorder presents itself.
- Describe how delusional disorder presents itself.
- Be able to distinguish the five disorders from one another.

8.1.1: Symptoms of Schizophrenia Spectrum Disorders

Individuals diagnosed with a schizophrenia spectrum disorder experience **psychosis,** which is defined as a loss of contact with reality and is manifested by delusions and/or hallucinations. These episodes of psychosis can make it difficult for individuals to perceive and respond to environmental stimuli, which can cause significant disturbances in everyday functioning. While there are a number of symptoms displayed in schizophrenia spectrum disorders, presentation of symptoms varies greatly among individuals, as there are rarely two cases similar in presentation, triggers, course, or responsiveness to treatment (APA, 2013). We will now turn our attention to the five major symptoms associated with these disorders: delusions, hallucinations, disorganized speech, disorganized behavior, and negative symptoms.

8.1.1.1: Delusions

Delusions are defined as "fixed beliefs that are not amenable to change in light of conflicting evidence" (APA, 2013, pp. 87). This means that despite evidence contradicting one's thoughts, the individual is unable to distinguish them from reality. There are a variety of delusions that can present in many different ways:

- **Delusions of grandeur –** beliefs they have exceptional abilities, wealth, or fame; the belief they are God or other religious saviors
- **Delusions of persecution –** beliefs they are going to be harmed, harassed, plotted or discriminated against by either an individual or an institution
- **Delusions of reference –** beliefs that specific gestures, comments, or even larger environmental cues (e.g., an ad in the newspaper, a terrorist attack) are directed at them
- **Delusions of control –** beliefs that their thoughts/feelings/actions are controlled by others
- **Delusions of thought broadcasting –** beliefs that one's thoughts are transparent and everyone knows what they are thinking
- **Delusions of thought withdrawal –** belief that one's thoughts have been removed by another source

The most common delusion is delusions of persecution (APA, 2013). It is believed that the presentation of the delusion is largely related to the social, emotional, educational, and cultural background of the individual (Arango & Carpenter, 2010). For example, an individual with schizophrenia who comes from a highly religious family is more likely to experience religious delusions (e.g., *delusions of grandeur)* than another type of delusion.

8.1.1.2: Hallucinations

Hallucinations can occur in any of the five senses including as hearing (auditory hallucinations), seeing (visual hallucinations), smelling (olfactory hallucinations), touching (tactile hallucinations), or tasting (gustatory hallucinations). Additionally, they can occur in a single modality or present across a combination of modalities (i.e. experiencing both auditory and visual hallucinations). For the most part, individuals recognize that their hallucinations are not real and attempt to engage in normal behavior while simultaneously combating ongoing hallucinations.

According to various research studies, nearly half of all people with schizophrenia report auditory hallucinations, 15% report visual hallucinations, and 5% report tactile hallucinations (DeLeon, Cuesta, & Peralta, 1993). Among the most common types of auditory hallucinations are voices talking to the individual or various voices talking to one another. Generally, these hallucinations are not attributable to any one person that the individual knows. However, they are usually clear, objective, and definite (Arango & Carpenter, 2010). Additionally, the auditory hallucinations can be pleasurable, providing comfort to the individuals; however, in other individuals, the auditory hallucinations can be unsettling as they produce commands or have malicious intent.

8.1.1.3: Disorganized Speech

Among the most common cognitive impairments displayed in individuals with schizophrenia are disorganized speech, communication, and thoughts. More specifically, thoughts and speech patterns may appear to be *circumstantial* or *tangential*. For example, individuals with **circumstantial speech** may give unnecessary details in response to a question before they finally produce the desired response. While the question is eventually answered by individuals with circumstantial speech, those with **tangential speech** never reach the point or answer the question. Another common cognitive symptom is **speech retardation** where the individual may take a long period of time before answering a question. **Derailment**, or the illogical connection in a chain of thoughts, is another common type of disorganized thinking. The most severe form of disorganized speech is **incoherence** or word salad which is where speech is completely incomprehensible and meaningful sentences are not produced.

These type of distorted thought patterns are often related to concrete thinking. That is, the individual is focused on one aspect of a concept or thing, and neglects all other aspects. This type of thinking makes treatment difficult as individuals lack insight into their illness and symptoms (APA, 2013).

8.1.1.4: Disorganized Behavior

Psychomotor symptoms can also be observed in individuals with schizophrenia spectrum disorders. These behaviors may manifest as awkward movements or even ritualistic/repetitive behaviors. They are often unpredictable and overwhelming, severely impacting the ability to perform daily activities (APA, 2013). **Catatonic behavior**, or the decrease or even lack of reactivity to the environment, is among the most commonly seen disorganized motor behavior in schizophrenia spectrum disorders. These catatonic behaviors include:

- **Negativism –** resistance to instruction
- **Mutism –** complete lack of verbal responses
- **Stupor –** complete lack of motor responses
- **Rigidity –** maintaining a rigid or upright posture while resisting efforts to be moved
- **Posturing –** holding odd, awkward postures for long periods of time

On the opposite side of the spectrum is **catatonic excitement,** where the individual experiences a hyperactivity of motor behavior. This can include **echolalia** (mimicking the speech of others) and **echopraxia** (mimicking the movement of others) but may also simply be manifested through excessive and/or purposeless motor behaviors.

8.1.1.5: Negative Symptoms

Up until this point, all the schizophrenia symptoms can be categorized as **positive symptoms** or symptoms that involve the presence of something that should not be there (e.g., hallucinations and delusions) or **disorganized symptoms** (disorganized speech and behavior). The final symptom included in the diagnostic criteria of several of the schizophrenia spectrum disorders is **negative symptoms**, which are defined as the inability, or decreased ability, to initiate actions, speech, express emotion, or to feel pleasure (Barch, 2013). Negative symptoms are typically present before positive symptoms and often remain once positive symptoms remit. They account for much of the morbidity in schizophrenia but not as prominent in the other spectrum disorders (indeed, as you will see, they are not included as a symptom in some of these other disorders). Because of their prevalence through the course of the schizophrenia, they are also more indicative of prognosis, with more negative symptoms suggestive of a poorer prognosis. The poorer prognosis may be explained by the lack of effect that traditional antipsychotic medications have in addressing negative symptoms (Kirkpatrick, Fenton, Carpenter, & Marder, 2006).

There are five main types of negative symptoms seen in individuals with schizophrenia:

- **Affective flattening –** reduction in emotional expression (i.e., a reduced display of emotional expression)
- **Alogia –** poverty of speech or speech content
- **Anhedonia –** decreased ability to experience pleasure
- **Asociality –** lack of interest in social relationships
- **Avolition –** lack of motivation of goal-directed behavior

8.1.2: Types of Schizophrenia Spectrum Disorders

8.1.2.1: Schizophrenia

As stated above, the hallmark symptoms of schizophrenia include the presence of at least *two* of the following symptoms for at least one month: delusions, hallucinations, disorganized speech, disorganized/abnormal behavior, negative symptoms. These

symptoms must create significant impairment in the individual's ability to engage in normal daily functioning such as work, school, relationships with others, or self-care. It should be noted that presentation of schizophrenia varies greatly among individuals, as it is a heterogeneous clinical syndrome (APA, 2013).

While the presence of active phase symptoms must persist for a minimum of one month to meet criteria for a schizophrenia diagnosis, the total duration of symptoms must persist for at least six months before a diagnosis of schizophrenia can be made. This six month period can comprise a combination of active, prodromal, and residual phase symptoms. **Active phase** symptoms represent the "full-blown" symptoms previously described. **Prodromal** symptoms are "subthreshold" symptoms that precede the active phase of the disorder and **residual** symptoms are subthreshold symptoms that follow the active phase. These prodromal and residual symptoms are milder forms of symptoms that do not cause significant impairment in functioning, with the exception of negative symptoms (Lieberman et al., 2001). Due to the severity of psychotic symptoms, mood disorder symptoms are also common among individuals with schizophrenia; however, to diagnose schizophrenia either there must be no mood symptoms or if mood symptoms have occurred they must be present for only a minority of the total duration of the illness. The latter helps to distinguish schizophrenia from a mood disorder with psychotic features for which psychotic symptoms are limited to the context of the mood episodes and do not extend beyond those episodes.

8.1.2.2: Schizophreniform Disorder

Schizophreniform disorder is similar to schizophrenia with the exception of the length of presentation of symptoms and the requirement for impairment in functioning. As described above, a diagnosis of schizophrenia requires impairment in functioning and a six-month minimum duration of symptoms. In contrast, impairment in functioning is not required to diagnose schizophreniform disorder. While many individuals with schizophreniform disorder do display impaired functioning, it is not essential for diagnosis. Moreover, symptoms must last at least one month but less than six-months do diagnose schizophreniform disorder. In this way, the duration of schizophreniform disorder is considered an "intermediate" disorder between schizophrenia and brief psychotic disorder (which we will consider next).

Approximately two-thirds of individuals who are initially diagnosed with schizophreniform disorder will have symptoms that last longer than six months, at which time their diagnosis is changed to schizophrenia (APA, 2013). The other one-third will recover within the six month time period and schizophreniform disorder will be their final diagnosis.

Finally, as with schizophrenia, any major mood episodes that are present concurrently with the psychotic features must only be present for a small period of time, otherwise, a diagnosis of schizoaffective disorder may be more appropriate.

8.1.2.3: Brief Psychotic **Disorder**

A diagnosis of brief psychotic disorder requires one or more of the following symptoms: delusions, hallucinations, disorganized speech, disorganized behavior. Moreover at least one of these symptoms must be delusions, hallucinations, or disorganized speech. Notice that negative symptoms are not included in this list. Also notice that while schizophrenia and schizophreniform disorder require a minimum of two symptoms, only one is required for a diagnosis of brief psychotic disorder. To diagnose brief psychotic disorder symptom(s) must be present for at least one day but less than one month (recall: one month is the minimum duration of symptoms required to diagnose schizophreniform disorder). After one-month individuals return to their full premorbid level of functioning. Also, while there is typically very severe impairment in functioning associated with brief psychotic disorder it is not required for a diagnosis.

8.1.2.4: Schizoaffective Disorder

Schizoaffective disorder is characterized by two or more of the symptoms of schizophrenia (delusions, hallucinations, disorganized speech, disorganized behavior, negative symptoms) *and* a concurrent uninterrupted period of a major mood episode—either a depressive or manic episode. Those who experience only depressive episodes are diagnosed with the **depressive type** of schizoaffective disorder while those who experience manic episodes (with or without depressive episodes) are diagnosed with the **bipolar type** of schizoaffective disorder. It should be noted that because a loss of interest in pleasurable activities is a common symptom of schizophrenia, to meet criteria for a depressive episode within schizoaffective disorder, the individual must present with a pervasive depressed mood (not just anhedonia). While schizophrenia and schizophreniform disorder do *not* have a significant mood component, schizoaffective disorder requires the presence of a depressive or manic episode for the majority, if not the total duration of the disorder. While psychotic symptoms are sometimes present in depressive episodes, they often remit once the depressive episode is resolved. For individuals with schizoaffective disorder, psychotic symptoms should continue for at least two weeks in the absence of a major mood disorder (APA, 2013). This is the key distinguishing feature between schizoaffective disorder and major depressive disorder with psychotic features.

8.1.2.5: Delusional Disorder

As suggestive of its title, delusional disorder requires the presence of at least one delusion that lasts for at least one month in duration. It is important to note that any other symptom of schizophrenia (i.e., hallucinations, disorganized behavior, disorganized speech, negative symptoms) rules out a diagnosis of delusional disorder. Therefore the only symptom that can be present is delusions. Unlike most other schizophrenia-related disorders, daily functioning is not overtly impacted in individuals with delusional disorder. Additionally, if symptoms of depressive or manic episodes present during delusions, they are typically brief in duration compared to the duration of the delusions.

The DSM 5 (APA, 2013) has identified several subtypes of delusional disorder in efforts to better categorize the symptoms of the individual's disorder. When making a diagnosis of delusional disorder, one of the following specifiers is included.

