Abnormal Psychology

Discovering Psychology Series

Abnormal Psychology

1st edition

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Lee W. Daffin Jr., Ph.D.

Washington State University

Version 1.03

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Pullman, WA

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Title Page

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2

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3

Table of Contents

4

Record of Changes

Edition	As of Date	Changes Made
1.0	Fall 2017	Initial writing; feedback pending
1.01	Spring 2018	Addition of Modules 2, 3, and 15
1.02	Summer 2018	Addition of Index, Glossary, and Preface; made minor edits based on student feedback.
1.03	Summer 2019	Proofreading edits

I

Part I. Setting the Stage

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Module 1: What is Abnormal Psychology?

Module Overview

Cassie is an 18 year old female from suburban Seattle, WA. She was a successful student in high school, graduating valedictorian and 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 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 worsened. 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 for college and stays in bed most of the day. Concerned, they 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 this 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 the same ordeal. 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 Module 1, we will explore what it means to display abnormal behavior, what mental disorders are and how society views it both today and has throughout history, and then we will overview research methods used by psychologists in general and how they are adapted to study abnormal behavior/mental disorders. We will conclude with an overview of what mental health professionals do.

Module Outline

- 1.1. Understanding Abnormal Behavior
- 1.2. Classifying Mental Disorders
- 1.3. The Stigma of Mental Illness
- 1.4. The History of Mental Illness
- 1.5. Research Methods in Psychopathology
- 1.6. Mental Health Professionals, Societies, and Journals

Module Learning Outcomes

- Explain what it means to display abnormal behavior.
- 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.
- Describe research methods used to study abnormal behavior and mental illness.
- Identify types of mental health professionals, societies they may join, and journals they can publish their work in.

1.1. Understanding Abnormal Behavior

Section Learning Objectives

- Describe the disease model and its impact on the field of psychology throughout history.
- Describe positive psychology.
- Define abnormal behavior.
- 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.
- Clarify the cost of mental illness on society.
- Define abnormal psychology, psychopathology, and mental disorders.

1.1.1. Understanding Abnormal Behavior

To understand what abnormal behavior is, we have to first understand what normal behavior is. This really is in the eye of the beholder and most psychologists have found it easier to explain what is wrong with people then what is right. How so?

Psychology worked with the disease model for over 60 years, from about the late 1800s into the middle part of the 19th century. The focus was simple – curing mental disorders – and included such pioneers as Freud, Adler, Klein, Jung, and Erickson. These names are synonymous with the psychoanalytical school of thought. In the 1930s, behaviorism, under B.F. Skinner, presented a new view of human behavior. Simply, human behavior could be modified if the correct combination of reinforcements and punishments were used. This view point espoused the dominant worldview still present at the time – mechanism – and that the world could be seen as a great machine and explained through the principles of physics and chemistry. In it, human beings were smaller machines in the larger machine of the universe.

Moving into the mid to late 1900s, we developed a more scientific investigation of mental illness which allowed us to examine the roles of both nature and nurture and to develop drug and psychological treatments to “make miserable people less miserable.” Though this was good, there were three consequences as pointed out by Martin Seligman in his 2008 TED Talk entitled, “The new era of positive

psychology." These are:

- "The first was moral; that psychologists and psychiatrists became victimologists, pathologizers; that our view of human nature was that if you were in trouble, bricks fell on you. And we forgot that people made choices and decisions. We forgot responsibility. That was the first cost."
- "The second cost was that we forgot about you people. We forgot about improving normal lives. We forgot about a mission to make relatively untroubled people happier, more fulfilled, more productive. And "genius," "high-talent," became a dirty word. No one works on that."
- "And the third problem about the disease model is, in our rush to do something about people in trouble, in our rush to do something about repairing damage, it never occurred to us to develop interventions to make people happier — positive interventions."

One attempt to address the limitations of both psychoanalysis and behaviorism came from 3rd force psychology – humanistic psychology – under such figures as Abraham Maslow and Carl Rogers starting in the 1960s. As Maslow, said, "The science of psychology has been far more successful on the negative than on the positive side; it has revealed to us much about man's shortcomings, his illnesses, his sins, but little about his potentialities, his virtues, his achievable aspirations, or his full psychological height. It is as if psychology had voluntarily restricted itself to only half its rightful jurisdiction, and that the darker, meaner half (Maslow, 1954, p. 354)." Humanistic psychology instead addressed the full range of human functioning and focused on personal fulfillment, valuing feelings over intellect, hedonism, a belief in human perfectibility, emphasis on the present, self-disclosure, self-actualization, positive regard, client centered therapy, and the hierarchy of needs. Again, these topics were in stark contrast to much of the work being done in the field of psychology up to and at this time.

In 1996, Martin Seligman became the president of the American Psychological Association (APA) and called for a **positive psychology** or one that had a more positive conception of human potential and nature. Building on Maslow and Roger's work, he ushered in the scientific study of such topics as happiness, love, hope, optimism, life satisfaction, goal setting, leisure, and subjective well-being. Though positive and humanistic psychology have similarities, it should be pointed out their methodology was much different. While humanistic psychology generally relied on qualitative methods, positive psychology utilizes a quantitative approach and aims to make the most out of life's setbacks, relate well to others, find fulfillment in creativity, and finally helping people to find lasting meaning and satisfaction (http://www.positivepsychologyinstitute.com.au/what_is_positive_psychology.html)

So to understand what normal behavior is do we look to positive psychology for an indication or do we first define abnormal behavior and then reverse engineer a definition of what normal is? Our preceding discussion gave suggestions about what normal behavior is but could the darker elements of our personality also make up what is normal, to some extent? Possibly. The one truth is that no matter what behavior we display, if taken to the extreme, it can become disordered – whether trying to control others through social influence or helping people in an altruistic fashion. As such, we can consider **abnormal behavior** to be a combination of personal distress, psychological dysfunction, deviance from social norms, dangerousness to self and others, and costliness to society.

1.1.2. How do we determine what is abnormal behavior?

In the previous section we showed that what we might consider normal behavior is difficult to define. Equally difficult is understanding what abnormal behavior is which may be surprising to you. The

American Psychiatric Association, in its publication which you will become intimately familiar with throughout this book, 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. These include:

- **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). Abnormal behavior, therefore, 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. As such, a good employee who suddenly demonstrates poor performance may be experiencing an environmental demand leading to stress and ineffective coping mechanisms. Once the demand resolves itself the person's performance should return to normal according to this principle.
- **Distress** – When the person experiences a disabling condition "in social, occupational, or other important activities" (pg. 20). Distress can take the form of psychological or physical pain, or both concurrently. 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. 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.
- **Deviance** – Closer examination of the word abnormal shows that it indicates a move away from what is normal, or the mean (i.e. what would be considered average and in this case in relation to behavior), and so is behavior that occurs infrequently (sort of an outlier in our data). 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, homosexuality was considered taboo in the U.S. just a few decades ago 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 while attending the Route 91 Harvest Festival, 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 conceptualization of what abnormal behavior is, many clinicians add **dangerousness** to this list, or when behavior represents a threat to the safety of the person or others. It is important to note that having a mental disorder does not mean you are also automatically dangerous. The depressed or anxious individual is often no more a threat than someone who is not depressed and as Hiday and Burns (2010) showed, dangerousness is more the exception than the rule. Still, mental health professionals have a duty to report to law enforcement when a mentally disordered individual expresses intent to harm another person or themselves. It is important to point out that people seen as dangerous are also not automatically mentally ill.

This leads us to wonder what the cost of mental illness is 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 earning 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 due to 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 both the United States and other countries.

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

https://www.washingtonpost.com/news/wonk/wp/2012/12/17/seven-facts-about-americas-mental-health-care-system/?utm_term=.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, and has the potential to harm others or the individual, is abnormal behavior. Armed with this understanding, let's discuss what mental disorders are.

1.1.3. Definition of abnormal psychology or psychopathology

Our discussion so far has concerned what is normal and abnormal behavior. We saw that the study of normal behavior falls under the providence of positive psychology. Similarly, 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 is what we refer to as **abnormal psychology**. Abnormal behavior can become pathological in nature and so leads to the scientific study of psychological disorders, or **psychopathology**. This begs the question of what the accepted definition of a psychological or mental disorder is. From our previous discussion we can fashion the following definition – **mental disorders** are 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.

1.2. 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 any subtypes.
- Define comorbidity.
- Define etiology.
- Define course.
- Define prognosis.
- Define treatment.

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.

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, and the world. **Psychiatric or mental health epidemiology** refers to the 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 or can be viewed as the number of cases per some number of people. For instance, if 20 people out of 100 have bipolar disorder, then the prevalence rate is 20%. Prevalence can be measured in several ways:

- **Point prevalence** indicates the proportion of a population that has the characteristic at a specific point in time. In other words, it is the number of active cases.
- **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.
- **Lifetime prevalence** indicates the proportion of a population that has had the characteristic 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 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.

Finally, **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 should be noted that the first study was conducted from 1980 to 1985 and surveyed 20,000 people from five different geographical regions in the U.S. A second study followed from 1990-1992 and was called the National Comorbidity Survey (NCS). The third study, the NCS-R, used a new nationally representative sample of the U.S. population, and found 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 great as services to treat substance abuse and mental disorders are often separate, despite their appearing together.

The **etiology** is the cause of the disorder. There may be social, biological, or psychological explanations for the disorders beginning 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 *acute* meaning that it lasts a short period of time, or *chronic,* meaning it lasts a long period of time. It 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 suffering from 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 prescribe medication or utilize psychotherapy to bring about this change. Treatment may be sought from the primary care provider, in an outpatient fashion, or through inpatient care or hospitalization at a mental hospital or psychiatric unit of a general hospital.

1.3. The Stigma of Mental Illness

Section Learning Objectives

- Clarify the importance of social cognition theory in understanding why people do not seek care.
- Define categories and schemas.
- Define stereotypes and heuristics.
- Describe social identity theory and its consequences.
- Differentiate between prejudice and discrimination.
- Contrast implicit and explicit attitudes.
- Explain the concept of stigma and its three forms.
- Define courtesy stigma.
- Describe what the literature shows about stigma.

In the previous section we discussed the fact 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 determine the psychological processes underlying this phenomena in the individual.

Social cognition is the process through which we collect information from the world around us and then interpret it. The collection process occurs through what we know as *sensation* – or detecting physical energy emitted or reflected by physical objects. Detection occurs courtesy of our eyes, ears, nose, skin and mouth; or via vision, hearing, smell, touch, and taste, respectfully. Once collected, the information is relayed to the brain through the neural impulse where it is processed and interpreted, or meaning is added to this raw sensory data which we call *perception*.

One way meaning is added is by taking the information we just detected and using it to assign people to **categories**, or groups. For each category, we have a **schema**, or a set of beliefs and expectations about a group of people, believed to apply to all members of the group, and based on experience. You might think of them as organized ways of making sense of experience. So it is during our initial interaction with someone that we collect information about him/her, assign the person to a category for which we have a schema, and then use that to affect how we interact with her or him. First impressions, called the *primacy effect*, are important because even if we obtain new information that should override an incorrect initial assessment, the initial impression is unlikely to change. We call this the *perseverance effect*, or *belief perseverance*.

Stereotypes are special types of schemas that are very simplistic, very strongly held, and not based on firsthand experience. They are **heuristics**, or mental shortcuts, that allow us to assess this collected information very quickly. One piece of information, such as skin color, can be used to assign the person to a schema for which we have a stereotype. This can affect how we think or feel about the person and behave toward them. Again, human beings have a tendency to imply things about this individual solely due to a distinguishing feature and disregard anything inconsistent with the stereotype.

Social identity theory (Tajfel, 1982; Turner, 1987) states that people categorize their social world into

meaningfully simplistic representations of groups of people. These representations are then organized as *prototypes*, or "fuzzy sets of a relatively limited number of category defining features that not only define one category but serve to distinguish it from other categories" (Foddy and Hogg, as cited in Foddy et al., 1999). We construct in-groups and out-groups and categorize the self as an in-group member. The self is assimilated into the salient in-group prototype, which indicates what cognitions, affect, and behavior we may exhibit. Stereotyping, out-group homogeneity, in-group/out-group bias, normative behavior, and conformity are all based on self-categorization. How so? *Out-group homogeneity* occurs when we see all members of a group outside our immediate one as the same. This leads to a tendency to show favoritism to, and exclude or hold a negative view of members outside of, one's immediate group, called the *in-group/out-group bias*. The negative view or set of beliefs about a group of people is what we call *prejudice* and this can result in acting in a way that is negative against a group of people, called *discrimination*. It should be noted that a person can be prejudicial without being discriminatory since most people do not act on their attitudes toward others due to social norms against such behavior. Likewise, a person or institution can be discriminatory without being prejudicial such as if a company requires that a person have a certain education level or be able to lift 80 pounds as part of normal job responsibilities. Individuals without a degree or ability to lift will be removed from consideration for the job, but this discriminatory act does not mean that the company has negative views of people without degrees or the inability to lift heavy weight. You might even hold a negative view towards a certain group of people and not be aware of it. An attitude we are unaware of is called an *implicit attitude***,** which stands in contrast to *explicit attitudes,* which are the ones we publicly state.

We have spent quite a lot of space and time understanding background information on how people gather information about the world and people around them, process this information, use it to make snap judgements about others, form groups for which stereotypes may exist, and then potentially hold negative views of this group and behave negatively toward them as a result. Just one piece of information can be used to set this series of mental events into motion. Outside of skin color, the label associated with having a mental disorder can be used. Stereotypes about people with a mental disorder can quickly and easily transform into prejudice when people in a society determine the schema to be correct and form negative emotions and evaluations of this group (Eagly & Chaiken, 1993). This in turn can lead to discriminatory practices such as an employer refusing to hire, a landlord refusing to rent an apartment, or a romantic relationship being avoided, all due to the person having a mental illness.

Overlapping with prejudice and discrimination in terms of how people with mental disorders are treated is **stigma**, or when negative stereotyping, labeling, rejection, and loss of status occur. Stigma 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 all together 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" or "nuts" 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 a family member had a serious mental illness (SMI). Due to this they felt hurt and betrayed and an important source of social support during the difficult time had disappeared, 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 versus the relative's place. 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 desiring to maintain personal boundaries. There was also a need to understand responses of others and to attribute it 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 "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 intention in a university sample (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 life span suggesting that anti-stigma campaigns should focus on older people primarily (Schomerus, et al., 2015).

One potentially disturbing trend is that mental health professionals have been shown to hold negative attitudes toward the people they are there to serve. Hansson et al. (2011) 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 a 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 work places 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 a stigma 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 in the public.

Self-stigma has also been shown to affect self-esteem, which then affects hope, which then affects quality of life among people with SMI. 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 stigma change. 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 though 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). More on this in Module 15.

1.4. 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.
- Describe the status of mental illness today.
- Outline the use of psychoactive drugs throughout time and their impact.
- Clarify the importance of managed health care for the treatment of mental illness.
- Define and clarify the importance of multicultural psychology.
- State the issue surrounding prescription rights for psychologists.
- Explain the importance of prevention science.

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.

1.4.1. 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 often occurred 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. Through it, the evil spirits could escape 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 spirts were cast out through prayer, magic, flogging, starvation, having the person ingest horrible tasting drinks, or noise-making.

1.4.2. 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, 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 brain 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 and too much black bile causing melancholia or 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. It was the responsibility of the community and their families to care for them. Greek physician, Galen (A.D. 129-199) said mental disorders had either physical or mental causes and 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 or warm baths, the hope being that their patients would be as comfortable as they could be. They practice 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.

1.4.3. 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 his influence. In extreme cases, the afflicted were exposed to confinement, beatings, and even execution. Scientific and medical explanations, such as those proposed by Hippocrates, were discarded.

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. 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 called upon to explain psychopathology.

1.4.4. 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, 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).

1.4.5. 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. Stressing affording the mentally ill respect, moral guidance, and humane treatment, all while considering their individual, social, and occupational needs, its earliest proponent was Francis Pinel (1745-1826) who was assigned as the superintendent of la Bicetre, a hospital for mentally ill men in Paris. 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 Country Asylums Act of 1845 which required that every county 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 and places where those afflicted with mental illness could recover. Its success was responsible for its decline. 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 but waves of immigrants arriving in the U.S. after the Civil War were overwhelming the same 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 as native citizens, even when the resources were available to treat them.

The moral treatment movement also fell due to the rise of the **mental hygiene movement**, which

focused on the physical well-being of patients. Its main proponent in the United States 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. Over the next 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.

"In the early 1950s, Mental Health America issued a call to asylums across the country for their discarded chains and shackles. On April 13, 1953, at the McShane Bell Foundry in Baltimore, Md., Mental Health America melted down these inhumane bindings and recast them into a sign of hope: the Mental Health Bell.

Now the symbol of Mental Health America, the 300-pound Bell serves as a powerful reminder that the invisible chains of misunderstanding and discrimination continue to bind people with mental illnesses. Today, the Mental Health Bell rings out hope for improving mental health and achieving victory over mental illnesses."

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

1.4.6. 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.

1.4.6.1. 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 a belief that everyone is plotting against you or that you are God (a delusion of grandeur), and

were termed *general paresis* by French physician A.L.J. Bayle. In 1897, Viennese psychiatrist Richard von Krafft-Ebbing injected patients suffering from 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 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 suffering a severe shock his memories had changed and in published work, suggested physicians study electric shock as a treatment for melancholia.

1.4.6.2. 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. 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 suffering from 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 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 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 relived 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.

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. Sigmund Freud's development of psychoanalysis followed on the heels of the work of Bruner, and others who came before him.

1.4.7. Current Views/Trends

1.4.7.1. Mental illness today. An article published by the Harvard Medical School in March 2014 called, "The Prevalent and Treatment of Mental Illness Today," presented the results of the aforementioned National Comorbidity Study Replication of 2001-2003 including a sample of more than 9,000 adults. The results showed that nearly 46% of the participants had a psychiatric disorder at some time in their lives. The most commonly reported disorders were:

- Major depression – 17%
- Alcohol abuse – 13%
- Social anxiety disorder – 12%
- Conduct disorder – 9.5%

Also of interest was that women were more likely to have had anxiety and mood disorders while men showed higher rates of impulse control disorders. Comorbid anxiety and mood disorders were common and 28% reported having more than one co-occurring disorder (Kessler, Berglund, et al., 2005; Kessler, Chiu, et al., 2005; Kessler, Demler, et al., 2005).

About 80% of the sample reported seeking treatment for their disorder, but with as much as a 10-year gap after symptoms first appeared. Women were more likely than men to seek help while whites were more likely than African and Hispanic Americans (Wang, Berglund, et al., 2005; Wang, Lane, et al., 2005). Care was sought primarily from family doctors, nurses, and other general practitioners (23%), followed by social workers and psychologists (16%), psychiatrists (12%), counselors or spiritual advisers (8%), and complementary and alternative medicine providers (CAMs; 7%).

In terms of the quality of the care, the article states:

"Most of this treatment was inadequate, at least by the standards applied in the survey. The researchers defined minimum adequacy as a suitable medication at a suitable dose for two months, along with at least four visits to a physician; or else eight visits to any licensed mental health professional. By that definition, only 33% of people with a psychiatric disorder were treated adequately, and only 13% of those who saw general medical practitioners."

In comparison to the original study conducted from 1991-1992, the use of mental health services has increased over 50% during this decade. This may be attributed to treatment becoming more widespread and increased attempts to educate the public about mental illness. Stigma, discussed in Section 1.3, has reduced over time, diagnosis is more effective, community outreach programs have increased, and most importantly, general practitioners have been more willing to prescribe psychoactive medications which themselves are more readily available now. The article concludes, "Survey researchers also suggest that we need more outreach and voluntary screening, more education about mental illness for the public and physicians, and more effort to treat substance abuse and impulse control disorders." We will explore several of these issues in the remainder of this section to include the use of psychiatric drugs and deinstitutionalization, managed health care, private psychotherapy, positive psychology and prevention science, Multicultural psychology, and prescription rights for psychologists.

For more on the Harvard article, please see:

https://www.health.harvard.edu/mind-and-mood/the-prevalence-and-treatment-of-mental-illness-today

1.4.7.2. Use of psychiatric drugs and deinstitutionalization. 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 and increase the effectiveness of treatments such as psychotherapy. 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 anti-anxiety drugs 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, followed closely by anti-anxiety. 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 leading suffers to believe 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. When people with severe mental illness do need inpatient care, it is typically in the form of short term hospitalization.

1.4.7.3. Managed health care. Managed health care is a term used to describe a type of health insurance in which the insurance company determines the cost of services, providers a person may see, and the number of visits a subscriber can have within a year. This is regulated through contracts with providers and medical facilities. The plans pay the providers directly so subscribers do not have to pay out-of-pocket or complete claim forms, though most require co-pays paid directly to the provider at the time of service. Exactly how much the plan costs depends on how flexible the subscriber wants it to be; the greater the flexibility, the greater the cost. Managed health care takes three forms:

- *Health Maintenance Organizations (HMO)* – Typically only pay for care within the network. The subscriber chooses a primary care physician (PCP) who coordinates the majority of their care. The PCP refers the subscriber to specialists or other health care providers as is necessary. This is the most restrictive option.
- *Preferred Provider Organizations (PPO)* – Usually pay more if the subscriber obtains care within the network but if care outside the network is sought, they pay part of it.
- *Point of Service (POS)* – These plans provide the most flexibility and allow the subscriber to choose between an HMO or a PPO each time care is needed.

In relation to the treatment needed for mental illness, managed care programs regulate the pre-approval of treatment via referrals from the PCP, which mental health providers can be seen, and oversight of which conditions can be treated and what type of treatment can be delivered. This system was developed in the 1980s to combat the rising cost of mental health care and took care out of the hand of single practitioners or small groups who could charge what they felt was appropriate. The actual impact of managed care on mental health services is still questionable at best.

1.4.7.4. Multicultural psychology. As our society becomes increasingly diverse, medical practitioners and psychologists alike have to take into account the patient's gender, age, race, ethnicity, socioeconomic (SES) status, and culture and how these factors shape the individual's thoughts, feelings, and behaviors. Additionally, we need to understand how the various groups, whether defined by race, culture, or gender, differ from one another. This approach is called **multicultural psychology**.

In August 2002, the American Psychological Association's (APA) Council of Representatives put forth six guidelines based on the understanding that "race and ethnicity can impact psychological practice and interventions at all levels" and that there is a need for respect and inclusiveness. They further state, "psychologists are in a position to provide leadership as agents of prosocial change, advocacy, and social justice, thereby promoting societal understanding, affirmation, and appreciation of multiculturalism against the damaging effects of individual, institutional, and societal racism, prejudice, and all forms of oppression based on stereotyping and discrimination." The guidelines from the 2002 document are as follows:

- "Guideline #1: Psychologists are encouraged to recognize that, as cultural beings, they may hold attitudes and beliefs that can detrimentally influence their perceptions of and interactions with individuals who are ethnically and racially different from themselves.
- Guideline #2: Psychologists are encouraged to recognize the importance of multicultural sensitivity/responsiveness, knowledge, and understanding about ethnically and racially different individuals.
- Guideline #3: As educators, psychologists are encouraged to employ the constructs of multiculturalism and diversity in psychological education.
- Guideline #4: Culturally sensitive psychological researchers are encouraged to recognize the importance of conducting culture–centered and ethical psychological research among persons from ethnic, linguistic, and racial minority backgrounds.
- Guideline #5: Psychologists strive to apply culturally–appropriate skills in clinical and other applied psychological practices.
- Guideline #6: Psychologists are encouraged to use organizational change processes to support culturally informed organizational (policy) development and practices."

Source: https://apa.org/pi/oema/resources/policy/multicultural-guidelines.aspx

This type of sensitivity training is important because bias on the basis of ethnicity, race, and culture has been found in the diagnosis and treatment of autism (Harrison et al., 2017; Burkett, 2015), borderline personality disorder (Jani et al., 2016), and schizophrenia (Neighbors et al., 2003'; Minsky et al., 2003). Despite these findings, Schwartz and Blankenship (2014) states, "It should also be noted that although clear evidence supports a longstanding trend in differential diagnoses according to consumer race, this trend does not imply that one race (*e.g.*, African Americans) actually demonstrate more severe symptoms or higher prevalence rates of psychosis compared with other races (*e.g.*, Euro-Americans). Because clinicians are the diagnosticians and misinterpretation, bias or other factors may play a role in this trend caution should be used when making inferences about actual rates of psychosis among ethnic minority persons." Additionally, white middle-class help seekers were offered appointments with psychotherapists almost three times as often as their black working-class counterparts while women were offered an appointment time in their preferred time range more than men were, though average appointment offer rates were similar between genders (Kugelmass, 2016). These findings collectively show that though we are becoming more culturally sensitive, we have a lot more work to do.

1.4.7.5. Prescription rights for psychologists. To reduce inappropriate prescribing as described in 1.4.7.2, 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)."

1.4.7.6. Prevention science. As a society, we used to wait for a mental or physical health issue to emerge and then we would scramble to treat it. More recently, medicine and science has taken a **prevention** stance, or 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 & Beer, 2007), in the workplace (Czabała, Charzyńska, & Mroziak, B., 2011), with undergraduate and graduate students (Conley et al., 2017; Bettis et al., 2016), 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). The growth of positive psychology in the late 1990s has further propelled this movement forward. For more on positive psychology, please see Section 1.1.1.

1.5. Research Methods in Psychopathology

Section Learning Objectives

- Define scientific method.
- Outline and describe the steps of the scientific method, defining all key terms.
- Identify and clarify the importance of, the three cardinal features of science.
- List the five main research methods used in psychology.
- Describe observational research, listing its advantages and disadvantages.
- Describe case study research, listing its advantages and disadvantages.
- Describe survey research, listing its advantages and disadvantages.
- Describe correlational research, listing its advantages and disadvantages.
- Describe experimental research, listing its advantages and disadvantages.
- State the utility and need for multimethod research.

1.5.1. The Scientific Method

Psychology is the “scientific study of behavior and mental processes.” We will spend quite a lot of time on the behavior and mental processes part throughout this book and in relation to mental disorders, but before we proceed, it is prudent to elaborate more on what makes psychology scientific. In fact, it is safe to say that most people not within our discipline or a sister science, would be surprised to learn that psychology utilizes the scientific method at all. That may be even truer of clinical psychology, especially in light of the plethora of self-help books that can be found at any bookstore. But yes, the treatment methods used by mental health professionals are based on empirical research and the scientific method.

As a starting point, we should expand on what the scientific method is.

The **scientific method** is a systematic method for gathering knowledge about the world around us.

The key word here is that it is systematic meaning there is a set way to use it. What is that way? Well, depending on what source you look at it can include a varying number of steps. I like to use the following:

Table 1.1: The Steps of the Scientific Method

Step	Name	Description
0	Ask questions and be willing to wonder.	To study the world around us you have to wonder about it. This inquisitive nature is the hallmark of **critical thinking,** or 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.
1	Generate a research question or identify a problem to investigate.	Through our wonderment about the world around us and why events occur as they do, we begin to ask questions that require further investigation to arrive at an answer. This investigation usually starts with a **literature review**, or 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, so that we can identify **gaps** or holes in this body of work.
2	Attempt to explain the phenomena we wish to study.	We now attempt to formulate an explanation of why the event occurs as it does. This systematic explanation of a phenomenon is a **theory** and our specific, testable prediction is the **hypothesis.** We will know if our theory is correct because we have formulated a hypothesis which we can now test.
3	Test the hypothesis.	It goes without saying that if we cannot test our hypothesis, then we cannot show whether our prediction is correct or not. Our plan of action of how we will go about testing the hypothesis is called our **research design**. In the planning stage, we will select the appropriate research method to answer our question/test our hypothesis.
4	Interpret the results.	With our research study done, we now examine the data to see if the pattern we predicted exists. We need to see if a cause and effect statement can be made, assuming our method allows for this inference. More on this in Section 2.3. For now, it is important to know that the statistics we use take on two forms. First, there are **descriptive statistics** which provide a means of summarizing or describing data, and presenting the data in a usable form. You likely have heard of the mean or average, median, and mode. Along with standard deviation and variance, these are ways to describe our data. Second, there are **inferential statistics** which allow for the analysis of two or more sets of numerical data to determine the **statistical significance** of the results. Significance is an indication of how confident we are that our results are due to our manipulation or design and not chance.
5	Draw conclusions carefully.	We need to accurately interpret our results and not overstate our findings. To do this, we need to be aware of our biases and avoid emotional reasoning so that they do not cloud our judgment. How so? In our effort to stop a child from engaging in self-injurious behavior that could cause substantial harm or even death, we might overstate the success of our treatment method.
6	Communicate our findings to the larger scientific community.	Once we have decided on whether or hypothesis was correct or not, we need to share this information with others so that they might comment critically on our methodology, statistical analyses, and conclusions. Sharing also allows for **replication** or repeating the study to confirm its results. Communication is accomplished via scientific journals, conferences, or newsletters released by many of the organizations mentioned in Module 1.6.

Science has at its root three *cardinal features* that we will see play out time and time again throughout this book. They are:

1. *Observation* – In order to know about the world around us we have to be able to see it firsthand. When an individual is afflicted by a mental disorder, we can see it through the overt behavior they make. An individual with depression may withdraw from activities he/she enjoys, those with social anxiety disorder will avoid social situations, people with schizophrenia may express concern over being watched by the government, and individuals with dependent personality disorder may wait to make any decision in life until trusted others tell them what to do. In these examples, and numerous others we can suggest, the behaviors that lead us to a diagnosis of a specific disorder can easily be observed by the clinician, the patient, and/or family and friends.
2. *Experimentation* – To be able to make *causal* or cause and effect statements, we must isolate variables. We have to manipulate one variable and see the effect of doing so on another variable. Let's say we want to know if a new treatment for bipolar disorder is as effective as existing treatments…or more importantly, better. We could design a study with three groups of bipolar patients. One group would receive no treatment and serve as a control group. A second group would receive an existing and proven treatment, and would also be considered a control group. Finally, the third group would receive the new treatment and be the experimental group. What we

are manipulating is what treatment the groups get – no treatment, the older treatment, and the newer treatment. The first two groups serve as controls since we already know what to expect from their results. There should be no change in bipolar disorder symptoms in the no treatment group, a general reduction in symptoms for the older treatment group, and the same or better performance for the newer treatment group. As long as patients in the newer treatment group don't perform worse than their older treatment counterparts, we can say the new drug is a success. You might wonder why we would get excited about the performance of the new drug being the same as the old drug. Does it really offer any added benefit? It terms of a reduction of symptoms, maybe not, but it could cost less money than the older drug and so that would be of value to patients.

3. *Measurement* – How do we know that the new drug has worked? Simply, we can measure the person's bipolar disorders symptoms before any treatment was implemented, and then again once the treatment has ran its course. This pre-post test design is typical in drug studies.

1.5.2. Research Methods

Step 3 called on the scientist to test his or her hypothesis. Psychology as a discipline uses five main research designs. They are:

1.5.2.1. Naturalistic and laboratory observation. In terms of **naturalistic observation**, the scientist studies human or animal behavior in its natural environment which could include the home, school, or a forest. The researcher counts, measures, and rates behavior in a systematic way and at times uses multiple judges to ensure accuracy in how the behavior is being measured. The advantage of this method is that you see behavior as it occurs and it is not tainted by the experimenter. The disadvantage is that it could take a long time for the behavior to occur and if the researcher is detected then this may influence the behavior of those being observed. **Laboratory observation** involves observing people or animals in a laboratory setting. The researcher might want to know more about parent-child interactions and so brings a mother and her child into the lab to engage in preplanned tasks such as playing with toys, eating a meal, or the mother leaving the room for a short period of time. The advantage of this method over naturalistic method is that the experimenter can use sophisticated equipment and videotape the session to examine it at a later time. The problem is that since the subjects know the experimenter is watching them, their behavior could become artificial. Clinical observation is a commonly employed research method to study psychopathology and we will talk about it more throughout this course.

1.5.2.2. Case studies. Psychology can also utilize a detailed description of one person or a small group based on careful observation. This was the approach the found of psychoanalysis, Sigmund Freud, took to develop this theories. The advantage of this method is that you arrive at a rich description of the behavior being investigated but the disadvantage is that what you are learning may be unrepresentative of the larger population and so lacks **generalizability**. Again, bear in mind that you are studying one person or a very small group. Can you possibly make conclusions about all people from just one or even five or ten? The other issue is that the case study is subject to the bias of the researcher in terms of what is included in the final write up and what is left out. Despite these limitations, case studies can lead us to novel ideas about the cause of abnormal behavior and help us to study unusual conditions that occur too infrequently to study with large sample sizes and in a systematic way.

1.5.2.3. Surveys/Self-Report data. This is a questionnaire consisting of at least one scale with some number of questions which assess a psychological construct of interest such as parenting style, depression, locus of control, or sensation seeking behavior. It may be administered by paper and pencil or computer. Surveys allow for the collection of large amounts of data quickly but the actual survey could be tedious for the participant and **social desirability**, when a participant answers questions dishonestly so that he/she is seen in a more favorable light, could be an issue. For instance, if you are asking high school students about their sexual activity they may not give genuine answers for fear that their parents will find out. You could alternatively gather this information via an interview in a structured or unstructured fashion.

1.5.2.4. Correlational research. This research method examines the relationship between two variables or two groups of variables. A numerical measure of the strength of this relationship is derived, called the *correlation coefficient*, and can range from -1.00, a perfect inverse relationship meaning that as one variable goes up the other goes down, to 0 or no relationship at all, to +1.00 or a perfect relationship in which as one variable goes up or down so does the other. In terms of a negative correlation we might say that as a parent becomes more rigid, controlling, and cold, the attachment of the child to parent goes down. In contrast, as a parent becomes warmer, more loving, and provides structure, the child becomes more attached. The advantage of correlational research is that you can correlate anything. The disadvantage is that you can correlate anything. Variables that really do not have any relationship to one another could be viewed as related. Yes. This is both an advantage and a disadvantage. For instance, we might correlate instances of making peanut butter and jelly sandwiches with someone we are attracted to sitting near us at lunch. Are the two related? Not likely, unless you make a really good PB&J but then the person is probably only interested in you for food and not companionship. The main issue here is that correlation *does not* allow you to make a causal statement.

A special from of correlational research is the **epidemiological study** in which the prevalence and incidence of a disorder in a specific population are measured (See Section 1.2 for definitions).

1.5.2.5. Experiments. This is a controlled test of a hypothesis in which a researcher manipulates one variable and measures its effect on another variable. The variable that is manipulated is called the **independent variable (IV)** and the one that is measured is called the **dependent variable (DV)**. In the example above, the treatment for bipolar disorder was the IV while the actual intensity or number of symptoms serves as the DV. A common feature of experiments is to have a **control group** that does not receive the treatment or is not manipulated and an **experimental group** that does receive the treatment or manipulation. If the experiment includes **random assignment** participants have an equal chance of being placed in the control or experimental group. The control group allows the researcher (or teacher) to make a *comparison* to the experimental group, make our causal statement possible, and stronger. In our experiment, the new treatment should show a marked reduction in the intensity of bipolar symptoms compared to the group receiving no treatment, and perform either at the same level as, or better than, the older treatment. This would be the hypothesis we begin the experiment with.

There are times when we begin a drug study and to ensure participant expectations have no effect on the final results through giving the researcher what he/she is looking for (in our example, symptoms improve whether or not a treatment is given or not), we use what is called a **placebo**, or a sugar pill made to look exactly like the pill given to the experimental group. This way, participants all are given something, but cannot figure out what exactly it is. You might say this keeps them honest and allows the results to speak for themselves.

Finally, the study of mental illness does not always afford us a large sample of participants to study and so we have to focus on one individual This is called a **single-subject experimental design** and differs from a case study in the sheer number of strategies that can be used to reduce potential **confounding variables**, or variables not originally part of the research design but contribute to the results in a meaningful way. One type of single-subject experimental design is the **reversal** or **ABAB design**. Kuttler, Myles, and Carson (1998) used social stories to reduce tantrum behavior in two social environments in a 12-year old student diagnosed with autism, Fragile-X syndrome, and intermittent explosive disorder. Using an ABAB design, they found that precursors to tantrum behavior decreased when the social stories were available (B) and increased when the intervention was withdrawn (A). A more recent study (Balakrishnan & Alias, 20174) also established the utility of social stories as a social learning tool for children with autism spectrum disorder (ASD) using an ABAB design. Four students were included in the study and during the baseline phase (A) they were observed and data recorded on an observation form. During the treatment phase (B), they were read the social story and data recorded in the same manner. Upon completion of the first B, the student was returned to A which was followed one more time by B and the reading of the social story. Once the second treatment phase ended, the participation was monitored again to obtain a final outcome. All students showed improvement during the treatment phases in terms of the number of positive peer interactions they had and then dropped back down in terms of the number of such interactions. From this the researchers concluded that the social story led to the increase in positive peer interactions of children with ASD.

1.5.2.6. Multi-method research. As you have seen above, no single method alone is perfect. All have their strengths and limitations. As such, for the psychologist to provide the most clear picture of what is affecting behavior or mental processes, several of these approaches are typically employed at different stages of the research study. This is called **multi-method research.**

1.6. Mental Health Professionals, Societies, and Journals

Section Learning Objectives

- Identify and describe the various types of mental health professionals.
- Clarify what it means to communicate findings.
- Identify professional societies in clinical psychology.
- Identify publications in clinical psychology.

1.6.1. Types of Professionals

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

Table 1.2: 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. or Ph.D.	Has specialized training in the diagnosis and treatment of mental disorders	Yes
Psychiatric Nurse Practitioner	R.N.	Has specialized treatment in the care and treatment of psychiatric patients	Yes
Occupational Therapist	B.S.	Has training with individuals suffering from physical or psychological handicaps and helps them acquire needed resources	No
Pastoral Counselor	Clergy	Has training in pastoral education and can make diagnoses and can provide individual and group therapy	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

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

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

1.6.2. Professional Societies and Journals

One of the functions of science is to communicate findings. Testing hypotheses, developing sound methodology, accurately analyzing data, and drawing cogent conclusions are important, but you must tell others what you have done too. This is accomplished via joining professional societies and submitting articles to peer reviewed journals. Below are some of the societies and journals important to applied behavior analysis.