- **Erotomanic delusion –** the individual reports a delusion of another person being in love with them. Generally speaking, the individual whom the convictions are about are of higher status such as a celebrity.
- **Grandiose delusion –** involves the conviction of having a great talent or insight. Occasionally, individuals will report they have made an important discovery that benefits the general public. Grandiose delusions may also take on a religious affiliation, as some people believe they are prophets or even God, himself.
- **Jealous delusion –** revolves around the conviction that one's spouse or partner is/has been unfaithful. While many individuals may have this suspicion at some point in their relationship, a jealous delusion is much more extensive and generally based on incorrect inferences that lack evidence.
- **Persecutory delusion –** involves beliefs that they are being conspired against, spied on, followed, poisoned or drugged, maliciously maligned, harassed, or obstructed in pursuit of their long-term goals (APA, 2013). Of all subtypes of delusional disorder, those experiencing persecutory delusions are the most at risk of becoming aggressive or hostile, likely due to the persecutory nature of their distorted beliefs.
- **Somatic delusion –** involves delusions regarding bodily functions or sensations. While these delusions can vary significantly, the most common beliefs are that the individual emits a foul odor despite attempts to rectify their smell; there is an infestation of insects on the skin; or that they have an internal parasite (APA, 2013).
- Mixed delusions – there are several themes of delusions (e.g., jealous and persecutory)
- **Unspecified delusion** – these are delusions that don't fit into one of the categories above (e.g., referential delusions without a persecutory or grandiose nature to them).
- **Bizarre delusion –** delusions that are clearly not plausible and do not stem from ordinary experience (e.g., the delusion that one is an alien/vampire hybrid).

8.2: Epidemiology and Comorbidity

Section Learning Objectives

- Indicate the prevalence of schizophrenia spectrum disorders.
- Describe the sex ratios for these disorders.
- Identify the disorders that are commonly comorbid with schizophrenia spectrum disorders.

8.2.1: Epidemiology

Schizophrenia occurs in approximately 0.3%-0.7% of the general population (APA, 2013). There is some discrepancy between the rates of diagnosis between genders; these differences appear to be related to the emphasis of various symptoms. For example, men typically present with more negative symptoms whereas women present with more mood-related symptoms. Despite gender differences in presentation of symptoms, there appears to be an equal risk for both genders to develop the disorder.

Schizophrenia typically occurs between late teens and mid-30's, with the onset of the disorder typically occurring slightly earlier for males than for females (APA, 2013). Earlier onset of the disorder is generally predictive of worse overall prognosis. The onset of symptoms is typically gradual, with initial symptoms presenting similarly to depressive disorders; however, some individuals will present with an abrupt presentation of the disorder. Negative symptoms appear to be more predictive of poorer prognosis than other symptoms. This may be due to negative symptoms being the most persistent, and therefore, most difficult to treat. Overall, an estimated 20% of individuals who are diagnosed with schizophrenia report complete recovery of symptoms (APA, 2013).

Schizoaffective disorder, schizophreniform disorder, brief psychotic disorder, and delusional disorder prevalence rates are all significantly less than that of schizophrenia, occurring in less than 0.3% of the general population. While the depressive type of schizoaffective disorder is diagnosed more in females than males, schizophreniform and delusional disorder appear to be diagnosed equally between genders. The gender discrepancy in schizoaffective disorder is likely due to the higher rate of depressive symptoms as seen in females than males because this sex discrepancy is not evident in the bipolar type of the disorder (APA, 2013).

8.2.2: Comorbidity

There is a high comorbidity rate between schizophrenia spectrum disorders and substance abuse disorders. Furthermore, there is some evidence to suggest that the use of various substances (specifically marijuana) may place an individual at an increased risk to develop schizophrenia if the genetic predisposition is also present (Corcoran et al., 2003). Additionally, there appears to be an increase in anxiety-related disorders—specifically obsessive-compulsive disorder and panic disorder—among individuals with schizophrenia than compared to the general public.

It should also be noted that individuals diagnosed with a schizophrenia spectrum disorder are also at an increased risk for associated medical conditions such as weight gain, diabetes, metabolic syndrome, and cardiovascular and pulmonary disease (APA, 2013). This predisposition to various medical conditions is likely related to medications and poor lifestyle choices, and also place individuals at risk for a reduced life expectancy.

8.3: Etiology

Section Learning Objectives

- Describe the biological causes of schizophrenia spectrum disorders.
- Describe the psychological causes of schizophrenia spectrum disorders.
- Describe the sociocultural causes of schizophrenia spectrum disorders.

8.3.1: Biological

8.3.1.1: Genetics

Twin and family studies consistently support the biological theory. More specifically, if one identical twin develops schizophrenia, there is a roughly a 50% that the other will also develop the disorder within their lifetime (Coon & Mitter, 2007). This percentage drops to 17% in fraternal twins. Similarly, family studies have also found similarities in brain abnormalities among individuals with schizophrenia and their relatives; the more similarities, the higher the likelihood that the family member also developed schizophrenia (Scognamiglio & Houenou, 2014).

8.3.1.2: Neurobiological

There is consistent and reliable evidence of a neurobiological component in the transmission of schizophrenia. More specifically, neuroimaging studies have found a significant reduction in overall and specific brain regions volumes, as well as in tissue density of individuals with schizophrenia compared to healthy controls (Brugger, & Howes, 2017). Additionally, there has been evidence of ventricle enlargement as well as volume reductions in the medial temporal lobe. As you may recall, structures such as the amygdala (involved in emotion regulation), the hippocampus (involved in memory), as well as the neocortical surface of the temporal lobes (processing of auditory information) are all structures within the medial temporal lobe (Kurtz, 2015). Additional studies also indicate a reduction in the orbitofrontal regions of the brain, a part of the frontal lobe that is responsible for response inhibition (Kurtz, 2015).

8.3.1.3: Stress Cascade

The stress-vulnerability model suggests that individuals have a genetic or biological predisposition to develop the disorder; however, symptoms will not present unless there is a stressful precipitating factor that elicits the onset of the disorder. Researchers have identified the HPA axis and its consequential neurological effects as the likely responsible neurobiological component responsible for this stress cascade.

The HPA axis is one of the main neurobiological structures that mediates stress. It involves the regulation of three chemical messengers (corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and glucocorticoids) as they respond to a stressful situation (Corcoran et al., 2003). Glucocorticoids, more commonly referred to as cortisol, is the final neurotransmitter released which is responsible for the physiological change that accompanies stress to prepare the body to “fight” or “flight.”

It is hypothesized that in combination with abnormal brain structures, persistently increased levels of glucocorticoids in brain structures may be the key to the onset of psychosis in individuals in a prodromal phase (Corcoran et al., 2003). More specifically, the stress exposure (and increased glucocorticoids) affects the neurotransmitter system and exacerbates psychotic symptoms due to changes in dopamine activity (Walker & Diforio, 1997). While research continues to explore the relationship between stress and the onset of schizophrenia spectrum disorders, evidence for the implication of stress and symptom relapse is strong. More specifically, individuals with schizophrenia experience more stressful life events leading up to a relapse of symptoms. Similarly, it is hypothesized that the worsening or exacerbation of symptoms is also a source of stress as symptoms interfere with daily functioning (Walker & Diforio, 1997). This stress alone may be enough to initiate a relapse.

8.3.2: Psychological

The cognitive model utilizes some of the aspects of the diathesis-stress model in that it proposes that premorbid neurocognitive impairment places individuals at risk for aversive work/academic/interpersonal experiences. These experiences in return lead to dysfunctional beliefs and cognitive appraisals, ultimately leading to maladaptive behaviors such as delusions/hallucinations (Beck & Rector, 2005).

Beck proposed a diathesis-stress model of development of schizophrenia. Based on his theory, an underlying neurocognitive impairment makes an individual more vulnerable to experience aversive life events such as homelessness, conflict within the family, etc. Individuals with schizophrenia are more likely to evaluate these aversive life events with a dysfunctional attitude and maladaptive cognitive distortions. The combination of the aversive events and negative interpretations of them, produces a stress response in the individual, thus igniting hyperactivation of the HPA axis. According to Beck and Rector (2005), it is the culmination of these events leads to the development of schizophrenia.

8.3.3: Sociocultural

8.3.3.1: Expressed Emotion

Research in support of a supportive family environment suggests that families high in expressed emotion, meaning families that have highly hostile, critical, or overinvolved family members, are predictors of relapse (Bebbington & Kuipers, 2011). In fact, individuals who return to families post hospitalization with high criticism and emotional involvement are twice as likely to relapse compared to those who return to families with low expressed emotion (Corcoran et al., 2003). Several meta-analyses have concluded that family atmosphere is causally related to relapse in individuals with schizophrenia and that these outcomes can be improved when the family environment is improved (Bebbington & Kuipers, 2011). Therefore, one major treatment goal in families of people with schizophrenia is to reduce expressed emotion within family interactions.

8.3.3.2: Family Dysfunction

Even for families with low levels of expressed emotion, there is often an increase in family stress due to the secondary effects of schizophrenia. Having a family member who has been diagnosed with schizophrenia increases the likelihood of a disruptive family environment due to managing their symptoms and ensuring their safety while they are home (Friedrich & Wancata, 2015). Because of the severity of symptoms, families with a loved one diagnosed with schizophrenia often report more conflict in the home as well as more difficulty communicating with one another (Kurtz, 2015).

8.4: Treatment

Section Learning Objectives

- Describe psychopharmacological treatment options for schizophrenia spectrum disorders.
- Describe psychological treatment options for schizophrenia spectrum disorders.
- Describe family interventions for schizophrenia spectrum disorders.

8.4.1: Psychopharmacological

Among the first antipsychotic medications used for the treatment of schizophrenia was Thorazine. Developed as a derivative of antihistamines, Thorazine was the first line of treatment that produced a calming effect on even the most severely agitated individuals and allowed for the organization of thoughts. Despite their effectiveness in managing psychotic symptoms, *conventional* or *first-generation* antipsychotics (such as Thorazine and Chlorpromazine) also produced significant negative side effects similar to that of neurological disorders. Therefore, psychotic symptoms were replaced with muscle tremors, involuntary movements, and muscle rigidity. Additionally, these conventional antipsychotics also produced **tardive dyskinesia**, which includes involuntary movements isolated to the tongue, mouth, and face (Tenback et al., 2006). While only 10% of clients reported the development of tardive dyskinesia, this percentage increased the longer they were on the medication, as well as the higher the dose (Achalia, Chaturvedi, Desai, Rao, & Prakash, 2014). In efforts to avoid these symptoms, clinicians have been cognizant of not exceeding the clinically effective dose of conventional antipsychotic medications. Should the management of psychotic symptoms not be resolved at this level, alternative medications are often added to produce a synergistic effect (Roh et al., 2014).

Due to the harsh side effects of conventional antipsychotic drugs, newer, arguably more effective *second generation* or *atypical* antipsychotic drugs have been developed. The atypical antipsychotic drugs appear to act on both dopamine and serotonin receptors, as opposed to only dopamine receptors in the conventional antipsychotics. Because of this, common atypical antipsychotic medications such as clozapine (Clozaril), risperidone (Risperdal), and aripiprazole (Abilify), appear to be more effective in managing *both* the positive *and* negative symptoms. While there does continue to be a risk of developing side effects such as tardive dyskinesia, recent studies suggest it is much lower than that of the conventional antipsychotics (Leucht, Heres, Kissling, & Davis, 2011). Due to their effectiveness and minimal side effects, atypical antipsychotic medications are typically the first line of treatment for schizophrenia (Barnes & Marder, 2011).

It should be noted that because of the harsh side effects of antipsychotic medications in general, many individuals, nearly one-half to three-quarters, discontinue use of antipsychotic medications (Leucht, Heres, Kissling, & Davis, 2011). Because of this, it is also important to incorporate psychological treatment along with psychopharmacological treatment to both address medication adherence, as well as provide additional support for symptom management.

8.4.2: Psychological Interventions

8.4.2.1: Cognitive Behavioral Therapy (CBT)

CBT has been thoroughly discussed in previous chapters and it should be clear that the goal of this treatment is to identify the negative biases and attributions that influence an individual's interpretations of events and the subsequent consequences of these thoughts and behaviors. When used in the context of a schizophrenia spectrum disorder, CBT focuses on the maladaptive emotional and behavioral responses to psychotic experiences, which is directly related to distress and disability. Therefore, the goal of CBT is *not* on symptom reduction, but rather to improve the interpretations and understandings of these symptoms (and experiences) which will reduce associated distress (Kurtz, 2015). Common features of CBT in this context include: psychoeducation about their disorder, the course of their symptoms (i.e. ways to identify coming and going of delusions/hallucinations), challenging and replacing the negative thoughts/behaviors to more positive thoughts/behaviors associated with their delusions/hallucinations, and finally, learning positive coping strategies to deal with their unpleasant symptoms (Veiga-Martinez, Perez-Alvarez, & Garcia-Montes, 2008).

Findings from studies exploring CBT as a supportive treatment have been promising. One study conducted by Aaron Beck (the founder of CBT) and colleagues (Grant, Huh, Perivoliotis, Stolar, & Beck, 2012) found that recovery-oriented CBT produced a marked improvement in overall functioning as well as symptom reduction in clients diagnosed with schizophrenia. This study suggests that by focusing on targeted goals such as independent living, securing employment, and improving social relationships,

individuals were able to slowly move closer to these targeted goals. By also including a variety of CBT strategies such as role-playing, scheduling community outings, and addressing negative cognitions, individuals were also able to address cognitive and social skill deficits.

8.4.2.2: Family Interventions

Family interventions have been largely influenced by the diathesis-stress model of schizophrenia. As previously discussed, the emergence of the disorder and/or exacerbation of symptoms is likely related to environmental stressors and psychological factors. While the degree in which environmental stress stimulates an exacerbation of symptoms varies among individuals, there is significant evidence to conclude that overall stress *does* impact illness presentation (Haddock & Spaulding, 2011). Therefore, the overall goal of family interventions is to reduce the stress on the individual that is likely to elicit the relapse of symptoms.

Unlike many other psychological interventions, there is not a specific outline for family-based interventions related to schizophrenia. However, the majority of programs include the following three components: psychoeducation, problem-solving skills, and cognitive-behavioral therapy.

- *Psychoeducation* is important for both the client and family members as it is reported that more than half of those recovering from a psychotic episode reside with their family (Haddock & Spaulding, 2011). Therefore, educating families on the course of the illness, as well as ways to recognize the onset of psychotic symptoms is important to ensure optimal recovery.
- *Problem-solving* is a very important component in the family intervention model. Seeing as family conflict can increase stress within the home, which in return can lead to exacerbation and relapse of psychotic symptoms, family members benefit from learning effective methods of problem-solving to address family conflicts. Additionally, teaching positive coping strategies for dealing with the symptoms and their direct effect on the family environment may also alleviate some conflict within the home
- *CBT* is similar to that described above. The goal of family-based CBT is to reduce negativity among family member interactions, as well as help family members adjust to living with someone with psychotic symptoms. These three components within the family intervention program have been shown to reduce re-hospitalization rates, as well as slow the worsening of schizophrenia-related symptoms (Pitschel-Walz, Leucht, Baumi, Kissling, & Engel, 2001).

8.4.2.3: Social Skills Training

Given the poor interpersonal functioning among individuals with schizophrenia, social skills training is another type of treatment that is commonly suggested to improve psychosocial functioning. Research has indicated that poor interpersonal skills not only predate the onset of the disorder but also remain significant even with the management of symptoms via antipsychotic medications. Impaired ability to interact with individuals in social, occupational, or recreational settings is related to poorer psychological adjustment (Bellack, Morrison, Wixted, & Mueser, 1990). This can lead to greater isolation and poorer social support among individuals with schizophrenia. As previously discussed, social support has been identified as a protective factor against symptom exacerbation, as it buffers psychosocial stressors that are often responsible for exacerbation of symptoms. Learning how to appropriately interact with others (i.e. establish eye contact, engage in reciprocal conversations, etc.) through role play in a group therapy setting is one effective way to teach positive social skills.

8.4.2.4: Inpatient Hospitalizations

More commonly viewed as community-based treatments, inpatient hospitalization programs are essential in stabilizing individuals experiencing acute psychotic episodes. Generally speaking, individuals will be treated on an outpatient basis, however, there are times when their symptoms exceed the needs of an outpatient service. *Short-term* hospitalizations are used to modify antipsychotic medications and implement additional psychological treatments so that the individual can safely return to his/her home. These hospitalizations generally last for a few weeks as opposed to a long-term treatment option that would last months or years (Craig & Power, 2010).

In addition to short-term hospitalizations, there are also *partial* hospitalizations where an individual enrolls in a full-day program but returns home for the evening/night. These programs provide individuals with intensive therapy, organized activities, and group therapy programs that enhance social skills training. Research supports the use of partial hospitalizations as individuals enrolled in these programs tend to do better than those who enroll in outpatient care (Bales et al., 2014).

While a combination of psychopharmacological, psychological, and family interventions is the most effective treatment in managing symptoms of schizophrenia spectrum disorders, rarely do these treatments restore the individual to premorbid levels of functioning (Kurtz, 2015; Penn et al., 2004). Although more recent advancements in treatment for schizophrenia spectrum disorders appear promising, the disorder itself is viewed as one that requires lifelong treatment and care.

Chapter Recap

In Chapter 8, we discussed the schizophrenia spectrum disorders to include schizophrenia, schizophreniform disorder, brief psychotic disorder, schizoaffective disorder, and delusional disorder. We started by describing the common symptoms of such disorders to include delusions, hallucinations, disorganized speech, disorganized behavior, and negative symptoms. We then identified how the various schizophrenia spectrum disorders are distinguished from one another. This then led to our normal discussion of the epidemiology, comorbidity, etiology, and treatment options of the disorders. In our final chapter, we will discuss personality disorders.

CHAPTER OVERVIEW

9: Personality Disorders

Learning Objectives

- Describe how personality disorders present and be able to distinguish between each.
- Identify the disorders included in each cluster and the characterization of each cluster.
- Describe the epidemiology of personality disorders.
- Describe comorbidity in relation to personality disorders.
- Describe the etiology of personality disorders.
- Describe treatment options for personality disorders.

In Chapter 9, we will discuss matters related to personality disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include Cluster A personality disorders of paranoid, schizoid, and schizotypal; Cluster B personality disorders of antisocial, borderline, histrionic, and narcissistic; and Cluster C personality disorders of avoidant, dependent, and obsessive-compulsive. As always, be sure you refer Chapters 1-3 for explanations of key terms (Chapter 1), an overview of the various models to explain psychopathology (Chapter 2), and descriptions of the various therapies (Chapter 3).

9.1: Overview of Clusters and Personality Disorders

Section Learning Objectives

- Describe the symptoms associated with each cluster A personality disorder.
- Describe the epidemiology of cluster A personality disorders.
- Describe the treatments for cluster A personality disorders.

9.1.1: Overview

In order to be diagnosed with any personality disorder, the individual must exhibit a pervasive and long-lasting pattern of inflexible behavior that violates cultural norms and is manifested in at least two of the following four areas: *distorted thinking patterns*, *problematic emotional responses*, *over- or under-regulated impulse control*, and *interpersonal difficulties*. While these four core features are common among all ten personality disorders, the DSM-5 divides the personality disorders into three different clusters based on symptom similarities. The pattern of behavior must persist since adolescence or early adulthood and must result in significant distress or impairment. Without distress or impairment, the pattern should be considered a personality *trait* rather than a *disorder.*

9.1.2: Cluster A

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). Often these behaviors are similar to those seen in schizophrenia. In fact, there is a strong relationship between cluster A personality disorders among individuals who have a relative diagnosed with schizophrenia (Chemerinksi & Siever, 2011). However, the symptoms of cluster A personality disorders tend to be less extensive and less impactful on daily functioning relative to those experienced in schizophrenia.

9.1.3: Cluster B

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). Due to the dramatic, emotional, and erratic nature of these disorders, it is nearly impossible for individuals to establish healthy relationships with others.

9.1.4: Cluster C

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. Likely due to the similarity in symptoms with mental health disorders that have effective treatment options, cluster C disorders have the most treatment options of all personality disorders.

9.2: Cluster A Personality Disorders

Section Learning Objectives

- Describe the symptoms associated with each cluster A personality disorder.
- Describe the epidemiology of cluster A personality disorders.
- Describe the treatments for cluster A personality disorders.

9.2.1: Clinical Descriptions

9.2.1.1: Paranoid Personality Disorder

Paranoid personality disorder is characterized by a severe distrust or suspicion of others. Individuals interpret and believe that other's motives and interactions are intended to harm them, and therefore, they are skeptical about establishing close relationships outside of family members — although at times even family members' actions are believed to be malevolent (APA, 2013). Individuals with paranoid personality disorder often feel as though they have been deeply and irreversibly hurt by others even though there is little to no evidence to support that others intended to, or actually did, hurt them. Because of these persistent suspicions, they will doubt relationships that show true loyalty or trustworthiness.

Individuals with paranoid personality disorder are also hesitant to share any personal information or confide in others as they fear the information will be used against them (APA, 2013). Additionally, benign remarks or events are often interpreted as demeaning or threatening. For example, if an individual with paranoid personality disorder was accidentally bumped into at the store, they would interpret this action as intentional, with the purpose of causing them injury. Because of this, individuals with paranoid personality disorder are quick to hold grudges and unwilling to forgive insults or injuries – whether intentional or not (APA, 2013). They are known to quickly, and angrily counterattack either verbally or physically in situations where they feel they were insulted.

9.2.1.2: Schizoid Personality Disorder

Individuals with schizoid personality disorder display a persistent pattern of avoidance from social relationships along with a limited range of emotion among social relationships (APA, 2013). Similar to those with paranoid personality disorder, individuals with schizoid personality disorder do not have many close relationships; however, unlike paranoid personality disorder, this lack of relationship is not due to suspicious feelings, but rather, the lack of desire to engage with others and the preference to engage in solitary behaviors. Individuals with schizoid personality disorder are often viewed as "loners" and prefer activities where they do not have to engage with others (APA, 2013). Established relationships rarely extend outside that of the family as those diagnosed with schizoid personality disorder make no effort to start or maintain friendships. This lack of establishing social relationships also extends to sexual behaviors, as those with schizoid personality disorder report a lack of interest in engaging in sexual experiences with others.

With regard to the limited range of emotion, individuals with schizoid personality disorder are often indifferent to criticisms or praises of others and appear to not be affected by what others think of them (APA, 2013). They will rarely show any feelings or expression of emotions and are often described as having a "bland" exterior (APA, 2013). In fact, individuals with schizoid personality disorder rarely reciprocate facial expressions or gestures typically displayed in normal conversations such as smiles or nods. Because of this lack of emotions, there is limited need for attention or acceptance.

9.2.1.3: Schizotypal Personality Disorder

Schizotypal personality disorder is characterized by a range of impairment in social and interpersonal relationships due to discomfort in relationships, along with odd cognitive and/or perceptual distortions and eccentric behaviors (APA, 2013). Similar to those with schizoid personality disorder, these individuals also seek isolation and have few, if any established relationships outside of family members.

One of the most prominent features of schizotypal personality disorder is **ideas of reference** or the belief that unrelated events pertain to them in a particular and unusual way. This is a milder version of the delusions of reference that were discussed in the previous chapter. Ideas of reference also lead to superstitious behaviors or preoccupation with paranormal activities that are not generally accepted in their culture (APA, 2013). The perception of special or magical powers such as the ability to mind read or control other's thoughts has also been documented in individuals with schizotypal personality disorder. Unusual perceptual experiences such as sensing the presence of another person or hearing one's name (subthreshold hallucinations), as well as unusual speech patterns such as derailment or incoherence are also symptoms of this disorder.

Similar to the other personality disorders within cluster A, there is also a component of paranoia or suspiciousness of other's motives in schizotypal personality disorder. Additionally, individuals with this disorder also display inappropriate or restricted affect, thus impacting their ability to appropriately interact with others in a social context. Significant social anxiety is often also present in social situations, particularly in those involving unfamiliar people. The combination of limited affect and social anxiety contributes to their inability to establish and maintain personal relationships; most individuals with schizotypal personality disorder prefer to keep to themselves in efforts to reduce this anxiety.

9.2.2: Epidemiology

The cluster A personality disorders have a prevalence rate of around 3-5%. More specifically, paranoid personality disorder is estimated to affect approximately 4.4% of the general population, with no reported diagnosis discrepancy between genders (APA, 2013). Schizoid personality disorder occurs in 3.1% of the general population, whereas the prevalence rate for schizotypal personality disorder is 3.9%. Both schizoid and schizotypal personality disorders are more commonly diagnosed in males than females, with males also reportedly being more impaired by the disorder than females (APA, 2013).

Note: Due to the overlap among comorbidities and etiological factors we will reserve our discussion of those until the end of the chapter and will proceed directly to the treatment of the cluster A personality disorders.

9.2.3: Treatment

Individuals with personality disorders within cluster A often do not seek out treatment as they do not identify themselves as someone who needs help (Millon, 2011). Of those that do seek treatment, the majority do not enter it willingly. Furthermore, due to the nature of these disorder, individuals in treatment often struggle to trust the clinician as they are suspicious of the clinician's intentions (paranoid and schizotypal personality disorder) or are emotionally distant from the clinician as they do not have a desire to engage in treatment due to a lack of overall emotion and desire for relationships (schizoid personality disorder; Kellett & Hardy, 2014, Colli, Tanzilli, Dimaggio, & Lingiardi, 2014). Because of this, treatment is known to move very slowly, with many clients dropping out of treatment before any resolution of symptoms can be met.