1.6.2.1. Professional Societies

- **Society of Clinical Psychology – Division 12 of the American Psychological Association**
    - Website – http://www.apa.org/about/division/div12.aspx
    - Mission Statement – "The mission of the Society of Clinical Psychology is to represent the field of Clinical Psychology through encouragement and support of the integration of clinical psychological science and practice in education, research, application, advocacy and public policy, attending to the importance of diversity."
    - Publications – *Clinical Psychology: Science and Practice and the newsletter Clinical Psychology: Science and Practice*(quarterly)
    - *Other Information – Members and student affiliates may join one of eight division sections*

such as emergencies and crises, clinical psychology of women, assessment, and clinical geropsychology

- **Society of Clinical Child and Adolescent Psychology – Division 53 of the American Psychological Association**
    - Website – https://www.clinicalchildpsychology.org/
    - Mission Statement – "The purpose of Division 53: Society of Clinical Child and Adolescent Psychology is to encourage the development and advancement of clinical child and adolescent psychology through integration of its scientific and professional aspects. The division promotes scientific inquiry, training, professional practice, and public policy in clinical child and adolescent psychology as a means of improving the welfare and mental health of children, youth, and families. In the service of these goals, the division promotes the general objectives of the American Psychological Association."
    - Publication – Journal of *Clinical Child and Adolescent Psychology*
- **American Academy of Clinical Psychology**
    - Website – https://www.aacpsy.org/
    - Mission Statement – "The American Academy of Clinical Psychology is an organization of Board Certified psychologists in the specialty of Clinical Psychology who have joined together to promote high quality services in Clinical Psychology, through encouraging high standards and ethical practice in the field. The Academy also provides member services, promotes the value and recognition of Board Certification in the specialty of Clinical Psychology, and encourages those qualified by training and experience to become candidates for Board Certification."
    - Publication – Bulletin of the American Academy of Clinical Psychology (newsletter)
- **The Society for a Science of Clinical Psychology (SSCP)**
    - Website – http://www.sscpweb.org/
    - Mission Statement – "**The Society for a Science of Clinical Psychology (SSCP)** was established in 1966. Its purpose is to affirm and continue to promote the integration of the scientist and the practitioner in training, research, and applied endeavors. Its members represent a diversity of interests and theoretical orientations across clinical psychology. The common bond of the membership is a commitment to empirical research and the ideal that scientific principles should play a role in training, practice, and establishing public policy for health and mental health concerns. SSCP has organizational affiliations with both the American Psychological Association (Section III of Division 12) and the Association for Psychological Science."
    - *Other Information – Offers ten awards ranging from early career award, outstanding mentor award, outstanding student teacher award, and outstanding student clinician award.*
- **American Society of Clinical Hypnosis**
    - Website – http://www.asch.net/
    - Mission Statement – "The American Society of Clinical Hypnosis is the largest U.S. organization for health and mental health care professionals using clinical hypnosis. Founded by Milton H. Erickson, MD in 1957, ASCH promotes greater acceptance of hypnosis as a clinical tool with broad applications. Today, ASCH offers professional hypnosis

training workshops, certification, and networking opportunities that can enhance both professional and personal lives. ASCH is unique among organizations for professionals using hypnosis. Members must be licensed healthcare workers and, at a minimum, have obtained a master's degree."
- Publication – American Journal of Clinical Hypnosis
- *Other Information – Offers certification in clinical hypnosis*

1.6.2.2. Professional Journals

- **Clinical Psychology: Science and Practice**
  - Website – http://onlinelibrary.wiley.com/journal/10.1111/(ISSN)1468-2850
  - Published by – American Psychological Association, Division 12
  - Description – "*Clinical Psychology: Science and Practice* presents cutting-edge developments in the science and practice of clinical psychology by publishing scholarly topical reviews of research, theory, and application to diverse areas of the field, including assessment, intervention, service delivery, and professional issues."
- **Journal of Clinical Child and Adolescent Psychology**
  - Website – https://www.clinicalchildpsychology.org/JCCAP
  - Published by – American Psychological Association, Division 53
  - Description – "It publishes original contributions on the following topics: (a) the development and evaluation of assessment and intervention techniques for use with clinical child and adolescent populations; (b) the development and maintenance of clinical child and adolescent problems; (c) cross-cultural and sociodemographic issues that have a clear bearing on clinical child and adolescent psychology in terms of theory, research, or practice; and (d) training and professional practice in clinical child and adolescent psychology, as well as child advocacy."
- **American Journal of Clinical Hypnosis**
  - Website – http://www.asch.net/Public/AmericanJournalofClinicalHypnosis.aspx
  - Published by – American Society of Clinical Hypnosis
  - Description – "The *Journal* publishes original scientific articles and clinical case reports on hypnosis, as well as reviews of related books and abstracts of the current hypnosis literature."

Module Recap

In Module 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 illness as causing dysfunction, distress, deviance, and at times, being dangerous for the afflicted and others

around him/her. From this we classified mental disorders in terms of their occurrence, cause, course, prognosis, and treatment. We acknowledged that mental illness is stigmatized in our society and provided a basis for why this occurs and what to do about it. This involved a discussion of the history of mental illness and current views and trends. Psychology is the scientific study of behavior and mental processes. The word *scientific* is key as psychology adheres to the strictest aspects of the scientific method and uses five main research designs in its investigation of mental disorders – observation, case study, surveys, correlational research, and experiments. These designs are used by various mental health professionals and societies and journals provide additional means to communicate findings or to be good consumers of psychological inquiry. It is with this foundation in mind that we move to examine models of abnormality in Module 2.

Module 2: Models of Abnormal Psychology

Module Overview

In Module 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.

Module Outline

- 2.1. Uni- vs. Multi-Dimensional Models of Abnormality
- 2.2. The Biological Model
- 2.3. Psychological Perspectives
- 2.4. The Sociocultural Model

Module Learning Outcomes

- 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.

2.1. Uni- vs. Multi-Dimensional Models of Abnormality

Section Learning Objectives

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

2.1.1. Uni-Dimensional

In order to effectively treat a mental disorder, we have 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 multiple

causes. 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 psychopathology 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 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). For mental health professionals, models help us to understand mental illness since diseases 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 as having dysthymia, paranoid schizophrenia, avoidant personality disorder, or illness anxiety disorder. Five out of nine symptoms may be enough to labeled as having one of the disorders, for example. There will be some variability in terms of what symptoms the afflicted displays, but in general all people with a specific psychopathology 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, determines 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 module?

- **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.
- **Socicultural** – 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 the role of viral infections in mental illness.
- Describe commonly used treatments for mental illness.
- Evaluate the usefulness of the biological model.

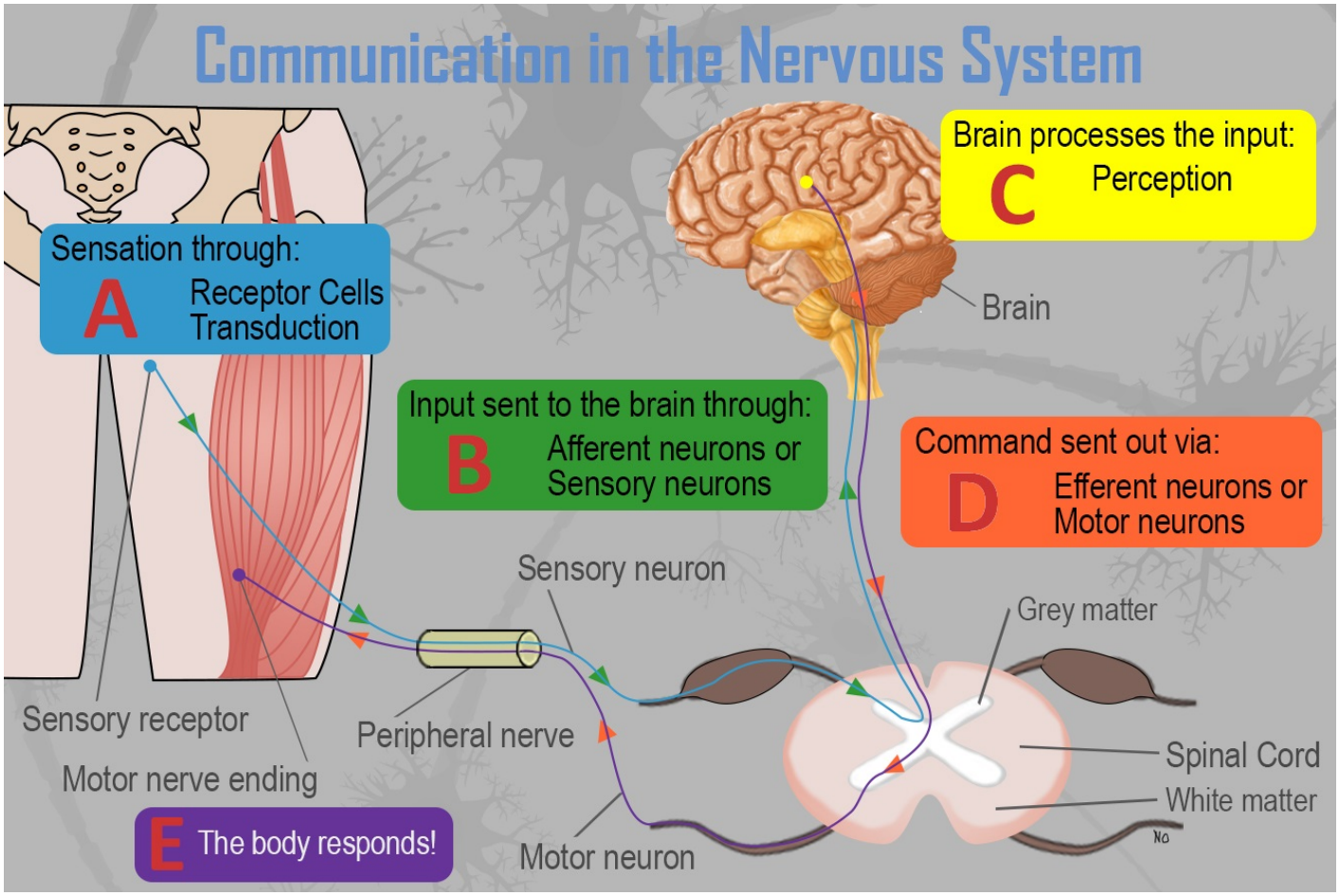
Proponents of the biological model view mental illness as being a result of a malfunction in the body to include issues with brain anatomy or chemistry. As such, we will need to establish a foundation for how communication in the nervous system occurs, what the parts of the nervous system are, what a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

2.2.1. Brain Structure and Chemistry

2.2.1.1. 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. See Figure 2.1 below. 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 (Step E), also via the peripheral nervous system.

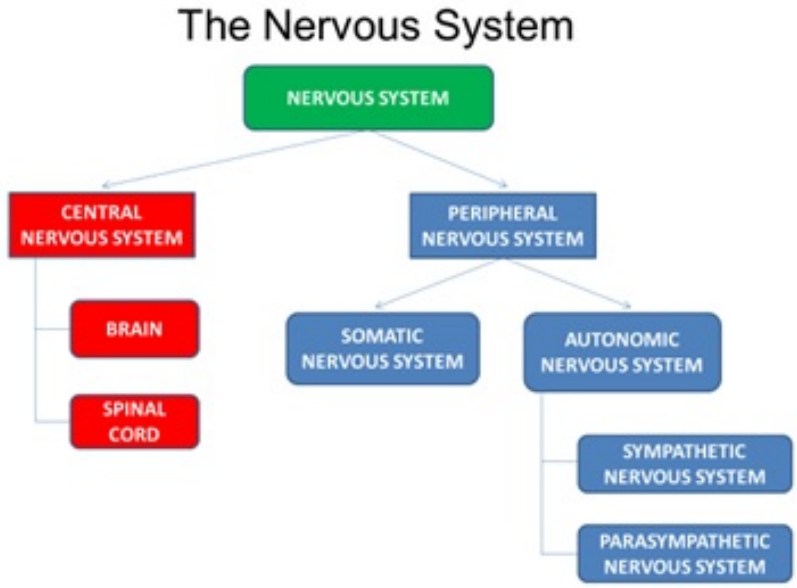
Figure 2.1. Communication in the Nervous System



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

2.2.1.2. 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 carries sensory information to the CNS. The **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. 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 instinct). Eventually the response brought about by the sympathetic nervous system must end. The **parasympathetic nervous system** calms the body.

Figure 2.2. The Structure of the Nervous System



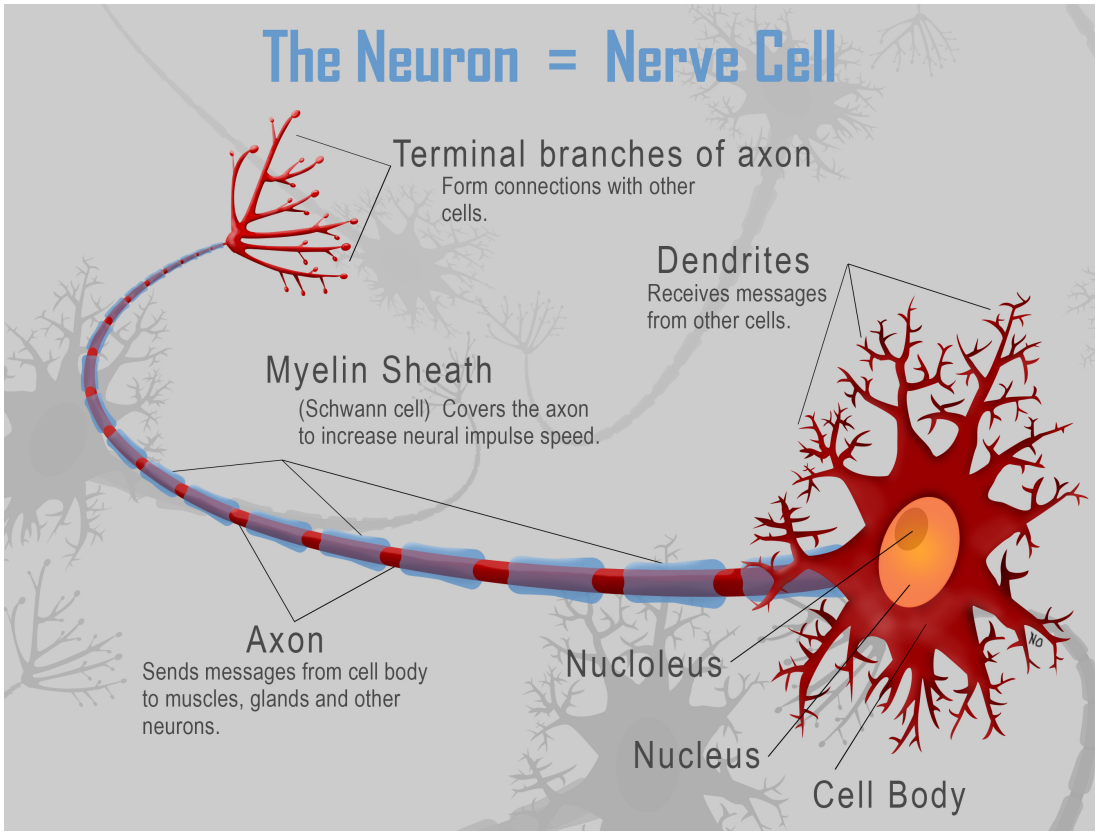
2.2.1.3. The neuron. The fundamental unit of the nervous system is the neuron, or nerve cell (See Figure 2.3). It has several structures in common with all cells in the body. The **nucleus** is the control center of the body and the **soma** is the cell body. It terms of structures that make it different, these focus on the ability of a neuron to send and receive information. The **axon** sends signals/information to neighboring neurons 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 passed to an adjacent neuron.

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 2.3. The Structure of the Neuron



2.2.1.4. 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? See Figure 2.4 below. We will cover this process in three parts.

Part 1. The Axon and Neural Impulse

Figure 2.4. The Neural Impulse

The Neural Impulse

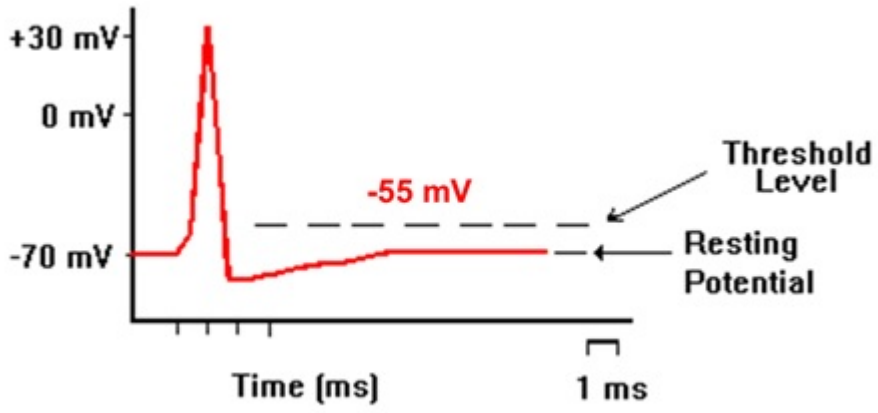
Step	Description	Type of Potential	Condition of the neuron in terms of charge	Polarity INSIDE the Neuron	Polarity OUTSIDE the Neuron
1	The neuron is normally in this state……	Resting	Polarization	−	+
2	When it is adequately stimulated it experiences an……	Action	Depolarization	+	−
				Na What ion ENTERS the axon to cause the shift in polarity?????	
3	As the electrical charge passes along the length of the axon, previous segments undergo this….		Repolarization	− (Inside the previous segments)	+ (Inside the previous segments)
				K What ion LEAVES the axon to cause the shift in polarity in each section?	
4	Immediately after firing the neuron enters this **period** in which it **will not fire** no matter how powerful the incoming message is	Absolute Refractory			
5	Then it enters this **period** in which the neuron **will fire** again only if the incoming message is extremely powerful	Relative Refractory	Polarization	−	+
6	Then it returns to the state it is normally in (as in Step 1)	Resting			

- Step 1 – Neurons waiting to fire are said to be in **resting potential** and **polarized**, or having 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 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 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**. Think of it as the neuron ABSOLUTELY will not fire, no matter what.
- 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. We start at resting potential in Step 1 and notice that Step 6 is the same as Step 1.

Part 2. The Action Potential

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

Figure 2.5. 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 we return to resting potential, as you saw in Figure 2.4

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* of the sending neuron; the *space* in between called the **synaptic space**, **gap**, or **cleft**; and the *dendrite* of the receiving 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 the process of the presynaptic neuron taking up excess neurotransmitters in the synaptic space for future use or **enzymatic**

degradation when enzymes are used to destroy excess neurotransmitters in the synaptic space.

2.2.1.5. Neurotransmistters. 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
- **Endorphins** – involved in reducing pain and making the person calm and happy
- **Norepinephrine** – increases the heart rate and blood pressure and regulates mood
- **GABA** – responsible for blocking the signals of excitatory neurotransmitters responsible for anxiety and panic.
- **Glutamate** – 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.

2.2.1.6. 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** – The major sensory relay center for all senses but 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 about facts and events
- 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.

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 structure is responsible for it. What is important to mental health professionals is that for some disorders, specific areas of the brain are involved. For instance, Parkinson's disease is a brain disorder which results in a gradual loss of muscle control and arises when cells in the **substantia nigra**, a long nucleus considered to be part of the basal ganglia, stop making dopamine. As these cells die, the brain fails to receive messages about when and how to move. In the case of depression, low levels of serotonin are responsible, at least partially. New evidence suggests "nerve cell connections, nerve cell growth, and the functioning of nerve circuits have a major impact on depression…and areas that play a significant role in depression are the amygdala, the thalamus, and the hippocampus." Also, 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 for those suffering from 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* for which we have 23 pairs or 46 total. Twenty-two of the 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 suffering from OCD are at higher risks to develop the disorder themselves. The same is true of borderline personality disorder.

WebMd adds, "Experts believe many mental illnesses are linked to abnormalities in many genes rather than just one or a few and that how these genes interact with the environment is unique for every person (even identical twins). That is why a person inherits a susceptibility to a mental illness and doesn't necessarily develop the illness. Mental illness itself occurs from the interaction of multiple genes and other factors — such as stress, abuse, or a traumatic event — which can influence, or trigger, an illness in a person who has an inherited susceptibility to it" (https://www.webmd.com/mental-health/mental-health-causes-mental-illness#1).

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** helps regulate the sleep-wake cycle while the **thyroid gland** regulates the body's rate of metabolism and so how energetic people are.

Of importance to mental health professionals are the **adrenal glands**, located on top of the kidneys, and which release *cortisol* to help the body deal with stress. Elevated levels of this hormone can lead to increased weight gain, interfere with learning and memory, reduce bone density, increase cholesterol, etc… But the hormone also can cause an increased risk of depression. Also, overproduction of the hormone melatonin can lead to SAD.

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 under five major categories.

The *antidepressants* are used to treat depression, but also anxiety, insomnia, or pain. The most common types of antidepressants are SSRIs or selective serotonin reuptake inhibitors and include Citalopram, Paroxetine, and Fluoxetine (Prozac). 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 Clonazepam, Alprazolam, and Lorazepam. “Anti-anxiety medications such as benzodiazepines are effective in relieving anxiety and take effect more quickly than the antidepressant medications (or buspirone) often prescribed for anxiety. However, people can build up a tolerance to benzodiazepines if they are taken over a long period of time and may need higher and higher doses to get the same effect.” 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. Stimulants are generally effective and produce a calming effect. Possible side effects includeloss of appetite, headache, motor tics or verbal tics, and personality changes such as appearing emotionless.

Antipsychotics are used to treat psychosis or, “conditions that affect the mind, and in which there has been some loss of contact with reality, often including delusions (false, fixed beliefs) or hallucinations (hearing or seeing things that are not really there).” They can 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 to

include severe depression, acute mania, suicidality, and some forms of schizophrenia. 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 1960s and 1970s.

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 some personality and anxiety disorders, though critics cite obvious ethical issues with conducting such surgeries as well as scientific issues.

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 Perspectives

Section Learning Objectives

- Describe psychodynamic theory.
- Outline the structure of personality and how it develops over time.
- Describe ways to deal with anxiety.
- Clarify what psychodynamic techniques are used.
- Evaluate the usefulness of psychodynamic theory.
- Describe learning.
- Outline respondent 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 schemas on creating abnormal behavior.
- Exemplify the effect of attributions on creating abnormal behavior.
- 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. 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 and 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 to include symptoms as 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 Module 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

2.3.1.1. 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 majors schools 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, and part of it becomes our conscience. The three parts of personality generally work together well and compromise, leading to a healthy personality, but if the conflict is not resolved, intrapsychic conflicts can arise and lead to mental disorders.

Personality develops over the course of five distinct stages, 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.

2.3.1.2. The development of personality. Freud's psychosexual stages of personality development are listed below. Please note 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 and

children develop an attachment to the parent of the opposite sex and are jealous of the same sex parent. The Oedipus complex develops in boys and results in the son faalling 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 during lovemaking.

2.3.1.3. Dealing with anxiety. 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. **Ego-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 such as we are angry with our boss but cannot lash out at him, and so are super friendly instead. Another example is having lustful thoughts to a coworker than you cannot express because you are married, and so you are extremely hateful 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.
- **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.
- **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

2.3.1.4. 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 inner most 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.

2.3.1.5. 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 study 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.

2.3.2. The Behavioral Model

2.3.2.1. 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**, or 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 panics 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).

2.3.2.2. Respondent 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 the 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 respondent 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.

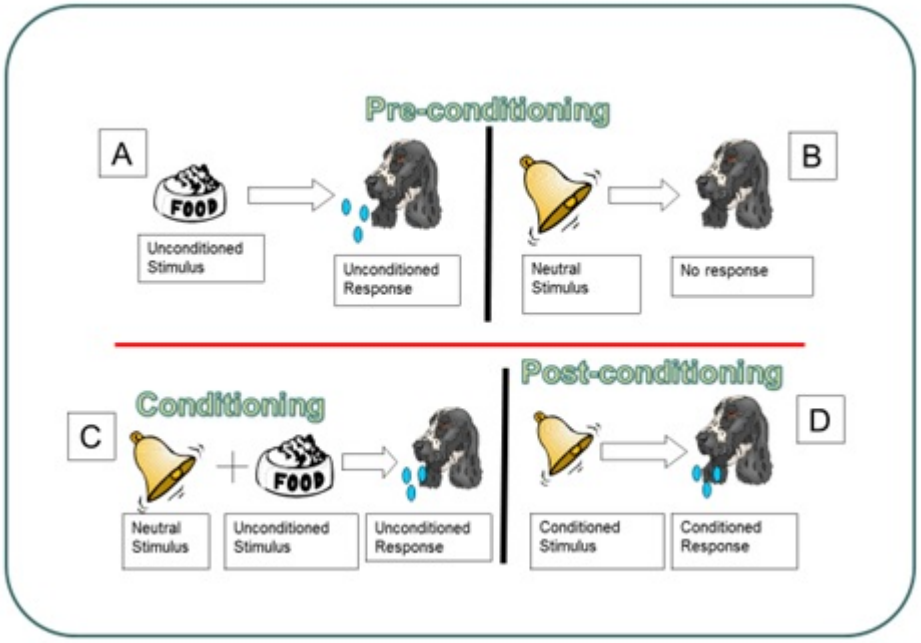
Respondent conditioning (also called classical or Pavlovian conditioning) occurs when we link a previously neutral stimulus with a stimulus that is unlearned or inborn, called an unconditioned stimulus. In respondent conditioning, learning occurs in three phases: preconditioning, conditioning, and postconditioning. See Figure 2.6 for an overview of Pavlov's classic experiment.

Preconditioning. Notice that preconditioning has both an A and a B panel. Really, all 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 Panel B, we 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 a pairing of 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).

Figure 2.6. Pavlov's Classic Experiment

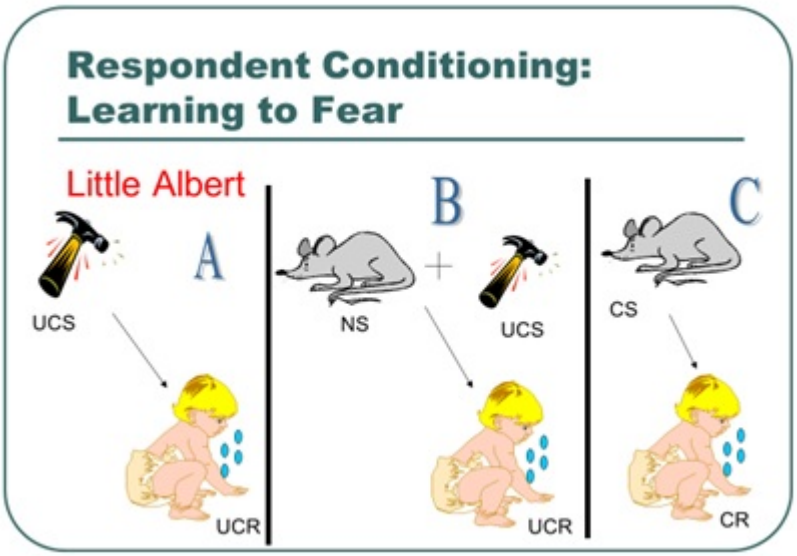


Postconditioning. Postconditioning, or *after* learning has occurred, establishes a *new* and not naturally occurring relationship of a conditioned stimulus (CS; previously the NS) and conditioned response (CR; the same response). So the dog now reliably salivates at the sound of the bell because he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

One of the most famous studies in psychology was conducted by Watson and Rayner (1920). Essentially, they wanted to explore "the possibility of conditioning various types of emotional response(s)." 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 to which no response was made outside of curiosity (NS–NR not shown). In Panel A of Figure 2.7, we have the naturally occurring response to the stimulus of a loud sound. On later

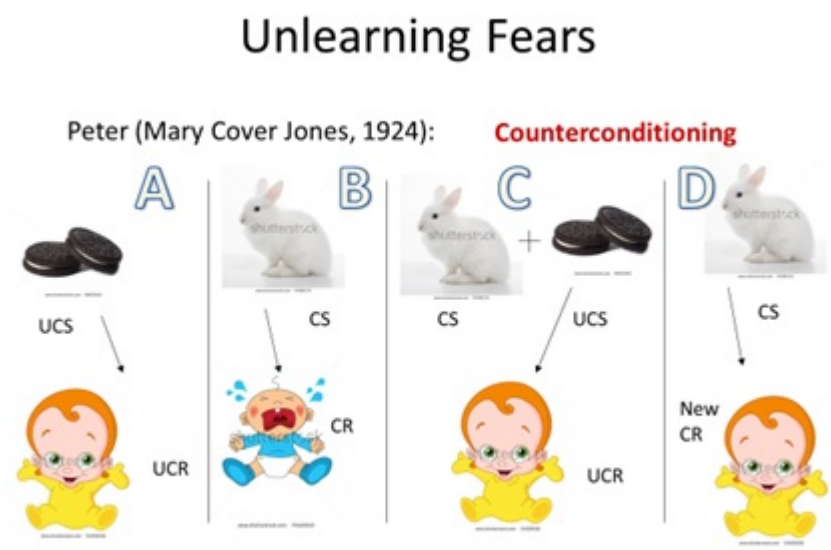
trials, the rat was presented (NS) and followed closely by a loud sound (UCS; Panel B). After several conditioning trials, the child responded with fear to the mere presence of the white rat (Panel C).

Figure 2.7. Learning to Fear



As fears can be learned, so too they can be unlearned. Considered the follow-up to Watson and Rayner (1920), Jones (1924; Figure 2.8) wanted to see if a child who learned to be afraid of white rabbits (Panel B) could be conditioned to become unafraid of them. Simply, she placed the child 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 the child some pleasant food (i.e., something sweet such as cookies [Panel C]; remember the response to the food is unlearned, i.e., Panel A). The procedure in Panel C continued with the rabbit being brought in a bit closer each time until eventually the child did not respond with distress to the rabbit (Panel D).

Figure 2.8. Unlearning Fears



This process is called **counterconditioning**, or the reversal of previous learning.

Another respondent conditioning way to unlearn a fear is what 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. That is the idea at least and if you were afraid of clowns, you would be thrown into a room full of clowns. Hmm….

Finally, several properties of respondent conditioning should be mentioned:

- **Respondent Generalization** – When a number of similar CSs or a broad range of CSs elicit the same CR. An example is the sound of a whistle eliciting salivation the same as the sound of a bell, both detected via audition.
- **Respondent Discrimination** – When the CR is elicited by a single CS or a narrow range of CSs. Teaching the dog to not respond to the whistle but only to the bell, and just that type of bell. Other bells would not be followed by food, eventually leading to….
- **Respondent Extinction** – When the CS is no longer paired with the UCS. The sound of a school bell ringing (new CS that was generalized) is not followed by food (UCS), and so eventually the dog stops salivating (the CR).
- **Spontaneous recovery** – When the CS elicits the CR after extinction has occurred. Eventually, the school bell will ring making the dog salivate. If no food comes, the behavior will not continue on. If food comes, the salivation response will be re-established.

2.3.2.3. 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. He 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 2.9. 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, or are given, 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 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. 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. NR can either result in current escape behavior or future avoidance behavior. What does this mean? *Escape* occurs when we are presently experiencing an aversive event and want it to end. We make a behavior and if the aversive event, like the headache, goes away, we will repeat the taking of Tylenol in the future. This future action is an *avoidance* event. We might start to feel a headache coming on and run to take Tylenol right away. By doing so we have removed the possibility of the aversive event occurring and this behavior demonstrates that learning has occurred.
- **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.

The type of reinforcer or punisher we use is important. Some are naturally occurring while some need to be learned. We describe these as primary and secondary reinforcers and punishers. *Primary* refers to reinforcers and punishers that have their effect without having to be learned. Food, water, temperature, and sex, for instance, are primary reinforcers while extreme cold or hot or a punch on the arm are inherently punishing. A story will illustrate the latter. When I was about 8 years old I would walk up the street in my neighborhood saying, "I'm Chicken Little and you can't hurt me." Most ignored me but some gave me the attention I was seeking, a positive reinforcer. So I kept doing it and doing it until one day, another kid was tired of hearing about my other identity and punched me in the face. The pain was enough that I never walked up and down the street echoing my identity crisis for all to hear. This was a positive punisher and did not have to be learned. That was definitely not one of my finer moments in life.

Secondary or conditioned reinforcers and punishers are not inherently reinforcing or punishing, but must be learned. An example was the attention I received for saying I was Chicken Little. Over time I learned that attention was good. Other examples of secondary reinforcers include praise, a smile, getting money for working or earning good grades, stickers on a board, points, getting to go out dancing, and getting out of an exam if you are doing well in a class. Examples of secondary punishers include a ticket for speeding, losing television or video game privileges, being ridiculed, or a fee for paying your rent or credit card bill late. Really, the sky is the limit with reinforcers in particular.

In operant conditioning, the rule for determining when and how often we will reinforce a desired behavior is called the **reinforcement schedule.** Reinforcement can either occur *continuously* meaning every time the desired behavior is made the person or animal will receive some reinforcer, or *intermittently/partially* meaning reinforcement does not occur with every behavior. Our focus will be on partial/intermittent reinforcement.

Figure 2.10. Key Components of Reinforcement Schedules

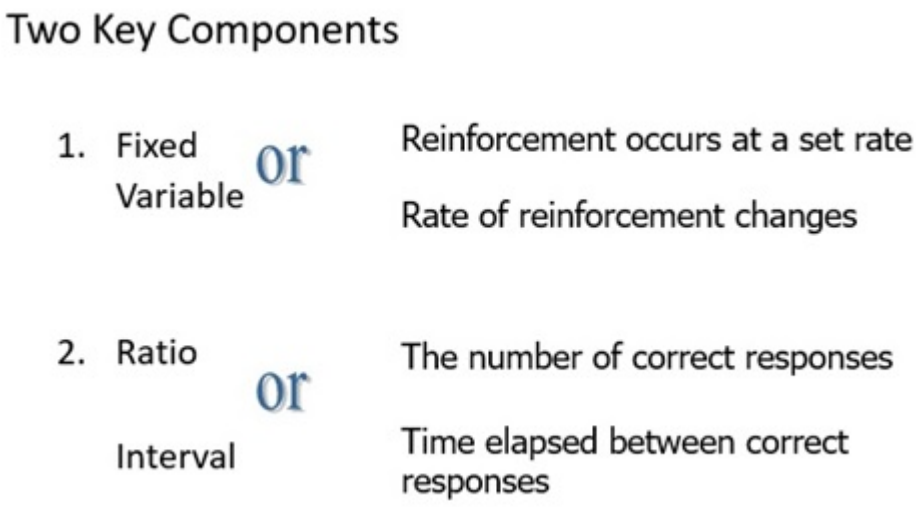


Figure 2.10 shows that that are two main components that make up a reinforcement schedule – when you will reinforce and what is being reinforced. In the case of when, it will be either fixed or at a set rate, or variable and at a rate that changes. In terms of what is being reinforced, we will either reinforce responses or time. These two components pair up as follows:

- **Fixed Ratio schedule (FR)** – With this schedule, we reinforce some set number of responses. For instance, every twenty problems (fixed) a student gets correct (ratio), the teacher gives him an extra credit point. A specific behavior is being reinforced – getting problems correct. Note that if we reinforce each occurrence of the behavior, the definition of continuous reinforcement, we could also describe this as a FR1 schedule. The number indicates how many responses have to be made and in this case it is one.
- **Variable Ratio schedule (VR)** – We might decide to reinforce some varying number of responses such as if the teacher gives him an extra credit point after finishing between 40 and 50 correct problems. This is useful after the student is obviously learning the material and does not need regular reinforcement. Also, since the schedule changes, the student will keep responding in the absence of reinforcement.
- **Fixed Interval schedule (FI)** – With a FI schedule, you will reinforce after some set amount of time. Let's say a company wanted to hire someone to sell their product. To attract someone, they could offer to pay them $10 an hour 40 hours a week and give this money every two weeks. Crazy idea but it could work. J Saying the person will be paid *every* indicates fixed, and *two weeks* is time or interval. So, FI.
- **Variable Interval schedule (VI)** – Finally, you could reinforce someone at some changing amount of time. Maybe they receive payment on Friday one week, then three weeks later on Monday, then two days later on Wednesday, then eight days later on Thursday. Etc. This could work, right? Not for a job but maybe we could say we are reinforced on a VI schedule if we are.

Finally, four properties of operant conditioning – extinction, spontaneous recovery, stimulus generalization, and stimulus discrimination – are important. These are the same four discussed under respondent conditioning. First, **extinction** is when something that we do, say, think/feel has not been reinforced for some time. As you might expect, the behavior will begin to weaken and eventually stop when this occurs. Does extinction just occur as soon as the anticipated reinforcer is not there? The answer is yes and no, depending on whether we are talking about continuous or partial reinforcement. With which type of reinforcement would you expect a person to stop responding to immediately if reinforcement is not there?

Do you suppose continuous? Or partial?

The answer is continuous. If a person is used to receiving reinforcement every time the correct behavior is made and then suddenly no reinforcer is delivered, he or she will cease the response immediately. Obviously then, with partial, a response continues being made for a while. Why is this? The person may think the schedule has simply changed. 'Maybe I am not paid weekly now. Maybe it changed to biweekly and I missed the email.' Due to this we say that intermittent or partial reinforcement shows *resistance to extinction*, meaning the behavior does weaken, but gradually.