When clients are enrolled in treatment, cognitive behavioral strategies are most commonly used with the primary intention of reducing anxiety-related symptoms. Additionally, attempts at cognitive restructuring – both identifying and changing maladaptive thought patterns – are also helpful in addressing the misinterpretations of other's words and actions, particularly in those with paranoid personality disorder (Kellett & Hardy, 2014). Clients with schizoid personality disorder may be engaged in CBT techniques to help them experience more positive emotions and engage in more satisfying social experiences; whereas the goal of CBT for schizotypal personality disorder is to evaluate unusual thoughts or perceptions objectively and to ignore the inappropriate thoughts (Beck & Weishaar, 2011). Finally, behavioral techniques such as social-skills training may also be implemented to address ongoing interpersonal problems displayed in the disorders.

9.3: Cluster B Personality Disorders

Section Learning Objectives

- Describe the symptoms of each cluster B personality disorder.
- Describe the epidemiology of cluster B personality disorders.
- Describe the treatments for cluster B personality disorders.

9.3.1: Antisocial Personality Disorder

9.3.1.1: Clinical Description

The defining feature of antisocial personality disorder is a consistent pattern of disregard for, and violation of, the rights of others (APA, 2013). While antisocial personality disorder can only be diagnosed in individuals who are 18 years of age or older, a diagnosis can only be made if there is evidence of conduct disorder prior to the age of 15. Although not discussed in this book, conduct disorder is a disorder of childhood that involves a repetitive and persistent pattern of behaviors that violate the rights of others (APA, 2013). Common behaviors exhibited by individuals with conduct disorder that go on to develop antisocial personality disorder are aggression toward people or animals, destruction of property, deceitfulness or theft, or serious violation of rules (APA, 2013).

While commonly referred to as “psychopaths” or “sociopaths” these are both separate (but related) disorders that are not recognized by the DSM. However, much like those with psychopathy and sociopathy, individuals with antisocial personality disorder fail to conform to social norms. This also includes legal rules as individuals with antisocial personality disorder are often repeatedly arrested for crimes such as property destruction, harassing/assaulting others, stealing, etc. (APA, 2013). Deceitfulness is another hallmark symptom of antisocial personality disorder as individuals often lie repeatedly, generally as a means to gain profit or pleasure. There is also a pattern of impulsivity, in that decisions are made spontaneously without forethought of personal consequences or consideration for others (Lang et al., 2015). This impulsivity also contributes to their inability to maintain employment as they are more likely to impulsively quit their jobs (Hengartner et al., 2014). Employment instability, along with impulsivity, also impacts their ability to manage finances; it is not uncommon to see individuals with antisocial personality disorder accumulate large debts that they are unable to pay (Derefinko & Widiger, 2016).

While also likely related to impulsivity, individuals with antisocial personality disorders tend to be extremely irritable and aggressive, repeatedly getting into fights. Their disregard for their own safety, as well as the safety of others, is also observed in reckless behavior such as speeding, driving under the influence, and engaging in sexual and substance abuse behavior that may put themselves and others at risk (APA, 2013).

Of course, one of the better-known symptoms of antisocial personality disorder is the lack of remorse for the consequences of their actions, regardless of how severe they may be (APA, 2013). Individuals with this disorder often rationalize their actions at the fault of the victim, minimize the harmfulness of the consequences of their behaviors, or display indifference (APA, 2013). Overall, individuals with antisocial personality disorder have limited personal relationships due to their selfish desires and lack of moral conscious.

9.3.1.2: Epidemiology

Antisocial personality disorder has an estimated prevalence rate of up to 3.3% of the population with men comprising 75% of the cases (APA, 2013). It is more commonly diagnosed in men, particularly those with substance abuse disorders. It is also observed more commonly in those from disadvantaged socioeconomic settings. While the majority of individuals with antisocial personality disorder end up incarcerated at some point throughout their lifetime, criminal activities appear to decline after the age of 40 (APA, 2013).

9.3.1.3: Treatment

Treatment options for antisocial personality disorder are limited, and generally not effective (Black, 2015). Like cluster A disorders, many individuals are forced to participate in treatment, thus impacting their ability to engage in and continue with treatment. Cognitive therapists have attempted to address the lack of moral conscious and encourage clients to think about the needs of others (Beck & Weishaar, 2011). Medications including lithium, atypical antipsychotics and SSRIs are sometimes prescribed to help reduce impulsive and aggressive behaviors but there is very little research on this topic and medication compliance can be a major issue.

9.3.2: Borderline Personality Disorder

9.3.2.1: Clinical Description

Individuals with borderline personality disorder display a persistent pattern of instability in interpersonal relationships, self-image, and affect (APA, 2013). The key characteristic of borderline personality disorder is unstable and/or intense relationships. For example, individuals may idealize or experience intense feelings for another person immediately after meeting them and then switch to devaluing them. It is not uncommon for people with borderline personality disorder to experience intense fluctuations in mood (i.e., mood lability), often observed as volatile interactions with family and friends (Herpertz & Bertsch, 2014). Those with borderline personality disorder may be friendly one day and hostile the next. The combination of these symptoms causes significant impairment in establishing and maintaining personal relationships.

Individuals with this disorder will often go to great lengths to avoid real or imagined abandonment. Fears related to abandonment can lead to inappropriate anger as they often interpret the abandonment as a reflection of their own behaviors. In efforts to prevent abandonment, individuals with borderline personality disorder will often engage in impulsive behaviors such as self-harm and suicidal behaviors. In fact, individuals with borderline personality disorder engage in more suicidal attempts and completion of suicide is higher among these individuals than the general public (Linehan et al., 2015). Other impulsive behaviors such as non-suicidal self-injury (cutting) and sexual promiscuity are often seen within this population, typically occurring during high-stress periods (Sansone & Sansone, 2012). Occasionally, hallucinations and delusions are present, particularly of a paranoid nature; however, these symptoms are often transient and recognized as unacceptable by the individual (Sieswerda & Arntz, 2007).

9.3.2.2: Epidemiology

Borderline personality disorder, one of the more commonly diagnosed personality disorders, is observed in 1.6% –5.9% of the general population, with women making up 75% of the diagnoses (APA, 2013). Approximately 10% of individuals with borderline personality disorder have been seen in an outpatient mental health clinic, and nearly 20% have sought treatment in a psychiatric inpatient unit (APA, 2013). This high percentage of inpatient treatment is likely related to the high incidence of suicidal and self-harm behaviors.

9.3.2.3: Treatment

Borderline personality disorder is the one personality disorder with the most effective treatment option – *Dialectical Behavioral Therapy (DBT)*. DBT is a form of cognitive behavioral therapy developed by Marsha Linehan (Linehan, Armstrong, Suarez, Allmon, & Heard, 1991). There are four main goals of DBT: reduce suicidal behavior, reduce therapy interfering behavior, improve quality of life, and reduce post-traumatic stress symptoms.

Within DBT, there are five main treatment components that together help reduce harmful behaviors (i.e. self-mutilation and suicidal behaviors) and replace them with effective, life-enhancing behaviors (Gonidakis, 2014). The first component is *skills training*. Generally performed in a group therapy setting, individuals engage in **mindfulness, distress tolerance**, **interpersonal effectiveness**, and **emotion regulation.** Second, individuals focus on *enhancing motivation* and applying skills learned in the previous component to specific challenges and events in their everyday life. The third, and often the most distinctive component of DBT, is the use of *telephone and in vivo coaching*. It is not uncommon for clients to have the cell phone number of their clinician for 24/7 availability of in-the-moment support. The fourth component, *case management*, consists of allowing the client to become their own "case manager" and effectively use the learned DBT techniques to problem solve ongoing issues. Within this component, the clinician will only intervene when absolutely necessary. Finally, the *consultation team*, which is a service for the clinicians providing the DBT treatment. Due to the high demands of clients with borderline personality disorder, the consultation team provides support to the providers in their work to ensure they remain motivated and competent in DBT principles in an effort to provide the best treatment possible.

Support for the effectiveness of DBT in the treatment of borderline personality disorder has been implicated in a number of randomized control trials (Harned, Korslund, & Linehan, 2014; Neacsiu, Eberle, Kramer, Wisemeann, & Linehan, 2014). More specifically, DBT has shown to significantly reduce suicidality and self-harm behaviors in those with borderline personality disorders. It also reduces anger and hospitalizations as well as improves emotional regulation and interpersonal functioning. Additionally, the drop-out rates for treatment are extremely low, suggesting that clients value the treatment components and find them effective in managing symptoms.

9.3.3: Histrionic Personality Disorder

9.3.3.1: Clinical Description

Histrionic personality disorder is characterized by a persistent and excessive need for attention from others. Individuals with this disorder are uncomfortable in social settings *unless* they are the center of attention. In efforts to gain attention, they are often very lively and dramatic, using emotional displays, physical gestures, and mannerisms along with grandiose language. These behaviors are initially very charming to their audience; however, they begin to wear due to the constant need for attention to be on them.

If their theatrical nature does not gain the attention they desire, individuals with histrionic personality disorder may go to great lengths to gain that attention such as make-up a story or create a dramatic scene (APA, 2013). Similarly, they often dress and engage in sexually seductive or provocative ways. These sexually charged behaviors are not only directed at those with whom they have a sexual or romantic interest but to the general public as well (APA, 2013). They often spend significant amounts of time on their physical appearance to gain the attention they desire.

Individuals with histrionic personality disorder are easily suggestible. Their opinions and feelings are influenced by not only their friends but also by current fads (APA, 2013). They also have a tendency to over exaggerate relationships, considering casual acquaintanceships as more intimate in nature than they really are.

9.3.3.2: Epidemiology

Histrionic personality disorder is one of the most uncommon personality disorders, occurring in only 1.84% of the general population (APA, 2013). While it was once believed to be more commonly diagnosed in females than males, more recent findings suggest the diagnosis rate is equal between genders.

9.3.3.3: Treatment

Individuals with histrionic personality disorder are actually *more* likely to seek out treatment than other those with other personality disorders. Unfortunately, due to the nature of the disorder, they are very difficult to treat as they are quick to employ their demands and seductiveness within the treatment setting. The overall goal for treatment of histrionic personality disorder is to help the individual identify their dependency and become more self-reliant. Cognitive therapists utilize techniques to help clients change their helpless beliefs and improve problem-solving skills (Beck & Weishaar, 2011).

9.3.4: Narcissistic Personality Disorder

9.3.4.1: Clinical Description

The key features of narcissistic personality disorder are a need for admiration, a pattern of grandiosity, and a lack of empathy for others (APA, 2013). The grandiose sense of self often leads to an overvaluation of their abilities and accomplishments. They often come across as boastful and pretentious, repeatedly proclaiming their superior achievements. These proclamations may also be fantasized as a means to enhance their success or power. Oftentimes they identify themselves as “special” and will only interact with others of high status.

Given the grandiose sense of self, it is not surprising that individuals with narcissistic personality disorder need excessive admiration from others. While it appears that their self-esteem is extremely inflated, it is actually very fragile and dependent on how others perceive them (APA, 2013). Because of this, they may constantly seek out compliments and expect favorable treatment from others. When this sense of entitlement is not upheld, they can become irritated or angry that their needs are not being met.

A lack of empathy is also displayed in individuals with narcissistic personality disorder as they often fail to recognize the desires or needs of others. This lack of empathy also leads to exploitation of interpersonal relationships, as they are unable to empathize other’s feelings (Marcoux et al., 2014). They often become envious of others who achieve greater success or have nicer possessions than them. Conversely, they believe everyone should be envious of their achievements, regardless of how small they may actually be.

9.3.4.2: Epidemiology

Finally, narcissistic personality disorder is reportedly diagnosed in 0 – 6.2% of the general public, with 75% of these individuals being men (APA, 2013).

9.3.4.3: Treatment

Of all the personality disorders, narcissistic personality disorders are among the most difficult to treat (with maybe the exception of antisocial personality disorder). In fact, most individuals with narcissistic personality disorder only seek out treatment for those

disorders secondary to their personality disorder, such as depression (APA, 2013). The focus of treatment is to address the grandiose, self-centered thinking, while also trying to teach clients how to empathize with others (Beck & Weishaar, 2014).

9.4: Cluster C Personality Disorders

Section Learning Objectives

- Describe the symptoms associated with each of the cluster C personality disorders.
- Describe the epidemiology of cluster C personality disorders.
- Describe the treatment for cluster C personality disorders.

9.4.1 Avoidant Personality Disorder

9.4.1.1 Clinical Description

Individuals with avoidant personality disorder display social anxiety due to feelings of inadequacy and increased sensitivity to negative evaluations (APA, 2013). The fear of being rejected drives their reluctance to engage in social situations, in efforts to prevent others from evaluating them negatively. This fear extends so far that it prevents individuals from maintaining employment due to their intense fear of a negative evaluation or rejection.