As you might expect, if reinforcement "mistakenly" occurs after extinction has started, the behavior will re-emerge. Consider your parents for a minute. To stop some undesirable behavior you made in the past surely they took away some privilege. I bet the bad behavior ended too. But did you ever go to your grandparent's house and grandma or grandpa, or worse, BOTH….. took pity on you and let you play your video games for an hour or two (or something equivalent)? I know my grandmother used to. What happened to that bad behavior that had disappeared? Did it start again and your parents could not figure out why? Don't worry. Someday your parents will get you back and do the same thing with your

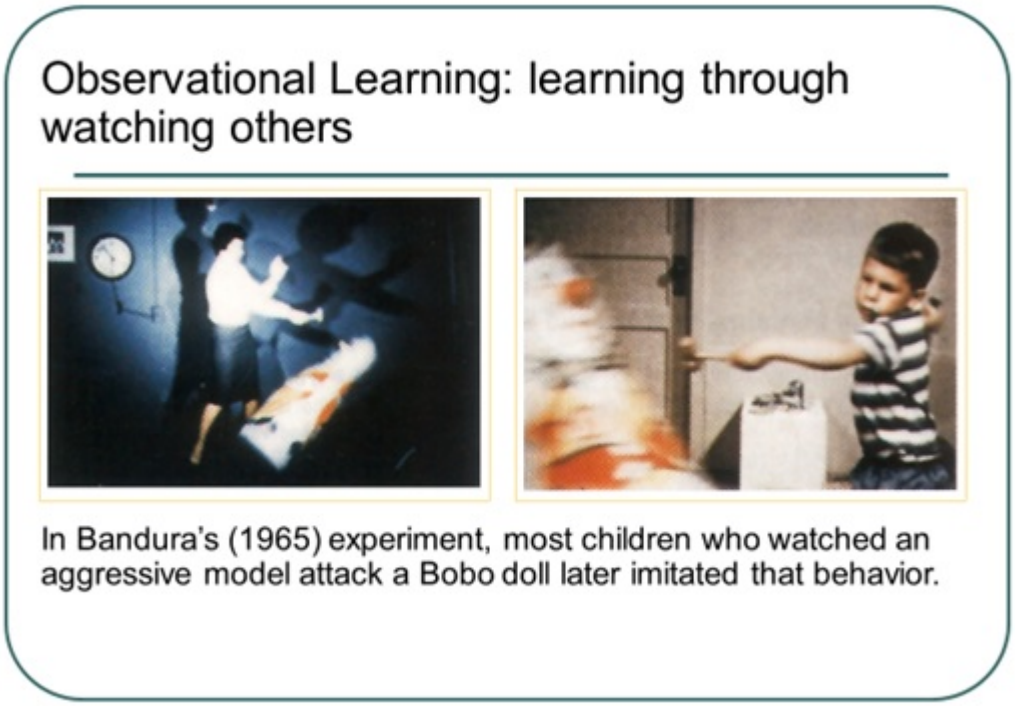
kid(s). J

Second, you might have wondered if the person or animal will try to make the response again in the future even though it stopped being reinforced in the past. The answer is yes and one of two outcomes is possible. First, the response is made and nothing happens. In this case extinction continues. Second, the response is made and a reinforcer is delivered. The response re-emerges. Consider a rat that has been trained to push a lever to receive a food pellet. If we stop delivering the food pellets, in time, the rat will stop pushing the lever. The rat will push the lever again sometime in the future and if food is delivered, the behavior spontaneously recovers. Hence why this phenomenon is called **spontaneous recovery.**

2.3.2.4. 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 unlike enactive. As you can learn desirable behaviors such as watching how your father bags groceries at the grocery store (I did this and still bag the same way today) 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 father seems genuinely happy and pleased with himself after bagging groceries his way, 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 the pivotal research on observational learning and you likely already know all about it. Check out Figure 2.11 to see if you do. In Bandura's experiment, children were first brought into a room to watch a video of an adult playing nicely or aggressively with a Bobo doll. This was a model. Next, the children are placed in a room with a lot of toys in it. In the room is a highly prized toy but they are told they cannot play with it. All other toys are fine and a Bobo doll is in the room. Children who watched the aggressive model behaved aggressively with the Bobo doll while those who saw the nice model, played nice. Both groups were frustrated when deprived of the coveted toy.

Figure 2.11. Bandura's Classic 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 subjects observe a model in a situation that usually causes them some anxiety. By seeing the model interact nicely with the fear evoking stimulus, their fear should subside. This form of behavior therapy is widely used in clinical, business, 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. In the business setting, a model or trainer demonstrates how to use a computer program or run a register for a new employee.

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. 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.

2.3.2.5. Evaluating the behavioral model. Within the context of abnormal behavior or psychopathology, the behavioral perspective is useful because it says that maladaptive behavior occurs when learning goes awry. The good thing is that what is learned can be unlearned or relearned and **behavior modification** is 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 strategies arise from all three learning models. In terms of operant conditioning, strategies include antecedent

manipulations, prompts, punishment procedures, differential reinforcement, habit reversal, shaping, and programming. Flooding and desensitization are typical respondent conditioning procedures used with phobias and modeling arises from social learning theory/observational learning. 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 and with the rise of cognitive psychology. This lead to a cognitive-behavioral perspective which combines concepts from the behavioral and cognitive models, the latter discussed in the next section.

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. This criticism would be true of behaviorism's first two stages, though sociobehaviorism steered away from this proposition and even fought against any mechanistic leanings of behaviorists.

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 Souza, 2016) and limitedly with the treatment of depression when co-morbid with OCD (Masoumeh and Lancy, 2016).

2.3.3. The Cognitive Model

2.3.3.1. What is it? As noted earlier, the idea of people being machines, called **mechanism**, 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, show that people can create their own problems by how they come to interpret events experienced in the world around them. How so?

2.3.3.2. Schemas and cognitive errors. First, consider the topic of **social cognition** or the process of collecting and assessing information about others. So what do we do with this information? Once *collected* or sensed (**sensation** is the cognitive process of detecting the physical energy given off or emitted by physical objects), the information is sent to the brain. This occurs through the neural impulse. Once in the brain, it is processed and interpreted. This is where *assessing information about others* comes in and involves the cognitive process of **perception,** or adding meaning to raw sensory

data. We take the information just detected and use it to assign people to **categories**, or groups. For each category, we have a **schema**, or a set of beliefs and expectations about a group of people, presumed to apply to all members of the group, and based on experience.

Can our schemas lead us astray, or be false? Consider where students sit in a class. It is generally understood that the students who sit in the front of the class are the overachievers and want to earn an A in the class. Those who sit in the back of the room are underachievers and really don't care. Right? Where do you sit in class, if you are on a physical campus and not an online student? Is this correct? What about other students in the class that you know? What if you found out that a friend who sits in the front row is a C student but sits there because he cannot see the screen or board, even with corrective lenses? What about your friend or acquaintance in the back? This person is an A student but does not like being right under the nose of the professor, especially if he/she has a tendency to spit when lecturing. The person in the back could also be shy and prefer sitting there so that she does not need to chat with others as much, or is easily distracted and so sits in the back so that all stimuli are in front of her. Again, your schema about front row and back row students is incorrect and causes you to make certain assumptions about these individuals. This might even affect how you interact with them. Would you want notes from the student in the front or back of the class?

2.3.3.3. Attributions and cognitive errors. Second, consider the very interesting topic from social psychology of **attribution theory**, or 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. Like schemas, the attributions we make can lead us astray. How so? The **fundamental attribution error** occurs when we automatically assume a dispositional reason for another person's actions and ignore situational factors. In other words, we assume the person who cut us off is an idiot (dispositional) and do not consider that maybe someone in the car is severely injured and this person is rushing them to the hospital (situational). Then there is the **self-serving bias** which is when we attribute our success to our own efforts (dispositional) and our failures to outside causes (situational). Obviously, our attribution in these two cases is in error but still, it comes to affect how we see the world and our subjective well-being.

2.3.3.4. Maladaptive cognitions. Irrational 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.
- Mind Reading – Assuming others know what you are thinking without any evidence.
- What if? – Asking yourself what if? Something happens without being satisfied by any of the answers.
- Blaming – You focus on someone else as the source of your negative feelings and do not take 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

2.3.3.5. Cognitive therapies. According to the National Alliance on Mental Illness (NAMI), **cognitive behavioral therapy** "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, you can use **cognitive restructuring**, also called rational restructuring, in which maladaptive cognitions are replaced 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, help the client stop thinking these thoughts and 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 over outcomes are possible.
2. Track the accuracy of the thought as if you believe focusing on a problem generates a solution. 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 by figuring out if you really do not have time to go to the gym. Recording what you do each day and then look at open times of the day. Add them up and see if you make some minor, or major, adjustments to your schedule if you can allow yourself an hour to get in valuable 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 teaches social skills, communication, and assertiveness through direct instruction, role playing, and modeling. For social skills, identify appropriate social behavior such as making eye contact, saying no to a request, or starting up a conversation with a stranger and whether the client is inhibited from

making this behavior due to anxiety. For communication, determine if the problem is with speaking, listening, or both and then develop a plan for use in various interpersonal situations. Finally, assertiveness training aids the client protect their rights and obtain what they want from others. Those who are not assertive are often overly passive and never get what they want, or are overly aggressive and only get what they want. Treatment starts with determining situations in which assertiveness is lacking and coming up with 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. Techniques might include weighing the pros of fighting uncertainty against the cons of doing so. The cons should outweigh the pros and help you to end the struggle and accept what is unknown. Chances are you are already accepting the unknown in some areas of life and identifying these can help you to see why it is helpful in these areas, and how you can also think like this in more difficult areas. Finally, does uncertainty unnecessarily lead to a negative end? We may think so but review of the evidence for and against this statement will show that it does not and reduce how threatening it seems.

2.3.3.6. Evaluating the cognitive model. The cognitive model made up for an obvious deficit in the behavioral model – overlooking the role cognitive processes play in our thoughts, feelings, and behaviors. Right before his death, Skinner (1990) reminded psychologists that the only thing we can truly know and study was the observable. Cognitive processes cannot be empirically and reliably measured and so should be ignored. Is there merit to this view? **Social desirability** states that sometimes participants 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 anything about controversial matters? 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, accepts 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*. 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 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. 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 substance abuse, "excessive anxiety, apathy, alienation, nihilism, avoidance, shame, addiction, despair, depression, guilt, anger, rage, resentment, embitterment, purposelessness, psychosis, and violence. They also focus on life-enhancing experiences like relationships, love, caring, commitment, courage, creativity, power, will, presence, spirituality, individuation, self-actualization, authenticity, acceptance, transcendence, and awe" (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 so 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 the perspectives tend to work best with people suffering from adjustment issues and not as well with severe mental illness. The perspectives do offer hope to people suffering 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**. How so? We will explore a few of these factors in this section.

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 then leads 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., 2012) and group differences for personality disorders have been found between African and European Americans (Ryder, Sunohara, and Kirmayer, 2015).

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 being more muscular.
- OCD has an earlier age of onset in girls than boys, with most people being diagnosed by age 19.
- Females 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. Interestingly younger adults are more likely to develop SAD than older adults.

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 experiences 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 lead to anxiety disorders.
- Trauma, stress, and other extreme stressors are predictive of depression.
- Malnutrition before birth, exposure to viruses, and other psychosocial factors are potential causes of schizophrenia.
- SAD occurs with greater frequency for those living far north or south from 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.

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 illness. 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 this can lead to disordered behavior (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). These therapies have proven efficacy for the treatment of depression (Kalibatseva & Leong, 2014) and schizophrenia (Naeem et al., 2015).

2.4.5. Evaluation of the Model

The sociocultural model has contributed greatly to our understanding of the nuances of mental illness diagnosis, prognosis, course, and treatment for other races, cultures, genders, ethnicities. In Module 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 socio-cultural model suffers from issues with the findings being difficult to interpret and not allowing for the establishment of causal relationships; a reliance on more qualitative data gathered from case studies and ethnographic analyses (one such example is Zafra, 2016), and an inability to make predictions about abnormal behavior for individuals.

Module Recap

In Module 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 illness and three treatment options were given. In terms of psychological perspectives, Freud’s psychodynamic theory; learning to include the work of Watson, Skinner, and Bandura and Rotter; the cognitive model; and the humanistic and existential perspectives were discussed. Finally, the sociocultural model indicated the role of socioeconomic, gender, environmental, and multicultural factors on abnormal behavior.

Module 3: Clinical Assessment, Diagnosis, and Treatment

Module Overview

Module 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. As this is the last module in Part 1, please make sure you are preparing for your first exam.

Module Outline

- 3.1. Clinical Assessment of Abnormal Behavior
- 3.2. Diagnosing and Classifying Abnormal Behavior
- 3.3. Treatment of Mental Disorders – An Overview

Module Learning Outcomes

- 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.

3.1. Clinical Assessment of Abnormal Behavior

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 seven methods of assessment.

3.1.1. What is Clinical Assessment?

In order for a mental health professional to be able to effectively help treat a client and know that the treatment selected actually worked (or is working), he/she first must engage in the **clinical**

assessment of the client, or 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 in terms of environmental stressors that are faced, and cultural factors particular to them such as the language that is spoken or ethnicity. Clinical assessment is not just conducted in 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 a treatment is needed, our second reason to engage in clinical assessment will be to determine what treatment will work best. As you will see later in this module, there are numerous approaches to treatment. These include Behavior Therapy, Cognitive and Cognitive-Behavioral Therapy (CBT), Humanistic-Experiential Therapies, Psychodynamic Therapies, Couples and Family Therapy, and biological treatments (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 figure this out. Finally, we need to know if the treatment we employed worked. This will involve measuring before any treatment is used and then measuring the behavior while the treatment is in place. We will even want to measure after the treatment ends to make sure symptoms of the disorder 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 that is agreed upon happens more so in the middle, and then making sure the treatment produces the desirable 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 suffering from a mental disorder. If one mental health professional says the person suffers from major depressive disorder and another says the issue is borderline personality disorder, then there is an issue with the assessment tool being used (in this case the DSM and more on that in a bit). Ensuring that two different raters are consistent in their assessment of patients 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 MMPI on Tuesday and then the same test on Friday. 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. What does identical mean? The score at test and the score at retest are 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 comparable to 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 observation, psychological tests, neurological tests, the clinical interview, and a few others in this section.

3.1.3.1. Observation. In Section 1.5.2.1 we talked about two types of observation – naturalistic, or observing the person or animal in their environment, and laboratory, or observing the organism in a more controlled or artificial setting where the experimenter can use sophisticated equipment and videotape the session to examine it at a later time. One-way mirrors can also be used. A limitation of this method is that the process of recording a behavior causes 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 the football game and not at home. This is a form of validity is called **cross-sectional validity.** We also need our rates to observe and record behavior in the same way or to have high inter-rater reliability.

3.1.3.2. 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 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. 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.

3.1.3.3. 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 in paper or oral fashion. **Projective tests c**onsist 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 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. From the response, the clinician gains perspective on the patient's worries, needs, emotions, conflicts and the individual always connects with one of the people on the card. 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 do not ever completely know the individual.

3.1.3.4. 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 chemistry and begins by injecting the patient with a radionuclide which collects in the brain and then having them 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. It can detect 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 and then combined and is used to diagnose brain damage cause by head injuries or brain tumors.

3.1.3.5. 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.

3.1.3.6. Behavioral assessment. Within the realm of behavior modification and applied behavior analysis, we talk about what is called **behavioral assessment** which simply is the measurement of a target behavior. The **target behavior** is 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. During behavioral assessment we learn about the ABCs of behavior in which **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 which is called self-modification and the person does their own measuring and recording of the ABCs which is called **self-monitoring.** In the context of psychopathology, behavior modification can be useful in treating phobias, reducing habit disorders, and ridding the person of maladaptive cognitions.

3.1.3.7. Intelligence tests. Intelligence testing is 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. An example is the *Stanford-Binet Intelligence test* which is used to assess fluid reasoning, knowledge, quantitative reasoning, visual-spatial processing and working memory. Intelligence tests have been criticized for not predicting future behaviors such as achievement and reflecting social or cultural factors/biases and not actual intelligence. Also, can we really assess intelligence through one dimension, or are there multiple dimensions?

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 determine 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, 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** 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 suffering from 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 suffer from a schizophrenia spectrum disorder while the latter suffer a NCD or neurocognitive disorder. 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 and 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). The DSM evolved through four major editions after World War II into a diagnostic classification system to be used psychiatrists and physicians, but also other mental health professionals. The Herculean task of revising the DSM 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 work group 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 “both within and across the domains of proposed disorders” (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 – *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 sleep walking 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. Somatic symptom disorder has a specifier for severity, if with predominant pain, and/or if persistent. 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. It 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.

3.2.2.3. 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	Module
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	Not covered
Schizophrenia Spectrum	Disorders characterized by one or more of the following: delusions, hallucinations, disorganized thinking and speech, disorganized motor behavior, and negative symptoms	12
Bipolar and Related	Characterized by mania or hypomania and possibly depressed mood; includes Bipolar I and II, cyclothymic disorder	4
Depressive	Characterized by sad, empty, or irritable mood, as well as somatic and cognitive changes that affect functioning; includes major depressive and persistent depressive disorders	4
Anxiety	Characterized by excessive fear and anxiety and related behavioral disturbances; Includes phobias, separation anxiety, panic attack, generalized anxiety disorder	7
Obsessive-Compulsive	Characterized by obsessions and compulsions and includes OCD, hoarding, and body dysmorphic disorders	9
Trauma- and Stressor-Related	Characterized by exposure to a traumatic or stressful event; PTSD, acute stress disorder, and adjustment disorders	5
Dissociative	Characterized by a disruption or disturbance in memory, identity, emotion, perception, or behavior; dissociative identity disorder, dissociative amnesia, and depersonalization/derealization disorder	6
Somatic Symptom	Characterized by prominent somatic symptoms to include illness anxiety disorder somatic symptom disorder, and conversion disorder	8
Feeding and Eating	Characterized by a persistent disturbance of eating or eating-related behavior to include bingeing and purging	10
Elimination	Characterized by the inappropriate elimination of urine or feces; usually first diagnosed in childhood or adolescence	Not covered
Sleep-Wake	Characterized by sleep-wake complaints about the quality, timing, and amount of sleep; includes insomnia, sleep terrors, narcolepsy, and sleep apnea	Not covered
Sexual Dysfunctions	Characterized by sexual difficulties and include premature ejaculation, female orgasmic disorder, and erectile disorder	Not covered
Gender Dysphoria	Characterized by distress associated with the incongruity between one's experienced or expressed gender and the gender assigned at birth	Not covered
Disruptive, Impulse-Control, Conduct	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.	Not covered
Substance-Related and Addictive	Characterized by the continued use of a substance despite significant problems related to its use	11
Neurocognitive	Characterized by a decline in cognitive functioning over time and the NCD has not been present since birth or early in life	14
Personality	Characterized by a pattern of stable traits which are inflexible, pervasive, and leads to distress or impairment	13
Paraphilic	Characterized by recurrent and intense sexual fantasies that can cause harm to the individual or others; includes exhibitionism, voyeurism, and sexual sadism	Not covered

3.2.3. The ICD-10

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

3.2.4. Harmonization of DSM-5 and ICD-11

As noted earlier, the ICD-11 is currently in development with an expected publication date of 2018. According to the DSM-5, there is an effort to harmonize the two classification systems so that there can be 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 issue of DSM-IV and ICD-10 diagnoses not agreeing (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 least some point in their lives" and that though the signs you need to seek help are obvious at times, we often try "to sustain your 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 you have stopped doing the things you 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/where-science-meets-the-steps/201303/5-signs-its-time-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 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. So what exactly is psychotherapy? APA states (article quoted below can be found at: http://www.apa.org/helpcenter/understanding-psychotherapy.aspx) 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.

3.3.1.4. 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 to see the mental health professional. 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 you takes depends on the reason for the visit.

In terms of what you should 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.

3.3.1.5. 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 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.

3.3.1.6. 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/understanding-psychotherapy.aspx

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Module Recap

That's it. With the conclusion of Module 3 you now have the necessary foundation to understand each of the groups of disorders we discuss beginning in Module 4 and through Module 14. In Module 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 observation, 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-centered relationship, and how well psychotherapy works. We discussed some of the specific therapies in Module 2 but will cover others throughout this book and in terms of the disorders they are used to treat. You should be getting ready for your first exam in the class now. We wish you luck and to prepare adequately, please use the learning objectives in each section. Also, ask your instructor if you have questions.

II

Part II. Mental Disorders – Block 1

Part II. Mental Disorders – Block 1

Module 4: Mood Disorders

Module Overview

In Module 4, 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 Dysthymia), Bipolar I disorder, Bipolar II disorder, and Cyclothymic disorder. Throughout our discussion, we will also cover major depressive, manic, and hypomanic episodes. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 4.1. Clinical Presentation – Depressive Disorders
- 4.2. Clinical Presentation – Bipolar Disorders
- 4.3. Epidemiology
- 4.4. Comorbidity
- 4.5. Etiology
- 4.6. Treatment

Module Learning Outcomes

- Describe how depressive disorders present.
- Describe how bipolar disorders present.
- 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.

4.1. Clinical Presentation – Depressive Disorders

Section Learning Objectives

- Identify and describe the two types of depressive disorders.
- Classify symptoms of depression.

Within mood disorders are two distinct groups- individuals with depressive disorders and individuals

with bipolar disorders. The key difference between the two groups is that those in the depressive disorder category *only* experience symptoms of depression, while those in the bipolar disorder category have periods of mania/hypomania that alternate with periods of depression.

The two most common types of depressive disorders are **Major Depressive Disorder** and **Persistent Depressive Disorder**. Persistent Depressive Disorder, previously known as Dysthymia, is thought to be a more chronic, less severe depression. Like previously mentioned, in order to be diagnosed with either major depressive disorder or persistent depressive disorder, the individual must *never* have had a manic or hypomanic episode.

Symptoms of depression can generally be categorized into four categories to include mood, behavioral, cognitive, and physical 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 for most of the day and/or feelings of anhedonia, which is the loss of interest in previously interesting activities.

Behavioral. Behavioral issues such as decreased physical activity and reduce productivity- both at home and at work- are often observed in individuals with depression. This is typically where a disruption in daily functioning is observed as individuals with depressive disorders are unable to maintain their social interactions and employment responsibilities.

Cognitive. It should not come as a surprise that there is a serious disruption 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. They often feel worthless, which creatives a negative feedback loop to their overall depressed mood. Individuals with depressive disorder also report difficulty concentrating on tasks, as they are easily distracted from outside stimuli. 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). Finally, thoughts of suicide and self-harm do occasionally occur in those with depressive disorders. This is covered in more detail in the epidemiology section.

Physical. Changes in sleep patterns are often common in those experiencing depression. Excessive sleeping is typically reported, often impacting an individual's daily functioning (i.e. meeting up with friends, getting to work on time). There can also be a report of insomnia. This can occur at various points throughout the night- either difficulty falling asleep, staying asleep all night, or even waking too early and not being able to fall back asleep before having to wake for the day. Although it is unclear whether symptoms of fatigue or loss of energy are related to insomnia issues, the fact that those experiencing hypersomnia also report symptoms of fatigue suggest that these symptoms are a component of the disorder rather than a secondary symptom of sleep disturbance.

Additional physical symptoms such as change in weight or eating behaviors are also observed. 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.

Finally, psychomotor agitation or retardation, which is the purposeless or slowed physical movement of the body (i.e. pacing around a room, tapping toes, restlessness etc.) is also reported in individuals with depressive disorders.

According to the DSM-5 (APA, 2013), major depressive disorder and persistent depressive disorder are diagnosed according to the listed criteria. Although symptoms for both are nearly identical, the time frame of symptoms are significantly different, with symptoms presenting for a 2-week period for major depressive disorder and symptoms present for majority of 2-years for persistent depressive disorder.

4.2. Clinical Presentation – Bipolar Disorders

Section Learning Objectives

- Distinguish the forms bipolar disorder takes.
- Describe manic episode.
- Define cyclothymic disorder.

According to the DSM-5 (APA, 2013), there are two types of Bipolar Disorder- Bipolar I and Bipolar II. A diagnosis of Bipolar I Disorder is made when there is at least one manic episode. This manic episode can be preceded by or followed by a hypomanic or major depressive episode. A diagnosis of Bipolar II Disorder is made when there is a current or history of a **hypomanic episode** *and* a current or past major depressive episode. In more simpler words, if an individual has ever experienced a manic episode, they qualify for a Bipolar I diagnosis; however, if the criteria has only been met for a hypomanic episode, the individual qualifies for a Bipolar II diagnosis.

So, what defines a **manic episode**? The key feature of a manic episode is a specific period of time in which an individual experiences abnormally, persistently, expansive or irritable mood for nearly all day, every day, for at least one week (APA, 2013). Additionally, the individual will display increased activity or energy during this same time. 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 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 laws, or even at times think they are God.

Despite their increased activity level, individuals experiencing a manic episode also require 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 story telling. 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 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 and flights of ideas. Because of these rapid thoughts, speech may become disorganized or incoherent.

It should be noted that there are a subclass of individuals who experience periods of hypomanic symptoms and *mild* depressive symptoms (i.e. do not fully meet criteria for a depressive episode). These individuals are diagnosed with **cyclothymic disorder** (APA, 2013). Presentation of these symptoms occur for two or more years, and are typically interrupted by periods of normal moods. While only a small percentage of the population develop cyclothymic disorder, it can eventually progress into bipolar I or bipolar II disorder (Zeschel et al., 2015).

Like we stated before, Bipolar I and Bipolar II disorder also require the presence of a Major Depressive Episode. The Major Depressive Episode can occur before or after the manic/hypomanic episode, as the two types of episodes will alternate or "cycle" throughout one's life. To review the criteria for a Major Depressive Episode, please see above under Major Depressive Disorder.

4.3. Epidemiology

Section Learning Objectives

- Describe the epidemiology of depressive disorders.
- Describe the epidemiology of bipolar disorders.
- Describe the epidemiology of suicidality.

4.3.1. Depressive Disorders

According to the DSM-5 (APA, 2013) the prevalence rate for major depression is approximately 7% within the United States. The prevalence rate for persistent depressive disorder is much lower, with a 0.5% rate among adults in the United states. 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 males. The estimated lifetime prevalence for major depressive disorder in women is 21.3% compared to 12.7% in men (Nolen-Hoeksema, 2001).

4.3.2. Bipolar Disorders

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% and 0.8% in the United States for bipolar I and bipolar II, respectively (APA, 2013). As for gender differences, there are no apparent differences in the frequency of men and women diagnosed with bipolar I; however, bipolar II appears to be more common in women, with approximately 80-90% of individuals with rapid-cycling episodes being women (Bauer & Pfenning, 2005). Women are also more likely to experience rapid cycling between manic/hypomanic episodes and depressive episodes.

4.3.3. Suicidality

Suicidality in depressive disorders, particularly bipolar disorder, is much higher than the general public. In depressive disorders, males and those with a past history of suicide attempts/threats are most at risk for attempting suicide. Individuals with bipolar disorder are approximately 15 times greater than the general population to attempt suicide. Prevalence rates of suicide attempts in bipolar patients is estimated to be 33%. Furthermore, bipolar disorder may account for one-quarter of all completed suicides (APA, 2013).

4.4. Comorbidity

Section Learning Objectives

- Describe the comorbidity of depressive disorders.
- Describe the comorbidity of bipoloar disorders.

4.4.1. Depressive Disorders

As I’m sure it does not come as a surprise, studies exploring depression symptoms among the general population show a substantial pattern of comorbidity between depression and other mental disorders, particularly substance use disorders (Kessler, Berglund, et al., 2003). In fact, nearly three-fourths of participants with lifetime MDD in a large scale research study 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. Majority of the studies have identified most depression cases occur secondly to another mental health disorder meaning that the onset of depression is a direct result to the onset of another disorder (Gotlib & Hammen, 2009).

4.4.2. Bipolar Disorders

Bipolar disorder also has a high comorbidity rate with other metal disorders, particularly anxiety

disorders and any 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 meeting criteria for three or more co-occurring mental disorders (APA, 2013).

4.5. Etiology

Section Learning Objectives

- Describe the biological causes of mood disorders.
- Describe the cognitive causes of mood disorders.
- Describe the behavioral causes of mood disorders.
- Describe the sociocultural causes of mood disorders.

4.5.1. Biological

Research throughout the years continues to provide evidence that depressive disorders have some biological cause. While it does not explain every depressive case, it is safe to say that some individuals may at least have a predisposition to develop a depressive disorder. Among the biological factors are genetic factors, biochemical factors and brain structure.

4.5.1.1. Genetics. Like with any disorder, researchers often explore the prevalence rate of depressive disorders among family members, in efforts to determine if there is some genetic component, whether it be a direct link or a predisposition. 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. Researchers support this with regards to depressive disorders as there is nearly a 30 percent increase in relatives diagnosed with depression, compared to 10 percent of the general population (Levinson & Nichols, 2014). Similarly, there is also an elevated prevalence among first-degree relatives for both bipolar I and bipolar II disorders as well.

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 scaled 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%. Despite the fraternal twin rate still being higher than that of a firs-degree relative, 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 as a cause to developing a depressive disorder. While much of the

research is speculation 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).

Bipolar disorders share a similar genetic predisposition to developing the disorder. Twin studies within bipolar disorder yield concordance rates for identical twins at as high as 72%, and 5-15% for fraternal twins, siblings, and other close relatives. 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).

4.5.1.2. Biochemical. As you will read in the treatment section, there is strong evidence of a biochemical deficit in depression and bipolar disorders. More specifically, low activity levels of norepinephrine and serotonin, have long been documented as contributing factors to developing depressive disorders. This was actually discovered accidently in the 1950's when MAOI's were given to tuberculosis patients, and miraculously, their depressive 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 effected development of depressive features, they did provide insight as to *what* neurotransmitters were involved in this system. 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 on other neurotransmitters (Ding et al., 2014).

Due to the close nature of depression and bipolar disorder, researchers initially believed that both norepinephrine and serotonin were both implicated in the development of bipolar disorder; however, the idea was that there was a drastic *increase* in serotonin during mania episodes. Unfortunately, research actually supports the opposite. It is believed that mania episodes may in fact be explained by low levels of serotonin and *high levels* of norepinephrine (Soreff & McInnes, 2014). Additional research with this area is needed to conclusively determine exactly what is responsible for the manic episodes within bipolar disorder.

4.5.1.3. Endocrine system. As you may know, 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 hormone released as a stress response, in the development of depression (Owens et al, 2014). Additionally, **melatonin**, a hormone released when it is dark outside to assist with the transition to sleep, may also be related to depressive symptoms, particularly during the winter months (seasonal affective disorder).

4.5.1.4. Brain anatomy. Seeing as neurotransmitters are involved in depressive disorders, it should not be a surprise that the 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 has been linked with depressive symptoms. Similarly, a smaller hippocampus, and consequently, fewer number of neurons, have also been linked to depressive symptoms. Finally, heightened activity and blood flow in the amygdala, the brain area responsible for our fight or flight emotions, is also consistently found in individuals with depressive symptoms.

There are a number of structural abnormalities in individuals with bipolar disorder; however, what or why these structures are abnormal is yet to be determined. Researchers continue to focus on areas of basal ganglia and cerebellum, which appear to be much smaller in individuals with bipolar disorder as opposed to the general public. Additionally, there appears to be a decrease in brain activity in regions associated with regulating emotions, as well as increase in brain activity among structures related to emotional responsiveness (Houenou et al., 2011). Additional research is still needed to determine exactly how each of these brain structures may be implicated in the development of bipolar disorder.

4.5.2. Cognitive

The cognitive model, arguably the most conclusive model with regards to depressive disorders, focuses on the negative thoughts and perceptions of an individual. One theory often equated with the cognitive model of depression is **learned helplessness.** Coined by Martin Seligman (1975), learned helplessness was developed based on his 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 shock, their learned helplessness carried over and they continued to believe they were unable to escape the shock.

This study has been linked to humans through the research in **attributional style** (Nolen-Hoeksema, Girgus & Seligman, 1992). There are two types of attributional style- positive and negative. A negative attributional style focuses on the *internal, stable* and *global* influence of daily lives, whereas a positive attributional style focuses on the *external, unstable,* and *specific* influence 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 conclude that it is *their* fault (internal), bad things *always* happen to them (stable), and bad things happen *all* day to them. Unfortunately, this maladaptive thinking style often takes over an individual's daily view, 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 thoughts- all of which combine to explain the cognitive model of depressive disorders.

Maladaptive attitudes, or negative attitudes about one self, others, and the world around them, are often present in those with depressive symptoms. 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. Individual's 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 their *experiences, themselves*, and their *futures.*

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 (see end of chapter). Among the most common are catastrophizing, 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 perceives this to be true.

Finally, **automatic thoughts**, or 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. 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 continuley supported Beck's maladaptive thoughts, attitudes, and errors in thinking as fundamental issues in those with 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.

4.5.3. Behavioral

The behavioral model explains depression as a result of 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 occurred in most people due to the reduced positive rewards in their life. Because they were not being positively rewarded, their constructive behaviors occurred 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 their exam despite studying for hours. Over time, the individual will reduce the amount of time they are studying, thus continuing to earn poor grades.

4.5.4. Sociocultural

In the sociocultural theory, the role of family and one's social environment play a strong role in the development of depressive disorders. There are two sociocultural views- the *family-social perspective* and the *multi-cultural perspective*.

4.5.4.1. Family-social perspective. Similar to that of the behavioral theory, the family-social perspective of depression suggests that depression is related to the unavailability of social support. This is often supported from research studies that show separated and divorced individuals are three times more likely to experience depressive symptoms than those that 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 family-social perspective can also be viewed in the opposite way, with stress and martial discord leading to increased rates of depression in one or both spouses (Nezlek et al., 2000). 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 division of work and discipline differences. 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 that other mothers to become depressed (Brown, 2002).

4.5.4.2. 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 hallmark in most societies, other symptoms may be more specific to one's nationality. More specifically, individuals from non-Western countries (China and other Asian countries) often focus on the physical symptoms of depression- tiredness, weakness, sleep issues, and 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 (American Psychiatric Association, 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 ethnical 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 lack of treatment opportunities as a possible explanation. According to Gonzalez and colleagues (2010), approximately 54 percent of depressed white American seek out treatment, compared to the 34 percent and 40 percent Hispanic and African Americans, respectively. The fact that there is such a large discrepancy in the use of treatment between non-white Americans and minority Americans suggests that these individuals are not receiving the effective treatment necessary to resolve the disorder, thus leaving them more vulnerable for repeated depressive episodes.

4.5.4.3. Gender differences. As previously discussed, there is a significant difference between gender and rates of depression, with women twice as likely to experience an episode of depression than man (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*- suggests that the difference between genders is due to clinician 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 will present with more traditionally "masculine" symptoms of anger or aggression. While this theory is often 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 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 fails 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, 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 their male counterparts do.

The final theory- *rumination theory*– suggests that women are more likely than men to ruminate, or intently focus, 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 depression symptoms (Hankin, 2009).

While there are many theories trying to explain the gender discrepancy in depressive episodes, 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 one of the most researched topic within depression, while simultaneously being the least understood phenomena in the clinical psychology world.

4.6. Treatment of Mood Disorders

Section Learning Objectives

- Describe treatment options for depressive disorders.
- Describe treatment options for bipolar disorders.
- Determine the efficacy of treatment options for depressive disorders.
- Determine the efficacy of treatment options for bipolar disorders.

4.6.1. Depressive Disorders

Given that Major Depressive Disorder is among the most frequent and debilitating psychiatric disorders, it should not be surprising that the research on this disorder 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 Major Depressive Disorder, there is minimal evidence to support one treatment modality

over the other; treatment is generally dictated by therapist competence, availability, and patient preference (Craighhead & Dunlop, 2014).

4.6.1.1. Psychopharmacology – Antidepressant medications. Antidepressants are often the most common first line attempt at treatment for MDD for a few reasons. Oftentimes an individual will present with symptoms to their primary caregiver (a medical doctor) who will prescribe them 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 fail to take prescription medication as indicated by their physician. Given the biological functions of neurotransmitters and their involvement in maintaining depressive symptoms, it makes sense that this is an effective type of treatment.

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

4.6.1.2. Psychopharmacology – 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, and reduced sex drive.

SSRI's improve depression symptoms by blocking the reuptake of norepinephrine and/or serotonin in presynaptic neurons, thus allowing more of these neurotransmitters to be available for postsynaptic neuron. While this is the general mechanism through which all SSRI's work, there are minor biological differences among different types of medications within the SSRI family. These minor differences are actually beneficial to patients in that there are a few treatment options to maximize medication benefits and minimize side effects.

4.6.1.3. Psychopharmacology – 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: three rings.

Tricyclic Antidepressants are similar to SSRIs in that they work by effecting the brain chemistry, altering the number of neurotransmitters available for neurons. More specifically, they block the absorption or reuptake of serotonin and norepinephrine, thus increasing their availability for post synaptic neurons. While effective, tricyclic antidepressants have been increasingly replaced by SSRIs due to their reduced side effects. However, tricyclic antidepressants have been show to be more effective in treating traditionally resistant depression and dysthymia.

While majority of the side effects are minimal- dry mouth, blurry vision, constipation, others can be serious- sexual dysfunction, tachycardia, cognitive and/or memory impairment, to name a few. Due to the potential impact on the heart, tricyclic antidepressants should not be used in cardiac patients as they have been shown to exacerbate cardiac arrhythmias (Roose & Spatz, 1999).

4.6.1.4. Psychopharmacology – Monoamine oxidase inhibitors (MAOIs). While the use of MAOI's were found serendipitously after it produced antidepressant effects in a tuberculosis patient in the early 1950's, it has been effective in treating depression in adults. Although they are still prescribed, they are not typically first line medications due to their safety concerns with hypertensive crises. Because of this, individuals on MAOI's 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 neurotransmitters norepinephrine, serotonin, and dopamine. MAOI's essentially prevent the monoamine oxidase (hence the name monoamine oxidase *inhibitors*) from removing these neurotransmitters, thus having an increase in these brain chemicals (Shulman, Herman & Walker, 2013). As previously discussed, norepinephrine, serotonin, and dopamine are all involved in the biological mechanisms of maintaining depressive symptoms.

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 patients 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.

4.6.1.5. 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 (and other disorders as well). 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, effect one another. It is believed that CBT can improve emotions in depressed patients by changing both cognitions (thoughts) and behaviors, which in return will improve mood. Common cognitive interventions with CBT include thought monitoring and recording, identifying cognitive errors, examining 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 four phases of treatment:

- **Phase 1: Increasing pleasurable activities.** Similar to behavioral activation (read below), the clinician encourages the patient to identify and engage in activities that are pleasurable to the individual. The clinician is able to help the patient during session to identify the activity, as well as help them plan out during the week when they will engage in that activity.
- **Phase 2: Challenging automatic thoughts.** During this stage, the clinician provides psychoeducation about the negative automatic thoughts that can maintain depressive symptoms. The patient will learn to identify these thoughts on their own during the week, and maintain a thought journal of these cognitions to review with the clinician in session.
- **Phase 3: Identifying negative thoughts.** Once the individual is consistently able to identify these negative thoughts on a daily basis, the clinician is able to help the patient identify *how* these

thoughts are maintaining their depressive symptoms. It is at this point that the patient begins to have direct insight as to how their cognitions contribute to their disorder.
- **Phase 4: Changing thoughts.** The final stage of treatment involves challenging the negative thoughts the patient has been identifying in the last two phases of treatment and replacing them with positive thoughts.

4.6.1.6. Psychotherapy – 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 colleages (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 patient by developing a list of pleasurable activities that they can engage in outside of treatment (i.e. going for a walk, going shopping, having dinner with a friend). Additionally, the clinician assists the patient in identifying their negative behaviors- crying, sleeping in, avoiding friends- and monitoring them so that do not impact the outcome of their pleasurable activities. Finally, the clinician works with the patient on effective social skills. The thought is 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.

4.6.1.7. Psychotherapy – 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 (Weissman, 1995). The treatment was created based off 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 depressive episodes compromise 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 *not* the patient'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 solving this crisis so the patient can improve their life situation while relieving depressive symptoms.

4.6.1.8. Multimodal treatment. While both pharmacological and psychological treatment alone is very effective in treating depression, a combination of the two treatments may offer additional benefits, particularly in the maintenance of wellness. Additionally, multimodal treatment options 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 done concurrently, treatments can be done sequentially, or treatments can be offered within stepped treatment (McGorry et al., 2010). With a stepped treatment, pharmacological therapy is often used initially to treat depressive symptoms. Once the patient 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 patients to their treatment preference may produce better outcomes than clinician driven treatment decisions.

4.6.2. Bipolar Disorder

4.6.2.1. Psychopharmacology. Unlike treatment for MDD, there is some controversy to the effective treatment of 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 antidepressants 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, the combination approach is believed to help treat both the 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 are often known to trigger a manic or hypomanic episode in bipolar patients. Because of this, the first line treatment option for Bipolar Disorder is mood stabilizers, particularly Lithium.

4.6.2.2. 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. Majority of psychological interventions are aimed at medication adherence, as many bipolar patients stop taking their mood stabilizers when they “feel better” (Advokat et al., 2014). Social skills training and problem-solving skills are also helpful techniques to address in the therapeutic setting as individuals with bipolar disorder often struggle in this area.

4.6.3. Outcome of Treatment

4.6.3.1. Depressive treatment. As we have discussed, major depressive disorder has a variety of treatment options- all found efficacious; however, research supports that while psychopharmacological interventions are more effective in rapidly reducing symptoms, psychotherapy or even a combination treatment approach are more effective in establishing long-term relief of symptoms.

Rates of relapse for major depressive disorder 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).

4.6.3.2. Bipolar treatment. Lithium and other mood stabilizers are very effective in managing symptoms of patients with bipolar disorder. Unfortunately, it is the adherence to the medication regimen that is often the issue with these patients. The euphoric highs that are associated with manic and hypomanic episodes are often desired by bipolar patients, thus often leading them to forgo their medication. Combination of psychopharmacology and psychotherapy aimed at increasing rate of adherence to medical treatment may be the most effective treatment option for bipolar I and II disorder.

Module 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, epidemiology, comorbidity, and etiology. This will help you with understanding what treatment options there are and their efficacy. In the next module we will discuss trauma- and stressor-related disorders.

Module 5: Trauma- and Stressor-Related Disorders

Module Overview

In Module 5, we will discuss matters related to trauma- and stressor-related disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include PTSD, acute stress disorder, and adjustment disorder. Before getting started with all this, we will explain what stressors are. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

Module Learning Outcomes

- Explain what stressors are.
- Describe how trauma- and stressor-related disorders present.
- Describe the epidemiology of trauma- and stressor-related disorders.
- Describe comorbidity in relation to trauma- and stressor-related disorders.
- Describe the etiology of trauma- and stressor-related disorders.
- Describe treatment options for trauma- and stressor-related disorders.

5.1. Stressors

Section Learning Objectives

- Define stressor.
- Describe common stressors.

Before we get into clinical presentations for the three most common trauma and stress related disorders, let's discuss a little about common events that precipitate a stress related diagnosis. A stress disorder occurs when an individual has difficulty coping with or adjusting to a recent **stressor**. Stressors can be any event- either witnessed firsthand, experienced personally or experienced by a close family member- that increases physical or psychological demands on an individual. These events are significant enough that they pose a threat, whether real or imagined, to the individual. While many people experience similar stressors throughout their lives, only a small percentage of individuals experience significant maladjustment to the event that psychological intervention is warranted.

Among the most commonly studied triggers for trauma related disorders are combat and physical/sexual assault. Symptoms of combat related trauma date back to World War I, when soldiers would return home with "shell shock" (Figley, 1978). Unfortunately, it wasn't until after the Vietnam War that significant progress was made in both identifying and treating war-related psychological difficulties (Roy-Byrne et al., 2004). With the more recent wars in Iraq and Afghanistan, attention was again brought to posttraumatic stress disorder (PTSD) symptoms due to the large number of service members returning from deployments and reporting significant trauma symptoms.

Physical assault, more specifically sexual assault, is another commonly studied traumatic event. **Rape,** or forced sexual intercourse or other sexual act committed without an individual's consent, occurs in one out of every five women and one in every 71 men (Black et al., 2011). Unfortunately, this statistic is likely an underestimate of the actual number of cases that occur due to the reluctance of many individuals to report their sexual assault. Of the reported cases, it is estimated that nearly 81% of female and 35% of male rape victims report both acute stress disorder and posttraumatic stress disorder symptoms (Black et al., 2011).

Now that we've discussed a little about some of the most commonly studied traumatic events, let's take a look further at the presentation for posttraumatic stress disorder, acute stress disorder, and adjustment disorder.

5.2. Clinical Presentation and DSM Criteria

Section Learning Objectives

- Describe how PTSD presents itself.
- Describe how acute stress disorder presents itself.
- Describe how adjustment disorder presents itself.

5.2.1. Posttraumatic Stress Disorder

Posttraumatic stress disorder, or more commonly known as PTSD, is identified by the development of physiological, psychological, and emotional symptoms following exposure to a traumatic event.

Individuals must have been exposed to a situation where actual or threatened death occurred. Examples of these situations include but are not limited to: witnessing a traumatic event as it occurred to someone else; learning about a traumatic event that occurred to a family member or close friend; or being exposed to repeated events where one experiences an aversive event (e.g. victims of child abuse/neglect, ER physicians in trauma center, etc.). It should be understood that while the presentation of these symptoms varies among individuals, to meet criteria for a diagnosis of PTSD, individuals need to report symptoms among the four different categories of symptoms.

The first category involves *recurrent experiences* of the traumatic event. This can occur via flashbacks, distinct memories (which may be voluntary or involuntary), or even distressing dreams. In order to meet criteria for PTSD, these recurrent experiences must be specific to the traumatic event or the moments immediately following. Regardless of the method, the recurrent experiences can last a short time-several seconds- or extend for several days. They are often initiated by physical sensations similar to those experienced during the traumatic events, or even environmental triggers such as a specific location. Because of these triggers, individuals with PTSD are known to avoid stimuli (i.e. activities, objects, people, etc.) associated with the traumatic event.

The second category involves *avoidance* of stimuli that is related to the traumatic event. Individuals with PTSD may be observed trying to avoid the distressing thoughts and/or feelings related to the memories of the traumatic event. One way individuals will avoid these memories is by avoiding physical stimuli such as locations, individuals, activities, or even specific situations that trigger the memory of the traumatic event.

The third category experienced by individuals with PTSD is *negative alterations in cognitions or mood*. This is often reported as difficulty remembering an important aspect of the traumatic event. It should be noted that this amnesia is not due to a head injury, loss of consciousness, or substances, but rather, due to the traumatic nature of the event. The impaired memory may also leading individuals to have false beliefs about the causes of the traumatic event, often blaming themselves or others. An overall persistent negative state, including a generalized negative belief about oneslf or others is also reported by those with PTSD. Similar to those with depression, individuals with PTSD may report a reduced interest in participation of previously enjoyable activities, as well as the desire to socially engage with others.

The fourth and final category is *alterations in arousal and reactivity*. Because of the negative mood and increased irritability, individuals with PTSD may be quick tempered and act out in aggressive manners, both verbally and physically. While these aggressive responses may be provoked, they are also sometimes unprovoked. It is believed these behaviors occur due to the heightened sensitivity to potential threats, especially if the threat is similar in nature to their traumatic event. More specifically, individuals with PTSD have a heightened startle response and easily jump or respond to unexpected noises just as a telephone ringing or a car backfiring. Given this heightened arousal state, it should not be surprising that individuals with PTSD also experience significant sleep disturbances, with difficulty falling asleep, as well as staying asleep due to nightmares.

Although somewhat obvious, these symptoms likely cause significant distress in social, occupational, and other (i.e. romantic, personal) areas of functioning. Duration of symptoms is also important, as PTSD cannot be diagnosed unless symptoms have been present for **at least one month**. If they have *not* been present for a month, the individual may meet criteria for Acute Stress Disorder (see below).

5.2.2. Acute Stress Disorder

Acute stress disorder is very similar to PTSD except for the fact that symptoms must be present from **3 days to 1 month** following exposure to one or more traumatic events. If the symptoms are present after 1 month, the individual would then meet criteria for PTSD. Additionally, if symptoms present immediately following the traumatic event but resolve by day 3, an individual would not meet criteria for acute stress disorder.

Symptoms of acute stress disorder follow that of PTSD with a few exceptions. PTSD requires symptoms within each of the four categories discussed above; however, acute stress disorder requires that the individual experience nine symptoms across five different categories (intrusion symptoms, negative mood, dissociative symptoms, avoidance symptoms, and arousal symptoms). For example, an individual may experience several arousal and reactivity symptoms such as sleep issues, concentration issues, and hypervigilance, but does not experience issues regarding a negative mood. Regardless of the category of the symptoms, so long as nine symptoms are present and the symptoms cause significant distress or impairment in social, occupational, and other functioning, an individual will meet criteria for acute stress disorder.

5.2.3. Adjustment Disorder

Adjustment disorder is the least intense of the three stress related disorders discussed in this chapter. An adjustment disorder occurs following an identifiable stressor that has occurred within the past 3 months. This stressor can be a single event (loss of job, death of a family member) or a series of multiple stressors (cancer treatment, divorce/child custody issues).

Unlike PTSD and acute stress disorder, adjustment disorder does not have a set of specific symptoms an individual must meet for diagnosis, rather, whatever symptoms the individual is experiencing must be related to the stressor and must be significant enough to impair social, occupational, or other important areas of functioning. It should be noted that bereavement can be diagnosed as an adjustment disorder in extreme cases where an individual’s grief exceeds the intensity or persistence that is expected.

It should be noted that there are modifiers associated with adjustment disorder. Due to the variety of behavioral and emotional symptoms that can be present with an adjustment disorder, clinicians are expected to classify a patient’s adjustment disorder as one of the following: with depressed mood; with anxiety; with mixed anxiety and depressed mood; with disturbance of conduct; with mixed disturbance of emotions and conduct; or unspecified for behaviors that do not meet criteria for one of the aforementioned categories. Depending on the individual’s presenting symptoms, the clinician will determine which category best classifies the patient’s symptoms. These modifiers are also important in determining treatment options for patients.

5.3. Epidemiology

Section Learning Objectives

- Describe the epidemiology of PTSD.
- Describe the epidemiology of acute stress disorder.
- Describe the epidemiology of adjustment disorders.

5.3.1. PTSD

The prevalence rate for PTSD in the US is 8.7% (APA, 2013). It should not come as a surprise that the rates of PTSD are higher among veterans and others who work in fields with high traumatic experiences (i.e. firefighters, police, EMTs, emergency room providers). In fact, PTSD rates for combat veterans are estimated to be as high as 30% (NcNally, 2012). Between one-third and one-half of all PTSD cases consist of rape survivors, military combat and captivity, and ethically or politically motivated genocide (APA, 2013).

With regards to gender, PTSD is more prevalent among females than males, likely due to their greater likelihood of exposure to traumatic experiences such as rape, domestic abuse, and other forms of interpersonal violence (APA, 2013). Gender differences are not found in populations where both males and females are exposed to significant stressors suggesting that both genders are equally predisposed to developing PTSD. Prevalence rates vary slightly across cultural groups, which may reflect differences in exposure to traumatic events. (Hinton & Lewis-Fernandez, 2011). More specifically, prevalence rates of PTSD are highest for African Americans, followed by Latino/Hispanic Americans and European Americans, and lowest for Asian Americans (Hinton & Lewis-Fernandez, 2011).

5.3.2. Acute Stress Disorder

The prevalence rate for acute stress disorder varies across the country and by traumatic event. While accurate prevalence rates for acute stress disorder are difficult to determine seeing as patients must seek treatment within 30-days of the traumatic event, it is estimated that anywhere between 7-30% of individuals experiencing a traumatic event will develop acute stress disorder (National Center for PTSD). While acute stress disorder is not a good predictor of who will develop PTSD, approximately 50% of those with acute stress disorder do eventually develop PTSD (Bryant, 2010; Bryant, Friedman, Speigel, Ursano, & Strain, 2010).

Similar to PTSD, acute stress disorder is more common in females than males; however, unlike PTSD, there may be some neurobiological differences in the stress response that contribute to females developing acute stress disorder more often than males (APA, 2013). With that said, the increased exposure to traumatic events among females may also be a strong reason why women are more likely to developing acute stress disorder than males.

5.3.3. Adjustment Disorder

Adjustment disorders are fairly common as they describe individuals who are having difficulty adjusting to life after a significant stressor. In fact, in a psychiatric hospital, adjustment disorders accounts for roughly 50% of the admissions, ranking number one for the most common diagnosis (APA, 2013). As for the general public, it is estimated that anywhere from 5-20% of outpatient referrals are due to an adjustment disorder (APA, 2013).

5.4. Comorbidity

Section Learning Objectives

- Describe the comorbidity of PTSD.
- Describe the comorbidity of acute stress disorder.
- Describe the comorbidity of adjustment disorder.

5.4.1. PTSD

Given the traumatic nature of the disorder, it should not be surprising that there is a high comorbidity rate between PTSD and other psychological disorders. In fact, individuals with PTSD are 80% more likely than those without PTSD to report clinically significant levels of depressive, bipolar, anxiety, or substance abuse related symptoms (APA, 2013).

There is also a strong relationship between PTSD and major neurocognitive disorders, which may be due to the overlapping symptoms between these disorders (Neurocognitive Disorders will be covered in Module VI). There has also been an increase in PTSD and traumatic brain injuries (TBI) due to the recent wars in Afghanistan and Iraq. US military personnel and combat veterans report a comorbidity rate between PTSD and TBI at nearly 50% (APA, 2013).

5.4.2. Acute Stress Disorder

Due to the fact that 30 days after the traumatic event, ASD becomes PTSD (or the symptoms remit), the comorbidity of ASD with other psychological disorders has not been studied. While ASD and PTSD cannot be comorbid disorders, several studies have explored the relationship between ASD and PTSD in efforts to identify individuals most at risk for developing PTSD. Research studies indicate roughly 80% of motor vehicle accident survivors, as well as assault victims, who met criteria for ASD went on to develop PTSD (Brewin, Andrews, Rose, & Kirk, 1999; Bryant & Harvey, 1998; Harvey & Bryant, 1998). While some researchers indicated ASD is a good predictor of PTSD, others argue further research between the two and confounding variables should be further explored to determine more consistent findings.

5.4.3. Adjustment Disorder

Unlike most of the disorders we have reviewed thus far, adjustment disorders actually have a high comorbidity rate with various other medical conditions (APA, 2013). Often following a critical or terminal medical diagnosis, and individual will meet criteria for adjustment disorder as they process the news about their health and the impact their new medical diagnosis will have on their life. Other psychological disorders are also diagnosed with adjustment disorder; however, symptoms of adjustment disorder must be met independently of the other psychological condition (APA, 2013). For example, and individual with adjustment disorder with depressive features must not meet criteria for a major depressive episode, otherwise, the diagnosis of major depression should be made over the adjustment disorder.

5.5. Etiology

Section Learning Objectives

- Describe the biological causes of trauma- and stressor-related disorders.
- Describe the cognitive causes of trauma- and stressor-related disorders.
- Describe the social causes of trauma- and stressor-related disorders.
- Describe the sociocultural causes of trauma- and stressor-related disorders.

5.5.1. Biological

HPA axis. One theory for the development of trauma and stress related disorders is the over involvement of the **hypothalamic-pituitary-adrenal (HPA) axis.** The HPA axis is involved in the fear producing response and some speculate that a dysfunction within this axis is to blame for the development of trauma symptoms. Within the brain, the **amygdala** serves as the integrative system that essentially elicits the physiological response to a traumatic/stressful environmental situation. The amygdala sends this response to the HPA axis in effort to prepare the body to "fight or flight." The HPA axis then releases hormones- **epinephrine** and **cortisol**– to help the body to prepare to respond to a dangerous situation (Stahl & Wise, 2008). While epinephrine is known to cause physiological symptoms such as increased blood pressure, increased heart rate, increased alertness, and increased muscle tension to name a few, cortisol is responsible for returning the body back to homeostasis once the dangerous situation is resolved.

Researchers have studied the amygdala and HPA axis in individuals with PTSD, and have identified heightened amygdala reactivity in stressful situations, as well as excessive reactivity to stimuli that is related to one's specific traumatic event (Sherin & Nemeroff, 2011). Additionally, studies have indicated that individuals with PTSD also show a diminished fear extinction, suggesting an overall higher level of stress during non-stressful times. These findings may explain why individuals with PTSD experience an increased startle response and exaggerated sensitivity to stimuli associated with their trauma (Schmidt, Kaltwasser, & Wotjak, 2013).

5.5.2. Cognitive

Preexisting conditions of depression and/or anxiety may predispose an individual to develop PTSD or other stress disorders. One theory is that these individuals may ruminate, or over analyze the traumatic event, thus bringing more attention to the traumatic event which in return leads to the development of stress related symptoms. Furthermore, negative cognitive styles or maladjusted thoughts about themselves and the environment may also contribute to PTSD symptoms. For example, individuals who identify life events as “out of their control” report more severe stress symptoms than those who feel as though they have some control over their lives (Catanesi et al., 2013).

5.5.3. Social

While this may hold true for many psychological disorders, social and family support have been identified as protective factors for individuals prone to develop PTSD. More specifically, rape victims who are loved and cared for by their friends and family members as opposed to judged for their actions prior to the rape, report fewer trauma symptoms and faster psychological improvement (Street et al., 2011).

5.5.4. Sociocultural

As was mentioned previously, different ethnicities report different prevalence rates of PTSD. While this may be due to increased exposure to traumatic events, there is some evidence to suggest that cultural groups also interpret traumatic events differently, and therefore, may be more vulnerable to the disorder. Hispanic Americans have routinely been identified as a cultural group that experiences a higher rate of PTSD. Studies ranging from combat related PTSD to on-duty police officer stress, as well as stress from a natural disaster, all identify Hispanic Americans as the cultural group experiencing the most traumatic symptoms (Kaczkurkin et al., 2016; Perilla et al., 2002; Pole et al., 2001).

Women also report a higher incidence of PTSD symptoms than men. Some possible explanations for this discrepancy are stigmas related to seeking psychological treatment, as well as a greater risk of exposure to traumatic events that are related to PTSD (Kubiak, 2006). Studies exploring rates of PTSD symptoms for military and police veterans have failed to report a significant gender difference in the diagnosis rate of PTSD suggesting that there is not a difference in the rate of occurrence of PTSD in males and females in these settings (Maguen, Luxton, Skopp, & Madden, 2012).

5.6. Treatment

Section Learning Objectives

- Describe the treatment approach of the psychological debriefing.
- Describe the treatment approach of exposure therapy.
- Describe the treatment approach of CBT.
- Describe the treatment approach of Eye Movement Desensitization and Reprocessing (EMDR).

- Describe the use of psychopharmacological treatment.

5.6.1. Psychological Debriefing

One way to negate the potential development of PTSD symptoms is thorough **psychological debriefing.** Psychological debriefing is considered 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 (Kinchin, 2007). While there are a few different methods to a psychological debriefing, they all appear to follow relatively the same format:

1. Identifying the facts (what happened?)
2. Evaluating the individual's thoughts and emotional reaction to the events leading up to the event, during the event, and then immediately following
3. Normalizing the individual's reaction to the event
4. Discussing how to cope with these thoughts and feelings, as well as creating a designated social support system (Kinchin, 2007).

Throughout the last few decades, there has been a debate on the effectiveness of psychological debriefing. Those within the field argue that psychological debriefing is not a means to cure or prevent PTSD, but rather, psychological debriefing is a means to assist individuals with a faster recovery time post traumatic event (Kinchin, 2007). Research across a variety of traumatic events (natural disasters, burns, war) routinely suggests that psychological debriefing is *not* helpful in either the reduction of posttraumatic symptoms, nor the recovery time of those with PTSD (Tuckey & Scott, 2014). One theory is these early interventions may in fact encourage patients to ruminate on their symptoms or the event itself, thus maintaining PTSD symptoms (McNally, 2004). In efforts to combat these negative findings of psychological debriefing, there has been a large movement to provide more structure and training to professionals employing psychological debriefing, thus ensuring that those who were providing treatment were properly trained to do so.

5.6.2. Exposure Therapy

While exposure therapy is predominately used in anxiety disorders, it has also shown great assistance in PTSD related symptoms as it helps individuals extinguish fears associated with the traumatic event. There are several different types of exposure techniques- **imaginal, in vivo,** and **flooding** are among the most common types (Cahill, Rothbaum, Resick, & Follette, 2009).

In imaginal exposure, the individual is asked to re-create, or imagine, specific details of the traumatic event. The patient is then asked to repeatedly discuss the event in more and more detail, providing more information regarding their thoughts and feelings at each step of the event. With in-vivo exposure, the individual is reminded of the traumatic event through the use of videos, images, or other tangible objects related to the traumatic event, that induces a heightened arousal response. While the patient is re-experiencing cognitions, emotions, and physiological symptoms related to the traumatic experience, they are encouraged to utilize positive coping strategies, such as relaxation techniques to reduce their overall level of anxiety.

Imaginal exposure and in vivo exposure are generally done in a gradual process, with imaginal exposure beginning with less details of the event, and slowly gaining more and more information over time; in vivo starts with images/videos that elicit lower levels of anxiety, and then the patient slowly works their way up a fear hierarchy, until they are able to be exposed to the most distressing images. Another type of exposure therapy, flooding, involves disregard for the fear hierarchy, presenting the most distressing memories or images at the beginning of treatment. While some argue that this is a more effective treatment method, it is also the most distressing- thus placing patients at risk for dropping out of treatment (Resick, Monson, & Rizvi, 2008).

5.6.3. Cognitive Behavioral Therapy (CBT)

Cognitive Behavioral Therapy, as discussed in the mood disorders chapter, has been proven to be an effective form of treatment for trauma/stress related disorders. It is believed that this type of treatment is effective in reducing trauma related symptoms due to its ability to identify and challenge the negative cognitions surrounding the traumatic event, and replacing them with positive, more adaptive cognitions (Foa et al., 2005).

Trauma-focused cognitive-behavioral therapy (TF-CBT) is an adaptation of CBT, that utilizes both CBT techniques, as well as trauma sensitive principles to address the trauma related symptoms. According to the Child Welfare Information Gateway (CWIG; 2012), TF-CBT can be summarized via the acronym PRACTICE:

- **P:** Psycho-education about the traumatic event. This includes discussion about the event
- itself, as well as typical emotional and/or behavioral responses to the event.
- **R**: Relaxation Training. Teaching the patient how to engage in various types of relaxation techniques such as deep breathing and progressive muscle relaxation.
- **A:** Affect. Discussing ways for the patient to effectively express their emotions/fears
- related to the traumatic event.
- **C:** Correcting negative or maladaptive thoughts.
- **T:** Trauma Narrative. This involves having the patient relive the traumatic event (verbally or written), including as many specific details as possible.
- **I:** In vivo exposure (see above).
- **C:** Co-joint family session. This is to provide the patient with a strong social support and sense of security. It also allows family members to learn about the treatment so that they are able to assist the patient if necessary.
- **E:** Enhancing Security. Patients are encouraged to practice the coping strategies they learn in TF-CBT so that they are prepared when they experience these triggers out in the real world, as well as any future challenges that may come their way.

5.6.4. Eye Movement Desensitization and Reprocessing (EMDR)

EMDR is a controversial treatment for a few reasons; however, the fact that the treatment emerged from a personal observation over a theory is among the most argued reasons. In the late 1980's psychologist Francine Shapiro found that by focusing her eyes on the waving leaves during her daily walk, her troubling thoughts resolved on their own. From this observation, she concluded that lateral

eye movements facilitate cognitive processing of traumatic thoughts (Shapiro, 1989). While EMDR has evolved somewhat since Shapiro's first claims, the basic components of EMDR consist of lateral eye movement induced by the therapist moving their index finger back and forth, approximately 35 cm from the client's face, as well as components of cognitive behavioral therapy and exposure therapy. The following 8-step approach to treatment has been identified as the standard treatment approach of EMDR (Shapiro & Maxfield, 2002):

1. Patient History and Treatment Planning- Identify trauma symptoms and potential barriers to treatment.
2. Preparation- Psychoeducation of trauma and treatment.
3. Assessment- Careful and detailed assessment of the traumatic event. Patient identifies images, cognitions, and emotions related to the traumatic event, as well as trauma related physiological symptoms.
4. **Desensitization and Reprocessing-** While holding trauma image, cognition, and emotion in mind, while simultaneously assessing their physiological symptoms, the patient must track the clinicians finger movement for approximately 20 seconds. At this time the patient must "blank it out" and let go of the memory.
5. **Installation of Positive Cognitions-** Once the negative image, cognition, and emotions are reduced, the patient must hold onto a positive image or thought while again tracking the clinician's finger movement for approximately 20 seconds.
6. **Body Scan-** Patient must identify any lingering bodily sensations while again tracking the clinician's fingers for a third time to essentially rid any remaining trauma symptoms.
7. Closure- Patient is provided with positive coping strategies and relaxation techniques to assist with any recurrent cognitions or emotions related to the traumatic experience.
8. Reevaluation- Clinician assesses if treatment goals were met. If not, schedules another treatment session and identifies remaining symptoms.

As you can see from above, only steps 4-6 are specific to EMDR; the remaining treatment is essentially a combination of exposure therapy and cognitive behavioral techniques. Because of the high overlap between treatment techniques, there have been quite a few studies comparing the treatment efficacy of EMDR to TF-CBT and exposure therapy. While research initially failed to identify a superior treatment, often citing EMDR and TF-CBT as equally efficacious in treating PTSD symptoms (Seidler & Wagner, 2006), more recent studies have found that EMDR may be superior to that of TF-CBT, particularly in psycho-oncology patients (Capezzani et al., 2013; Chen, Zang, Hu & Liang, 2015). While meta-analytic studies continue to debate which treatment is the most effective in treating PTSD symptoms, the World Health Organization's (2013) publication on the *Guidelines for the Management of Conditions That Are Specifically Related to Stress,* identified TF-CBT and EMDR as the only recommended treatment for individuals with PTSD.

5.6.5. Psychopharmacological Treatment

While psychopharmacological interventions have been shown to provide some relief, particularly to veterans with PTSD, most clinicians agree that resolution of symptoms cannot be accomplished without implementing exposure and/or cognitive techniques that target the physiological and maladjusted thoughts maintaining the trauma symptoms. With that said, clinicians agree that psychopharmacology interventions are an effective second line of treatment, particularly when psychotherapy alone does not

produce relief from symptoms.

Among the most common types of medications used to treat PTSD symptoms are selective serotonin reuptake inhibitors (SSRIs; Bernardy & Friedman, 2015). As previously discussed in the depression chapter, SSRI's work by increasing the amount of serotonin available to neurotransmitters. Tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs) are also recommended as second-line treatments. Their effectiveness is most often observed in individuals who report co-occurring major depressive disorder symptoms, as well as those who do not respond to SSRIs (Forbes et al., 2010). Unfortunately, due to the effective CBT and EMDR treatment options, research on psychopharmacological interventions have been limited. Future studies exploring other medication options are needed to determine if there are alternative medication options for stress/trauma disorder patients.

Module Recap

In Module 5, we discussed trauma- and stressor-related disorders to include PTSD, acute stress disorder, and adjustment disorder. We clarified what stressors were and then discussed how these disorders present themselves. In addition, we clarified the epidemiology, comorbidity, and etiology of each disorder. Finally, we discussed potential treatment options for the trauma- and stressor-related disorders. Our discussion in Module 6 moves to dissociative disorders. Be sure you are preparing for your second exam in the class, which is the first to deal with psychological disorders.

Module 6: Dissociative Disorders

Module Overview

In Module 6, we will discuss matters related to dissociative disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include dissociative identity disorder, dissociative amnesia, and depersonalization/derealization. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 6.1. Clinical Presentation
- 6.2. Epidemiology
- 6.3. Comorbidity
- 6.4. Etiology
- 6.5. Treatment

Module Learning Outcomes

- Describe how dissociative disorders present.
- Describe the epidemiology of dissociative disorders.
- Describe comorbidity in relation to dissociative disorders.
- Describe the etiology of dissociative disorders.
- Describe treatment options for dissociative disorders.

6.1. Clinical Presentation

Section Learning Objectives

- Describe dissociative disorders.
- Describe how DID presents itself.
- Describe how dissociative amnesia presents itself.
- Describe how depersonalization/derealization presents itself.

Dissociative disorders are a group of disorders categorized by symptoms of disruption in consciousness, memory, identify, 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 who suffer from acute stress disorder and 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: *Dissociative Identity Disorder, Dissociative Amnesia,* and *Depersonalization/Derealization Disorder.*

6.1.1. Dissociative Identity Disorder (DID)

Dissociative Identity Disorder (DID) is what people commonly refer to as *multiple personality disorder.* 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, have different memories, and/or sensory-motor functioning.

The second main diagnostic criteria for DID is that there must be a gap in recall of events, information, and/or trauma due to the switching of personalities. These gaps are more excessive than typical forgetting one may experience due to lack of attention. It is important that these personalities are not a secondary effect of a substance or medical condition (I.e. gap of information due to seizure).

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 **subpersonalities**. Although it is hard to identify how many subpersonalities an individual may have at one time, it is believed that there are on average 15 subpersonalities for women and 8 for men (APA, 2000).

The presentation of *switching* between personalities 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 subpersonality best equipped to handle the current stressor will present. The relationship between subpersonalities varies between individuals- with some individuals reporting knowledge of other subpersonalities while others have a one-way amnesic relationship with subpersonalities, meaning they are not aware of other personalities (Barlow & Chu, 2014). These individuals will experience episodes of “amnesia” when the primary personality is not present.

6.1.2. Dissociative Amnesia Disorder

Dissociative amnesia disorder is identified by the inability to recall important autobiographical information. 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 within dissociative amnesia. **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.

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 amnesia experience deficits in both *semantic* and *procedural* knowledge. This means that individuals have no common knowledge of (i.e. cannot identify letters, colors, numbers) nor do they have the ability to engage in learned skills (i.e. typing shoes, driving car).

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 and rarely is there a relapse. 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).

6.1.3. Depersonalization/Derealization Disorder

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 *outer body experience* where you are an observer of your thoughts, feelings, and physical being. Furthermore, some patients 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. Emotionally, one may feel detached from their own feelings, lacking the ability to *feel* emotions despite knowing they have them.

Symptoms of **derealization** include feelings of unreality or detachment from the *world*—whether it be individuals, objects, or their surroundings. For example, an individual 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 your 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).

6.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of dissociative disorders.

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%, with an equal distribution between men and women (APA, 2013). Similarly, a large community sample suggested dissociative amnesia occurs in approximately 1.8% of the population. Unlike DID, females are twice as more likely to be diagnosed with dissociative amnesia 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.

While many individuals experience brief episodes of depersonalization/derealization throughout their life, 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).

Onset of dissociative disorders is generally late adolescence to early adulthood, with the exception of DID. Due to the high comorbidity between childhood abuse and DID, it is believed that symptoms actually begin in early childhood following the repeated exposure to abuse; however, full onset of the disorder is not observed (or noticed by others) until adolescence (Sar et al., 2014).

6.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of dissociative disorders.

Given that dissociative disorders are often precipitated by a traumatic experience, it should not be surprising that there is a high comorbidity between dissociative disorders and PTSD. Similarly, depressive disorders are also often found in combination with dissociative disorders, likely due to the impact the disorders have on social and emotional functioning. In individuals with dissociative amnesia, a wide range of emotions related to their inability to recall memories during the episode often present once the amnesia episode is in remission (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, particularly for those who experience dissociative amnesia. Furthermore, dependent, avoidant, and borderline personality disorders have all been suspected as co-occurring disorders among the dissociative disorder family.

6.4. Etiology

Section Learning Objectives

- Describe the biological causes of dissociative disorders.
- Describe the cognitive causes of dissociative disorders.
- Describe the sociocultural causes of dissociative disorders.
- Describe the psychodynamic causes of dissociative disorders.

6.4.1. Biological

While studies on the involvement of genetic underpinnings need additional research, there is some suggestion that 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).

6.4.2. Cognitive

One proposed cognitive theory of dissociative disorders, particularly dissociative amnesia, is a memory retrieval deficit. More specifically, Kopelman (2000) theorizes 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 that are responsible for the retrieval difficulties, as research studies have reported mixed findings.

Specific to DID, neuroimaging studies have shown differences in hippocampus activation between subpersonalities (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).

6.4.3. 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 subpersonalities (Goff & Simms, 1993).

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

6.4.4. Psychodynamic

The psychodynamic theory of dissociative disorders assumes that the dissociative disorders are caused by and 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 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).

6.5. Treatment

Section Learning Objectives

- Clarify why treatment for dissociative disorders is limited.
- Describe treatment options for dissociative identity disorder.
- Describe treatment options for dissociative amnesia.
- Describe treatment options for depersonalization/derealization.

Treatment for dissociative disorders is limited for a few reasons. First, with respects to dissociative amnesia, 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. Second, the rarity of these disorders has offered limited opportunities for research of both the development and effectiveness of treatment methods. Due to the differences in dissociative disorders the disorders, treatment options will be discussed specific to each disorder.

6.5.1. Dissociative Identity Disorder

The ultimate treatment goal for DID is **integration** of subpersonalities to a point of **final fusion** (Chu et al., 2011). Integration refers to the ongoing process of merging subpersonalities into one personality. Psychoeducation is paramount for integration, as the individual must have an understanding of their disorder, as well as acknowledge their subpersonalities. Like mentioned above, many individuals have a one-way amnesic relationship with the subpersonalities, meaning they are not aware of one another. Therefore, the clinician must first make the individual aware of the various subpersonalities 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 subpersonalities. In making the subpersonalities aware of one another, as well as addressing their conflicts, is an essential component of integration of subpersonalities, 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 complete loss of separateness. Depending on the number of subpersonalities, this process can take quite a while. Once all subpersonalities 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 patients 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 subpersonalities 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 prevent future relapses.

6.5.2. Dissociative Amnesia

As previously mentioned, many individuals regain memory without the need for treatment; however, there is a small population who do require additional treatment. While there is no evidenced based treatment for dissociative amnesia, both hypnosis and phasic treatment have been shown to produce some positive effects in patients 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). Because of 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 patient 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).

6.5.3. Depersonalization/Derealization Disorder

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.

Module Recap

In this module, we discussed the dissociative disorders of Dissociative Identity Disorder, Dissociative Amnesia, and Depersonalization/Derealization Disorder in terms of their clinical presentation, epidemiology, comorbidity, etiology, and treatment approaches. This represents the final class of disorders in this unit so make sure you are getting ready for your exam. In our next block of disorders we will discuss anxiety disorders, somatic symptom, and obsessive-compulsive disorders. If you have questions about any of the disorders discussed in this module or the prior two, be sure you ask your instructor. Good luck with the exam.

III

Part III. Mental Disorders – Block 2

Part III. Mental Disorders – Block 2

Module 7: Anxiety Disorders

Module Overview

In Module 7, we will discuss matters related to anxiety disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include Generalized Anxiety Disorder, Specific Phobias, Agoraphobia, Social Anxiety Disorder, and Panic Disorder. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 7.1. Clinical Presentation
- 7.2. Epidemiology
- 7.3. Comorbidity
- 7.4. Etiology
- 7.5. Treatment

Module Learning Outcomes

- Describe how anxiety disorders present.
- Describe the epidemiology of anxiety disorders.
- Describe comorbidity in relation to anxiety disorders.
- Describe the etiology of anxiety disorders.
- Describe treatment options for anxiety disorders.

7.1. Clinical Presentation

Section Learning Objectives

- Describe how Generalized Anxiety Disorder presents itself.
- Describe how Specific Phobias presents itself.
- Describe how Agoraphobia presents itself.
- Describe how Social Anxiety Disorder presents itself.
- Describe how Panic Disorder presents itself.

The hallmark symptoms of anxiety related disorders are excessive fear or anxiety related to behavioral disturbances. Fear is considered an adaptive response, as it often prepares your body for an impending threat. Anxiety, however, is more difficult to identify as it is often the response to a *vague* sense of threat. The two can be distinguished from one another as fear is related to either a real or a perceived threat, while anxiety is the *anticipation* of a future threat (APA, 2013).

As you will see throughout the chapter, individuals may experience anxiety in many different forms. *Generalized anxiety disorder*, the most common of the anxiety disorders, is characterized by a global and persistent feeling of anxiety. A *specific phobia* is observed when an individual experiences anxiety related to a specific object or subject. Similarly, individuals may also experience *agoraphobia* when they experience fear specific to leaving their home and traveling to public places. *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. And finally, there is *panic disorder*, where an individual experiences recurrent panic attacks consisting of physical and cognitive symptoms.

7.1.1. Generalized Anxiety Disorder (GAD)

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. Individuals with GAD will also experience somatic symptoms during intensive periods of anxiety as listed below.

7.1.2. Specific Phobia

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.

7.1.3. Agoraphobia

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.

7.1.4. Social Anxiety Disorder

For **social anxiety disorder**, 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, boring, 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, and 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 in 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.

7.1.5. Panic Disorder

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 occurs 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. Intensity of symptoms also varies among individuals, with some patients 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.

7.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of Generalized Anxiety Disorder.
- Describe the epidemiology of Specific Phobias.
- Describe the epidemiology of Agoraphobia.
- Describe the epidemiology of Social Anxiety Disorder.
- Describe the epidemiology of Panic Disorder.

7.2.1. Generalized anxiety disorder

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).

7.2.2. Specific phobia

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.

7.2.3. Agoraphobia

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.

7.2.4. Social anxiety disorder

The overall prevalence rate of social anxiety disorder is significantly higher in the United States than other countries would wide, with an estimated 7% of the US population diagnosed with a 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.

7.2.5. Panic disorder

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.

7.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of Generalized Anxiety Disorder.

- Describe the comorbidity of Specific Phobias.
- Describe the comorbidity of Agoraphobia.
- Describe the comorbidity of Social Anxiety Disorder.
- Describe the comorbidity of Panic Disorder.

7.3.1. Generalized anxiety disorder

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.

7.3.2. Specific phobia

Seeing as the onset of specific phobias occurs at a younger age than most other anxiety disorders, it is generally the primary diagnosis with the occasional generalized anxiety disorder 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.

7.3.3. Agoraphobia

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.

7.3.4. Social anxiety disorder

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, and individual with social anxiety disorder may consume larger amounts of alcohol in social settings in efforts to alleviate the anxiety of the social situation.

7.3.5. Panic disorder

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, cardia 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.

7.4. 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.

7.4.1. Biological

7.4.1.1. Biological – Genetic influences. While genetics have been known to contribute to the presentation of anxiety symptoms, the interaction between genetics and stressful environmental influences appears to actually account 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).

7.4.1.2. Biological – 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**, for determination if 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.