Individuals with this disorder have very few if any friends, despite their desire to establish social relationships. They actively avoid social situations in which they can establish new friendships out of the fear of being disliked or ridiculed. Similarly, they are cautious of new activities or relationships as they often exaggerate the potential negative consequences and embarrassment that may occur; this is likely a result of their ongoing preoccupation of being criticized or rejected by others.

You may recall that schizoid personality disorder is also associated with social isolation but avoidant personality disorder differs from schizoid personality disorder because while those with schizoid personality disorder do not desire social connections, those with avoidant personality very much want relationships with others, they avoid them only because of their feelings of inadequacy, fears of criticism, and negative evaluation.

9.4.1.2 Epidemiology

Avoidant personality disorder occurs in 2.4% of the general population and is diagnosed equally among men and women (APA, 2013).

9.4.1.3 Treatment

While many individuals with avoidant personality disorder seek out treatment to address their anxiety or depressive-like symptoms, it is often difficult to keep them in treatment due to fear of rejection from the clinician. Treatment goals for avoidant personality disorder are similar to that of social anxiety disorder. CBT techniques such as identifying and challenging distressing thoughts have been effective in reducing anxiety-related symptoms (Weishaar & Beck, 2006). Behavioral treatments such as gradual exposure to various social settings, along with a combination of social skills training, has been shown to improve individuals' confidence prior to engaging in social outings (Herbert, 2007). Anti-anxiety and antidepressant medications commonly used to treat anxiety disorders have also been used with minimal efficacy; furthermore, symptoms resume as soon as the medication is discontinued.

9.4.2 Dependent Personality Disorder

9.4.2.1 Clinical Description

Dependent personality disorder is characterized by a persistent and excessive need to be taken care of by others (APA, 2013). This intense need leads to submissive and clinging behaviors as they fear they will be abandoned or separated from their parent, spouse, or another person whom they feel dependent on. They are so dependent on this other individual that they cannot make even the smallest decisions without first consulting with them and gaining their approval or reassurance. They often allow others to assume complete responsibility of their life, making decisions in nearly all aspects of their lives. Rarely will they challenge these decisions as their fear of losing this relationship greatly outweighs their desire to express their own opinion. Should the relationship end, they experience significant feelings of helplessness and quickly and indiscriminately seek out another relationship to replace the old one (APA, 2013).

Individuals with dependent personality disorder express difficulty initiating and engaging in tasks on their own. They lack self-confidence and feel helpless when they are left to care for themselves or engage in tasks on their own. In efforts to not have to

engage in tasks alone, individuals will go to great lengths to seek out support of others, often volunteering for unpleasant tasks if it means they will get the reassurance they need (APA, 2013).

9.4.2.2 Epidemiology

Dependent personality disorder occurs in less than 1% of the population (APA, 2013). Women are more frequently diagnosed with dependent personality disorder than men (APA, 2013) but this may reflect biases in clinicians making the diagnoses more than a true difference in the prevalence of the disorders in men and women.

9.4.2.3 Treatment

Unlike other personality disorders where individuals avoid treatment and are skeptical of the clinician, individuals with dependent personality disorder are likely to seek treatment and to place a large emphasis of their treatment on the clinician. Therefore, one of the main treatment goals for individuals with dependent personality disorder is to teach them to accept responsibility for themselves, both in and outside of treatment (Colli, Tanzilli, Dimaggio, & Lingiardi, 2014). Cognitive strategies such as challenging and changing thoughts on helplessness and their inability to care for themselves have been minimally effective in establishing independence. Additionally, behavioral techniques such as assertiveness training have also shown some promise in teaching individuals how to express themselves within a relationship. Some argue that family or couples therapy would be particularly helpful for those with dependent personality disorder due to the dysfunctional relationship between the individual and the person whom they are dependent on; however, research on this treatment method has not yielded consistently positive results (Nichols, 2013).

9.4.3 Obsessive-Compulsive Personality Disorder

9.4.3.1 Clinical Description

Obsessive-Compulsive Personality Disorder (OCPD) is defined by a preoccupation with orderliness, perfectionism, and control at the expense of flexibility, openness, and efficiency in everyday life (APA, 2013). Their preoccupation with details, rules, lists, orders, organizations or schedules overshadows the larger picture of the task or activity. In fact, their self-imposed high standards and need to complete tasks perfectly often prevent these tasks from ever being completed. Their desire to complete tasks perfectly often causes them to spend excessive amounts of time on the tasks, occasionally repeating them in an attempt to reach some perfectionistic standard. Due to repetition and attention to fine detail, individuals with OCPD often feel like they do not have time to engage in leisure activities or engage in social relationships. Despite the excessive amount of time spent on activities or tasks, individuals with OCPD will not seek help from others, as they are convinced that the others are incompetent and will not complete the tasks to their standard.

Personally, individuals with OCPD are rigid and stubborn, particularly with their morals, ethics, and values. Not only do they hold these standards for themselves, but they also expect others to have high standards, thus causing significant disruption in their social interactions. Their rigid and stubborn behaviors are also seen in their financial status, as they are known to live significantly below their means, in order to prepare financially for potential catastrophes (APA, 2013). Similarly, they may have difficulty discarding worn-out or worthless items, despite their lack of sentimental value.

Unfortunately, the term OCPD leads many to believe this is a similar disorder to OCD, but there is a distinct difference in that OCPD lacks the obsessions and compulsions that characterize and define OCD (APA, 2013). Although many individuals are diagnosed with both OCD and OCPD, research indicates that individuals with OCPD are more likely to be diagnosed with major depression, generalized anxiety disorder, or substance abuse disorder than OCD (APA, 2013).

9.4.3.2 Epidemiology

OCPD is the most commonly diagnosed personality disorder, occurring in 7.9% of individuals. Men are twice as likely to be diagnosed with OCPD than women (APA, 2013).

9.4.3.3 Treatment

Individuals with OCPD often seek out treatment to address their anxiety or depressive-like symptoms. Cognitive techniques aimed at changing dichotomous thinking (see etiology), perfectionism, and chronic worry are helpful in managing symptoms of OCPD. CBT may also be used to try to challenge and reduce perfectionistic beliefs and standards as well as rigid behaviors. They are often

taught relaxation techniques to overcome the anxiety that manifests from attempts to break their rigid schedules and other behaviors.

9.5: Comorbidity and Etiology

Section Learning Objectives

- Describe the comorbidity of personality disorders.
- Describe the various factors that contribute to personality disorders

9.5.1: Comorbidity

Among the most common comorbid diagnoses with personality disorders are other personality disorders, mood disorders, anxiety disorders, and substance abuse disorders (Lenzenweger, Lane, Loranger, & Kessler, 2007). Indeed, many individuals are diagnosed with more than one personality disorders.

A large meta-analysis exploring the data on the comorbidity of personality disorders and mood disorders indicated a high level of comorbid diagnoses of MDD, PDD, and bipolar disorder (Friborg, Martinsen, Martinussen, Kaiser, Overgard, & Rosenvinge, 2014). Further exploration of MDD suggested the lowest rate of diagnosis in cluster A disorders, higher rate in cluster B disorders, and the highest rate in cluster C disorders. While the relationship between bipolar disorders and personality disorders has not been consistently clear, the most recent findings report a high comorbidity with OCPD as well as with the cluster B personality disorders.

Clear relationships between personality disorders and anxiety disorders have also been established (Skodol, Geier, Grant, & Hasin, 2014). More specifically, individuals diagnosed with borderline and schizotypal personality disorders were found to have elevated rates of additional diagnoses of each of the four main anxiety disorders. Individuals with narcissistic personality disorders were more likely to be diagnosed with GAD and panic disorder. Schizoid and avoidant personality disorders reported significant rates of GAD; avoidant personality disorder had a higher diagnosis rate of social phobia.

Finally, substance abuse disorders are frequently found in individuals diagnosed with antisocial, borderline, and schizotypal personality disorders (Grant et al., 2015).

9.5.2: Etiology

Research regarding the development of personality disorders is limited compared to that of other mental disorders. The following is a general overview of contributing factors to personality disorders as a whole. While there is some research lending itself to specific causes of specific personality disorders, we will review the overall contribution of biological, psychological, and social factors globally for all of the personality disorders.

9.5.2.1: **Biological**

Research across the personality disorders suggests some underlying biological or genetic component. However, identification of specific mechanisms has not been identified for most personality disorders, with the exception of the cluster A personality disorders which show a genetic link with schizophrenia. Because of the lack of specific evidence of biological causes, researchers argue that it is difficult to determine what role genetics plays in the development of these disorders compared to that of environmental influences. Therefore, while there is likely a biological predisposition to personality disorders, exact causes cannot be determined at this time.

Research on the development of schizotypal personality disorder has identified similar biological causes to that of schizophrenia, specifically, high activity of dopamine and enlarged brain ventricles (Lener et al., 2015). Similar differences in neuroanatomy may explain the high similarity of behaviors in both schizophrenia and schizotypal personality disorder.

Antisocial personality disorder and borderline personality disorder are also related to neurological dysfunctions. More specifically, individuals with both disorders reportedly show deficits in serotonin activity (Thompson, Ramos, & Willett, 2014). These low levels of serotonin activity in combination with deficient functioning of the frontal lobes, particularly the prefrontal cortex which is used in planning, self-control, and decision making, as well as an overly reactive amygdala, may explain the impulsive and aggressive nature of individuals with antisocial and borderline personality disorder (Stone, 2014).

9.5.2.2: **Psychological**

Psychodynamic, cognitive, and behavioral theories are among the most common models used to explain the development of personality disorders. Although much is still speculation, the following are general etiological views with regards to each specific

theory.

9.5.2.2.1: Psychodynamic

The psychodynamic theory places a large emphasis on negative early childhood experiences and their impact on an individual's ability to establish healthy relationships in adulthood. More specifically, individuals with personality disorders report higher levels of childhood stress such as living in impoverished environments, exposure to family/domestic violence, and experiencing repeated abuse and maltreatment (Kumari et al., 2014). Additionally, high levels of neglect and parental rejection are observed in people with personality disorders, with early parental loss and rejection leading to fears of abandonment throughout life (Caligor & Clarkin, 2010; Newnham & Janca, 2014; Roepke & Varter, 2014).

Psychodynamic theorists believe that because of these negative early experiences, their sense of self, and consequently, their beliefs of others are negatively impacted, thus leading to the development of a personality disorder. For example, an individual who was neglected as a young child and deprived of love may report a lack of trust in others as an adult, a characteristic of paranoid and antisocial personality disorders (Meloy & Yakeley, 2010). Difficulty trusting others or beliefs that they are unable to be loved may also impact their ability or desire to establish social relationships as seen in many personality disorders, particularly schizoid, avoidant and dependent personality disorders. Because of these early childhood deficits, individuals may also overcompensate in their relationships in efforts to convince themselves that they are worthy of love and affection as may be the case in histrionic and narcissistic personality disorders (Celani, 2014). Conversely, individuals may respond to their early childhood experiences by becoming emotionally distant, using relationships as a sense of power and destructiveness.

9.5.2.2.2: Cognitive

While psychodynamic theory places an emphasis on early childhood experiences, cognitive theorists focus on the maladaptive thought patterns and cognitive distortions displayed by those with personality disorders. Overall deficiencies in thinking place individuals with personality disorders in a position to develop inaccurate perceptions of others (Beck, 2015). These dysfunctional beliefs likely originate from the interaction between a biological predisposition and undesirable environmental experiences. Maladaptive thought patterns and strategies are strengthened during aversive life events as a protective mechanism and ultimately come together to form patterns of behaviors displayed in personality disorders (Beck, 2015).

Cognitive distortions such as **dichotomous thinking**, also known as all or nothing thinking, are observed in several personality disorders. More specifically, dichotomous thinking helps to explain rigidity and perfectionism in OCPD, and the lack of independence observed in those with dependent and borderline personality disorders (Weishaar & Beck, 2006). **Discounting the positive** helps explain the underlying mechanisms for avoidant personality disorder (Weishaar & Beck, 2006). For example, individuals who have been routinely criticized or rejected during childhood may have difficulty accepting positive feedback from others, expecting to only receive rejection and harsh criticism. In fact, they may employ these misattributions to support their ongoing theory that they are constantly rejected and criticized by others.

9.5.2.2.3: Behavioral

There are three major behavioral theories of the etiology of personality disorders: modeling, reinforcement, and lack of social skills. With regards to modeling, personality disorders are explained by an individual learning maladaptive social relationship patterns and behaviors due to directly observing family members engaging in similar behaviors (Gaynor & Baird, 2007). While we cannot discredit the biological component of the familial influence, research does support an additive modeling or imitating component to the development of personality disorders (especially antisocial personality disorder; APA, 2013).