7.4.2. Psychological

7.4.2.1. Psychological – Cognitive. The cognitive perspective on the development of anxiety related disorders centers around dysfunctional thought patters. As seen in depression, **maladaptive assumptions** are routinely observed in individuals with anxiety related disorders, as they often engage in interpreting events as dangerous or overreacting to potential 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 symptoms as a potential treat. 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 to 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. This overestimation of social performance negatively reinforces future avoidance of social situations.

7.4.2.2. Psychological – 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 ground breaking 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.

7.4.2.3. Psychological – Modeling is another behavioral explanation of the development of specific and social phobias. In modeling, an individual acquires a far though 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 will present, as opposed to a specific phobia.

7.4.3. Sociocultural

Seeing how prominent the biological and psychological constructs are in explaining the development of anxiety related disorders, we also need to review the social constructs that contribute 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 great attention, particularly because due to the epidemiological nature of the disorder.

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 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 more often with anxiety related disorders.

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.

7.5. Treatment

Section Learning Objectives

- Describe treatment options for Generalized Anxiety Disorder.
- Describe treatment options for Specific Phobias.
- Describe treatment options for Agoraphobia.
- Describe treatment options for Social Anxiety Disorder.
- Describe treatment options for Panic Disorder.

7.5.1. Generalized Anxiety Disorder

7.5.1.1. Psychopharmacology. Benzodiazepines, a class of sedative-hypnotic drugs that will be discussed in more detail in the Substance Abuse chapter, 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 done on benzodiazepines, serious side effects as well as physical dependence of benzodiazepines at large dosages has 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.

7.5.1.2. 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 effort to provide relief to those suffering from 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).

7.5.1.3. Cognitive Behavioral Therapy (CBT). CBT is discussed in great detail in the Depression chapter; however, it is also among the most effective treatment options for a variety of anxiety related disorders, including GAD. In fact, findings suggest 60 percent 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).

7.5.1.4. Biofeedback. Biofeedback provides a visual representation of a patient's physiological arousal. To achieve this feedback, a patient is connected to a computer that provides continuous information of their physiological states. There are several ways a patient 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 patient's skin just above a major muscle group- commonly the forearm or the forehead. Other common areas 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 patient is connected to the biofeedback machine, the clinician is able to walk the patient 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 patient 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, patients do report a positive experience with the treatment due to the visual feedback of their physiological arousal (Brambrink, 2004).

7.5.2. Specific Phobias

7.5.2.1. 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 in several different approaches: *systematic desensitization, flooding, and modeling.*

Systematic desensitization is an exposure technique that is 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 patient, 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 all objects on the list.

7.5.2.2. Flooding. 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.

7.5.2.3. Modeling. Finally, **modeling** is another common technique that is used to treat phobia disorders (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.

7.5.3. Agoraphobia

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 form nearly 60 to 80 percent of patients with agoraphobia; however, there is a relatively high rate of partial relapse suggesting that long-term treatment or booster sessions at minimum should be continued for several years (Craske & Barlow, 2014).

7.5.4. Social Anxiety Disorder

7.5.4.1. Exposure. A hallmark treatment approach for all anxiety related 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 patient so that the patient can practice social interactions in a safe, controlled environment (Rodebaugh, Holaway, & Heimberg, 2004). As the patient becomes habituated to the interaction with the clinician, the clinician and patient 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 patient is encouraged to continue with these exposure based social interactions outside of treatment to help reduce anxiety related to social situations.

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

7.5.4.3. 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. Similar to cognitive restructuring previously discussed in the Depression chapter, the clinician will work with the therapist to identify negative, automatic thoughts that contribute to the distress in social situations. The clinician can then help the patient 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).

7.5.5. Panic Disorder

7.5.5.1. 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).

7.5.5.2. Psychoeducation. Treatment begins by educating the patient 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.

7.5.5.3. 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 patient is then

encouraged to identify and document 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 patient is learning the fundamental processes in which they can manage their panic symptoms (Craske & Barlow, 2014).

7.5.5.4. Relaxation training. Similar to that in exposure based treatment for phobias, prior to engaging in exposure training, the individual must learn a relaxation technique to apply during 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).

7.5.5.5. Progressive muscle relaxation. To replace the breathing retraining, Craske & Barlow (2014) suggest **progressive muscle relaxation** (PMR). In progressive muscle relaxation, the patient learns to tense and relax various large muscle groups throughout the body. Generally speaking, the patient 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).

7.5.5.6. 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. Similar to the discussion in the Depression chapter, cognitive restructuring involves identifying the role of thoughts in generating and maintaining emotions. The clinician encourages the patient to view these thoughts as “hypotheses” as opposed to fact, which allows the thoughts to be questioned and challenged. This is where the detailed recordings in the self-monitoring section of treatment is helpful. By discussing specifically what the patient has recorded for the relationship between physiological arousal and thoughts/behaviors, the clinician is able to help the patient restructure the maladaptive thought processes to more positive thought processes which in return, helps to reduce fear and anxiety.

7.5.5.7. Exposure. As discussed in detail above in the specific phobia section, the patient is next 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 patient 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 patient 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.

7.5.5.8. Pharmacological interventions. According to Craske & Barlow (2014), nearly half of patients 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 medication was discontinued post treatment, the CBT+ medication groups fared worse than the CBT treatment along groups, thus supporting the theory that immersion in interoceptive exposure is limited due to 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).

Module Recap

Module 7 was the first of Unit 3 and we covered the topic of anxiety disorders. This discussion included Generalized Anxiety Disorder, Specific Phobias, Agoraphobia, Social Anxiety Disorder, and Panic Disorder. As with other modules in this book, we discussed the clinical presentation, epidemiology, comorbidity, and etiology of the anxiety disorders. Treatment options were also given and included biological, psychological, and sociocultural options. In Module 8 we will discuss somatic symptom and related disorders.

Module 8: Somatic Symptom and Related Disorders

Module Overview

In Module 8, we will discuss matters related to somatic symptom disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include Somatic Symptom Disorder, Illness Anxiety Disorder, Conversion Disorder, and Factitious Disorder. We also will discuss psychological factors affecting other medication conditions in relation to their clinical presentation, diagnostic criteria, common types of psychophysiological disorders, and treatment. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 8.1. Clinical Presentation
- 8.2. Epidemiology
- 8.3. Comorbidity
- 8.4. Etiology
- 8.5. Treatment
- 8.6. Psychological Factors Affecting Other Medical Conditions

Module Learning Outcomes

- Describe how somatic symptom disorders present.
- Describe the epidemiology of somatic symptom disorders.
- Describe comorbidity in relation to somatic symptom disorders.
- Describe the etiology of somatic symptom disorders.
- Describe treatment options for somatic symptom disorders.
- Describe psychological factors affecting other medical conditions in terms of their clinical presentation, diagnostic criteria, common types of psychophysiological disorders, and treatment.

8.1. Clinical Presentation

Section Learning Objectives

- Describe how Somatic Symptom Disorder presents itself.
- Describe how Illness Anxiety Disorder presents itself.
- Describe how Conversion Disorder presents itself.

- Describe how Factitious Disorder presents itself.

Psychological disorders that feature somatic symptoms are often very difficult to diagnose due to the internalizing nature of the disorder, meaning there is no real way for a clinician to measure the somatic symptom. Furthermore, the somatic symptoms could take on many forms. For example, the individual may be *faking* the physical symptoms, *imagining* the symptoms, *over exaggerating* the symptoms, or, they could in fact be real and triggered by external factors such as stress or other psychological disorders. Let's not also forget the symptoms also may be part of a real medical illness or disorder, and therefore, the symptoms should be treated medicinally.

All of the disorders within this chapter share a common feature: there is a presence of somatic symptoms associated with significant distress and/or impairment. Oftentimes, individuals with a somatic disorder will present to their primary care physician with their physical complaints. Occasionally, they will be referred to clinical psychologists after an extensive medical evaluation concludes that their current symptoms cannot be explained by a medical diagnosis. As you will read further, despite their similarities, there are key features among the various disorders, that distinguish them from one another.

8.1.1. Somatic Symptom Disorder

Individuals with somatic symptom disorder often present with multiple somatic symptoms at one time. These symptoms are significant enough to impact their daily functioning, such as preventing them from attending school, work, or family obligations. The symptoms can be localized (i.e. in one spot) or diffused (i.e. entire body), and may be specific or nonspecific (i.e. fatigue). For somatic symptom disorder, a lack of medical explanation is not needed for diagnosis, as it is assumed that the individual's suffering *is authentic.* In fact, somatic symptom disorder is often diagnosed when another medical condition is present, as these two diagnoses are not mutually exclusive.

Somatic symptom disorder patients generally present with significant worry about their illness. Their interpretation of symptoms are often viewed as threatening, harmful, or troublesome (APA, 2013). Because of these negative appraisals, they often fear that their medical status is more serious than it typically is, and high levels of distress are often reported. Oftentimes these patients will "shop" at different physician offices to confirm the seriousness of their symptoms.

8.1.2. Illness Anxiety Disorder

Illness anxiety disorder, previously known as hypochondriasis, involves the excessive preoccupation with having or acquiring a serious medical illness. The key distinction between illness anxiety disorder and somatic symptom disorder is that an individual will illness anxiety disorder does *not* typically present with any somatic symptoms. Occasionally an individual will present with a somatic symptom; however, the intensity of the symptom is mild, and does not drive the anxiety- acquiring a serious illness drives the concerns.

Individuals with illness anxiety disorder generally have been cleared medically; however, there are some individuals who are diagnosed with a medical illness. In this case, their anxiety surrounding the severity of their disorder is excessive or disproportionate to their actual medical diagnosis. While an individual's concern for an illness may be due to a physical sign or sensation, most individual's concerns are derived not from a physical complaint, but their actual anxiety related to a suspected medical disorder. This excessive worry often expands to a general anxiety regarding one's health and disease. Unfortunately, this anxiety does not appease even after reassurance from a medical provider or negative test results, even when provided my multiple physicians and diagnostic tests.

As one can imagine, the preoccupation and anxiety associated with attaining a medical illness severely impacts daily functioning. Individual's will often spend copious amounts of type scanning and analyzing their body for "clues" of potential illnesses. Additionally, excessive amount of time is often spent on internet searches related to symptoms and rare illnesses. Although extreme, some cases of invalidism have been reported due to illness anxiety disorder (APA, 2013).

8.1.3. Conversion Disorder

According to the DSM-5 (APA, 2013), many clinicians describe the symptoms of conversion disorder as either *functional* or *psychogenic*. Functional symptoms would be those of abnormal central nervous system functioning, and are often assumed to be associated with a neurological disorder. Psychogenic symptoms have no biological basis for the symptoms, and therefore, are psychological in nature.

There are a multitude of symptoms that may present in conversion disorder, as indicated by the many specifiers. Common motor symptoms include weakness or paralysis, abnormal movements (i.e. tremors), and gait abnormalities (i.e. limping). Additionally, sensory symptoms such as altered, reduced, or absent skin sensations, and vision or hearing impairment are also reported in many individuals. Less commonly seen are epileptic seizures and episodes of unresponsiveness resembling fainting or coma (Marshall et al., 2013).

The most difficult aspect of conversion disorder is the complex relationship with a medical evaluation. While a diagnosis of conversion disorder requires that the symptoms *not* be explained by a neurological disease, just because a medical provider fails to provide evidence that it is not a specific medical disorder is not sufficient. Therefore, there must be evidence of *incompatibility* of the medical disorder and the symptoms. For example, and individual experiencing attacks resembling epilepsy would require a normal simultaneous electroencephalogram (EEC), indicating that there is not epileptic activity during what was previously thought of as an epileptic seizure.

8.1.4. Factitious Disorder

Factitious disorder, which is commonly referred to as *Munchausen syndrome,* is different from the three previously discussed somatic disorders in that there is intentional falsification of medical or psychological symptoms of oneself or another, with the overall intention of deception. While a medical condition may be present, the severity or impairment related to the medical condition is more excessive due to the individual's need to deceive those around them. Even more alarming is that this disorder is not only observed in the individual leading the deception— it can also be present in another individual,

oftentimes a child or an individual with a compromised mental status who is not aware of the deception behind their illness (aka *Munchausen by Proxy*).

Some examples of factitious disorder behaviors include but are not limited to altering a urine or blood test, falsifying medical records, ingesting a substance that would indicate abnormal laboratory results, and physically injure oneself or induce an illness by injecting or ingesting a harmful substance (APA, 2013). While it is unclear why an individual would want to fake their own (or someone else's) physical well-being, there is some evidence suggesting that factors such as depression, lack of parental support during childhood, or an excessive need for social support may contribute to this disorder (McDermott, Leamon, Feldman, & Scott, 2012; Ozden & Canat, 1999; Feldman & Feldman, 1995).

8.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of somatic disorders.

The prevalence rates for somatic disorders are often difficult to determine; however, overall estimates of somatic symptom disorder is estimated to be around 5-7% (APA, 2013). There is a trend that females report more somatic symptoms than males, thus more females are diagnosed with somatic symptom disorder than males (APA, 2013).

Seeing as illness anxiety disorder is a newer diagnosis (replacing hypochondriasis), prevalence rates are largely based on the previous disorder. Previous findings suggest that illness anxiety disorder occurs in 1-10% of the general population. It is equal in males and females.

Prevalence rates of factitious disorder could not be obtained; however, the illness is incredibly rare. More recent research has indicated that nearly 8% of individuals admitted to a psychiatric inpatient unit present with factitious symptoms (Catalina, Gomez, de Cos, 2008). It is believed that these symptoms are likely related to physical symptoms felt in the past and are therefore exaggerated, as opposed to deliberately feigning the symptoms.

8.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of somatic disorders.

Given that half of psychiatric patients also have an additional medical disorder, 35% have an undiagnosed medical condition, and approximately 20% reported medical problems *caused* their mental condition, it should not come as a surprise that somatic disorders in general have a high comorbidity with other psychological disorders (Felker, Yazel, Short, 1996). More specifically, anxiety and depression are among the most commonly co-diagnosed disorders for somatic disorders. While there is not a lot of information regarding specific comorbidities among somatic related disorders, there is some evidence to suggest that those with illness anxiety disorder are at-risk of developing somatic symptom and personality disorders (APA, 2013). Similarly, personality disorders are more common in individuals with conversion disorder than the general public, with approximately two-thirds of individuals with illness anxiety disorder are likely to have at least one other psychological disorder (APA, 2013).

There is also a high comorbidity between somatic disorders and other physical disorders classified as *central sensitivity syndromes (CSSs)*, due to their common central sensitization symptoms, yet medically unexplained symptoms (McGeary, Harzell, McGeary, & Gatchel, 2016). Disorders included in this group are fibromyalgia, irritable bowel syndrome, and chronic fatigue syndrome. Comorbidity rates are estimated at 60% for these functional syndromes and somatic pain disorder (Egloff et al., 2014).

8.4. Etiology

Section Learning Objectives

- Describe the psychodynamic causes of somatic disorders.
- Describe the cognitive causes of somatic disorders.
- Describe the behavioral causes of somatic disorders.
- Describe the sociocultural causes of somatic disorders.

8.4.1. Psychodynamic

Psychodynamic theory suggests that somatic symptoms present as a response against unconscious emotional issues. There are two factors that initiate and maintain somatic symptoms: *primary gain* and *secondary gain*. Primary gains produce *internal* motivators, whereas secondary gains produce *external motivators* (Jones, Carmel & Ball, 2008). When you relate this to somatic disorders, the primary gain, according to psychodynamic theorists, provides protection from the anxiety or emotional symptoms and/or conflicts. This need for protection is expressed via the physical symptom such as pain, headache, etc. The secondary gain, the external experiences from the physical symptoms that maintain these physical symptoms, can range from attention and sympathy, to missed work, obtain financial assistance, or psychiatric disability, to name a few.

8.4.2. Cognitive

Cognitive theorists often believe that somatic related disorders are a result of negative beliefs or exaggerated fears of physiological sensations. Individuals with somatic related disorders may have a heightened sensitivity to bodily sensations. This combined with their maladaptive thought patterns may lead individuals to overanalyze and interpret their physiological symptoms in a negative light.

For example, an individual with a headache may *catastrophize* the symptoms and believe that their headache is the direct result of a brain tumor, as opposed to maybe stress or other inoculate reason. When this diagnosis is not confirmed by their medical provider, the individual may then catastrophize even further, believing they have an extremely rare disorder that requires an evaluation from a specialist.

8.4.3. Behavioral

Keeping true with the behavioral approach to psychological disorders, behaviorists propose that somatic related disorders develop and are maintained by *reinforcers.* More specifically, individuals experiencing significant somatic symptoms are often rewarded by gaining attention from other people (Witthoft & Hiller, 2010). These rewards may also extend to more significant factors such as receiving disability.

While the behavioral theory of somatic disorders appears to be similar to the psychodynamic theory of secondary gains, there is a clear distinction between the two—behaviorists view these gains as the *primary* reason for the development and maintenance of the disorder, whereas psychodynamic theorists view these gains as secondary, only after the underlying conflicts create the disorder.

8.4.4. Sociocultural

There are a couple of different ways that sociocultural factors contribute to somatic related disorders. First, there is the social factor of familial influence that likely plays a large role in the attention to somatic symptoms. Individuals with somatic symptom disorder are more likely to have a family member or close friend who is overly attentive to their somatic symptoms or report high anxiety related to their health (Watt, O'Connor, Stewart, Moon, & Terry, 2008; Schulte, Petermann, & Noeker, 2010).

Culturally, Western countries express less of a focus on somatic complaints compared to those in the Eastern part of the world. This may be explained by the different evaluations of the relationship between mind and body. For example, Westerners tend to have a view that psychological symptoms *sometimes* influence somatic symptoms, whereas Easterners focus more heavily on the mind/body relationship and how psychological and somatic symptoms interact with one another. These different cultural beliefs are routinely seen in research where Asian populations are more likely to report the physical symptoms related to stress than the cognitive or emotional problems that many in the United States report (Sue & Sue, 2016).

8.5. Treatment

Section Learning Objectives

- Describe treatment options for somatic disorders.

Treatment for these disorders are often difficult as individuals see their problems as completely medical, and therefore, do not think psychological intervention is necessary (Lahmann, Henningsen, & Noll-Hussong, 2010). Generally speaking, once an individual does not find relief from their symptoms after meeting with several different physicians, they often do willingly engage in psychotherapy, psychopharmacology, or both (Raj et al., 2014).

Among the most effective treatment approaches is the *biopsychosocial model* of treatment. This approach takes into account the various biological, psychological, and social factors that influence the illness and presenting symptoms (Gatchel et al., 2007). This treatment is often achieved through a *multidisciplinary* approach where the symptoms are managed my many providers, often times including a physician, psychiatrist, and psychologist. Another approach, the *interdisciplinary* approach involves a higher level of care as the multiple disciplines interact with one another and identify a treatment goal (Gatchel et al., 2007). This approach, although more difficult to find, particularly in more rural settings, is presumed to be more effective due to the integration of health care providers and their ability to uniformly work together to treat the patient.

8.5.1. Psychotherapy

8.5.1.1. Psychodynamic. Interpersonal psychotherapy, a type of psychodynamic therapy, has been found to be efficacious in treating somatic related disorders. Interpersonal psychotherapy focuses on the relationship between self-experience and the unconscious, and how these factors contribute to body dysfunction. This type of treatment has been shown to reduce anxiety, depression, and improve overall quality of life immediately following treatment; however, effects appear to diminish over time (Abass et al., 2014; Steinert et al., 2015).

8.5.1.2. CBT. Traditional cognitive-behavioral therapies (CBT) have been employed to address the cognitive attributions and maladaptive coping strategies that are responsible for the development and maintenance of the disorder. The most often misattribution for these disorders is *catastrophic thinking*, or the rumination about worst-case scenario outcomes. Additionally, goals of CBT treatment are the acceptance of the medical condition, addressing avoidance behaviors, and mediating expectations of treatment (Gatchel et al., 2014).

8.5.1.3. Behavioral. Behavioral therapies have also been shown to effectively manage complex chronic somatic symptoms, particularly pain. The behavioral approach involves bringing attention to physiological symptoms, the individuals attribution to those symptoms, and the subsequent anxiety produced by the negative attributions (Looper & Kirmayer, 2002).

8.5.2. Psychopharmacology

Psychopharmacological interventions are rarely used due to the possible side effects and unknown efficacy. Given that these individuals already have a heightened reaction to their physiological symptoms, there is a high likelihood that the side effects of a medication would produce more harm than help. With that said, psychopharmacological interventions may be helpful for those individuals who have comborbid psychological disorders such as depression or anxiety, which may negatively impact their ability to engage in psychotherapy (McGeary, Harzell, McGeary, & Gatchel, 2016).

8.6. Psychological Factors Affecting Other Medical Conditions

Section Learning Objectives

- Describe how psychological factors affecting other medical conditions presents itself.
- List and describe the most common types of psychophysiological disorders.
- Describe treatment options for psychological factors affecting other medical conditions.

Although previously identified as psychosomatic disorders, the DSM-5 has identified physical illnesses that are caused or exacerbated by biopsychosocial factors as *psychological factors affecting other medical conditions.* This disorder is different than all the previously mentioned somatic related disorders as the primary focus of the disorder is not the mental disorder, but rather the physical disorder. It is believed that lack of positive coping strategies, psychological distress, or maladaptive health behaviors exacerbate these physical symptoms (McGeary, Harzell, McGeary, & Gatchel, 2016).

8.6.1. Psychophysiological Disorders

The most common types of psychophysiological disorders are: headaches (migraines and tension), Gastrointestinal (ulcer and irritable bowel), Insomnia, and Cardiovascular related disorders (coronary heart disease and hypertension). We will briefly review these disorders and discuss the associated psychological features believed to exacerbate symptoms.

8.6.1.1. Headaches. Among the most common types of headaches are **migraines** and **tension headaches** (Williamson, 1981). Migraine headaches are often more severe and are explained by a throbbing pain localized to one side of the head. It is often accompanied by nausea, vomiting, sensitivity to light, and vertigo. It is believed that migraines are caused by the blood vessels in the brain narrowing, thus reducing the blood flow to various parts of the brain, followed by the same vessels later expanding, thus rapidly changing the blood flow. It is estimated that 23 million people in the US alone suffer from migraines (Williamson, Barker, Veron-Guidry, 1994).

Tension headaches are 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. Unlike migraines, nausea, vomiting, and sensitivity to light do not often occur with tension headaches. Tension headaches, as well as migraines, are believed to be largely caused by stress as they are in response to sustained muscle contraction that is often exhibited by those under extreme stress or emotion (Williamson, Barker, Veron-Guidry, 1994). In efforts to reduce the frequency and intensity of both migraines and tension headaches, individuals have found relief in relaxation techniques, as well as the use of biofeedback training to help encourage the relaxation of muscles.

8.6.1.2. Gastrointestinal. Among the two most common types of gastrointestinal psychophysiological disorders are **ulcers** and **irritable bowel syndrome (IBS).** 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. Among the most common types of ulcers are peptic ulcers, which is caused by the bacteria *H. pylori* (Sung, Kuipers, El-Serag, 2009). While there is evidence to support the involvement of stress in the development of dyspeptic symptoms, the evidence to support the involvement of stress in peptic ulcers is slowly growing. (Purdy, 2013). Researchers believe that while *H. pylori* must be present for a peptic ulcer to develop, increased stress levels may impact the amount of digestive acids present in the stomach lining, thus increased the frequency and intensity of symptoms (Sung, Kuipers, El-Serag, 2009).

IBS is a chronic, functional disorder of the gastrointestinal tract. Common symptoms of IBS include abdominal pain and extreme bowel habits (diarrhea and/or constipation. It affects up to a quarter of the population and is responsible for nearly half of all referrals to gastroenterologists (Sandler, 1990).

Because IBS is a functional disorder, there are no known structural, chemical, or physiological abnormalities responsible for the symptoms. However, there is conclusive evidence that IBS symptoms are related to psychological distress, particularly in those with anxiety and/or depression. Although more research is needed to better determine the timing between the onset of IBS and psychological disorders, preliminary evidence suggests that psychological distress is present before IBS symptoms, and therefore, IBS may be best explained as a somatic expression of associated psychological problems (Sykes, Blanchard, Lackner, Keefer, & Krasner, 2003).

8.6.1.3. Insomnia. Insomnia, the difficult falling or staying asleep occurs in more than one-third of the US population, with approximately 10% of patients reporting chronic insomnia (Perlis & Gehrman, 2013). While exact pathways of psychophysiological chronic insomnia are unclear, there is evidences of some biopsychosocial factors that may predispose an individual to developing insomnia such as anxiety, depression, and overactive arousal systems (Trauer et al, 2015). Part of the difficulty with insomnia is the fact that these psychological symptoms can impact one’s ability to fall asleep; however, we also know that lack of adequate sleep also predisposes individuals to increased psychological distress. Due to this cyclic nature of psychological distress and insomnia, intervention for both sleep issues as well as psychological issues is important to managing symptoms.

8.6.1.4. Cardiovascular. Heart disease has been the leading cause of death in the United States for the past several decades. Costs related to disability, medical procedures, and societal burdens are estimated to be $444 billion a year (Purdy, 2013). With this large financial burden, there has been great efforts to identify risk and protective factors in predicting cardiovascular mortality.

Researchers have identified that depression is a predictor of early onset **coronary heart disease**

(Ketterer, Knysk, Khanal, & Hudson, 2006). More specifically, there is a five-fold increase of depression in those with coronary heart disease than the general population (Ketterer, Knysk, Khanal, & Hudson, 2006). Additionally, anxiety and anger have also been identified as early predictor of cardiac events, suggesting psychological interventions aimed at reducing anxiety and establishing positive coping strategies for anger management may be effective in reducing future cardiac events (Ketterer, Knysk, Khanal, & Hudson, 2006).

8.6.1.5. Hypertension. Also called or chronically elevated blood pressure, is also found to be effected by psychological factors. More specifically, constant stress, anxiety, and depression have all been found to impact the likelihood of a cardiac event due to their impact on vasoconstriction (Purdy, 2013). Elevated inflammatory markers such as C-reactive protein, which is indicative of plaque instability, has been found in chronically depressed individuals thus predisposing them to potential heart attacks (Ketterer, Knysk, Khanal, & Hudson, 2006).

8.6.2. Treatments for Psychological Factors Affecting Other Medical Conditions

As more information regarding contributing factors to psychophysiological disorders is discovered, more psychological treatment approaches have been developed and applied to these medical problems. The most common types of treatments include relaxation training, biofeedback, hypnosis, traditional CBT treatments, group therapy, as well as a combination of the previous treatments.

8.6.2.1. Relaxation training. Relaxation training essentially teaches individuals how to relax their muscles on command. While relaxation is used in combination with other psychological interventions to reduce anxiety (as seen in PTSD and various anxiety related disorders), it has also been shown to be effective in treating physical symptoms such as headaches, chronic pain, as well as pain related to specific causes (ie. injection sites, side effects of medications, etc; McKenna et al., 2015).

8.6.2.2. Biofeedback. Biofeedback is a unique psychological treatment in which an individual is connected to a machine (usually a computer) that allows for continuous monitoring of involuntary physiological reactions. Measurements that can be obtained are heart rate, galvanic skin response, respiration, muscle tension, and body temperature to name a few.

There are a few different ways in which biofeedback can be administered. The first is clinician led. The clinician will actively guide the patient through a relaxation monologue, encouraging the patient to relax muscles associated near the pain region (or within the entire body). While going through the monologue, the clinician is provided with real time feedback about the patient's physiological response. Research studies have routinely supported the use of biofeedback, particularly for those with pain and headaches that have not been responsive to pharmacological interventions (McKenna et al., 2015).

Another option of biofeedback is through computer programs developed by psychologists. The most common, a program called Wild Devine (now Unyte) is an integrative relaxation program that encourages the use of breathing techniques while simultaneously measuring the patient's physiological responses. This type of programming is especially helpful for younger patients as there are various "games" the child can play that requires the awareness and control of their thoughts, feelings, and emotions.

8.6.2.3. Hypnosis. Hypnosis, which some argue is just an extreme sense of relaxation has been

effective in reducing pain and managing anxiety symptoms associated with medical procedures (Lang et al., 2000). Through extensive training, an individual can learn to engage in self-hypnosis or obtain recorded hypnosis monologues to assist with management of physiological symptoms outside of hypnosis sessions. While additional research is still needed within the field of hypnosis, studies have indicated that hypnosis is effective in not only treating chronic pain, but also assist with a reduction in anxiety, improved sleep, and improved overall quality of life. (Jensen et al., 2006).

8.6.2.4. Group Therapy. Group therapy is another effective treatment option for individuals with psychological distress related to physical disorders. These groups not only aim to reduce the negative emotions associated with chronic illnesses, but they also provide support from other group members that are experiencing the same physical and psychological symptoms. These groups are typically CBT based, and utilize cognitive and behavioral strategies in a group setting to encourage acceptance of disease while also addressing maladaptive coping strategies.

Module Recap

Well, that’s it. In Module 8, we discussed somatic disorders in terms of their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Somatic disorders included Somatic Symptom Disorder, Illness Anxiety Disorder, Conversion Disorder, and Factitious Disorder. We also discussed psychological factors affecting other medication conditions in relation to their clinical presentation, common types of psychophysiological disorders, and treatment. Module 9 will be our last in Part III of the course and will conclude Block 2. We will discuss obsessive-compulsive and related disorders. If you have questions, be sure to ask your instructor, and begin studying for your third exam if you have not already.

Module 9: Obsessive-Compulsive and Related Disorders

Module Overview

In Module 9, we will discuss matters related to obsessive-compulsive and related disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include obsessive compulsive disorder (OCD), body dysmorphic disorder (BDD), and hoarding. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 9.1. Clinical Presentation
- 9.2. Epidemiology
- 9.3. Comorbidity
- 9.4. Etiology
- 9.5. Treatment

Module Learning Outcomes

- Describe how obsessive-compulsive disorders present.
- Describe the epidemiology of obsessive-compulsive disorders.
- Describe comorbidity in relation to obsessive-compulsive disorders.
- Describe the etiology of obsessive-compulsive disorders.
- Describe treatment options for obsessive-compulsive disorders.

9.1. Clinical Presentation

Section Learning Objectives

- Describe how OCD presents itself.
- Describe how BDD presents itself.
- Describe how hoarding presents itself.

9.1.1. Obsessive Compulsive Disorder

Obsessive compulsive disorder, more commonly known as OCD, requires the presence of **obsessions, compulsions,** or both obsessions and compulsions together (the most common presentation of the disorder). Obsessions are defined as repetitive and persistent thoughts, urges, or images. These obsessions are intrusive, time consuming, and unwanted, often causing significant distress in an individual's daily functioning. Common obsessions are contamination (dirt on self or objects), errors of uncertainty regarding daily behaviors (locking 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 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), counting (i.e. flicking the lights off and on for 5 times), hand washing, symmetry, or repeating specific words. These compulsive behaviors essentially alleviate the anxiety associated with the obsessive thoughts. For example, an individual may feel as though their hands are dirty after using utensils at a restaurant. They may obsess over this thought for a period of time, impacting their ability to interact with others or complete a specific task. This obsession will ultimately lead to the individual performing a compulsion where they will wash their hands with extremely hot water to rid all the germs, or even wash their hands a specified number of times if they also have a counting compulsion. At this point, the individual's anxiety should 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. Additionally, they cause significant impairment in one's daily functioning. Given the example above, an individual with a fear of contamination may refuse to eat out at restaurants, or maybe bring their own utensils with them and insist on using them when they are not eating at home.

9.1.2. Body Dysmorphic Disorder

Body Dysmorphic Disorder (BDD) is another obsessive disorder, however, the focus of these obsessions are with ones perceived defects or flaws in their physical appearance. A key feature of these obsessions are that they are *not* observable to others. An individual who has a congenital facial defect or a burn victim who is concerned about their 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- such as acne, wrinkles, skin color, hair-particularly thinning or excessive body hair, or nose- size.

Due to the distressing nature of the obsessions regarding one's body, individuals with BDD also engage in compulsive behaviors that take up a considerable amount of time in one's day. For example, one may repeatedly compare their body to other people's bodies in the general public; repeatedly look at themselves in the mirror; engage in excessive grooming which includes using make-up to modify their appearance. Some individuals with BDD will go as far as having numerous plastic surgeries in attempts to obtain their "perfect" appearance. 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

their appearance (i.e on average 3-8 hours a day), as well as display significant impairment in social, occupational, or other areas of functioning.

9.1.2.1. 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).

9.1.3. Hoarding

Thanks to popular television shows, most of us have had some exposure to hoarding disorder, even if it has only been through commercials. In hoarding, the key feature is the *persistent* over accumulation of possessions. While we all obtain items throughout our life, individuals with hoarding disorder continue to accumulate items without discarding possessions, regardless of their value or sentiment. This lack of discarding occurs over a long period of time, and is not explained by a recent significant stressor (i.e. lost house in fire so now keeps everything). For example, last week's newspaper that had no relevance to me or any historical value- those with hoarding disorder would keep this newspaper despite the lack of value or sentiment.

The most common items that are hoarded are newspapers, magazines, clothes, bags, books, mail, and paperwork (APA, 2013). While these items may be stored in attics and garages, individuals with a hoarding disorder also have these items cluttering their living space, sometimes to the extent that they are unable to even utilize their furniture because it is covered in stuff. Cognitive factors contributing to the need to hold onto these non-sentimental items are fear of losing important information and fear of being wasteful. When asked to "clean out" their house or get rid of these items, individuals with hoarding disorder experience significant distress. One's hoarding behaviors also impacts their daily functioning, and causes impairment in social and occupational functioning.

9.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of OCD.
- Describe the epidemiology of BDD.
- Describe the epidemiology of hoarding.

9.2.1. OCD

The prevalence rate for OCD is approximately 1.2% both in the US, and worldwide (APA, 2013). Similar to other anxiety related disorders, women are diagnosed with OCD more often than males; however, in childhood, boys are actually 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).

9.2.2. BDD

The point prevalence rate for BDD among US 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 appears similar.

Gender based prevalence rates indicates that women are more likely to be diagnosed with BDD than males (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.

9.2.3. Hoarding

While national studies on the prevalence rate of hoarding within the US and Internationally are not available, surveys estimate clinically significant hoarding as occurring in 2-6% of the population (APA, 2013; Gilliam & Tolin, 2010). Epidemiological studies suggest that males report a higher incidence of hoarding behaviors; however, clinical samples are more highly represented by females. What does this mean? Either epidemiological studies are skewed, or, females seek out treatment for hoarding more often than males. It should be noted that older individuals (between ages 55-94) are three times more likely to be diagnosed with hoarding disorder than younger adults.

9.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of OCD.
- Describe the comorbidity of BDD.
- Describe the comorbidity of hoarding.

9.3.1. OCD

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, due to the nature of OCD and its symptoms, nearly 41% of those with OCD will also be diagnosed with a major depressive episode (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 psychological disorder triad, it is believed there is a neurobiological mechanism at fault for 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 patient diagnosed with one of the disorders above, should also routinely assess patients for OCD.

9.3.2. BDD

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). MDD 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).

9.3.3. Hoarding

Hoarding has an extremely high comorbidity rate with other mood and/or anxiety disorders, with approximately 75% of individuals meeting diagnostic criteria for either major depressive disorder, social anxiety disorder, or generalized anxiety disorder (APA, 2013). Additionally, nearly 20% also meet criteria for OCD, which is not surprising seeing the similarity in their etiology.

9.4. Etiology

Section Learning Objectives

- Describe the biological causes of obsessive compulsive disorders.
- Describe the cognitive causes of obsessive compulsive disorders.
- Describe the behavioral causes of obsessive compulsive disorders.

9.4.1. Biological

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

9.4.1.1. 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 patients diagnosed with OCD are at a 5-fold increase to develop OCD at some point throughout their lifespan (Nestadt, et al., 2000).

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).

Initial studies exploring genetic factors for BDD and hoarding also indicate a likely hereditary influence; however, environmental factors appear to play a larger role in the development of these disorders than that of OCD (Ahmed, et al., 2014; Lervolino et al., 2009).

9.4.1.2. 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 these patients report a significant reduction in their depressive symptoms, but also significant improvement in their OCD symptoms (Bokor & Anderson, 2014). Interestingly enough, antidepressant medications that do not effect 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).

9.4.1.3. 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 filters 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 over activity of the orbitofrontal cortex and a lack of filtering in the caudate nuclei, thus causing too many impulses 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).

9.4.2. 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 harm, control, or uncertainty in their life, thus leading them to over interpret potential negative outcomes of events. 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 to repeat the compulsive behavior multiple times to ensure it is done correctly. These cognitive biases are supported throughout research studies that repeatedly find 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 so often why does this happen? Everyone has times when they have repetitive or intrusive thoughts such as: "Did I shut 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 up on their thoughts once, or even forgo checking their thoughts 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 their 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).

9.4.3. Behavioral

The behavioral explanation of obsessive compulsive related disorders focuses on the explanation of compulsions rather than obsessions. Behaviorists believe that these compulsions begin with and are maintained by the **classical conditioning** theory. As you may remember, classical condition occurs

when an unconditioned stimulus is paired with a conditioned stimulus to produce a conditioned response. How does this 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 patients classical conditioning associated with the obsessions and compulsions by preventing the individual from engaging in the conditioned stimulus until anxiety is reduced.

9.5. Treatment

Section Learning Objectives

- Describe treatment options for OCD.
- Describe treatment options for BDD.
- Describe treatment options for hoarding.

9.5.1. OCD

9.5.1.1. Exposure and Response Prevention. Treatment of OCD has come a long way in the 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), as you might infer from the name, individuals are repeatedly exposed to their obsession, thus causing anxiety/fears, while simultaneously 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. For example, a fear that one's house would burn down if their compulsion was not carried out would obviously be done via imaginary exposure, as it would not be ethical to have a person burn their house down.

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

Within the hierarchy of obsessions, the individual is also gradually exposed to their obsession. For example, an individual obsessed with germs, may first watch a person sneeze on the computer in

session. Once anxiety is managed and compulsions are refrained 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 preventing them from engaging in their compulsive behavior. Once this level of their hierarchy was managed, they would move on to the next obsession and so forth until the entire list was complete.

Treatment outcome for 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 patients to commit to treatment, as the repeated exposures and prevention of compulsive behaviors can be extremely distressing to patients.

9.5.1.2. 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- clomipramine or SSRI's. Reportedly, up to 60% of patients 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 an added benefit (Foa et al., 2005).

9.5.2. BDD

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 patients 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 patients with BDD. Similar to OCD, medications such as clomipiaramine and other SSRIs are generally prescribed. While these are effective in reducing BDD symptoms, once 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 participations 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).

9.5.2.1. 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 deficits. Phillips and colleagues (2001) evaluated treatments of patients with BDD and found that 76.4% of the patients 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 psychological distress before completing any type of medical treatment.

9.5.3. Hoarding

Recent research has concluded that unlike OCD, many individuals with hoarding disorder do not experience intrusive thoughts, nor do they experience urges to perform rituals Because of this difference, treatment for hoarding disorder has moved away from exposure and response prevention, and more toward a traditional cognitive-behavioral approach.

Frost and Hartl (1996) believed that individuals with hoarding disorder engage in complex decision-making processes, overanalyzing the value and worth of possessions, thus leading to hoarding the possession as opposed to discarding it. Therefore, in addition to having the individual engage in exposure treatment, an added component of cognitive restructuring and motivational interviewing are added in efforts to address the complex-decision making that is involved in maintaining unnecessary possessions. By discussing motives for keeping items, as well as fears that may be associated with discarding items, clinicians can assist patients in their cognitive processes to ultimately determine the items actual worth (Williams & Viscusi, 2016). Unfortunately, due to the distressing nature of having to discard their possessions, many individuals in treatment for hoarding disorder prematurely end treatment, thus never reaching remission of symptoms (Mancebo, Eisen, Sibrava, Dyck, & Rasmussen, 2011).

Module Recap

As in all modules past, we have discussed the clinical presentation, epidemiology, comorbidity, etiology, and treatment options for a specific class of disorders – the obsessive compulsive and related disorders. This concludes Part III and the second block of disorders and an exam will now follow. In our next block of disorders we will cover eating and substance-related and addictive disorders.

Part IV. Mental Disorders – Block 3

Part IV. Mental Disorders – Block 3

Module 10: Eating Disorders

Module Overview

In Module 10, we will discuss matters related to eating disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include anorexia nervosa, bulimia nervosa, and binge eating disorder. We will also discuss changes in how eating disorders are classified from the DSM IV-TR to the DSM 5. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 10.1. Clinical Presentation
- 10.2. EDNOS and Changes from DSM IV-TR to DSM 5
- 10.3. Epidemiology
- 10.4. Comorbidity
- 10.5. Etiology
- 10.6. Treatment

Module Learning Outcomes

- Describe how eating disorders present.
- Describe the epidemiology of eating disorders.
- Describe comorbidity in relation to eating disorders.
- Describe the etiology of eating disorders.
- Describe treatment options for eating disorders.

10.1. Clinical Presentation

Section Learning Objectives

- Describe how Anorexia Nervosa presents itself.
- Describe how Bulimia Nervosa presents itself.
- Describe how Binge-Eating Disorder (BED) presents itself.

Eating disorders are very serious, yet relatively common mental health disorders, particularly in the Western society where there is a heavy emphasis on thinness and physical appearance. In fact, 13% of adolescents will be diagnosed with at least one eating disorder by their 20th birthday (Stice, Marti, & Rohde, 2013). Furthermore, a large number of adolescents will engage in significant disordered eating behaviors just below the clinical threshold (Culbert, Burt, McGue, Iacono & Klump, 2009).

While there is no exact cause for eating disorders, the combination of biological, psychological and sociocultural factors have been identified as major contributors in both the development and maintenance of eating disorders. This chapter serves as an introduction to three of the most common eating disorders, their etiology, and treatment.

Within the DSM-5 (APA, 2013) there are six disorders classified under the Feeding and Eating Disorders section: Pica, Rumination Disorder, Avoidant/Restrictive Food Intake Disorder, Anorexia Nervosa, Bulimia Nervosa, and Binge-Eating Disorder. For the purpose of this class, we will cover the latter three.

Diagnostic criteria for Eating Disorders is **mutually exclusive**, meaning that only one of these diagnoses can be assigned at any given time, with the exception of Pica, which can be given as a diagnosis along with any of the aforementioned eating disorders. Given how similar many eating disorders may present, it is important to routinely review diagnostic criteria to ensure the most appropriate diagnosis has been made.

10.1.1. **Anorexia Nervosa**

Anorexia nervosa involves the *restriction* of food which leads to significantly low body weight relative to the individual's age, sex, and development. This restriction is often secondary to an intense fear of gaining weight or becoming fat, despite the individual's low body weight. Altered perception of self and an over evaluation of one's body weight and shape contribute to this disturbance of body size (National Eating Disorder Association).

Typical warning signs/symptoms of an individual with anorexia nervosa are divided into two different categories- Emotional/Behavioral and Physical. Some emotional and behavioral symptoms include: dramatic weight loss, preoccupation with food, weight, calories, etc., frequent comments about feeling "fat", eats a restricted range of foods, makes excuses to avoid mealtimes, and often does not eat in public. Physical changes may include dizziness, difficulty concentrating, feeling cold, sleep problems, fine hair/hair loss, and muscle weakness to name a few.

The onset of disorder typically begins with mild dietary restrictions- eliminating carbs, or specific fatty foods. As weight gain is prevented, the dietary restrictions progress to more severe restrictions-e.g. under 500 calories/day. While symptoms typically present in mid-teenage years, there is a noticeable trend of younger girls- as young as 8 years old- who exhibit extreme dietary restrictive behaviors. While males are not immune from this disorder, the number of females diagnosed each year is overwhelmingly larger than that of males.

10.1.2. Bulimia Nervosa

Unlike Anorexia nervosa where there is solely restriction of food, bulimia nervosa involves a pattern of

recurrent binge eating behaviors. **Binge eating** can be defined as a discrete period of time where the amount of food consumed is significantly more than most people would eat during a similar time period. Individual's with bulimia nervosa often report a sense of lack of control over eating during these binge eating episodes. While not always, these binge eating episodes are often followed by a feeling of disgust with oneself, which leads to a **compensatory behavior** in attempts to rid the body of the excessive calories. These compensatory behaviors include vomiting, use of laxatives, fasting (or severe restriction), or excessive exercise. This cycle of binge eating and compensatory behaviors occurs on average, at least once a week for 3 months (National Eating Disorder Association).

It is important to note that while there are periods of severe calorie restriction like anorexia, the two disorders cannot be diagnosed simultaneously. Therefore, it is important to determine if an individual engages in a binge eating epsiode- if they do, they do *not* meet criteria for anorexia nervosa.

Sings and symptoms of bulimia nervosa are similar to anorexia nervosa. These symptoms include but are not limited to: hiding food wrappers or containers after a bingeing episode, feeling uncomfortable eating in public, developing food rituals/limited diet, disappearing to bathroom after eating meal, and drinking excessive amounts of water or non-caloric beverages. Additional physical changes include weight fluctuations- both up and down, difficulty concentrating, dizziness, sleep disturbance, and possible dental problems due to purging post binge eating episode.

Symptoms of bulimia nervosa typically present later in development- late adolescence or early adulthood. Similar to anorexia nervosa, bulimia nervosa initially presents with mild restrictive dietary behaviors; however, episodes of binge eating interrupt the dietary restriction, causing body weight to rise around normal levels. In response to weight gain, patients then being to engage in compensatory behaviors or purging episodes to reduce body weight. This cycle of restriction, binge eating, and calorie reduction often occurs for years prior to seeking help.

10.1.3. Binge-Eating Disorder (BED)

Binge-Eating Disorder is similar to Bulimia Nervosa in that it involves recurrent binge eating episodes along with feelings of lack of control during the binge eating episode; however, these episodes are *not* followed by a compensatory behavior to rid the body of calories. Despite the feelings of shame and guilt post binge, individual's with BED will not engage in vomiting, excessive exercises or other compensatory behaviors. These binge eating episodes occur on average, at least once a week for 3 months.

Because these binge eating episodes occur without compensatory behaviors, individuals with BED are at risk for obesity and related health disorders. Individual's with BED report feelings of embarrassment at the quantity of food consumed, and thus will often refuse to eat in public. Due to the restriction of eating around others, individuals with BED often engage in secret binge eating episodes in private, followed by discrete disposal of wrappers and containers.

While much is still being researched about binge-eating disorder, current research indicates that onset of BED is later than that of anorexia nervosa and bulimia nervosa. Most patients are middle-aged and approximately one third or more are male. Binge-eating disorder also appears to be more phasic rather than persistent, with individuals experiencing significant time periods where their binge-eating episodes are in control. The gender discrepancy in BED is much smaller than that of anorexia nervosa

and bulimia nervosa.

10.2. EDNOS and Changes from DSM IV-TR to DSM 5

Section Learning Objectives

- Outline changes in the DSM 5 in relation to eating disorders.

On a global scale, the new section- Feeding and Eating Disorders in the DSM-5- now covers all eating disorders as well as diagnoses previously belonging to 'Feeding and Eating Disorders of Infancy and Early Childhood.' The primary reason behind combining the two chapters was to decrease the frequency of individuals diagnosed with Eating Disorder- Not Otherwise Specified (EDNOS). By combining all eating/feeding disorders into one section, as well as making adjustments to individual diagnoses, the goal is to better identify eating disorder diagnoses.

10.2.1. EDNOS

The most notable change to the specific disorders within the eating disorder chapter is the elimination of Eating Disorder- Not Otherwise Specified (EDNOS). One of the main issues with the DSM-IV was the high rates of EDNOS diagnoses- ranging anywhere from 50% to 90% of all individuals seeking treatment for eating disorders in clinical settings (Call, Walsh & Attia, 2013). Given these high numbers, it was evident that revisions were needed to specific diagnoses (anorexia nervosa, bulimia nervosa) as well as the EDNOS diagnosis. How could over half of individuals presenting with an eating disorder be categorized as 'not otherwise specified'? Therefore, Binge-Eating Disorder was established as a separate eating disorder, followed by small changes to anorexia nervosa and bulimia nervosa.

10.2.2. BED

As previously stated, BED was couched under EDNOS prior to the DSM-5. Although it is now its own diagnosis, there was one modification to diagnostic criteria- changes to the frequency and duration of binge eating episodes. The DSM-IV-TR required binge eating episodes to occur at least twice per week for 6 months or more, whereas the DSM-5 only requires binge eating episodes to occur at least once per week for 3 months or more (Call, Walsh & Attia, 2013). While reducing the frequency and duration of binge eating episodes does not significantly increase the prevalence rate of those with BED, it does better classify individuals exhibiting binge-eating behaviors, thus eliminating the need for an EDNOS category.

10.2.3. Anorexia Nervosa

Anorexia nervosa also underwent several changes, with the most notable being the removal of the criteria that the individual must be under 85% of normal body weight. Not only was this criteria an issue for diagnosing individuals (what happens if they are at 87% below normal body weight?) but also for insurance companies. The new criteria requires the individual to be at 'low weight- less than minimally normal weight in adults (and minimally expected weight in adolescents and children; Call, Walsh & Attia, 2013).

Additionally, the anorexia nervosa diagnosis also removed the requirement of amenorrhea. This was another criteria that was often not met in individuals presenting with all other anorexia nervosa diagnostic criteria. The Eating Disorders Work Group identified several studies that suggest that women who met criteria for anorexia nervosa but still maintained menstrual cycles did not differ clinically from those that did not maintain their menstrual cycle ?(Call, Walsh & Attia, 2013). Additionally, by removing this criteria, it is also allows for inclusion of adolescents who may not have reached menarche, as well as men.

10.2.4. Bulimia Nervosa

Similar to that of BED, bulimia nervosa maintained its core criteria with the exception of the frequency and duration of binge eating episodes and compensatory behaviors. The DSM-5 requires binge eating and compensatory behaviors to occur a minimum of one time per week, on average over a 3-month period. Again, research supported the reduction in these behaviors as individuals exhibiting behaviors at higher frequencies and lower frequencies did not differ significantly in terms of clinical presentation and treatment response (Call, Walsh & Attia, 2013).

10.3. Epidemiology

Section Learning Objectives

- Describe the epidemiology of eating disorders.

According to the DSM-5 (APA, 2013), the prevalence rate for anorexia nervosa among young women is 0.4% whereas the prevalence rate for bulimia nervosa is 1%-1.5%. While BED is still a relatively new diagnosis, the estimated prevalence rate in females is 1.6%. Prevalence rates for males with anorexia or bulimia is unknown; however, research suggests the female-to-male ration is approximately 10:1 for both disorders (APA, 2015). Estimated prevalence rates for BED in males is 0.8%. The ration between females-to-males with BED is much less skewed than that in anorexia and bulimia.

10.4. Comorbidity

Section Learning Objectives

- Describe the comorbidity of anorexia nervosa.
- Describe the comorbidity of bulimia nervosa.
- Describe the comorbidity of BED.

10.4.1. Anorexia

Anorexia is rarely a single diagnosis. High rates of bipolar disorder, depressive symptoms, and anxiety disorders are also common among individuals with anorexia nervosa. Obsessive compulsive disorder is more often seen in those with restrictive type of anorexia nervosa, whereas alcohol use disorder and other substance use disorders are more commonly seen in those with anorexia who engage in binge-eating/purging behaviors. Unfortunately, there is also a high rate of suicidality, as many as 12 per 100,000 per year (APA, 2013).

10.4.2. Bulimia

Majority of individual's diagnosed with bulimia nervosa also present with at least one other mental disorder, if not more. Similar to anorexia nervosa, there is also a high frequency of depressive symptoms, as well as bipolar disorder. While some experience mood fluctuations as a result of their eating pattern, a large number of individuals will identify mood symptoms prior to the onset of bulimia nervosa (APA, 2013). Anxiety, particularly social anxiety is often present in those with bulimia nervosa. It should be noted that most mood and anxiety symptoms are resolved once effective treatment of bulimia is established. Alcohol use, as well as substance abuse is also prevalent in those with bulimia. The substance abuse tends to begin as a compensatory behavior- stimulant use is used to control appetite and weight- and over time, as the eating disorder progresses, so does the substance abuse. Finally, there is also a percentage of individuals with bulimia nervosa who also display personality characteristics consistent with a range of personality disorders.

10.4.3. BED

Seeing as binge-eating disorder is a new diagnosis, research regarding comorbidity with other mental disorders is still developing. Preliminary evidence suggests that binge-eating disorder shares similar comborbidities with anorexia nervosa and bulimia nervosa. Common comorbidities include (but are not limited to) bipolar disorder, depressive disorders and anxiety disorders. Although there is some evidence of substance abuse disorder, it is not as prevalent as that in bulimia nervosa and anorexia nervosa.

10.5. Etiology

Section Learning Objectives

- Describe the biological causes of eating disorders.
- Describe the cognitive causes of eating disorders.
- Describe the sociocultural causes of eating disorders.
- Describe how personality traits are the cause of eating disorders.

What causes eating disorders? While researchers have yet to identify a specific cause of eating disorders, the most compelling argument to date is that eating disorders are **multidimensional disorders**. This means there are many contributing factors that lead to the development of an eating disorder. While there is likely a genetic predisposition, there are also environmental, or external factors, such as family dynamics and cultural influences that impact the presentation of an eating disorder. Research supporting these influences is well documented for anorexia nervosa and bulimia nervosa; however, seeing as BED has only just recently been established as a formal diagnosis, research on the evolvement of BED is ongoing.

10.5.1. Biological

There is some evidence of a genetic predisposition to eating disorders, with relatives of those diagnosed with an eating disorder are up to six times more likely than other individuals to be diagnosed with an eating disorder (APA, 2013). Twin concordance studies also support the gene theory. If an identical twin is diagnosed with anorexia, there is a 70 percent chance the other twin will develop an anorexia in their lifetime (APA, 2013). The concordance rate for fraternal twins (who share less genes) is 20 percent. While not as strong for bulimia cases, identical twins still display a 23 percent concordance rate, compared to the 9 percent fraternal twins rate (APA, 2013).

In addition to hereditary causes, disruption in the neuroendocrine system is common in those with eating disorders (Culbert, Racine, & Klump, 2015). Unfortunately, it's difficult for researchers to determine if these disruptions *caused* the disorder, or are in fact are caused by the disorder as manipulation of eating patterns is known to cause changes in hormone production. With that said, researchers have explored the **hypothalamus** as a potential contributing factor. The hypothalamus is responsible for regulating body functions- particularly hunger and thirst (Fetissov & Mequid, 2010). Within the hypothalamus, the lateral hypothalamus is responsible for initiating hunger cues, causing the organism to eat, whereas the ventromedial hypothalamus is responsible for sending signals of satiation, telling the organism to stop eating. Clearly a disruption in either of these structures could explain why an individual may not take in enough caloric intake, or experience periods of eating excessively.

10.5.2. Cognitive

Some argue that eating disorders are in fact a variant of Obsessive Compulsive Disorder (OCD). The obsession with body shape and weight- the hallmark of an eating disorder- is likely a driving factor in anorexia nervosa. Distorted thought patterns and an over-evaluation of body size likely contribute to this obsession and one's desire for thinness. Research has identified high levels of Impulsivity, particularly in those with binge eating episodes, suggesting a temporary lack of control is responsible for these episodes. Post binge-eating episode, many individuals report feelings of disgust or even thoughts of failure. These strong cognitive factors are indicative as to why cognitive-behavioral therapy is the hallmark treatment for eating disorders.

10.5.3. Sociocultrual

Eating disorders are overwhelmingly found in Western countries where there is a heavy emphasis on thinness- a core feature of eating disorders. It is also found in countries where food is in abundance as in places of deprivation, round figures are more desirable (Polivy & Herman, 2002). While eating disorders were once thought of as disorder of higher SES, more recent research suggests that as our country becomes more homogenized, the more universal eating disorders become.

10.5.3.1. Media. One commonly discussed contributor to eating disorders is the media. Idealizing thin models and actresses sends the message to young women (and adolescents) that in order to be popular and attractive, you must be thin. These images are not isolated to magazines, but are also seen in television shows, movies, commercials, and large advertisements on billboards and hanging in store windows. With the immergence of social media (i.e. facebook, snapchat, Instagram), exposure to media images and celebrities is even easier. Couple this with the ability to alter images to make individuals even thinner, it is no wonder many young women (and men) become dissatisfied with their own weight and shape (Polivy & Herman, 2004).

10.5.3.2. Ethnicity. While eating disorders are not solely a "white woman" disorder, there are significant discrepancies when it comes to race, especially for anorexia nervosa. Why is this? Research indicates that black men prefer heavier women than do white men (Greenberg & Laporte, 1996). Given this preference, it should not be surprising that black women and children have larger ideal physiques than their white peers (Polivy & Herman, 2000). Since black women are less driven to thinness, black women are less likely to develop anorexia; however, more recent studies suggest that this may not remain. Caldwell and colleagues (1997) found that high-income black women were equally as dissatisfied as high-income white women with their physique, suggesting body image issues may be more closely related to SES than that of race. The race discrepancies are also less significant in BED, where the prominent feature of the eating disorder is not thinness (Polivy & Herman, 2002).

10.5.3.3. Gender. Males account for only a small percentage of eating disorders- roughly 5-10% (APA, 2013). While it is unclear as to why there is such a discrepancy, it is likely somewhat related to cultural desires of women being "thin" and women being "muscular" or "strong."

Of men diagnosed with an eating disorder, the overwhelming percentage of them identified a job or sport team as the primary reason for their eating behaviors (Strother, Lemberg, Stanford, & Turberville, 2012). Jockeys, distance runners, wrestlers, and body builders are some of the professions identified as most restrictive in body weight.

There is some speculation that males are not diagnosed as frequently as women due to the stigma attached to eating disorders. Eating disorders have routinely been characterized as a “white, adolescent female” problem. Due to this bias, young men may not seek help for their eating disorder in efforts to prevent labeling (Raevuoni, Keski-Rahkonen & Hoek, 2014).

10.5.3.4. Family. Family influences are one of the strongest external contributors to maintaining eating disorders. Often family members are praised/reinforced for their slenderness. Think about the last time you saw a family member or close friend- how often have you said, “you look great!” or commented on their appearance in some way? Odds are it is pretty high. While the intent of the family member is not to maintain maladaptive eating behaviors, by praising the physical appearance of someone struggling with an eating disorder, you are indirectly perpetuating the disorder.

While family involvement can help maintain the disorder, it can also contribute to the development of one as well. Families that emphasize thinness or place a large emphasis on physical appearance are more likely to have a child diagnosed with an eating disorder (Zerbe, 2008). In fact, mothers with eating disorders are more likely to have children who develop some type of feeding/eating disorder than mother’s without eating disorders (Whelan & Cooper, 2000). Additional family characteristics that are common in adolescents that present for treatment of eating disorders are: enmeshed, intrusive, critical, hostile, or overly concerned with parenting (Polivy & Herman, 2002). It should be noted that while there has been some correlation between these family dynamics and eating disorders, they are not evident in all families of people with eating disorders.

10.5.4. Personality

There are many personality characteristics that are common in individuals with eating disorders. While it is unknown if these characteristics are inherent in the individual’s personality, or a product of personal experiences, the thought is eating disorders develop due to the combination of the two.

10.5.4.1. Perfectionism. It should come as no surprise that perfectionism, or the belief that one must be perfect, is a contributing factor to disorders (particularly anorexia nervosa) related to eating, weight, and body shape. While an exact mechanism is unknown, it is believed that perfectionism magnifies normal body imperfections, leading an individual to go to excessive (i.e. restrictive) behaviors to remedy the imperfection (Hewitt, Flett, Ediger, 1995).

10.5.4.2. Self-Esteem. Self-esteem, or one’s belief in their own worth or ability, has routinely been identified as a moderator of many psychological disorders, and eating disorders are no exception. Low self-esteem not only contributes to the development of an eating disorder, but is also likely involved in the maintenance of the disorder. One theory, the **transdiagnostic model** of eating disorders, suggests that overall low self-esteem increases the risk for over-evaluation of body, which in turn, leads to negative eating behaviors that could lead to an eating disorder (Fairburn, Cooper, Shafran, 2003).

10.6. Treatment

Section Learning Objectives

- Describe treatment options for anorexia nervosa.
- Describe treatment options for bulimia nervosa.
- Describe treatment options for binge eating disorder.
- Discuss the outcome of treatment for eating disorders.

10.6.1. Anorexia

The immediate goal for treatment of anorexia nervosa is weight gain and recovery from malnourishment. This is often established via an intensive outpatient program, or if needed, through an inpatient hospitalization program where caloric intake can be managed and controlled. Both the inpatient and outpatient programs use a combination of therapies and support to help restore proper eating habits. Of the most common (and successful) treatments are Cognitive-Behavioral Therapy (CBT) and Family-Based Therapy (FBT).

10.6.1.1. CBT. Because anorexia nervosa requires changes to both eating behaviors as well as thought patterns, CBT strategies have been very effective in producing lasting changes to those suffering from anorexia nervosa. Some of the behavioral strategies include recording eating behaviors- hunger pains, quality and quantity of food, as well as emotional behaviors- feelings related to the food. In addition to these behavioral strategies, it is also important to address the maladaptive thought patters associated with their negative body image and desire to control their physical characteristics. Changing the *fear* associated with gaining weight is essential in recovery.

10.6.1.2. FBT. FBT is also an effective treatment approach, often used as a component of individual CBT, especially for children and adolescents with the disorder. FBT is an effective treatment, with reports of up to 50-60% of weight restoration in one year, as well as weight maintenance 2-4 years post treatment (Campbell & Peebles, 2014; LeGrange, Lock, Accurso, Agras, Darcy, Forsberg, et. al, 2014). Additionally, FBT has been shown to improve rapid weight gain, produce less hospitalizations, and is more cost effective than other types of therapies with family involvement (Agras, Lock, Brandt, Bryson, Dodge, Halmi, et al., 2014).

FBT typically involves 16-18 sessions which are divided into 3 phases: (1) Parents taking charge of weight restoration, (2) clients gradual control over eating, and (3) address developmental issues including fostering autonomy from parents (Chen, et al., 2016). While FBT has shown to be effective in treating adolescents with anorexia nervosa, the application for older eating patients (i.e. college students and above) is still undetermined. As with adolescents, the goal for a family based treatment program should be centered around helping the patient separate their feelings/needs from that of their family.

10.6.2. Bulimia

Just as anorexia nervosa treatment’s initial focus is on weight gain, bulimia nervosa’s initial goal of

treatment is to eliminate binge eating episodes, as well as eliminate compensatory behaviors. The goal is to replace both of these negative behaviors with positive eating habits. One of the most effective ways to establish this is through Cognitive Behavioral Therapy (CBT).

10.6.2.1. CBT. Similar to anorexia nervosa, individuals with bulimia nervosa are expected to keep a journal of their eating habits; however, with bulimia nervosa, it is also important that the journal include changes in sensations of hunger and fullness, as well as other feelings surrounding their eating patterns in efforts to identify triggers to their binging episodes (Agras, Fitzsimmons-Craft & Wilfley, 2017). Once these triggers are identified, psychologists will utilize specific behavioral or cognitive techniques to prevent the individual from engaging in binge episodes or compensatory behaviors.

One way this is done is through *Exposure and Response Prevention.* As previously discussed in the OCD chapter, this treatment is very effective in helping individuals stop performing their compulsive behaviors by literally preventing them from engaging in the behavior, while simultaneously engaging in relaxation strategies to reduce anxiety associated with not engaging in the negative behavior. Therefore, to prevent an individual from purging post-binge episode, the individual would be encouraged to partake in an activity that directly competes with their ability to purge- i.e., write their thoughts and feelings in a journal at the kitchen table. Research has indicated that this treatment is particularly helpful for individuals suffering from comorbid anxiety disorders (particularly OCD; Agras, Fitzsimmons-Craft & Wilfley, 2017).

In addition to changing behaviors, it's also important to change the maladaptive thoughts toward food, eating, weight, and shape. Negative thoughts such as "I am fat" and "I can't stop eating when I start" can be modified into more appropriate thoughts such as "My body is healthy" or "I can control my eating habits." By replacing these negative thoughts with more appropriate, positive thought patterns, individuals begin to control their mind, which in return, can help them control their behaviors.

10.6.2.2. Interpersonal Psychotherapy (IPT). IPT has also been established as an effective treatment for those with bulimia nervosa, particularly if an individual has not been successful with CBT treatment. The goal of IPT is to improve interpersonal functioning in those with eating disorders. Originally a treatment for depression, IPT-E was adapted to address the social isolation and self-esteem problems that contribute to the maintenance of negative eating behaviors.

IPT-E has 3 phases, which are typically covered in weekly sessions over the course of 4-5 months. *Phase One* consists of engaging the patient in treatment, and providing psychoeducation about their disease and the treatment program. This phase also includes identifying interpersonal problems that are maintaining the disease.

Phase Two is the main treatment component. In this phase, the primary focus is on problem solving the interpersonal issue. The most common types of interpersonal issues are: lack of intimacy and interpersonal deficits, interpersonal role disputes, role transitions, grief, and life goals. Once the primary interpersonal problem is identified, the clinician supports the patient in their pursuit to identify ways to change. A key component of IPT-E is the supportive role of the clinician, as opposed to the teaching role in other treatments. The idea is that by having the patient make changes, they are able to better understand their problems, and as a result, can make more profound changes (Murphy, Straebler, Basden, Cooper, & Fairburn, 2012).

Phase Three is the final stage. The goals of this phase are to ensure that the changes made in phase two are maintained. To achieve this, treatment sessions are spaced out, allowing patients more time to

engage in their changed behavior. Additionally, relapse prevention (i.e. problem solving ways *not* to relapse) is also discussed to ensure long term results. In doing this, the patient reviews the progress they have made over the course of treatment, as well as identifying potential interpersonal issues that may arise, and how their treatment can be adapted to address those issues.

Support for IPT-E is limited; however, two large studies suggest that IPT-E is effective in treating bulimia nervosa, and possibly BED. While treatment is initially slower than CBT, it is equally effective in long-term follow-up and maintenance of disorder (Fairburn, Marcus, & Wilson, 1993).

10.6.3. Binge Eating Disorder

Given the similar presentations of BED and bulimia nervosa, it should not be surprising that the most effective treatments for BED is similar to that of bulimia nervosa. CBT, along with antidepressant medications are among the most effective in treating BED. Interpersonal therapy, as well as dialectical behavioral therapy have also been effective in reducing binge-eating episodes, however, they have not been effective in weight loss (Guerdjikova, Mori, Casuto, & McElroy, 2017). Goals of treatment are of course to eliminate binge eating episodes, as well as reduce body weight as most individuals with BED are overweight. Seeing as BED has only recently been established as its own separate eating disorder, treatment research specific to this disorder is expected to grow.

10.6.3.1. Antidepressant medications. Given the high comborbidity between eating disorders and depressive symptoms, antidepressants have been a primary method of treatment for years. While they have been shown to improve depressive symptoms, which may help individuals make gains in their eating disorder treatment, research has not supported antidepressants as an effective treatment strategy for treating the eating disorder itself.

10.6.4. Outcome of Treatment

Now that we have discussed treatments for eating disorders, how effective are they? Research has indicated favorable prognostic features for anorexia nervosa are early age of onset and short history of the disorder. Conversely, unfavorable features are long history of symptoms prior to treatment, severe weight loss, and binge eating and vomiting. The mortality rate over the first 10 years from presentation is about 10% (APA, 2013). Majority of these deaths are from medical complications due to the disorder, or suicide.

Unfortunately, research has not identified any consistent predictors of positive outcomes for bulimia nervosa. However, there is some speculation that individuals with childhood obesity, low self-esteem, and those with a personality disorder have worse treatment outcomes (APA, 2013).

While treatment outcome for BED is still in it’s infancy, initial findings suggest that remission rates of BED are much higher than that for anorexia nervosa and bulimia nervosa.

Module Recap

Module 10 covered eating disorders in terms of their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. We also discussed changes in the DSM from IV-TR to 5. This was the first disorder covered in Part IV and Block 3. In Module 11 we will discuss substance-related and addictive disorders which will conclude this part. Be sure you are preparing for Exam 4.

Module 11: Substance-Related and Addictive Disorders

Module Overview

In Module 11, we will discuss matters related to substance-related and addictive disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include substance intoxication, substance use disorder, and substance withdrawal. We also list substances people can become addicted to. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 11.1. Clinical Presentation
- 11.2. Epidemiology
- 11.3. Comorbidity
- 11.4. Etiology
- 11.5. Treatment

Module Learning Outcomes

- Describe how substance-related and addictive disorders present.
- Describe the epidemiology of substance-related and addictive disorders.
- Describe comorbidity in relation to substance-related and addictive disorders.
- Describe the etiology of substance-related and addictive disorders.
- Describe treatment options for substance-related and addictive disorders.

11.1. Clinical Presentation

Section Learning Objectives

- Define substances and substance abuse.
- Describe properties of substance abuse.

11.1.1. Substance Abuse

Substance-related disorders are among the most prevalent psychological disorders with roughly 100 million people in the United States reporting the use of an illegal substance sometime throughout their life (SAMHSA, 2014). While this disorder was previously classified as "drug abuse," the evolvement of the disorder has sparked abuse of other substances such as alcohol, tobacco, and caffeine, thus better classifying the disorder as abuse of **substances.**

What are substances? Substances are any ingested materials that cause temporary cognitive, behavioral, and/or physiological symptoms within the individual. These changes that are observed directly after or within a few hours of ingestion of the substance are classified as **substance intoxication** (APA, 2013). Substance intoxication symptoms vary greatly, and are dependent on the type of substance ingested. Specific substances and their effects will be discussed later in the chapter.

Repeated use of these substances, or frequent substance intoxication can develop into a long-term problem known as **substance abuse.** 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. The need to continually increase the amount of ingested substance is also known as **tolerance.** As tolerance builds, additional physical and psychological symptoms present, often causing significant disturbances in an individual's personal and/or professional life. Individuals with substance abuse are often spending a significant amount of time engaging in activities that revolve around their substance use, thus spending less time in recreational activities that once consumed their time. Sometimes, there is a desire to reduce or abstain from substance use, however, cravings and **withdrawal** symptoms often prohibit this from occurring on one's own attempts. Common withdrawal symptoms include but are not limited to: cramps, anxiety attacks, sweating, nausea, tremors, and hallucinations. Depending on the substance and the tolerance level, most withdrawal symptoms last anywhere from a few days to a week. For those with extensive substance abuse- or multiple substances being abused- withdrawal should be closely monitored in a hospital setting to avoid serious possible consequences such as seizures, stroke, or even death.

According to the DSM-5 (APA, 2013), an individual is diagnosed with *Substance Intoxication, Use*, and/or *Withdrawal* specific to the substance(s) the individual is ingesting. While there are some subtle differences in symptoms, particularly psychological, physical, and behavioral symptoms, the general diagnostic criteria for *Substance Intoxication, Use*, and *Withdrawal* remains the same across substances. Therefore, the general diagnostic criteria for *Substance Intoxication, Use*, and *Withdrawal* is reviewed below, with more specific details of psychological, physical, and/or behavioral symptoms in the Types of Substances Abused section.

For a diagnosis of *Substance Intoxication,* the individual must have recently ingested a substance (APA, 2013). Immediately following the ingestion of this substance, significant behavioral and/or psychological change is observed. In addition, physical and physiological symptoms present as a direct result of the substance ingested. As stated above, these behavioral, physical and physiological symptoms are dependent on the type of substance that is ingested and therefore, discussed in more detail within each substance category (i.e. depressants, stimulants, hallucinogens/cannabis/combination).

In order to meet criteria for *Substance Use Disorder*, an individual must experience significant impairment or distress over the course of 12-months due to their use of a substance (APA, 2013). Distress or impairment can be described as any of the following: inability to complete or lack of participation in work, school or home obligations/activities; increased time spent on activities obtaining, using, or recovering from substance use; impairment in social or interpersonal relationships; use of

substance in a potentially hazardous situation; psychological problems due to recurrent substance abuse; craving for substance; an increase in the amount of substance used over time (i.e. tolerance); difficulty reducing the amount of substance used despite desire to reduce/stop using the substance; and/or withdrawal symptoms (APA, 2013). While the number of these symptoms may vary among individuals, only two symptoms are required to be present for a diagnosis of a *Substance Use Disorder.*

Finally, *Substance Withdrawal* is diagnosed when there is cessation or reduction of a substance that has been used for a long period of time. Individuals undergoing substance withdrawal will experience physiological and/or psychological symptoms within a few hours after cessation/reduction (APA, 2013). These symptoms cause significant distress or impairment in daily functioning. Similar to *Substance Intoxication*, physiological and/or psychological symptoms during substance withdrawal are often specific to the substance abused and are discussed in more detail within each substance category later in the module.

11.1.2. Types of Substances Abused

The substances that are most often abused can be divided into three categories based on how they impact one's physiological state: depressants, stimulants, and hallucinogens/cannabis/combination.

11.1.2.1. Depressants. 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. Unfortunately, when used in large amounts, they can also impair an individual's judgment and motor activity.

While **alcohol** is one of the only legal (over the counter) substances we will discuss, it is also the most commonly consumed substance. According to the 2015 National Survey on Drug Use and Health, approximately 70% of individuals drank an alcoholic beverage in the last year and nearly 56% of individuals drank an alcoholic beverage in the past month (SAMHSA, 2015). While the legal age of consumption in the United States is 21, approximately 78% of teens report that they have drank alcohol at some point in their life (SAMHSA, 2013).

Despite the legal age of consumption, many college-aged students engage in binge or heavy drinking. In fact, 45% of college age students report engaging in binge drinking, with 14% engaging in binge drinking at least 5 days per month (SAMHSA, 2013). In addition to these high levels of alcohol consumption, college age students also engage in other behaviors such as skipping meals which can impact the rate of alcohol intoxication, as well as place them at risk for dehydration, blacking out, and developing alcohol induced seizures (Piazza-Gardner & Barry, 2013).

The "effective" substance of alcohol, *ethyl alcohol*, is a chemical that is absorbed quickly into the blood via the lining of the stomach and intestine. Once in the blood stream, ethyl alcohol travels to the central nervous system (i.e. brain and spinal cord) and produces *depressive* symptoms such as impaired reaction time, disorientation, and slurred speech. These symptoms are produced due to the ethyl alcohol binding to GABA receptors, thus preventing GABA from providing inhibitory messages and allowing the individual to relax (Filip et al., 2015).

The effect of ethyl alcohol in moderation allows for an individual to relax, engage more easily in conversation, and in general, produces a confident and happy personality. However, when consumption is increased or excessive in nature, the central nervous system is unable to adequately metabolize the ethyl alcohol, and negative effects begin to present. Symptoms such as blurred vision, difficulty walking,

slurred speech, slowed reaction time, and sometimes, aggressive behaviors are observed.

The extent to which these symptoms present are directly related to the concentration of ethyl alcohol within the body, as well as the individual's ability to metabolize the ethyl alcohol. There are a lot of factors that contribute to how quickly one's body can metabolize ethyl alcohol. Food, gender, body weight, and medications are among the most common factors that affect alcohol absorption (NIAAA,1997). More specifically, recent consumption of food, particularly that high in fat and carbohydrates, slows the absorption rate of ethyl alcohol, thus reducing its effects. With regards to gender, women absorb and metabolize alcohol differently than man, likely due to the smaller amount of body water and the lower activity of an alcohol metabolizing enzyme in the stomach. Another factor related to gender is body weight- with individuals with more body mass metabolizing the alcohol at a slower rate than those who weigh less. Finally, various medications, both over the counter and prescription based can impact the livers ability to metabolize alcohol, thus impacting the severity of symptoms that present (NIAAA, 1997).

Sedative-Hypnotic drugs, more commonly known as **anxiolytic drugs,** have a calming and relaxing effect on individuals. When used at a clinically effective amount, they can have a sedative effect, thus making them an appropriate drug for treating anxiety related disorders. In the early 1900's, **barbiturates** were introduced as the main sedative and hypnotic drug; however, due to their addictive nature, as well as respiratory distress when consumed in large amounts, they have been largely replaced by **benzodiazepines** which are considered a safer alternative as they have less addictive qualities (Filip et al., 2014)

Commonly prescribed benzodiazepines— Xanex, Ativan, and Valium—have a similar effect to alcohol as they too bind to the GABA receptors and increase GABA activity (Filip et al., 2014). This increase in GABA produces a sedative and calming effect. Benzodiazepines can be prescribed for both temporary (relief anxiety on flight or prior to surgery) or long-term use (generalized anxiety disorder). While they do not produce respiratory distress in large dosages like benzodiazepines, they can cause intoxication and addictive behaviors due to their effects on tolerance.

Opioids are naturally occurring, derived from the sap of the opium poppy. In the early 1800's, **morphine** was isolated from opium by German chemist Friedrich Wihelm Adam Serturner. Due to its analgesic effect, it was named after the Greek god of dreams, Morpheus (Brownstein, 1993). Its popularity grew during the civil war as it was the primary medication given to soldiers with battle injuries. Unfortunately, this is also when the addictive nature of the medication was discovered, as many soldiers developed "Soldier's Disease" as a response to tolerance of the drug (Casey, 1978).