Second, reinforcement or rewarding of maladaptive behaviors can also help explain personality disorders. Parents may unintentionally reward aggressive behaviors by giving into a child's desires in efforts to cease the situation or prevent escalation of behaviors. When this is done repeatedly over time, children (and later as adults, particularly those with antisocial and borderline personality disorder) continue to display these maladaptive behaviors as they are effective in gaining their needs/wants. On the other side, there is some speculation that excessive reinforcement or praise during childhood may contribute to the grandiose sense of self-observed in individuals with narcissistic personality disorder (Millon, 2011).

Finally, a failure to develop normal social skills may explain the development of some personality disorders, such as avoidant personality disorder (Kantor, 2010). While there is some discussion as to whether lack of social skills leads to avoidance of social settings OR if social skills deficits develop as a result of avoiding social situations, most researchers agree that the avoidance of social situations contributes to the development of personality disorders, whereas, underlying deficits in social skills may contribute more to social anxiety disorder (APA, 2013).

9.5.2.3: Social

9.5.2.3.1: Family Dysfunction

High levels of psychological or social dysfunction within families have also been identified as a contributing factor to the development of personality disorders. High levels of poverty, unemployment, family separation, and witnessing domestic violence are routinely observed in individuals diagnosed with personality disorders (Paris, 1996). While formalized research has yet to further explore the relationship between socioeconomic status and personality disorders, correlational studies suggest a relationship between poverty, unemployment and poor academic achievement with increased levels of personality disorder diagnoses (Alwin, 2006).

9.5.2.3.2: Childhood Maltreatment

Childhood maltreatment is among the most influential arguments for the development of personality disorders in adulthood. Individuals with personality disorders often struggle with a sense of self and with the ability to relate to others — something that is generally developed during the first four to six years of a child's life and is affected by the emotional environment in which the child was raised. This sense of self is the mechanism in which individuals view themselves within their social context, while also informing attitudes and expectations of others. A child who experiences significant maltreatment, whether it be through neglect or physical, emotional, or sexual abuse, is at-risk for under or lack of development of a sense of self. Due to the lack of affection, discipline, or autonomy during childhood, these individuals are unable to engage in appropriate relationships as adults as seen across the spectrum of personality disorders.

Another way childhood maltreatment contributes to personality disorders is through the emotional bonds or **attachments** developed with primary caregivers. The relationship between attachment and emotional development was thoroughly researched by John Bowlby as he explored the need for affection in Harlow monkeys (Bowlby, 1998). Based on Bowlby's research, four attachment styles have been identified: **secure, anxious, ambivalent**, and **disorganized**. While securely attached children generally do not develop personality disorders, those with anxious, ambivalent, and disorganized attachment are at an increased risk to develop various disorders. More specifically, those with an anxious attachment are at-risk for developing internalizing disorders, those with an ambivalent attachment are at-risk for developing externalizing disorders, and those with disorganized attachment are at-risk for dissociative symptoms and personality disorders (Alwin, 2006).

Chapter Recap

Chapter 9 covered the personality disorders which are arranged in three clusters: cluster A which includes paranoid, schizoid, and schizotypal; cluster B which includes antisocial, borderline, histrionic, and narcissistic; and cluster C which includes avoidant, dependent, and obsessive-compulsive. We covered the clinical description, diagnostic criteria, epidemiology, treatment, comorbidity, and etiology of personality disorders.

Index

D

Glossary

Sample Word 1 | Sample Definition 1

Glossary 1

Abnormal behavior – behavior that involves a combination of personal distress, psychological dysfunction, deviance from social norms, dangerousness to self and others, and costliness to society

Abnormal psychology – The scientific study of abnormal behavior, with the intent to be able to reliably predict, explain, diagnose, identify the causes of, and treat maladaptive behavior

Absolute refractory period – After the neuron fires it will not fire again no matter how much stimulation it receives

Acceptance techniques – A cognitive therapy used to reduce a client's worry and anxiety

Action potential – When the neuron depolarizes and fires

Acute stress disorder – Though very similar to PTSD, symptoms must be present from 3 days to 1 month following exposure to one or more traumatic events

Adjustment disorder – Occurs following an identifiable stressor within the past 3 months; stressor can be a single event (loss of job) or a series of multiple stressors (marital discord that ends in a divorce); there is not a set of specific symptoms an individual must meet for diagnosis, rather, the symptoms must be significant enough that they impair social, occupational, or other important areas of functioning

Adrenal glands – Located on top of the kidneys, and which release *cortisol* to help the body deal with stress

Affective flattening – Reduction in emotional expression; reduced display of emotional expression

Agoraphobia – When a person experiences fear specific to leaving their home and traveling to public places

All-or-nothing principle – The neuron either hits -55mV and fires or it does not

Alogia – Poverty of speech or speech content

Amygdala – The part of the brain responsible for evaluating sensory information and quickly determining its emotional importance

Anal Stage – Lasting from 2-3 years, the libido is focused on the anus as toilet training occurs

Anhedonia – Inability to experience pleasure

Anorexia Nervosa – An eating disorder characterized by the restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health; intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, despite significantly low weight; and disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight

Antecedents – The environmental events or stimuli that trigger a behavior

Antisocial personality disorder – Characterized by the persistent pattern of disregard for, and violation of, the rights of others

Apathy – General lack of interest

Asociality – Lack of interest in social relationships

Asylums – Places of refuge for the mentally ill where they could receive care

Attribution theory – The idea that people are motivated to explain their own and other people's behavior by attributing causes of that behavior to personal reasons or *dispositional factors* that are in the person themselves or linked to some trait they have; or *situational factors* that are linked to something outside the person

Automatic thoughts – The constant stream of negative thoughts, also leads to symptoms of depression as individuals begin to feel as though they are inadequate or helpless in a given situation

Autonomic nervous system – Regulates functioning of blood vessels, glands, and internal organs such as the bladder, stomach, and heart; It consists of sympathetic and parasympathetic nervous systems

Avoidant personality disorder – Display a pervasive pattern of social anxiety due to feelings of inadequacy and increased sensitivity to negative evaluations

Avolition – Lack of motivation of goal-directed behavior

Axon – Sends signals/information to neighboring neurons

Axon terminals – The end of the axon where the electrical impulse becomes a chemical message and is passed to an adjacent neuron

B

Behavior modification – The process of changing behavior

Behavioral assessment – The measurement of a target behavior

Behaviors – What the person does, says, thinks/feels

Binge-Eating Disorder (BED) – An eating disorder characterized by recurrent episodes of binge eating associated with: significant distress regarding binge eating behaviors; binge eating occurring, on average, at least once a week for 3 months; and binge eating behaviors are not associated with compensatory behaviors such as that in bulimia nervosa

Biological Model – Includes genetics, chemical imbalances in the brain, the functioning of the nervous system, etc.

Bipolar Disorder I – A mood disorder characterized by a least one manic episode and the symptoms are not explained by a personality disorder

Bipolar Disorder II – A mood disorder characterized by having at least one hypomanic episode and at least one major depressive episode, never having had a manic episode, and the symptoms are not better explained by a personality disorder; Symptoms cause clinically significant distress or impairment in daily functioning

Body Dysmorphic Disorder (BDD) – is an obsessive disorder, the focus of the obsessions being on perceived defects or flaws in the person's physical appearance

Borderline personality disorder – Display a pervasive pattern of instability in interpersonal relationships, self-image, affect, and instability

Bulimia Nervosa – An eating disorder characterized by recurrent episodes of binge eating, recurrent compensatory behaviors to prevent weight gain, and the over-evaluation of shape and weight; the binge eating and compensatory behaviors both occur, on average, at least once a week for 3 months and these behaviors do not occur exclusively during an episode of anorexia nervosa

C

Catatonic behavior – The decrease or even lack of reactivity to the environment

Central nervous system (CNS) – The control center for the nervous system which receives, processes, interprets, and stores incoming sensory information

Cerebellum – The part of the brain involved in our sense of balance and for coordinating the body's muscles so that movement is smooth and precise; Involved in the learning of certain kinds of simple responses and acquired reflexes

Chronic traumatic encephalopathy (CTE) – A progressive, degenerative condition due to repeated head trauma

Civil commitment – When individuals with a mental illness behave in erratic or potentially dangerous ways, it is responsibility of the government to place the individual in involuntary commitment in a hospital or mental health facility to protect the individual

Classification – The way in which we organize or categorize things

Classification systems -Provide mental health professionals with an agreed upon list of disorders falling in distinct categories for which there are clear descriptions and criteria for making a diagnosis

Client-centered therapy – Stated that the humanistic therapist should be warm, understanding, supportive, respectful, and accepting of his/her clients

Clinical assessment – The collecting of information and drawing conclusions through the use of observation, psychological tests, neurological tests, and interviews to determine what the client's problem is and what symptoms he/she is presenting with

Clinical description – Includes information about the thoughts, feelings, and behaviors that constitute that mental disorder

Clinical diagnosis – The process of using assessment data to determine if the pattern of symptoms the person presents with is consistent with the diagnostic criteria for a specific mental disorder set forth in an established classification system such as the

DSM-5 or ICD-10

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

Cognitive coping skills training – Teaches social skills, communication, and assertiveness through direct instruction, role playing, and modeling

Cognitive restructuring – Also called rational restructuring, in which maladaptive cognitions are replaced with more adaptive ones

Comorbidity – When two or more mental disorders are occurring at the same time and in the same person

Compulsions – Repetitive behaviors or mental acts that an individual performs in response to an obsession

Concussion – Occurs when there is a significant blow to the head, followed by changes in brain functioning

Conditioning – A type of associative learning, occurs which two events are linked

Confounding variables – Variables not originally part of the research design but contribute to the results in a meaningful way

Consciousness – According to Freud, the level of personality that is the seat of our awareness

Consequences – The outcome of a behavior that either encourages it to be made again in the future or discourages its future occurrence

Contingencies – When one thing occurs due to another

Control group – The group in an experiment that does not receive the treatment or is not manipulated

Conversion Disorder – A somatic symptom and related disorders characterized by at least one voluntary motor or sensory dysfunction, lack of medical compatibility between symptom and neurological/medical condition, symptom(s) not better explained by another medical or mental disorder, and causes clinically significant distress or impairment in daily functioning

Cortisol – A hormone released as a stress response

Counterconditioning – The reversal of previous learning

Courtesy stigma – When stigma affects people associated with the person with a mental disorder

Course – The particular pattern a disorder displays

Criminal commitment – When people are accused of crimes but found to be mentally unstable, they are usually sent to a mental health institution for treatment

Critical thinking – Our ability to assess claims made by others and make objective judgments that are independent of emotion and anecdote and based on hard evidence, and required to be a scientist

Cross-sectional validity – When a behavior made in one environment happens in other environments as well

Culture – The totality of socially transmitted behaviors, customs, values, technology, attitudes, beliefs, art, and other products that are particular to a group, and determines what is normal

Culture-sensitive therapies – A sociocultural therapies that include increasing the therapist's awareness of cultural values, hardships, stressors, and/or prejudices faced by their client; the identification of suppressed anger and pain; and raising the client's self-worth

D

Dangerousness – When behavior represents a threat to the safety of the person or others

Degenerative – Meaning the symptoms and cognitive deficits become worse overtime

Deinstitutionalization – The release of patients from mental health facilities

Delirium – Characterized by a significant disturbance in attention or awareness and cognitive performance that is significantly altered from one's usual behavior

Dementia – A major decline in cognition and self-help skills due to a neurocognitive disorder

Dendrites – Receives information from neighboring neurons and look like little trees

Denial – Sometimes life is so hard all we can do is deny how bad it is

Dependent personality disorder – Characterized by pervasive and excessive need to be taken care of by others

Dependent variable (DV) – In an experiment, the variable that is measured

Depersonalization – Defined as a feeling of unreality or detachment from oneself

Depolarized – When ion gated channels open allowing positively charged Sodium ions to enter; This shifts the polarity to positive on the inside and negative outside

Depressant substances – Such as alcohol, sedative-hypnotic drugs, and opioids, are known to have a depressing, or inhibiting effect on one's central nervous system; therefore, they are often used to alleviate tension and stress

Derealization – Include feelings of unreality or detachment from the world—whether it be individuals, objects, or their surroundings

Descriptive statistics – Statistics which provide a means of summarizing or describing data, and presenting the data in a usable form

Deviance – A move away from what is normal, or the mean, and so is behavior that occurs infrequently

Displacement – When we satisfy an impulse with a different object because focusing on the primary object may get us in trouble

Dissociative disorders – A group of disorders categorized by symptoms of disruption in consciousness, memory, identify, emotion, perception, motor control, or behavior

Dissociative Amnesia Disorder – Dissociative disorder identified by the inability to recall important autobiographical information