In efforts to alleviate the addictive nature of morphine, **heroin** was synthesized by the German chemical company Bayer in 1898, and was offered in a cough suppressant (Yes, Bayer Promoted Heroin). For years, heroin remained in cough suppressants as well as other pain reducers until it was discovered that heroin was actually more addictive than morphine. In 1917, Congress identified that *all* drugs derived from opium were addictive, thus banning the use of opioids in over the counter medications.

Opioids are unique in that they provide both euphoria and drowsiness. Tolerance to these drugs builds quickly, thus resulting in an increased need of the medication to produce desired effects. This rapid tolerance is also likely responsible for opioids highly addictive nature. Opioid withdrawal symptoms can range from restlessness, muscle pain, fatigue, anxiety, and insomnia. Unfortunately, these withdrawal symptoms, as well as intense cravings for the drug can persist for several months, with some reports up

to years. Because of the intensity and longevity of these withdrawal symptoms, many individuals struggle to remain abstinent, and accidental overdoses are common (CDC, 2013).

The rise of abuse and misuse of opioid products in the early-to-mid 2000s is a direct result of the increased number of opioid prescription medications containing *oxycodone* and *hydrocodone* (Jayawant & Balkrishnana, 2005). The 2015 report estimated 12.5 million Americans were abusing prescription narcotic pain relievers in the past year (SAMHSA, 2016). In efforts to reduce the abuse of these medications, the FDA developed programs to educate prescribers about the risks of misuse and abuse of opioid medications.

11.1.2.2. Stimulants. The two most common types of stimulants abused are cocaine and amphetamines. Unlike depressants that reduce the activity of the central nervous system, stimulants have the opposite effect, increasing the activity in the central nervous system. Physiological changes that occur with stimulants are increased blood pressure, heart rate, pressured thinking/speaking, and rapid, often jerky behaviors. Because of these symptoms, stimulants are often used for their feelings of euphoria, to reduce appetite, and prevent sleep.

Similar to opioids, **cocaine** is extracted from a South American plant- the coca plant- and produces feelings of energy and euphoria. It is the most powerful natural stimulant known to date (Acosta et al., 2011). As stated, low doses can produce feelings of excitement, talkativeness, and euphoria; however, as the amount of ingested cocaine increases, physiological changes such as rapid breathing, increased blood pressure, and excessive arousal can be observed. The psychological and physiological changes from cocaine are due to an increase of *dopamine, norepinephrine* and *serotonin* in various brain structures (Haile, 2012; Hart & Ksir, 2014).

One key feature of cocaine use is the rapid high of *cocaine intoxication,* followed by the rapid letdown, or *crashing*, as the drug diminishes within the body. During the euphoric intoxication, individuals will experience poor muscle coordination, grandiosity, compulsive behavior, aggression, and possible hallucinations and/or delusions (Haile, 2012). Conversely, as the drug leaves the system, the individual will experience negative effects such as headaches, dizziness, and fainting (Acosta et al., 2011). These negative feelings often produce a negative feedback loop, encouraging individuals to ingest more cocaine to alleviate the negative symptoms. This also increases the chance of accidental overdose.

Cocaine is unique in that it can be ingested various different ways. While cocaine was initially snorted via the nasal cavity, individuals found that if the drug was smoked or injected, its effects were more powerful and longer lasting (Haile, 2012). The most common way cocaine is currently ingested is via **freebasing**, which involves heating cocaine with ammonia to extract the cocaine base. This method produces a form of cocaine that is almost 100 percent pure. Due to its low melting point, freebased cocaine is easy to smoke via a glass pipe. Inhaled cocaine is absorbed into the blood stream and brain within 10-15 seconds suggesting its effects are felt almost immediately (Addiction Centers of America).

Crack is a derivitative of cocaine that is formed by combining cocaine with water and another substance (commonly baking soda) to create a solid structure that is then broken into smaller pieces. Because of this process, it requires very little cocaine to make crack, thus making it a more affordable drug. Coined for the crackling sound that is produced when it is smoked, it is also highly addictive, likely due to the fast acting nature of the drug. While the effects of cocaine peak in 20-30 minutes and last for about 1-2 hours, the effects of crack peak in 3-5 minutes and last only for up to 60 minutes (Addiction Centers of America).

Amphetamines are manufactured in a laboratory setting. Currently, the most common amphetamines are prescription medications such as Ritalin, Adderall, and Dexedrine (prescribed for sleep disorders). These medications produce an increase in energy and alertness and reduce appetite when taken at clinical levels; however, when consumed at larger dosages, it can produce intoxication similar to psychosis, including violent behaviors. Due to the increased energy levels and appetite suppressant qualities, these medications are often abused by students studying for exams, athletes needing extra energy, and individuals seeking weight loss (Haile, 2012). Biologically, similar to cocaine, amphetamines affect the central nervous system by increasing the amount of dopamine, norepinephrine, and serotonin in the brain (Haile, 2012).

Methamphetamine, a derivative of amphetamine, is often abused due to its low cost and feelings of euphoria and confidence; however, it can have serious health consequences such as heart and lung damage (Hauer, 2010). Most commonly used intravenously or nasally, methamphetamine can also be eaten or heated to a temperature in which it can be smoked. The most notable effects of methamphetamine use are the drastic physical changes to one's appearance including significant teeth damage and facial lesions (Rusyniak, 2011).

While I'm sure you all are well aware of how **caffeine** is consumed, you may be surprised to learn that in addition to coffee, energy drinks, and soft drinks, caffeine can also be found in chocolate and tea. Because of the vast use of caffeine, it is the most widely consumed substance in the world, with approximately 90% of Americans consuming some type of caffeine *every day* (Fulgoni, Keast, & Lieberman, 2015). While caffeine is often consumed in moderate dosages, caffeine intoxication and withdrawal can occur. In fact, an increase in caffeine intoxication and withdrawal has been observed with the simultaneous popularity of energy drinks. Common energy drinks such as Monster and RedBull have nearly double the amount of caffeine of tea and coke (Bigard, 2010). While these drinks are commonly consumed by adults, a startling 30% of middle and high schoolers also report regular consumption of energy drinks to assist with academic and athletic responsibilities (Terry-McElrath, O'Malley, & Johnston, 2014). The rapid increase in caffeinated beverages has led to an increase in ER visits due to the intoxication effects (SAMHSA, 2013).

11.1.2.3. Hallucinogens/Cannabis/Combination. The final category includes both hallucinogens and cannabis- both of which produce sensory changes after ingestion. While hallucinogens are known for their ability to produce more severe delusions and hallucinations, cannabis also has the capability of producing delusions or hallucinations, however, this typically occurs only when large amounts of cannabis are ingested. More commonly, cannabis has been known to have stimulant and depressive effects, thus classifying itself in a group of its own due to the many different effects of the substance.

Hallucinogens come from natural sources and have been involved in cultural and religious ceremonies for thousands of years. Synthetic forms of hallucinogens have also been created- most common of which are *PCP*, *Ketamine, LSD* and *Ecstasy*. In general, hallucinogens produce powerful changes in sensory perception. Depending on the type of drug ingested, effects can range from hallucinations, changes in color perception, or distortion of objects. Additionally, some individuals report enhanced auditory, as well as changes in physical perception such as tingling or numbness of limbs and interchanging hot and cold sensations (Weaver & Schnoll, 2008). Interestingly, the effect of hallucinogens can vary both between individuals, as well as *within* the same individual. This means that the same amount of the same drug may produce a positive experience one time, but a negative experience the next time.

Overall, hallucinogens do not have addictive qualities; however, individuals can build a tolerance, thus needing larger quantities to produce similar effects (Wu, Ringwalt, Weiss, & Blazer, 2009). Furthermore, there is some evidence that long term use of these drugs results in psychosis, mood, or anxiety disorders due to the neurobiological changes after using hallucinogens (Weaver & Schnoll, 2008).

Similar to hallucinogens and a few other substances, **cannabis** is also derived from a natural plant- the hemp plant. While the most powerful of hemp plants is *hashish*, the most commonly known type of cannabis, marijuana, is a mixture of hemp leaves, buds, and tops of plants (SAMHSA, 2014). The potency of cannabis is impacted by many external factors such as the climate it was grown in, the way the cannabis was prepared, and the duration of storage. Of the active chemicals within cannabis, **tetrahydrocannabinol (THC)** appears to be the single component that determines the potent nature of the drug. Various strains of marijuana have varying amounts of THC; hashish contains a high concentration of THC, while marijuana has a small concentration.

THC binds to cannabinoid receptors in the brain which produces psychoactive effects. These effects vary depending on both an individual's body chemistry, as well as various strains and concentrations of THC. Most commonly, people report feelings of calm and peace, relaxation, increased huger, and pain relief. Occasionally, negative symptoms such as increased anxiety or paranoia, dizziness, and increased heart rate also occur. In rare cases, individuals develop psychotic symptoms or schizophrenia following cannabis use (Donoghue et al., 2014).

While nearly 20 million American's report regular use of marijuana, only ten percent of these individuals will develop a dependence on the drug (SAMHSA, 2013). Of particular concern is the number of adolescents engaging in cannabis use. One in eight 8th graders, one in four 10th graders, and one in three 12th graders report use of marijuana in the past year (American Academy of Child and Adolescent Psychiatry, 2013). Individuals who begin cannabis abuse during adolescence are at an increased risk to develop cognitive effects from the drug due to the critical period of brain development during adolescence (Gruber, Sagar, Dahlgren, Racine, & Lukas, 2012). Increased discussion about the effects of marijuana use, as well as psychoeducation about substance abuse in general is important in preventing marijuana use during adolescence.

It is not uncommon for substance abusers to consume more than one type of substance at a time. This **combination** of substance use can have dangerous results depending on the interactions between substances. For example, if multiple depressant drugs (i.e. alcohol and benzodiazepines and/or opiates) are consumed at one time, an individual is at risk for severe respiratory distress or even death, due to the compounding depressive effects on the central nervous system. Additionally, when an individual is under the influence of one substance, judgement may be impaired, and ingestion of a larger amount of another drug may lead to an accidental overdose. Finally, the use of one drug to counteract the effects of another drug—taking a depressant to combat the effects of a stimulant—is equally as dangerous as the body is unable to regulate homeostasis.

11.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of depressants.
- Describe the epidemiology of stimulants.
- Describe the epidemiology of hallucinogens.

It has been estimated that nearly 9 percent of teens and adults in the United States have a substance abuse disorder (SAMHSA, 2014). Asian/Pacific Islanders, Hispanics, and African Americans are less likely to develop a lifetime substance abuse disorder compared to non-Hispanic white individuals (Grant et al., 2016). Native Americans actually have the highest rate of substance abuse at nearly 22 percent (NSDUH, 2013). Additional demographic variables also suggest that overall substance abuse is greater in men than women, younger versus older individuals, unmarried/divorced individuals than married, and in those with an education level of a high school degree or lower (Grant et al., 2016). With regards to specific types of substances, the highest prevalence rates of substances abused are cannabis, opioids, and cocaine, respectively (Grant et al., 2016).

11.2.1. Depressants

With regards to depressant substances, men out-number women in alcohol abuse 2 to 1 (Johnston et al., 2014). Ethnically, Native Americans have highest rate of alcoholism, followed by White, Hispanic, African, Asian, respectively. With regards to opioid use, roughly 1 percent of the population have this disorder, with 80% of those being addicted to pain-reliever opioids such as oxycodone or morphine; the remaining 20% are heroin (SAMHSA, 2014).

11.2.2. Stimulants

Nearly 1.1 percent of all high school seniors have used cocaine within the past month (Johnston et al., 2014). Due to the high cost of cocaine, it is more commonly found in suburban neighborhoods where consumers have the financial means to purchase the drugs. Methamphetamine is used by men and women equally. It is popular among biker gangs, rural America, and urban gay communities, as well as in clubs and all night dance parties (aka raves; Hopfer, 2011).

A major discussion within the stimulant substance abuse is the abuse of stimulant medication among college students. This is a growing concern, with 17% of college students reportedly abusing stimulant medications. Greek organization membership, academic performance, and other substance use were the most highly correlated variables related to stimulant medication abuse.

11.2.3. Hallucinogens

Up to 14% of general population have used LSD or another hallucinogen. Nearly 20 million adults and adolescents report current use of marijuana. Men report more than women. Sixty-five percent of individuals report their first drug of use was marijuana- labeling it as a gateway drug to other illicit substances. Due to the increased research and positive effects of medicinal marijuana, the movement to legalize *recreational* marijuana has gained momentum, particularly in the Pacific Northwest of the United States.

11.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of substance-related and addictive disorders.

It should not come as a surprise that substance abuse in general has a high comorbidity rate within itself (meaning abuse of multiple different substances), as well as with other mental health disorders. Researchers believe that substance abuse disorders are often secondary to another mental health disorder, as the substance abuse develops as a means to "self-medicate" the underlying psychological disorder. In fact, several large surveys identified alcohol and drug dependence to be twice as more likely in individuals with anxiety, affective, and psychotic disorders than the general public (Hartz et al., 2014). While it is difficult to identify exact estimates of the relationship between substance abuse and serious mental health disorders, the general consensus among researchers is that there is a strong relationship between substance abuse and mood, anxiety, posttraumatic stress, and personality disorders (Grant et al., 2016).

11.4. Etiology

Section Learning Objectives

- Describe the biological causes of substance-related and addictive disorders.
- Describe the cognitive causes of substance-related and addictive disorders.
- Describe the behavioral causes of substance-related and addictive disorders.
- Describe the sociocultural causes of substance-related and addictive disorders.

11.4.1. Biological

11.4.1.1. Genetics. Similar to other mental health disorders, substance abuse is genetically influenced. With that said, it is different than other mental health disorders in that if the individual is

not exposed to the substance, they will not develop substance abuse.

Heritability of alcohol is among the most well studied substances, likely due to the fact that it is the only legal substance (with the exception of cannabis in some states). Twin studies have indicated a range of 50-60% heritability risk for alcohol disorder (Kendler et al., 1997). More recent studies exploring the heritability of other substance abuse, particularly drug use, suggests there may be a stronger heritability link than previously thought (Jang, Livesley, & Vernon, 1995). Twin studies indicate that the genetic component of drug abuse is stronger than drug use in general, meaning that genetic factors are more significant for abuse of a substance over nonproblematic use (Tsuang et al., 1996). Merikangas and colleagues (1998) actually found an 8-fold increased risk for developing a substance abuse disorder across a wide range of substances.

Unique to substance abuse is the fact that both genetic and familial influence are both at play. What does this mean? Well, biologically, the individual may be genetically predisposed to a substance abuse; additionally, the individual may also be at risk due to their familial environment where their parents and/or siblings are also engaging in substance abuse. Individuals whose parents abuse substances may have a greater opportunity to ingest substances, thus promoting drug-seeking behaviors. Furthermore, families with a history of substance abuse may have a more accepting attitude of drug use than families with no history of substance abuse (Leventhal & Schmitz, 2006).

11.4.1.2. Neurobiological. A longstanding belief about how drug abuse begins and is maintained is the *brain reward system*. A *reward* can be defined as any event that increases the likelihood of a response and has a pleasurable effect. Majority of research on the brain reward system has focused on the mesocorticolimbic dopamine system, as it appears this area is the primary reward system of most substances that are abused. As research has evolved in the field of substance abuse, five additional neurotransmitters have also been implicated in the reinforcing effect of addiction: dopamine, opioid peptides, GABA, serotonin, and endocannabinoids. More specifically, dopamine is less involved in opioid, alcohol, and cannabis. Alcohol and benzodiazepines lower the production of GABA, while cocaine and amphetamines are involved in the lowering of dopamine. Cannabis has been shown to reduce the production of endocannabinoids.

11.4.2. Cognitive

Cognitive theorists have focused on the beliefs regarding the anticipated effects of substance use. Defined as the *expectancy effect,* drug-seeking behavior is presumably motivated by the desire to attain a particular outcome by ingesting a substance. The expectancy effect can be defined in both positive and negative forms. Positive expectations are thought to increase drug-seeking behavior, while negative experiences would decrease substance use (Oei & Morawska, 2004). Several alcohol studies have examined the expectancy effect on the use of alcohol. Those with alcohol abuse reported expectations of tension reduction, enhanced sexual experiences, and improved social pleasure (Brown, 1985). Additionally, observing positive experiences, both in person and through television or social media also shapes our drug use expectancies.

While some studies have explored the impact of negative expectancy as a way to eliminate substance abuse, research has failed to continually support this theory, suggesting that positive experiences and expectations are a more powerful motivator of substance abuse than the negative experiences (Jones, Corbin, Fromme, 2001).

11.4.3. Behavioral

Operant conditioning has been implicated in the role of developing substance use disorders. As you may remember, operant conditioning refers to the increase or decrease of a behavior, due to a reinforcement or punishment. Since we are talking about increasing substance use, behavioral theorists suggest that substance abuse is *positively and negatively reinforced* due to the effects of a substance.

Positive reinforcement occurs when the substance use is increased due to the positive or pleasurable experiences of the substance. More specifically, the rewarding effect or pleasurable experiences while under the influence of various substances directly impacts the likelihood that the individual will use the substance again (Wise & Koob, 2013). Studies of substance use on animals routinely supports this theory as animals will work to receive injections of various drugs (Wise & Koob, 2013).

Negative reinforcement, or the increase of a given behavior due to the removal of a negative effect, also plays a role in substance abuse in two different ways. First, many people ingest a substance as an escape from their unpleasant life- whether it be physical pain, stress, or anxiety, to name a few. Therefore, the substance temporarily provides relief from a negative environment, thus reinforcing future substance abuse (Wise & Koob, 2013). The second way negative reinforcement is involved in substance abuse is during symptoms of withdrawal. As previously mentioned, withdrawal from a substance often produces significant negative symptoms such as nausea, vomiting, uncontrollable shaking, etc. To eliminate these symptoms, and individual will consume more of the substance, thus again escaping the negative symptoms and enjoying the “highs” of the substance.

11.4.4. Sociocultural

Arguably, one of the strongest influences of substance abuse is the impact of one’s friends and immediate environment. Peer attitudes, perception of one’s friends drug use, pressure from peers to use substances, and beliefs about substance use are among the strongest predictors of drug use patterns (Leventhal & Schmitz, 2006). This is particularly concerning during adolescence when patterns of substance use typically begin.

Additionally, research continually supports a strong relationship between second generation substance abusers (Wilens et al., 2014). The increased likelihood of family members substance abuse is likely related to both a genetic predisposition, as well as the accepting attitude of the familial environment (Chung et al., 2014). Not only does a child have early exposure to these substances if their parent has as substance abuse problem, but they are also less likely to have parental supervision which may impact their decision related to substance use (Wagner et al., 2010). One potential protective factor against substance use is religiosity. More specifically, families that promote religiosity may actually reduce substance use by promoting negative experiences (Galen & Rogers, 2004).

Another sociocultural view on substance abuse is stressful life events, particularly those related to financial stability. Prevalence rates of substance abuse is higher in poorer people (SAMHSA, 2014). Furthermore, additional stressors such as childhood abuse/trauma, negative work environments, as well as discrimination are also believed to contribute to the development of a substance use disorder (Hurd, Varner, Caldwell, & Zimmerman, 2014; McCabe, Wilsnack, West, & Boyd, 2010; Unger et al., 2014).

11.5. Treatment

Section Learning Objectives

- Describe biological treatment options for substance-related and addictive disorders.
- Describe behavioral treatment options for substance-related and addictive disorders.
- Describe cognitive-behavioral treatment options for substance-related and addictive disorders.
- Describe sociocultural treatment options for substance-related and addictive disorders.

Given the large number of the population that is effected by substance abuse, it is not surprising that there are many different approaches to treat substance use disorder. Overall, treatments for substance related disorders are only mildly effective, likely due in large part to the addictive qualities in many of these substances (Belendiuk & Riggs, 2014).

11.5.1. Biological

11.5.1.1. Detoxification. Detoxification refers to the medical supervision of a withdrawal of a specified drug. While most detoxification programs are inpatient for increased supervision, there are some programs that allow for outpatient detoxification, particularly if the addiction is not as severe. There are two main theories of detoxification—gradually decreasing the amounts of the substance until the individual is off the drug completely, or, eliminate the substance entirely while providing additional medications to manage withdrawal symptoms (Bisaga et al., 2015). Unfortunately, relapse rates are high for those engaging in detoxification programs, particularly if they lack any follow-up psychological treatment.

11.5.1.2. Agonist drugs. As researchers continue to learn more about both the mechanisms of substances commonly abused, as well as the mechanisms in which the body processes these substances, alternative medications are being created to essentially replace the drug in which the individual is dependent on. These **agonist drugs** provide the individual with a “safe” drug that has a similar chemical make-up to the addicted drug. One common example of this is *methadone,* an opiate agonist that is often used in the reduction of heroin use (Schwartz, Brooner, Montoya, Currens, & Hayes, 2010). Unfortunately, because methadone reacts to the same neurotransmitter receptors as heroin, the individual essentially replaces their addiction of heroin with the addiction to methadone. While this is not ideal, methadone treatment is highly regulated under safe medical supervision. Furthermore, it is taken by mouth, thus eliminating the potential negative effects of unsterilized needles in heroin use. While some argue that methadone maintenance programs are not an effective treatment because it simply replaces one drug for another, some argue that the combination of methadone with education and psychotherapy can successfully help individuals off both illicit drugs and methadone medications (Jhanjee, 2014).

11.5.1.3. Antagonist drugs. Unlike agonist drugs, **antagonist drugs** block or change the

effects of the addictive drug. Among the most commonly prescribed antagonist drugs are Disulfiram and Naloxone. Disulfiram is often given to individuals trying to abstain from alcohol as it produces significant negative effects (i.e. nausea, vomiting, increased heart rate, and dizziness) when coupled with alcohol consumption. While this can be an effective treatment to eliminate alcohol use, the individual must be motivated to take the medication as prescribed (Diclemente et al., 2008).

Similar to Disulfiram, Naloxone is used for individuals with opioid abuse. Naloxone acts by binding to endorphin receptors, thus preventing the opioids from having the intended euphoric effect. While in theory this treatment appears promising, it is in fact extremely dangerous as it can send the individual into immediate, severe withdrawal symptoms (Alter, 2014). This type of treatment requires high medical supervision to ensure the safety of the patient.

11.5.2. Behavioral

11.5.2.1. Aversion therapy. Based on classical conditioning principles, **aversion therapy** is a form of treatment for substance abuse that pairs the stimulus with some type of negative or aversive stimulus. For example, an individual may be given a shock every time they think about or attempt to drink alcohol. Through the use of pairing this aversive stimulus to the abused substance, the individual will begin to independently pain the substance with an aversive thought, thus reducing their craving/desire for the substance. Some argue that the use of agonist and antagonist drugs is a form of aversion therapy as these medications utilize the same treatment strategy as traditional aversion therapy.

11.5.2.2. Contingency management. Contingency management is a treatment approach that emphasizes *operant conditioning*—increasing sobriety/adherence to treatment program through rewards. Originally developed to increase adherence to medication and reinforce opiate abstinence in methadone patients, contingency management has been adapted to increase abstinence in many different substance abuse treatment programs. In general, patients are “rewarded” with vouchers or prizes in exchange for abstinence from substance use (Hartzler, Lash, & Roll, 2012). These vouchers allow for individuals to gain incentives specific to their own interest, thus increasing the chances of abstinence. Common vouchers include movie tickets, sports equipment, or even cash (Mignon, 2014).

Contingency management has been proven to be effective in treating various types of substance abuse, particularly alcohol and cocaine (Lewis & Petry, 2005). Not only has it been effective in reducing substance use in addicts, but it has also been effective in increasing the amount of time patients remain in treatment as well as compliance to the treatment program (Mignon, 2014). Despite its success, dissemination of this type of treatment has been rare. In efforts to rectify this, the federal government has provided financial resources through SAMHSA to the development, implementation, and evaluation of contingency management as a treatment to reduce alcohol and drug use (Mignon, 2014).

11.5.3. Cognitive-Behavioral

11.5.3.1. Relapse prevention training. Relapse prevention training is essentially what it sounds like- identifying potentially high-risk situations for relapse and then learning behavioral skills and cognitive interventions to prevent the occurrence of a relapse. Early in treatment, the clinician guides

the patient to identify any interpersonal, intrapersonal, environmental, and/or physiological risks for relapse. Once these triggers are identified, the clinician works with the patient on cognitive and behavioral strategies such as learning effective coping strategies, enhancing self-efficacy, and encouraging mastery of outcomes. Additionally, psychoeducation about how substance abuse is maintained, as well as identifying maladaptive thoughts and learning cognitive restructuring techniques helps the patient make informed choices during high-risk situations. Finally, role-playing these high-risk situations in session allows patients to become comfortable engaging in these effective coping strategies that enhance their self-efficacy, and ultimately reducing the chances of a relapse. Research for relapse prevention training appears to be somewhat effective for individuals with substance-related disorders (Marlatt & Donovan, 2005).

11.5.4. Sociocultural

11.5.4.1. Self-help. In 1935, two men suffering from alcohol abuse met and discussed their treatment options. Slowly, the group grew and by 1946 this group was known as **Alcoholics Anonymous (AA).** The two founders, along with other early members developed the Twelve Step Traditions as a way to help guide members in spiritual and character development. Due to the popularity of the treatment program, other programs such as Narcotics Anonymous and Cocaine Anonymous adopted and adapted the Twelve Steps for their respective substance abuse. Similarly, Al-Anon and Alateen are two support groups that offer support for families and teenagers of individuals struggling with alcohol abuse.

The overarching goal of AA is abstinence from alcohol. In order to achieve this, the participants are encouraged to “take one day at a time.” In using the 12 steps, participants are encouraged to admit that they have a disease, that they are powerless over this disease, and that their disease is more powerful than any man. Therefore, participants are encouraged to turn their addiction over to God and ask Him to right their wrong and to remove negative character defects and shortcomings. The final steps encourage participants to identify and make amends to those who they have wronged during their alcohol abuse.

While studies examining the effectiveness of AA programs are inconclusive, AA’s membership indicates that 27% of its members have been sober less than one year, 24% have been sober 1-5 years, 13% have been sober 5-10 years, 14% have been sober 10-20 years, and more than 22% have been sober more than 20 years (Alcoholics Anonymous, 2014). Some argue that this type of treatment is most effective for those who are willing and able to abstain from alcohol as opposed to those who can control their drinking to moderate levels.

11.5.4.2. Residential treatment centers. Another type of treatment similar to self-help is **residential treatment programs.** In this placement, individuals are completely removed from their environment and live, work, and socialize within a drug-free environment while also attending daily individual, group, and family therapy. The types of treatment used within a residential program varies from program to program, with most focusing on cognitive-behavioral and behavioral techniques. Many also incorporate 12-step programs into treatment as well, as many patients transition from a residential treatment center to a 12-step program post discharge. As one would expect, residential treatment programs goals are abstinence and any evidence of substance abuse during the program is grounds for immediate termination.

Studies examining the effectiveness of residential treatment centers suggest that these programs are effective in treating a variety of substance abuse disorders; however, many of these programs are very costly, thus limiting the availability of this treatment to the general public (Bender, 2004; Galanter, 2014). Additionally, many individuals are not able to completely remove themselves from their daily responsibilities for several weeks to months, particularly those with families. Therefore, while this treatment option is very effective, it is also not an option for most individuals struggling with substance abuse.

11.5.4.3. Community reinforcement. The goal for community reinforcement treatment is for patients to abstain from substance use by replacing the positive reinforcements of the substance with that of sobriety. This is done through several different techniques such as motivational interviewing, learning adaptive coping strategies, and encouraging family support (Mignon, 2014). Essentially, the community around the patient reinforces the positive choices of abstaining from substance use.

Surprisingly, community reinforcement has been found to be effective in both an inpatient and outpatient setting (Meyers & Squires, 2001). It is believed that the intrinsic motivation and the effective coping skills, in combination with the support of an individual's immediate community (friends and family) is responsible for the long-term positive treatment effects of community reinforcement.

Module Recap

And that concludes Part IV of the book and Block 3 of mental disorders. In this module we discussed substance-related and addictive disorders to include substance intoxication, substance use disorder, and substance withdrawal. Substances include depressants, sedative-hypnotic drugs, opioids, stimulants, and hallucinogens. As in past modules, we discussed the clinical presentation, epidemiology, comorbidity, and etiology of the disorders. We then also discussed biological, behavioral, cognitive-behavioral, and sociocultural treatment approaches. Be sure you prepare for your next exam. In Part V and Block 4 we will discuss schizophrenia spectrum and personality disorders.

V

Part V. Mental Disorders – Block 4

Part V. Mental Disorders – Block 4

Module 12: Schizophrenia Spectrum and Other Psychotic Disorders

Module Overview

In Module 12, we will discuss matters related to schizophrenia spectrum disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include schizophrenia, schizophreniform disorder, schizoaffective disorder, and delusional disorder. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 12.1. Clinical Presentation
- 12.2. Epidemiology
- 12.3. Comorbidity
- 12.4. Etiology
- 12.5. Treatment

Module Learning Outcomes

- Describe how schizophrenia spectrum disorders present.
- 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.

12.1. Clinical Presentation

Section Learning Objectives

- List and describe distinguishing features that make up the clinical presentation of schizophrenia spectrum disorders.
- Describe how schizophrenia presents itself.
- Describe how schizophreniform disorder presents itself.
- Describe how schizoaffective disorder presents itself.
- Describe how delusional disorder presents itself.

12.1.1. The Clinical Presentation of Schizophrenia Spectrum Disorders

The schizophrenia spectrum disorders, which for the purpose of this course consists of Schizophrenia, Schizophreniform disorder, Schizoaffective disorder, and Delusional disorder, are defined by one of the following main symptoms: delusions, hallucinations, disorganized thinking (speech), disorganized or abnormal motor behavior, and negative symptoms. Individuals diagnosed with a schizophrenia spectrum disorder experience **psychosis,** which is defined as a loss of contact with reality. Be psychosis episodes make it difficult for individuals to perceive and respond to environmental stimuli, which causes a significant disturbance in everyday functioning. While there are a vast 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).

12.1.1.1. 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, they are unable to distinguish them from reality, likely due to a lack of insight. There are a variety of delusions that can present in many different ways:

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

Among the most common delusions are delusions of persecution (Arango & Carpenter, 2010). 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 (*delusions of grandeur)* than another type of delusion.

12.1.1.2. Hallucinations. Hallucinations can occur in any of the five senses such 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. having 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 patients 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 patient or various voices talking to one another. Generally, these hallucinations are not attributable to

any one person that the individual knows. They are usually clear, objective, and definite (Arango & Carpenter, 2010). Additionally, the auditory hallucinations can be pleasurable, providing comport to the patient; however, in other individuals, the auditory hallucinations can be unsettling as they produce commands or malicious intent.

12.1.1.3. Disorganized thinking. Among the most common cognitive impairments displayed in patients with schizophrenia are disorganized thought, communication, and speech. More specifically, thoughts and speech patterns may appear to be *circumstantial* or *tangential*. For example, patients may give unnecessary details in response to a question before they finally produce the desired response. While the question is eventually answered in circumstantiality, in tangentially, the patient never reaches the point. 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. Although not always, derailment is often seen in *illogicality,* or the tendency to provide bizarre explanations for things.

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).

12.1.1.4. Disorganized/Abnormal motor behavior. *Psychomotor symptoms* can also be observed in individuals with schizophrenia. These behaviors may manifest as awkward movements or even ritualistic/repetitive behaviors. They are often unpredictable and overwhelming, severely impacting their ability to perform daily activities (APA, 2013).

12.1.1.5. Catatonic behavior. Catatonic behavior, or the decrease or even lack of reactivity to the environment, is among the most commonly seen disorganized motor behavior in schizophrenia. There runs a range of catatonic behaviors from *negativism* (resistance to instruction); *mutism* or *stupor* (complete lack of verbal and motor responses); *rigidity* (maintaining a rigid or upright posture while resisting efforts to be moved); or *posturing* (holding odd, awkward postures for long periods of time; APA, 2013). There is one type of catatonic behavior, *catatonic excitement*, where the individual experiences a hyperactivity of motor behavior, in a seemingly excited/delirious way.

12.1.1.6. Negative symptoms. Up until this point, all the schizophrenia symptoms can be categorized as **positive symptoms**, or symptoms that are an over-exaggeration of normal brain processes; these symptoms are also new to the individual. The final symptom included in the diagnostic criteria of schizophrenia is **negative symptoms**, which are defined as the inability or decreased ability to initiate actions, speech, expressed emotion, or to feel pleasure (Barch, 2013). Negative symptoms are often present before positive symptoms and remain once positive symptoms remit. Because of their prevalence through the course of the disorder, 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 effectiveness antipsychotic medications have in addressing negative symptoms (Kirkpatrick, Fenton, Carpenter, & Marder, 2006).

There are six main types of negative symptoms seen in patients with schizophrenia. Such symptoms include:

- **Affective flattening –** Reduction in emotional expression; reduced display of emotional expression
- **Alogia** – Poverty of speech or speech content

- **Anhedonia** – Inability to experience pleasure
- **Apathy –** General lack of interest
- **Asociality –** Lack of interest in social relationships
- **Avolition –** Lack of motivation of goal-directed behavior

12.1.2. Schizophrenia

As stated above, the hallmark symptoms of schizophrenia include the presentation of at least two of the following for at least one month: delusions, hallucinations, disorganized speech, disorganized/abnormal behavior, or negative symptoms. These symptoms create significant impairment in an 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 symptoms have to persist for a minimum of 6 months to meet criteria for a schizophrenia diagnosis, it is not uncommon to have **prodromal** symptoms that precede the active phase of the disorder and **residual** symptoms that follow it. These prodromal and residual symptoms are "subthreshold" forms of psychotic 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, these mood symptoms are distinct from a mood disorder diagnosis in that psychotic features will exist beyond the remission of depressive symptoms.

12.1.3. Schizophreniform Disorder

Schizophreniform disorder is similar to schizophrenia with the exception of the length of presentation of symptoms. Schizophreniform disorder is considered an "intermediate" disorder between schizophrenia and brief psychotic disorder as the symptoms are present for at least one month but *not* longer than 6 months. As you may recall, schizophrenia symptoms must be present for at least 6 months; A brief psychotic disorder is diagnosed when symptoms are present for *less* than 1 month. Approximately two-thirds of individuals who are initially diagnosed with schizophreniform disorder will have symptoms that last longer than 6 months, at which time their diagnosis is changed to schizophrenia (APA, 2013).

Another key distinguishing feature of schizophreniform disorder is the lack of criteria related to impaired functioning. While many individuals with schizophreniform disorder do display impaired functioning, it is not essential for diagnosis. Finally, any major mood episodes—either depressive or manic— 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.

12.1.4. Schizoaffective Disorder

Schizoaffective disorder is 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. It should be noted that because 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. While schizophrenia and schizophreniform disorder do *not* have a significant mood component, schizoaffective disorder requires the presence of a depressive or manic episode for 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 2 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.

12.1.5. 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 should an individual experience a delusion, but, has also experienced either hallucinations, disorganized speech, disorganized or catatonic behavior, or negative symptoms (additional criteria of schizophrenia), they should *not* be diagnosed with delusional disorder as their symptoms are more aligned with a schizophrenia diagnosis. Unlike most other schizophrenia related disorders, daily functioning is not overly impacted due to the delusions. 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-IV (APA, 2013) has identified five main subtypes of delusional disorder in efforts to better categorize the symptoms of the individuals disorder. When making a diagnosis of delusional disorder, one of the following modifiers (in addition to mixed presentation) is included. **Erotomanic delusion** occurs when an 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, patients will report they have made an important discovery that benefits the general public. Grandiose delusions may also take on a religious affiliation, as 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 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 (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. Finally, **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).

12.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of schizophrenia spectrum disorders.

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 onset of the disorder slightly earlier for males than females (APA, 2013). Earlier onset of disorder is generally predictive of worse overall prognosis. Onset of symptoms is typically gradual, with initial symptoms presenting similar to depressive disorders; however, some individuals will present with an abrupt presentation of the disorder. Negative symptoms appear to be more predictive of 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, and delusional disorder prevalence rates are all significantly less than that of schizophrenia, occurring in less than 0.3% of the general population. While schizoaffective disorder is diagnosed more in females than males (similar to schizophrenia), 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 (APA, 2013).

12.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of schizophrenia spectrum disorders.

There is a high comorbidity rate between schizophrenia related 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 (see diathesis-stress model below; 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 related 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.

12.4. 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.

12.4.1. Biological

12.4.1.1. Genetic/Family studies. Twin and family studies consistently support the biological theory. More specifically, if one identical twin develops schizophrenia, there is a 48 percent chance that the other will also develop the disorder within their lifetime (Coon & Mitter, 2007). This percentage drops to 17 percent 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).

12.4.1.2. 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 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).

12.4.1.3. 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 ilicits 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.

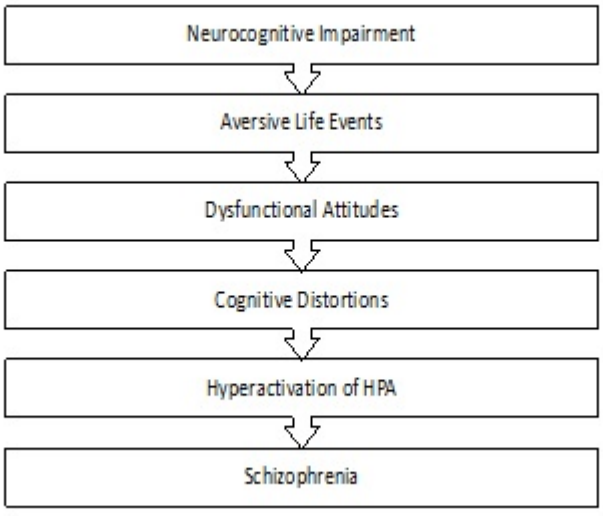
The HPA axis is one of the main neurobiological structures that mediates stress. It involves the regulation of three chemical messengers (cotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and glucocorticoids) as they respond to a stressful situation (Corcoran et al., 2003). Glucocorticoids, 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, persistent increased levels of glucocorticoids in brain structures may be the key to the onset of psychosis in prodromal patients (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 onset of disorder, evidence for the implication of stress and symptom relapse is strong. More specifically, schizophrenia patients 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 they interfere with daily functioning (Walker & Diforio, 1997). This stress alone may be enough to initiate the onset of a relapse.

12.4.2. Psychological

12.4.2.1. Cognitive. 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 the following diathesis-stress model of development of schizophrenia:



Adapted from Beck & Rector, 2005, pg. 580

Based on this theory, an underlying neurocognitive impairment (as discussed above) 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.

12.4.3. Sociocultural

12.4.3.1. Expressed emotion. Research in support of a supportive family environment suggests that families high in expressed emotion, meaning families that have high 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 are causally related to relapse in patients 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 patients with schizophrenia is to reduce expressed emotion within family interactions.