Dissociative fugue – Considered to be the most extreme type of dissociative amnesia where not only does an individual forget personal information, but they also flee to a different location

Dissociative Identity Disorder – Dissociative disorder characterized by the presence of two or more distinct personality states which causes discontinuity of self; difficulty recalling everyday events, personal information, or traumatic events due to lapse of memory; and causes significant distress or impairment in daily functioning

Distress – When a person experiences a disabling condition that can affect social, occupational, or other domains of life and takes psychological and/or physical pain

Dopamine – Neurotransmitter which controls voluntary movements and is associated with the reward mechanism in the brain

Dream analysis – In psychoanalytic theory, is an attempt to understand a person's inner most wishes as expressed in their dreams

Dysfunction – Includes "clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning" (APA, 2013)

E

Ego – According to Freud, the part of personality that attempts to mediate the desires of the id against the demands of reality, and eventually the moral limitations or guidelines of the superego

Ego-defense mechanisms – According to Freud, they protect us from the pain created by balancing both the will of the id and the superego, but are considered maladaptive if they are misused and become our primary way of dealing with stress

Enactive learning – Learning by doing

Endorphins – Neurotransmitters involved in reducing pain and making the person calm and happy

Eros – Our life instincts which are manifested through the libido and are the creative forces that sustain life

Erotomanic delusion – Occurs when an individual reports a delusion of another person being in love with them

Enzymatic degradation – When enzymes are used to destroy excess neurotransmitters in the synaptic space

Epidemiological study – A special from of correlational research in which the prevalence and incidence of a disorder in a specific population are measured

Epidemiology – The scientific study of the frequency and causes of diseases and other health-related states in specific populations such as a school, neighborhood, a city, country, and the world

Etiology – The cause of the disorder

Existential perspective – This approach stresses the need for people to continually re-create themselves and be self-aware, acknowledges that anxiety is a normal part of life, focuses on free will and self-determination, emphasizes that each person has a unique identity known only through relationships and the search for meaning, and finally, that we develop to our maximum potential

Exorcism – A procedure in which evil spirts were cast out through prayer, magic, flogging, starvation, having the person ingest horrible tasting drinks, or noise-making

Experimental group – In an experiment, the group that receives the treatment or manipulation

Extinction – When something that we do, say, think/feel has not been reinforced for some time

F

Factitious disorder – Commonly referred to as *Munchausen syndrome*, is characterized by intentional falsification of medical or psychological symptoms of oneself or another, with the overall intention of deception

Fixed Interval schedule (FI) – With a FI schedule, you will reinforce after some set amount of time

Fixed Ratio schedule (FR) – With this schedule, we reinforce some set number of responses

Flooding – Exposing the person to the maximum level of stimulus and as nothing aversive occurs, the link between CS and UCS producing the CR of fear should break, leaving the person unafraid

Forensic psychology/psychiatry – When clinical psychology is applied to legal arena in terms of assessment, treatment, and evaluation

Free association – In psychoanalytic theory, this technique involves the patient describing whatever comes to mind during the session

Frontal lobe – Part of the cerebrum that contains the motor cortex which issues orders to the muscles of the body that produce voluntary movement

Frontotemporal Lobar Degeneration (FTLD) – Causes progressive declines in language or behavior due to the degeneration in the frontal and temporal lobes of the brain; symptoms include significant changes in behavior and/or language

Fundamental attribution error – Occurs when we automatically assume a dispositional reason for another person's actions and ignore situational factor

G

GABA – Neurotransmitter responsible for blocking the signals of excitatory neurotransmitters responsible for anxiety and panic

Gaps – Holes in the literature of a given area

Generalizability – Begin able to apply your findings for the sample to the population

Generalized amnesia – A type of dissociative amnesia in which the person has a complete loss of memory of their entire life history, including their own identity

Generalized anxiety disorder – The most common anxiety disorder characterized by a global and persistent feeling of anxiety

Genital Stage – Beginning at puberty, sexual impulses reawaken and unfulfilled desires from infancy and childhood can be satisfied during lovemaking

Glial cells – The support cells in the nervous system that serve five main functions: as a glue and hold the neuron in place, form the myelin sheath, provide nourishment for the cell, remove waste products, and protect the neuron from harmful substances

Glutamate – Neurotransmitter associated with learning and memory

Grandiose delusion – Involves the conviction of having a great talent or insight

H

Habituation – When we simply stop responding to repetitive and harmless stimuli in our environment

Hippocampus – Our "gateway" to memory; Allows us to form spatial memories so that we can accurately navigate through our environment and helps us to form new memories about facts and events

Histrionic personality disorder – Addresses the pervasive and excessive need for emotion and attention from others; these individuals are often uncomfortable in social settings unless they are the center of attention

Hoarding – Focused on the persistent over-accumulation of possessions

Hypertension – -Chronically elevated blood pressure

Hypomanic episode – Persistently elevated, expansive, or irritable mood; May present as persistent increased activity or energy; Symptoms last at least 4 consecutive days and present most of the day, nearly every day; Includes at least three of the following: inflated self-esteem or grandiosity, decreased need for sleep, more talkative or pressured speech, flight of ideas, distractibility, increase in goal-directed activity or psychomotor agitation, or excessive involvement in activities that have a high potential for painful consequences

Hypothalamic-pituitary-adrenal (HPA) axis – Involved in the fear producing response and may be involved in the development of trauma symptoms

Hypothalamus – The part of the brain involved in drives associated with the survival of both the individual and the species; It regulates temperature by triggering sweating or shivering, and controls the complex operations of the autonomic nervous system

Hypothesis – A specific, testable prediction

Humanism – The worldview that emphasizes human welfare and the uniqueness of the individual

I

Id – According to Freud, is the impulsive part of personality that expresses our sexual and aggressive instincts

Ideas of reference – The belief that unrelated events pertain to them in a particular and unusual way

Identification – This is when we find someone who has found a socially acceptable way to satisfy their unconscious wishes and desires and we model that behavior

Illness anxiety disorder – Previously known as hypochondriasis, involves the excessive preoccupation with having or acquiring a serious medical illness

Incidence – The number of new cases in a population over a specific period of time

Independent variable (IV) – In an experiment, the variable that is manipulated

Inferential statistics – Statistics which allow for the analysis of two or more sets of numerical data

Insomnia – The difficult falling or staying asleep

Intellectualization– When we avoid emotion by focusing on intellectual aspects of a situation

Intelligence tests – Used to determine the patient's level of cognitive functioning and consists of a series of tasks asking the patient to use both verbal and nonverbal skills

Ions – Charged particles found both inside and outside the neuron

Irritable bowel syndrome (IBS) – A chronic, functional disorder of the gastrointestinal tract including symptoms such as abdominal pain and extreme bowel habits (diarrhea and/or constipation)

J

Jealous delusion – Revolves around the conviction that one's spouse or partner is/has been unfaithful

K

L

Laboratory observation – A research method in which the scientist observes people or animals in a laboratory setting

Latency Stage – From 6-12 years of age, children lose interest in sexual behavior and boys play with boys and girls with girls

Latent content – The hidden or symbolic meaning of a dream

Law of effect (Thorndike, 1905) – The idea that if our behavior produces a favorable consequence, in the future when the same stimulus is present, we will be more likely to make the response again, expecting the same favorable consequence

Learning – Any relatively permanent change in behavior due to experience

Libido – The psychic energy that drives a person to pleasurable thoughts and behaviors

Lifetime prevalence – Indicates the proportion of a population that has had the characteristic at any time during their lives

Literature review – When we conduct a literature search through our university library or a search engine such as Google Scholar to see what questions have been investigated already and what answers have been found

Localized amnesia – The most common type of dissociative amnesia, is the inability to recall events during a specific period of time

M

Major Depressive Disorder – A mood disorder characterized by depressed mood most of the day or decreased interest or pleasure in all or most activities most of the day, along with insomnia or hypersomnia, fatigue, feelings of worthlessness, or difficulty concentrating to name a few symptoms; symptoms occur during a two week period

Major neurocognitive disorder – Individuals with the disorder show significant decline in both overall cognitive functioning as well as the ability to independently meet the demands of daily living such as paying bills, taking medications, or caring for oneself

Manic episode – Persistent elevated, expansive, or irritable mood. May present as persistent increased goal-directed activity or energy; Symptoms **last at least 1 week** and present most of the day, nearly every day; includes three of the following: inflated self-esteem or grandiosity, decreased need for sleep, more talkative or pressured speech, flight of ideas, distractibility, increase in goal-directed activity or psychomotor agitation, or excessive involvement in activities that have a high potential for painful consequences

Manifest content – The person's actual retelling of the dream

Mass madness – or Group hysteria; When large numbers of people display similar symptoms and false beliefs; a term used during the Middle Ages

Medulla – The part of the brain that regulates breathing, heart rate, and blood pressure

Melatonin – A hormone released when it is dark outside to assist with the transition to sleep

Mental disorders – Characterized by psychological dysfunction which causes physical and/or psychological distress or impaired functioning and is not an expected behavior according to societal or cultural standards

Mental health epidemiology – Refers to the occurrence of mental disorders in a population

Mental hygiene movement – An idea arising in the late 18th century to the early 19th century with the fall of the moral treatment movement, it focused on the physical well-being of patients

Mental status examination – Used to organize the information collected during the clinical interview and systematically evaluates the patient through a series of questions assessing appearance and behavior to include grooming and body posture, thought processes and content to include disorganized speech or thought and false beliefs, mood and affect such that whether the person feels hopeless or elated, intellectual functioning to include speech and memory, and awareness of surroundings to include where the person is and what the day and time are

Migraine headaches – Headaches explained by a throbbing pain localized to one side of the head and often accompanied by nausea, vomiting, sensitivity to light, and vertigo

Model – A representation or imitation of an object

Modeling – Techniques used to change behavior by having subjects observe a model in a situation that usually causes them some anxiety

Moral treatment movement – An idea arising in Europe in the late 18th century and then in the United States in the early 19th century, it stressed affording the mentally ill respect, moral guidance, and humane treatment, all while considering their individual, social, and occupational needs

Myelin sheath – The white, fatty covering which: 1) provides insulation so that signals from adjacent neurons do not affect one another and, 2) increases the speed at which signals are transmitted

Multicultural psychology – The area of psychology which attempts to understand how the various groups, whether defined by race, culture, or gender, differ from one another

Multi-dimensional model – An explanation for mental illness that integrates multiple causes of psychopathology and affirms that each cause comes to affect other causes over time

N

Narcissistic personality disorder – Individuals display a pattern of grandiosity along with a lack of empathy for others

Naturalistic observation – A research method in which the scientist studies human or animal behavior in its natural environment which could include the home, school, or a forest

Negative Punishment (NP) – This is when something good is taken away or subtracted making a behavior less likely in the future

Negative Reinforcement (NR) – This is when something bad or aversive is taken away or subtracted due to your actions, making it that you will be more likely to make the same behavior in the future when the same stimuli presents itself

Negative symptoms – The inability or decreased ability to initiate actions, speech, expressed emotion, or to feel pleasure

Nerves – A group of axons bundled together like wires in an electrical cable

Neurological tests – Used to diagnose cognitive impairments caused by brain damage due to tumors, infections, or head injury; or changes in brain activity

Neuron – The fundamental unit of the nervous system

Neurotransmitter – When the actual code passes from one neuron to another in a chemical form

Nomenclature – A naming system

Norepinephrine – Neurotransmitter which increases the heart rate and blood pressure and regulates mood

Nucleus – The control center of the body

O

Observation – Observing others either naturalistically or in a controlled environment

Observational learning – When we learn by observing the world around us

Obsessions – Repetitive and persistent thoughts, urges, or images

Obsessive compulsive disorder – More commonly known as OCD, the disorder requires the presence of both obsessions and compulsions

Obsessive-Compulsive personality disorder – Defined by an individual's preoccupation with orderliness, perfectionism, and ability to control situations that they lose flexibility, openness, and efficiency in everyday life

Operant conditioning – A type of associate learning which focuses on consequences that follow a response or behavior that we make (anything we do, say, or think/feel) and whether it makes a behavior more or less likely to occur

Oral Stage – Beginning at birth and lasting to 24 months, the libido is focused on the mouth and sexual tension is relieved by sucking and swallowing at first, and then later by chewing and biting as baby teeth come in

P

Panic disorder – When an individual experiences recurrent panic attacks consisting of physical and cognitive symptoms

Paranoid personality disorder – Characterized by a marked distrust or suspicion of others

Parasympathetic nervous system – The part of the autonomic nervous system that calms the body after sympathetic nervous system arousal

Parietal lobe – The part of the cerebrum that contains the somatosensory cortex and receives information about pressure, pain, touch, and temperature from sense receptors in the skin, muscles, joints, internal organs, and taste buds

Peripheral nervous system – Consists of everything outside the brain and spinal cord; It handles the CNS's input and output and divides into the somatic and autonomic nervous systems

Period prevalence – Indicates the proportion of a population that has the characteristic at any point during a given period of time, typically the past year

Persecutory delusion – Involves the individual believing that they are being conspired against, spied on, followed, poisoned or drugged, maliciously maligned, harassed, or obstructed in pursuit of their long-term goals

Persistent Depressive Disorder – A mood disorder characterized by poor appetite or overeating, insomnia or hypersomnia, low self-esteem, low energy, and feelings of hopelessness lasting most of the day, for more days than not, for at least 2 years

Personality disorders – Have four defining features which include distorted thinking patterns, problematic emotional responses, over- or under- regulated impulse control, and interpersonal difficulties

Personality inventories – Ask clients to state whether each item in a long list of statements applies to them, and could ask about feelings, behaviors, or beliefs

Phallic Stage – Occurring from about age 3 to 5-6 years, the libido is focused on the genitals and children develop an attachment to the parent of the opposite sex and are jealous of the same sex parent

Pineal gland – Helps regulate the sleep-wake cycle

Pituitary gland – The "master gland" which regulates other endocrine glands; It influences blood pressure, thirst, contractions of the uterus during childbirth, milk production, sexual behavior and interest, body growth, the amount of water in the body's cells, and other functions as well

Placebo – Or a sugar pill made to look exactly like the pill given to the experimental group

Point prevalence – Indicates the proportion of a population that has the characteristic at a specific point in time

Polarized – When the neuron has a negative charge inside and a positive charge outside

Pons – The part of the brain that acts as a bridge connecting the cerebellum and medulla and helps to transfer messages between different parts of the brain and spinal cord

Posttraumatic stress disorder – More commonly known as PTSD, is identified by the development of physiological, psychological, and emotional symptoms following exposure to a traumatic even

Positive psychology – The position in psychology that holds a more positive conception of human potential and nature

Positive Punishment (PP) – If something bad or aversive is given or added, then the behavior is less likely to occur in the future

Positive Reinforcement (PR) – If something good is given or added, then the behavior is more likely to occur in the future

Positive symptoms – Symptoms that are an over-exaggeration of normal brain processes

Preconscious – According to Freud, the level of personality that includes all of our sensations, thoughts, memories, and feelings

Presenting problem – The issue the person displays

Prevalence – The percentage of people in a population that has a mental disorder or can be viewed as the number of cases per some number of people

Prevention – When we identify the factors that cause specific mental health issues and implement interventions to stop them from happening, or at least minimize their deleterious effects

Prognosis – The anticipated course the mental disorder will take

Projection – When we attribute threatening desires or unacceptable motives to others

Projective tests – A psychological test which consists of simple ambiguous stimuli that can elicit an unlimited number of responses

Psychoanalysis – Psychoanalytic therapy used to understand the personality of a therapist's patient and to expose repressed material

Psychological debriefing – A type of crisis intervention that requires individuals who have recently experienced a traumatic event to discuss or process their thoughts and feelings related to the traumatic event, typically within 72 hours of the event

Psychological model – includes learning, personality, stress, cognition, self-efficacy, and early life experiences and how they affect mental illness

Psychological or psychogenic perspective – States that emotional or psychological factors are the cause of mental disorders and represented a challenge to the biological perspective

Psychological tests – Used to assess the client's personality, social skills, cognitive abilities, emotions, behavioral responses, or interests and can be administered either individually or to groups in paper or oral fashion

Psychopathology – The scientific study of psychological disorders

Psychosis – A loss of contact with reality

Public stigma – When members of a society endorse negative stereotypes of people with a mental disorder and discriminate against them

Punishment – Due to the consequence, a behavior/response is less likely to occur in the future

R

Random assignment – When participants have an equal chance of being placed in the control or experimental group

Rape – Forced sexual intercourse or other sexual act committed without an individual's consent

Rationalization – When we offer well thought out reasons for why we did what we did but in reality these are not the real reason

Reaction formation – When an impulse is repressed and then expressed by its opposite

Reactivity – When the observed changes behavior due to realizing they are being observed

Receptor sites – Locations where neurotransmitters bind to

Reinforcement – Due to the consequence, a behavior/response is more likely to occur in the future

Reinforcement schedule – The rule for determining when and how often we will reinforce a desired behavior

Relative refractory period – After a short period of time, the neuron can fire again, but needs greater than normal levels of stimulation to do so

Regression – When we move from a mature behavior to one that is infantile in nature

Reliable – When our assessment is consistent

Replication – Repeating a study to confirm its results

Repolarization – When the Na channels close and Potassium channels open; K has a positive charge and so the neuron becomes negative again on the inside and positive on the outside, or polarizes

Repression – When unacceptable ideas, wishes, desires, or memories are blocked from consciousness

Research design – Our plan of action of how we will go about testing the hypothesis

Resistance – According to psychoanalytic theory, is the point during free association that the patient cannot or will not proceed any further

Respondent conditioning (also called classical or Pavlovian conditioning) – Occurs when we link a previously neutral stimulus with a stimulus that is unlearned or inborn

Respondent Discrimination – When the CR is elicited by a single CS or a narrow range of CSs

Respondent Extinction – When the CS is no longer paired with the UCS

Respondent Generalization – When a number of similar CSs or a broad range of CSs elicit the same CR

Resting potential – When the neuron is waiting to fire

Reticular formation – The part of the brain responsible for alertness and attention

Reuptake reuptake – The process of the presynaptic neuron taking up excess neurotransmitters in the synaptic space for future use

Reversal or **ABAB design** – A study in which the control is followed by the treatment, and then a return to control and second administration of the treatment condition; builds replication in to the design

S

Schema – A set of beliefs and expectations about a group of people, presumed to apply to all members of the group, and based on experience

Self-stigma – When people with mental illnesses internalize the negative stereotypes and prejudice, and in turn, discriminate against themselves

Schizoaffective disorder – Characterized by the psychotic symptoms included in criteria A of schizophrenia *and* a concurrent uninterrupted period of a major mood episode—either a depressive or manic episode

Schizoid personality disorder – Displays a persistent pattern of avoidance from social relationships along with a limited range of emotion among social relationships

Schizophrenia – A mental disorder that includes the presentation of at least two of the following for at least one month: delusions, hallucinations, disorganized speech, disorganized/abnormal behavior, or negative symptom

Schizophreniform Disorder – A mental disorder characterized by at least two of the following: delusions, hallucinations, disorganized speech, disorganized/abnormal behavior, and/or negative symptoms

Schizotypal personality disorder – Characterized by a range of impairment in social and interpersonal relationships due to discomfort in relationships, along with odd cognitive and/or perceptual distortions and eccentric behaviors

Scientific method – A systematic method for gathering knowledge about the world around us

Sedative-Hypnotic drugs – More commonly known as anxiolytic drugs**,** these drugs have a calming and relaxing effect on individuals

Selective amnesia – Is in a sense, a component of localized amnesia in that the individual can recall some, but not all, of the details during a specific time period

Self-monitoring – When the person does their own measuring and recording of the ABCs

Self-serving bias – When we attribute our success to our own efforts (dispositional) and our failures to outside causes (situational)

Sensitization – When our reactions are increased due to a strong stimulus

Serotonin – Neurotransmitter which controls pain, sleep cycle, and digestion; leads to a stable mood and so low levels leads to depression

Single-subject experimental design – When we have to focus on one individual in a study

Social anxiety disorder – Occurs when an individual experiences anxiety related to social or performance situations, where there is the possibility that they will be evaluated negatively

Social cognition – The process of collecting and assessing information about others

Social desirability – When a participant answers questions dishonestly so that he/she is seen in a more favorable light

Social norms – The stated and unstated rules of society

Sociocultural Model – includes factors such as one's gender, religious orientation, race, ethnicity, and culture that affect mental illness

Soma – The cell body

Somatic delusion – Involves delusions regarding bodily functions or sensations

Somatic nervous system – Allows for voluntary movement by controlling the skeletal muscles and carries sensory information to the CNS

Somatic Symptom Disorder – A somatic symptom or related disorder characterized by disproportionate and persistent thoughts of the seriousness of the symptom, high levels of anxiety about the symptom, and/or excessive time/energy spent focused on the symptom

Specific phobia – Observed when an individual experiences anxiety related to a specific object or subject

Spontaneous recovery – When the CS elicits the CR after extinction has occurred

Standardization – When we use clearly laid out rules, norms, and/or procedures in the process of assessing client's

Statistical significance – An indication of how confident we are that our results are due to our manipulation or design and not chance

Stigma – When negative stereotyping, labeling, rejection, and loss of status occur

Stressors – Any event- either witnessed firsthand, experienced personally or experienced by a close family member- that increases physical or psychological demands on an individual

Sublimation – When we find a socially acceptable way to express a desire

Substance abuse – Occurs when an individual consumes the substance for an extended period of time, or has to ingest large amounts of the substance to get the same effect a substance provided previously

Substance Intoxication – A substance use disorder characterized by recent ingestion of substance, significant behavioral or psychological changes immediately following the ingestion of substance, physical and physiological symptoms develop after ingestion of substance, and changes in behavior not attributable to a medical condition or other psychological disorder

Substance Use Disorder – A substance use disorder diagnosed when the individual presents with at least two criteria to include: substance is consumed in larger amounts over time, desire or inability to reduce quantity of substance use, cravings for substance use, use of the substance in potentially hazardous situations, tolerance of substance use, and withdrawal, to name a few (11 total criteria)

Substance Withdrawal – A substance use disorder characterized by cessation or reduction in substance that has been previously used for a long or heavy period of time, physiological and/or psychological symptoms within a few hours after cessation/reduction, physiological and/or psychological symptoms cause significant distress or impairment in functioning, and symptoms not attributable to a medical condition or other psychological disorder

Substances – Any ingested materials that cause temporary cognitive, behavioral, and/or physiological symptoms within the individual

Superego – According to Freud, the part of personality which represents society's expectations, moral standards, rules, and represents our conscience

Sympathetic nervous system – Involved when a person is intensely aroused; It provides the strength to fight back or to flee (fight-or-flight instinct)

Synapse – The point where the code passes from one neuron to another; Consists of three parts – the *axon* of the sending neuron; the *space* in between called the synaptic space, gap, or cleft; and the *dendrite* of the receiving neuron

Syndrome – Symptoms occurred regularly in clusters

T

Target behavior – Whatever behavior we want to change and it can be in excess or needing to be reduced, or in a deficit state and needing to be increased

Tension headaches – Often described as a dull, constant ache that is localized to one part of the head/neck; however, it can co-occur in multiple places at one time

Thalamus – The major sensory relay center for all senses but smell

Thanatos – Our death instinct which is either directed inward as in the case of suicide and masochism or outward via hatred and aggression

Thematic Apperception Test – A projective test which asks the individual to write a complete story about each of 20 cards shown to them and give details about what led up to the scene depicted, what the characters are thinking, what they are doing, and what the outcome will be

Theory – A systematic explanation of a phenomenon

Threshold of excitation – -55mV or the amount of depolarization that must occur for a neuron to fire; It rises from -70mV to -55mV

Thyroid gland – The endocrine gland which regulates the body's rate of metabolism and so how energetic people are.

Tolerance – The need to continually increase the amount of ingested substance

Transference – In psychoanalytic theory, this technique involves patients transfering to the therapist attitudes he/she held during childhood

Trauma-focused cognitive-behavioral therapy (TF-CBT) – An adaptation of CBT, that utilizes both CBT techniques, as well as trauma sensitive principles to address the trauma related symptoms

Treatment – Any procedure intended to modify abnormal behavior into normal behavior

Trephination – In which a stone instrument known as a *trephine* was used to remove part of the skull, creating an opening

Trial and error learning – Making a response repeatedly if it leads to success

U

Ulcers – Or painful sores in the stomach lining, occur when mucus from digestive juices are reduced, thus allowing digestive acids to burn a hole into the stomach lining

Unconscious – According to Freud, the level of personality not available to us

Uni-dimensional model – A single factor explanation for mental illness

V

Validity – When the test measures what it says it measures

Variable Interval schedule (VI) – Reinforcing at some changing amount of time

Variable Ratio schedule (VR) – Reinforcing some varying number of responses

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    - 9.2: Cluster A Personality Disorders - *CC BY-NC-SA 4.0*
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    - 9.4: Cluster C Personality Disorders - *CC BY-NC-SA 4.0*
    - 9.5: Comorbidity and Etiology - *CC BY-NC-SA 4.0*
  - Back Matter - *Undeclared*