12.4.3.2. 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 was diagnosed with schizophrenia increases the likelihood of a disruptive family environment due to managing the patients symptoms and ensuring their safety while they are home (Friedrich et al., 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).

12.5. 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.

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

12.5.1. 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 patients, and allowed for organization of thoughts. Despite their effectiveness in managing psychotic symptoms, *conventional* 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** in patients, which included involuntary movements isolated to the tongue, mouth, and face (Tenback et al., 2006). While only 10% of patients reported development of tardive dyskinesia, this percentage increased the longer patients 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 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 medications such as clozapine (Clozaril), risperidone (Risperdal), and aripiprazole (abilify), appear to be more effective in managing *both* 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). Thus, 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 of patients, 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.

12.5.2. Psychological Interventions

12.5.2.1. Cognitive Behavioral Therapy (CBT). While CBT has been thoroughly discussed in previous chapters, the goal of 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. With respect to schizophrenia, 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 for schizophrenia patients include: psychoeducation about their disease, 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, 2011) found that recovery-oriented CBT produced a marked improvement in overall functioning as well as symptom reduction in patients diagnosed with schizophrenia. This study suggests that by focusing on targeted goals such as independent living, securing employment, and improving social relationships, patients 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.

12.5.3. 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 onset of symptoms.

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

Psychoeducation is important for both the patient 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 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 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 of a mental illness and its direct effect on the family environment may also alleviate some conflict within the home

The third component, *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).

12.5.3.1. 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 management of symptoms via antipsychotic medications. Impaired ability to interact with individuals in a social, occupational, or recreational setting 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 of 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.

12.5.3.2. Inpatient Hospitalizations. More commonly viewed as community based treatments, inpatient hospitalization programs are essential in stabilizing patients in psychotic episodes. Generally speaking, patients 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 a patient can safely return to their 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).

Module Recap

In our first module of Part V – Block 4, we discussed the schizophrenia spectrum disorders to include schizophrenia, schizophreniform disorder, schizoaffective disorder, and delusional disorder. We started by describing common features of such disorders to include delusions, hallucinations, disorganized thinking, disorganized/abnormal motor behavior, catatonic behavior, and negative symptoms. This then led to our normal discussion of the epidemiology, comorbidity, etiology, and treatment options of the disorders. In our final module of Part V we will discuss personality disorders.

Module 13: Personality Disorders

Module Overview

In Module 13, 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 disorders of paranoid, schizoid, and schizotypal; Cluster B disorders of antisocial, borderline, histrionic, and narcissistic; and Cluster C personality disorders of avoidant, dependent, and obsessive-compulsive. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 13.1. Clinical Presentation
- 13.2. Epidemiology
- 13.3. Comorbidity
- 13.4. Etiology
- 13.5. Treatment

Module Learning Outcomes

- Describe how personality disorders present.
- 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.

13.1. Clinical Presentation

Section Learning Objectives

- List the defining features of personality disorders.
- Describe the three clusters.
- Describe how paranoid personality disorder presents itself.
- Describe how schizoid personality disorder presents itself.
- Describe how schizotypal personality disorder presents itself.
- Describe how antisocial personality disorder presents itself.

- Describe how borderline personality disorder presents itself.
- Describe how histrionic personality disorder presents itself.
- Describe how narcissistic personality disorder presents itself.
- Describe how avoidant personality disorder presents itself.
- Describe how dependent personality disorder presents itself.
- Describe how obsessive-compulsive personality disorder presents itself.

13.1.1. Overview of Personality Disorders

Personality disorders have four defining features which include *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.

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 between these three disorders is social awkwardness and social withdrawal (APA, 2013). Often these behaviors are similar to those seen in schizophrenia, however, they tend to be not as extensive or impactful of daily functioning as 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).

Cluster B is 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.

And finally, **Cluster C** is 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 the disorders, you will see an overlap with symptoms within the 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.

It should be noted that in order to meet criteria for any personality disorder, the individual must display the pattern of behaviors in *adulthood*. Children cannot be diagnosed with a personality disorder. Some children may present with similar symptoms in childhood such as poor peer relationships, odd or eccentric behaviors, or peculiar thoughts and language; however, a formal personality disorder diagnosis cannot be made until the child reaches age 18.

13.1.2. Cluster A

13.1.2.1. Paranoid personality disorder. Paranoid personality disorder is characterized by a marked 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 also 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 lacks 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 accidently 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.

13.1.2.2. 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 extends outside that of 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 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). Individuals 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 these lack of emotions, there is limited need for attention or acceptance.

13.1.2.3. 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, 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. 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. Similar to schizophrenia, unusual perceptual experiences such as auditory hallucinations, as well as unusual speech patterns of derailment or incoherence are also present.

Similar to the other personality disorder within cluster A, there is also a component of paranoia or suspiciousness of other's motives. Additionally, individuals with schizotypal personality 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.

13.1.3. Cluster B

13.1.3.1. Antisocial personality disorder. The essential feature of antisocial personality disorder is the persistent pattern of disregard for, and violation of, the rights of others. The pattern of this behavior begins in late childhood or early adolescence and continues throughout adulthood. While the presence of this behavior begins prior to age 15, the individual cannot be diagnosed with antisocial personality disorder until the age of 18. Prior to age 18, the individual would be diagnosed with *Conduct Disorder*. Although not discussed in this course as it is a disorder of childhood, conduct disorder involves repetitive and persistent pattern of behaviors that violate the rights of others or major age-appropriate norms (APA, 2013). Common behaviors of 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," 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 violation of property destruction, harassing/assaulting others, or stealing (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- decisions are made in the moment without forethought of personal consequences or consideration for others (Lang et al., 2015). This impulsivity also contributes to their inability to withhold jobs 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 to 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. The marked 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 at risk (APA, 2013).

Of course, the most known and devastating symptom 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 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 desire and lack of moral conscious.

13.1.3.2. Borderline personality disorder. Individuals with borderline personality disorder display a pervasive pattern of instability in interpersonal relationships, self-image, affect, and instability (APA, 2013). The combination of these symptoms cause significant impairment in establishing and maintaining personal relationships. They will often go to great lengths to avoid real or imagined abandonment. Fears related to abandonment often lead to inappropriate anger as they often interpret the abandonment as a reflection of their own behaviors. It is not uncommon to experience intense fluctuations in mood, 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.

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).

Another key characteristic of borderline personality disorder is the unstable and/or intense relationships. For example, individuals may idealize or experience intense feelings for another person immediately after meeting them. 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).

13.1.3.3. Histrionic personality disorder. Histrionic personality disorder is the first personality disorder that 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. In efforts to gain the attention, the individual is often very lively and dramatic, using 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 the theatrical nature does not gain the attention they desire, they may go to great lengths to gain the attention such as make-up a story or create a dramatic scene (APA, 2013).

To ensure they gain the attention they desire, individuals with histrionic personality disorder often dress and engage in sexually seductive or provocative ways. These sexually charged behaviors are not only directed at those in which they have a sexual or romantic interest, but to the general public as well (APA, 2013). They often spend significant amount of time on their physical appearance to gain the attention they desire.

Individuals with histrionic personality disorder are easily suggestable. 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.

13.1.3.4. Narcissistic personality disorder. Similar to histrionic personality disorder, narcissistic personality disorder also centers around the individual; however, with narcissistic personality disorder, individuals display a pattern of grandiosity along with a lack of empathy for others (APA, 2013). The grandiose sense of self 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 met.

A lack of empathy is also displayed in individuals with narcissistic personality disorder as they often have difficulty (or choose not to) recognizing 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 possessions than them. Conversely, they believe everyone should be envious of their achievements, regardless of how small hey may actually be.

13.1.4. Cluster C

13.1.4.1. Avoidant personality disorder. Individuals with avoidant personality disorder display a pervasive pattern of 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.

Socially, they 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.

Despite their view as socially inept, unappealing, or inferior, individuals with avoidant personality disorder do not typically suffer from social skills deficits, but rather from misattributions of their own behaviors (APA, 2013).

13.1.4.2. Dependent personality disorder. Dependent personality disorder is characterized by pervasive 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 other person whom they are in a dependent relationship with. 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, the individual experiences significant feelings of helplessness and quickly seeks out another relationship to replace the old one (APA, 2013).

When they are on their own, 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).

13.1.4.3. Obsessive-Compulsive personality disorder. OCPD is defined by an individual's preoccupation with orderliness, perfectionism, and ability to control situations that they lose flexibility, openness, and efficiency in everyday life (APA, 2013). One's preoccupation with details, rules, lists, orders, organizations or schedules overshadows the larger picture of the task or activity. In fact, the need to complete the task or activity is significantly impacted by the individuals self-imposed high standards and need to complete the task perfectly, that the task often does not get completed. The desire to complete the task perfectly often causes the individual to spend excessive amount of time on the task, occasionally repeating it until it is to their standard. Due to repetition and attention to fine detail, the individual often does 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 task up to their standard.

Personally, individuals with OCD 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 similar high standards, thus causing significant disruption to their social interactions. The 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 a potential catastrophe (APA, 2013). Similarly, they may have difficulty discarding worn-out or worthless items, despite their lack of sentimental value.

While some argue that OCPD and OCD are one in the same, others argue that there is a distinct difference in that the personality disorder lacks definitive obsessions and compulsions (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).

13.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of Cluster A personality disorders.
- Describe the epidemiology of Cluster B personality disorders.
- Describe the epidemiology of Cluster C personality disorders.

13.2.1. Cluster A

Disorders within Cluster A have a prevalence rate around 3-4%. More specifically, paranoid personality disorder is estimated to effect 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 prevalence rates for schizotypal personality disorders 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 diagnosis than females (APA, 2013).

13.2.2. Cluster B

Cluster B personality disorders have a wide range of occurrence in the general public. Antisocial personality disorder has an estimated prevalence rate of up to 3.3% of the population (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 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).

Borderline personality disorder, one of the more commonly diagnosed personality disorders, is observed in 5.9% of the general population, with women making up 75% of the diagnoses (APA, 2013). Among 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.

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.

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

13.2.3. Cluster C

Aside from OCPD, Cluster C personality disorders are rarely diagnosed in the general public. For example, avoidant personality disorder occurs in 2.4% of the general population whereas dependent personality disorder occurs in less than 1% of the population (APA, 2013). While avoidant personality is diagnosed equally among men and women, women are more frequently diagnosed with dependent personality disorder than men. (APA, 2013).

OCPD is the most commonly diagnosed personality disorder, occurring in 7.9% of individuals. Some argue that OCPD and OCD should actually be combined into one disorder; however, interestingly enough, more women are diagnosed with OCD than men, yet men are twice as likely to be diagnosed with OCPD than women (APA, 2013). This may suggest that there actually is a significant difference within the etiology of the two disorders.

13.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of personality disorders.

Among the most commonly comorbid diagnoses with personality disorders are mood disorders, anxiety disorders, and substance abuse disorders (Lenzenweger, Lane, Loranger, & Kessler, 2007). A large meta-analysis exploring the data on the comorbidity of major depressive disorder and personality disorders indicated a high diagnosis of major depressive disorder, bipolar disorder, and dysthymia (Friborg, Martinsen, Martinussen, Kaiser, Overgard, & Rosenvinge, 2014). Further exploration of major depressive disorder suggested a lowest rate of diagnosis in cluster A disorders, higher rate in cluster B disorders, and highest rate in cluster C disorders. While the relationship between bipolar disorder and personality disorders has not been consistently clear, the most recent findings report a high comorbidity rate between cluster B personality disorders, with the exception of OCPD (which is in Cluster C), which had the highest comorbidity rate than any other personality disorder. Overall analysis of dysthymia suggested that it is *the* most commonly diagnosed major depressive disorder among all personality disorders.

A more detailed analysis exploring the prevalence rates of the four main anxiety disorders (generalized anxiety disorder (GAD), specific phobia, social phobia, and panic disorder) among individuals with various personality disorders found a clear relationships specific to personality disorders and anxiety disorders (Skodol, Geier, Grant, & Hasin, 2014). More specifically, individuals diagnosed with borderline and schizotypal personality disorders were found to have an additional diagnosis 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.

Substance abuse disorders occur less frequently across the ten personality disorders but are frequently found in individuals diagnosed with antisocial, borderline, and schizotypal personality disorders (Grant et al., 2015).

13.4. Etiology

Section Learning Objectives

- Describe the biological causes of dissociative disorders.
- Describe the psychological causes of dissociative disorders.
- Describe the social causes of dissociative disorders.

Research regarding the development of personality disorders is limited compared to that of other mental health 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, the overall contribution of biological, psychological, and social factors will be reviewed for the purpose of this course.

13.4.1. Biological

Research across the personality disorders suggests some underlying biological or genetic component; however, identification of specific mechanisms have not been identified in most disorders, with the exception of those below. Because of this lack of specific evidence, researchers argue that it is difficult to determine what role genetics plays into 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—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.

Surprisingly, *antisocial personality disorder* and *borderline personality disorder* also have similar neurological changes. 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 both antisocial and borderline personality disorder (Stone, 2014).

13.4.2. Psychological

Psychodynamic, cognitive, and behavioral theories are among the most common psychological 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.

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

Because of these negative early maltreatment experiences, psychodynamic theorists believe an individual's sense of self, and consequently, their beliefs of others, is 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 antisocial personality disorder (Meloy & Yakeley, 2010). Difficulty trusting others or beliefs that they are unable to be loved may also impact one's ability or desire to establish social relationships as seen in many personality disorders, particularly schizoid personality disorder. 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 (Celani, 2014). Conversely, individuals may respond to their early childhood experiences by becoming emotionally distant, using relationships as a sense of power and destructiveness.

13.4.2.2. 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 explains rigidity and perfectionism in OCPD, and the lack of independence observed in dependent and borderline personality disorders (Weishaar & Beck, 2006). **Discounting the positive** also explains 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 positive feedback to support their ongoing theory that they are constantly rejected and criticized by others.

13.4.2.3. Behavioral. Behavioral theorists identify three major theories in their explanation of the development 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).

Similarly, reinforcement, or rewarding of maladaptive behaviors is another behavioral theory used to explain the development of personality disorders. Parents may unintentionally reward aggressive behaviors by giving in to 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) continue with 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, 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).

13.4.3. Social

13.4.3.1. Family dysfunction. High levels of psychological or social dysfunction within families has 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 SES and personality disorders, correlational studies suggest a relationship between poverty, unemployment and poor academic achievement with increased levels of personality disorder diagnoses (Alwin, 2006).

13.4.3.2. 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 the ability to relate to others—something that is generally developed during the first four to six years of a child's life and it is affected by the emotional environment of which that 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 though neglect or physical, emotional, or sexual abuse, is at-risk for under or lack of development of one's 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 of affection in Harlow monkeys (Bowlby, 1998). Based off 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, ambivalent are at-risk for

developing externalizing disorders, and disorganized are at-risk for dissociative symptoms and personality related disorders (Alwin, 2006).

13.5. Treatment

Section Learning Objectives

- Describe treatment options for personality disorders.

13.5.1. Cluster A

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, 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 lack of overall emotion (schizoid personality disorder; Kellett & Hardy, 2014, Colli, Tanzilli, Dimaggio, & Lingiardi, 2014). Because of this, treatment is known to move very slowly, with many patients dropping out of treatment before any resolution of symptoms can be met.

When patients 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 those with paranoid personality disorder (Kellett & Hardy, 2014). Schizoid personality disorder patients may engage in CBT techniques to help 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.

13.5.2. Cluster B

13.5.2.1. Antisocial personality disorder. 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 patients to think about the needs of others (Beck & Weishaar, 2011).

13.5.2.2. Borderline personality disorder. 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* for DBT patients from the DBT clinical team. It is not uncommon for patients 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 patient 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 demand of borderline personality disorder patients, the consultation team provides support to the providers in their work to ensure they remain motivated and competent in DBT principles in effort to provide the best treatment possible.

Support for the effectiveness of DBT in borderline personality disorder patients 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. Additionally, the drop-out rates for treatment are extremely low, suggesting that patients value the treatment components and find them effective in managing symptoms.

13.5.2.3. Histrionic personality disorder. Individuals with histrionic personality disorder are actually *more* likely to seek out treatment than other personality disorder patients. Unfortunately, due to the nature of the disorder, they are very difficult patients 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 patient identify their dependency and become more self-reliant. Cognitive therapists utilize techniques to help patients change their helpless beliefs and improve problem-solving skills (Beck & Weishaar, 2011).

13.5.2.4. Narcissistic personality disorder. 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 patients how to empathize with others (Beck & Weishaar, 2014).

13.5.3. Cluster C

While many individuals within avoidant and OCPD personality disorders seek out treatment to address their anxiety or depressive like symptoms, it is often difficult to keep them in treatment due to distrust

or 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). Specific to OCPD, cognitive techniques aimed at changing dichotomous thinking, perfectionism, and chronic worrying are helpful in managing symptoms of OCPD. 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 when treating avoidant personality disorder (Herbert, 2007). Antianxiety and antidepressant medications commonly used to treat anxiety disorders have also been used with minimal efficacy; furthermore, symptoms resume as soon as medication is discontinued.

Unlike other personality disorders where individuals are skeptical of the clinician, individuals with dependent personality disorder place a large emphasis of their treatment on the clinician. Therefore, one of the main treatment goals for dependent personality disorder patients 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 inability to care for oneself have been minimally effective in establishing independence. Additionally, behavioral techniques such as assertiveness training as 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 relationship between the patient and a family member/spouse being the primary issue; however, research on this treatment method has not yielded consistent positive results (Nichols, 2013).

Module Recap

Module 13 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, epidemiology, comorbidity, etiology, and treatment of personality disorders. This is the final module of Part V, Block 4. In Module 5 we cover one last set of personality disorders – neurocognitive – and then contemporary issues in psychopathology.

VI

Part VI. Mental Disorders – Block 5

Part VI. Mental Disorders – Block 5

Module 14: Neurocognitive Disorders

Module Overview

In Module 14, we will discuss matters related to neurocognitive disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include Delirium, Major Neurocognitive Disorder, and Mild Neurocognitive Disorder. We also discuss nine subtypes to include: Alzheimer's disease, Traumatic Brain Injury (TBI), Vascular Disorders, Substance Abuse, Dementia with Lewy Bodies, Frontotemporal Lobar Degeneration (FTLD), Parkinson's disease, Huntington's disease, and HIV infection. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 14.1. Clinical Presentation
- 14.2. Epidemiology
- 14.3. Etiology
- 14.4. Treatment

Module Learning Outcomes

- Describe how neurocognitive disorders present.
- Describe the epidemiology of neurocognitive disorders.
- Describe the etiology of neurocognitive disorders.
- Describe treatment options for neurocognitive disorders.

14.1. Clinical Presentation

Section Learning Objectives

- Describe how Delirium presents itself.
- Describe how Major Neurocognitive Disorder presents itself.
- Describe how Mild Neurocognitive Disorder presents itself.

Unlike many of the disorders we have discussed thus far, neurocognitive disorders often result from

disease processes or medical conditions. Therefore, it is important that individuals presenting with these symptoms complete a medical assessment to better determine the etiology behind the disorder.

There are three main categories of neurocognitive disorders- Delirium, Major Neurocognitive Disorder, and Mild Neurocognitive Disorder. Within major and minor neurocognitive disorders are several subtypes due to the etiology of the disorder. For the purpose of this course, we will review diagnostic criteria for both major and minor neurocognitive disorders, followed by a brief description of the various disease subtypes in the etiology section.

14.1.1. Delirium

Delirium is characterized by a significant disturbance in attention or awareness and cognitive performance that is significantly altered from one's usual behavior (APA, 2013). Disturbances in attention are often manifested as difficultly to sustain, shift, or focus attention. Additionally, an individual experiencing an episode of delirium will also have a disruption in cognition, including confusion of where they are. Disorganized thinking, incoherent speech, and hallucinations and delusions may also be observed during periods of delirium. Onset of delirium is abrupt, occurring over a period of several hours. Symptoms can range from mild to severe, and can last from days to several months.

14.1.2. Major Neurocognitive Disorder

Individuals with major neurocognitive 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 (APA, 2013). While it is not necessary, it is helpful to have documentation of the cognitive decline via neuropsychological testing within a controlled, standardized testing environment. Information from close family members or caregivers is also important in documenting the decline and impairment in areas of functioning.

Within the umbrella of major neurocognitive disorder is **dementia**, a major decline in cognition and self-help skills due to a neurocognitive disorder. The DSM-V (APA, 2013) refrained from using this term in diagnostic categories as it is often used to describe the natural decline in degenerative dementias that affect older adults; whereas neurocognitive disorder is the preferred term used to describe conditions affecting younger individuals such as impairment due to traumatic brain injuries or other medical conditions. Therefore, while dementia is accurate in describing those experiencing major neurocognitive disorder due to age, it is not reflective of those experiencing neurocognitive issues secondary to an injury or illness.

14.1.3. Mild Neurocognitive Disorder

Individuals with mild neurocognitive disorder demonstrate a *modest* decline in one of the listed cognitive areas. The decline in functioning is not as extensive as that seen in major neurocognitive disorder and the individual does *not* experience difficulty independently engaging in daily activities;

however, they may require assistance or extra time to complete these tasks, particularly if the cognitive decline continues to progress.

It should be noted that the primary difference between major and mild neurocognitive disorder is the severity of the decline and independent functioning. Some argue that the two are earlier and later stages of the same disease process (Blaze, 2013). Conversely, individuals can go from major to mild neurocognitive disorders following recovery from a stroke or traumatic brain injury (Petersen, 2011).

14.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of neurocognitive disorders.

Delirium often occurs among those hospitalized for other medical issues (up to 24%) and in older individuals. While the rate of occurrence is quite rare among the general public (1-2%), it significantly increases to 14% among individuals older than 85 years old (APA, 2013).

Major and mild neurocognitive disorder prevalence rates vary widely depending on the etiological nature of the disorder. Dementia occurs in 1-2% of individuals age 65 years old and up to 30% of individuals over age 85.

Alzheimer's disease, the most commonly diagnosed neurocognitive disorder, is observed in nearly 5.5 million Americans (Alzheimer's Association, 2017a) with 7% of those between ages 65 and 74; 53% between 75 and 84; and 40% older than 84 (APA, 2013). It should also be noted that somewhere between 60-90% of dementias are attributable to Alzheimer's disease (APA, 2013).

Nearly 1.7 million TBI's occur each year within the United States, with 59% of these injuries occurring in males (APA, 2013). The most common causes of TBI's are falls, automobile accidents, and accidental head strikes (APA, 2013). There has also been an increase in TBI's within the military due to the recent wars in Iraq and Afghanistan.

14.3. Etiology

Section Learning Objectives

- Define degenerative.
- Describe the symptoms and causes of Alzheimer's disease.
- Describe the symptoms and causes of Traumatic Brain Injury (TBI).
- Describe the symptoms and causes of Vascular Disorders.
- Describe the symptoms and causes of Substance Abuse.
- Describe the symptoms and causes of Dementia with Lewy Bodies.
- Describe the symptoms and causes of Frontotemporal Lobar Degeneration (FTLD).
- Describe the symptoms and causes of Parkinson's disease.
- Describe the symptoms and causes of Huntington's disease.
- Describe the symptoms and causes of HIV infection.

Neurocognitive disorders occur due to a wide variety of medical conditions or injury to the brain. Therefore, this section will focus on a brief description of the nine different etiologies of neurocognitive disorders per the DSM-V. As you will see, majority of these neurocognitive disorders are both **degenerative**, meaning the symptoms and cognitive deficits become worse overtime, as well as related to a medical condition or disease.

Per the DSM-V (APA, 2013), an individual will meet diagnostic criteria for *either* mild or major neurocognitive disorder as listed above. In order to specify the type of neurocognitive disorder, additional diagnostic criteria needs to be met specific to one of the following subtypes.

14.3.1. Alzheimer's Disease

Alzheimer's disease is the most prevalent neurodegerative disorder. While the primary symptom of Alzheimer's disease is the gradual progression of impairment in cognition, it is important to also identify concrete evidence of cognitive decline. This can be done in one of two ways: via genetic testing of the individual or a documented family history of the disease, or, through clear evidence of cognitive decline over time by repeated standardized neuropsychological evaluations (APA, 2013). It is important to identify these markers in making the diagnosis of Alzheimer's disease as there are some individuals who present with memory impairment but eventually show reversal of symptoms; this is not the case for individuals with Alzheimer's disease (APA, 2013).

14.3.1.1. Causes of Alzheimer's disease. Autopsies of individuals diagnosed with Alzheimer's disease identify two abnormal brain structures— **beta-amyloid plaques** and **neurofibrillary tangles**— both of which are responsible for neuron death, inflammation, and loss of cellular connections (Lazarov, Mattson, Perterson, Pimplika, & van Praag, 2010). It is believed that beta-amyloid plaques, large bundles of plaque that develop *between* neurons, appear before development of dementia symptoms. As these plaque bundles increase in size and number, cognitive symptoms and impaired daily functioning become evident to close family members. Neurofibrillary tangles are believed to appear after the onset of dementia symptoms, and are found *inside* of cells, affecting the protein that helps transport nutrients in healthy cells. Both beta-amyloid plaques and neurofibrillary tangles impact the health of neurons within the hippocampus, amygdala, and the cerebral cortex, areas associated with memory and cognition (Spires-Jones & Hyman, 2014).

Researchers have identified additional genetic and environmental influences in the development of

Alzheimer's disorder. Genetically, the **apolipoprotein E (ApoE)** that helps to eliminate beta-amyloid by-products from the brain has been implicated in the development of Alzheimer's disorder. More specifically, one of the three variants of this gene, the e4 allele, appears to reduce the production of ApoE, thus increasing the number of beta-amyloid plaques within the brain. It should be noted, however, that not all individuals with the e4 allele develop Alzheimer's disease; therefore, this explanation may better explain a vulnerability to Alzheimer's disease as opposed to the cause of the disease.

Various brain regions have also been implicated in the development of Alzheimer's disease. More specifically, neurons shrinking or dying within the hypothalamus, thalamus, and the locus ceruleus have been linked to declining cognition (Selkoe, 2011, 1992). Acetylcholine-secreting neurons within the basal forebrain also appear to shrink or die, contributing to Alzheimer's disease symptoms (Hsu et al., 2015).

Environmental toxins such as high levels of zinc and lead may also contribute to the development of Alzheimer's disease. More specifically, zinc has been linked to the clumping of beta-amyloid proteins, similar to those structures previously discussed in brains of individuals with Alzheimer's disease. Although lead has largely been phased out of environmental toxins due to negative health consequences, current elderly individuals were exposed to these toxic levels of lead in gasoline and paint as young children. There is some speculation that lead and other pollutants may impact cognitive functioning in older adults (Richardson et al., 2014).

14.3.1.2. Onset of Alzheimer's disease. Alzheimer's disease is defined by the onset of symptoms. *Early-onset* Alzheimer's disease occurs before the age of 65. While only a small percentage of individuals experience early-onset of the disease, those that do experience early disease progression appear to have more of a genetic influence of the disease and a higher rate of family members with the disease.

Late-onset Alzheimer's disease occurs after the age of 65 and has less of a familial influence. This onset appears to occur due to a combination of biological, environmental, and lifestyle factors (Chin-Chan, Navarro-Yepes, & Quintanilla-Vega, 2015). Nearly 30% of individuals within this class of diagnosis have the ApoE gene that fails to eliminate the beta-amyloid proteins from various brain structures. It is believed that the combination of the presence of this gene along with environmental toxins and lifestyle choices (i.e. more stress) impact the development of Alzheimer's disease.

14.3.2. Traumatic Brain Injury (TBI)

TBIs occur when an individual experiences a significant trauma or injury to the head. Neurocognitive disorder due to TBI is diagnosed when persistent cognitive impairment is observed immediately following the head injury, along with one or more of the following symptoms: loss of consciousness, posttraumatic amnesia, disorientation and confusion, or neurological impairment (APA, 2013).

The presentation of symptoms varies among individuals and depends largely on the location of the injury and the intensity of the trauma. Furthermore, the effects of the TBI can be temporary or permeant. Symptoms generally range from headaches, disorientation, confusion, irritability, fatigue, poor concentration, as well as emotional and behavioral changes. More severe injuries can result in more significant neurological symptoms such as seizures, paralysis and visual disturbances (APA, 2013).

The most common type of TBI is a concussion. A **concussion** occurs when there is a significant blow to the head, followed by changes in brain functioning. It often causes immediate disorientation or loss of consciousness, along with headaches, dizziness, nausea, and sensitivity to light (Alla, Sullivan, & McCrory, 2012). While symptoms of a concussion are usually temporary, there can be more permanent damage due to repeated concussions, particularly if they are within close time periods. The media has brought considerable attention to this with the recent discussions of **chronic traumatic encephalopathy (CTE)** which is a progressive, degenerative condition due to repeated head trauma. CTE's are most commonly seen in athletes (i.e. football players) and military personnel (Baugh et al., 2012). In addition to the neurological symptoms, psychological symptoms such as depression and poor impulse control have been observed in individuals with CTE. These individuals also appear to be at greater risk for development of dementia (McKee et al., 2013).

14.3.3. Vascular Disorders

Neurocognitive disorders due to vascular disorders can occur from a one-time event such as a stroke, or from ongoing subtle disruptions of blood flow within the brain (APA, 2013). Occurrence of these vascular disorders general begins with **atherosclerosis**, or the clogging of arteries due to a build-up of plaque. The **plaque** builds up over time, eventually causing the artery to narrow, thus reducing the amount of blood able to pass through to other parts of the body. When these arteries within the brain become completely obstructed, a **stroke** occurs. The lack of blood flow during a stroke results in the death of neurons and loss of brain function (APA, 2013). There are two types of strokes—a **hemorrhagic stroke** which occurs when a blood vessel bursts within the brain, and an **ischemic stroke** which is when a blood clot blocks the blood flow in an artery within the brain (American Stroke Association, 2017).

While strokes can occur at any age, majority of strokes occur after age 65 (Hall, Levant, & DeFrances, 2012). A wide range of cognitive, behavioral and emotional changes occur following a stroke. Symptoms are generally dependent on the location of the stroke within the brain as well as the extensiveness of damage to those brain regions (Poels et al., 2012). For example, strokes that occur on the left side of the brain tend to cause problems with speech and language, as well as physical movement on the *right* side of the body; whereas strokes that occur on the right side of the brain tend to cause problems with impulsivity and impair judgement, short-term memory loss, and physical movement on the *left* side of the body (Hedna et al., 2013).

14.3.4. Substance Abuse

As discussed in the Substance Abuse chapter, significant cognitive changes occur due to repetitive drug and alcohol abuse. Delirium can be observed in individuals with extreme substance intoxication, withdrawal, or even when multiple substances have been used within a close time period (APA, 2013). While delirium symptoms are often transient during these states, mild neurocognitive impairment due to heavy substance abuse may remain until a significant period of abstinence is observed (Stavro, Pelletier, & Potvin, 2013).

14.3.5. Dementia with Lewy Bodies

Symptoms associated with neurocognitive disorder due to Lewy bodies include significant fluctuations in attention and alertness; recurrent visual hallucinations; impaired mobility; and sleep disturbance (APA, 2013). While the trajectory of the illness develops more rapidly than Alzheimer's disease, the survival period is similar in that most individuals do not survive longer than 8 years post diagnosis (Lewy Body Dementia Association, 2017).

Lewy bodies are irregular brain cells that result from buildup of abnormal proteins in the nuclei of neurons. These brain cells deplete the cortex of **acetylcholine**, which causes the behavioral and cognitive symptoms observed in both dementia with Lewy bodies and Parkinson's disease. The motor symptoms observed in both these disorders occurs from the depletion of dopamine by the Lewy body brain cells that accumulate in the brain stem.

14.3.6. Frontotemporal Lobar Degeneration (FTLD)

FTLD causes progressive declines in language or behavior due to the degeneration in the frontal and temporal lobes of the brain (APA, 2013). Symptoms of FTLD include significant changes in behavior and/or language. Individuals may present with apathy or disinhibition. Additionally, they may lose interest in socialization as they often lose empathy and sympathy for others. Individuals may also engage in perseverative or compulsive behaviors. Cognitive decline is generally not as prominent as the behavioral and language changes, showing very little deficits in early stages of the disorder; however, they will present with a decline in executive functions such as poor planning and organization, distractibility, and poor judgement to name a few (APA, 2013).

The language deficits are often observed with fluency of speech and word meaning. Individuals often experience difficulties understanding words or naming objects (APA, 2013). Occasionally, muscle weakness and other physical abnormalities are present, although not necessary for diagnosis.

14.3.7. Parkinson's Disease

The awareness of Parkinson's disease has increased in recent years due in large part to Michael J. Fox's early diagnosis in 1991. It is the 2nd most common neurodegenerative disorder in the United States affecting approximately 630,000 individuals (Kowal, Dall, Chakrabarti, Storm, & Jain, 2013). While many are aware of the tremors of hands, arms, legs, or face, additional symptoms of rigidity of the limbs and trunk; slowness in initiating movement; and drooping posture or impaired balance and coordination, are the other three main symptoms of Parkinson's disease (National Institute of Neurological Disorders and Stroke, 2017). These motor symptoms are generally present at least one year prior to the beginning of cognitive decline, although severity and progression of symptoms varies significantly from person to person.

14.3.8. Huntington's Disease

Huntington's disease is a rare, genetic disorder in which involves involuntary movement, progressive dementia, and emotional instability. Due to the degenerative nature of the disorder, there is a shortened life-expectancy as death typically occurs 15-20 years post onset of symptoms (Clabough, 2013). Although symptoms can present at any time, the average age of symptom presentation is during middle adulthood (APA, 2013). Symptoms generally begin with neurocognitive decline along with changes in mood/personality. As symptoms progress, more physical symptoms present such as facial grimaces, difficulty speaking, and repetitive movements. Because there is no treatment for Huntington's disease, the severity of the cognitive and physical impairments ultimately lead to complete dependency and need for full-time care.

14.3.9. HIV Infection

Not many people are aware that cognitive impairment is sometimes the first symptom of untreated HIV. While symptoms vary among individuals, slower mental processing, difficulty with complex tasks, and difficulty concentrating/learning new information are among the most common early signs (APA, 2013). When HIV becomes active in the brain, significant alterations of mental processes occurs, thus leading to a diagnosis of *neurocognitive disorder due to HIV infection*. Significant impairment can also occur due to HIV-infection related inflammation throughout the central nervous system.

Fortunately, antiretroviral therapies used in treating HIV have been effective in reducing and preventing the onset of severe cognitive impairments; however, HIV-related brain changes still occur in nearly half of all patients on antiretroviral medication. There is hope that once antiretroviral therapies are able to cross the blood-brain barrier in the central nervous system, there will be significant improvement in the prevalence in HIV related neurocognitive disorder (Vassallo et al., 2014).

14.4. Treatment

Section Learning Objectives

- Describe treatment options for neurocognitive disorders.

Treatment options for those with neurocognitive disorders are minimal at best, with most attempting to treat secondary symptoms as opposed to the neurocognitive disorder itself. Furthermore, the degenerative nature of these disorders also make it difficult to treat, as many diseases will progress regardless of the treatment options.

14.4.1. Pharmacological

Pharmacological interventions, and more specifically medications designed to target acetylcholine and

glutamate, the main neurotransmitters affected by the disease, have been the most effective treatment options in alleviating symptoms and reducing speed of cognitive decline within individuals diagnosed with Alzheimer's disease. Specific medications such as *donepezil* (Aricept), *rivastigmine* (Exelon), *galantamine* (Razadyne), and *memantine* (Mamenda) are among the most commonly prescribed (Alzheimer's Association, 2017a). Due to possible negative side effects of the medications, these drugs are most commonly prescribed to individuals in early/middle stages of Alzheimer's as opposed to those with advanced disease. Researchers have also explored treatment options aimed at preventing the build-up of beta-amyloid and neurofibrillary tangles, however, this research is still in its infancy (Alzheimer's Association, 2017a)

Parkinson's disease has also found success in pharmacological treatment options. The medication *levodopa* increases dopamine availability, which provides relief to both physical and cognitive symptoms. Unfortunately, there are also significant side effects such as hallucinations and psychotic symptoms; therefore, the medication is often only used when the benefits outweigh the negatives of the potential risks (Poletti & Bonuccelli, 2013).

14.4.2. Psychological

Among the most effective psychological treatment options for individuals with neurocognitive disorders are the use of cognitive and behavioral strategies. More specifically, engaging in various cognitive activities such as computer based cognitive stimulation programs, reading books, and following the news, have been identified as effective strategies in preventing or delaying the onset of Alzheimer's disease (Szalavits, 2013; Wilson, Segawa, Boyle, & Bennett, 2012).

Engaging in social skills and self-care training are additional behavioral strategies used to help improve functioning in individuals with neurocognitive deficits. For example, by breaking down complex tasks into smaller, more attainable goals, as well as simplifying the environment (i.e. labeling location of items, removing clutter), individuals are able to successfully engage in more independent living activities.

14.4.3. Support for Caregivers

Supporting caregivers is an important treatment option to include as the emotional and physical toll on caring for an individual with a neurocognitive disorder is often underestimated. According to the Alzheimer's Association (2017b), nearly 90% of all individuals with Alzheimer's disease is cared for by a relative. The emotional and physical demands on caring for a family member who continues to cognitively and physically decline can lead to increased anger and depression in a caregiver (Kang et al. 2014). It is important that medical providers routinely assess caregivers psychosocial functioning, and encourage caregivers to participate in caregiver support groups, or individual psychotherapy to address their own emotional needs.

Module Recap

Our discussion in Module 14 turned to neurocognitive disorders to include the categories of Delirium, Major Neurocognitive Disorder, and Mild Neurocognitive Disorder**.** We also discussed the subtypes of Alzheimer's disease, Traumatic Brain Injury (TBI), Vascular Disorders, Substance Abuse, Dementia with Lewy Bodies, Frontotemporal Lobar Degeneration (FTLD), Parkinson's disease, Huntington's disease, and HIV infection. The clinical description, epidemiology, etiology, and treatment options for neurocognitive disorders was discussed. This is the final class of disorders we will discuss in this book. If you have questions, please let your instructor know. In Module 15 we will finish out our discussion of psychopathology by covering contemporary issues in psychopathology.

Module 15: Contemporary Issues in Psychopathology

Module Overview

In our final module we will tackle the issue of how clinical psychology interacts with law. Our discussion will include issues related to civil and criminal commitment, patient's rights, and the patient-therapist relationship. We end on an interesting note and discuss whether gaming can be addictive. Enjoy.

Module Outline

- 15.1. Legal Issues Related to Mental Illness
- 15.2. Patient's Rights
- 15.3. The Therapist-Client Relationship
- 15.4. Future Directions

Module Learning Outcomes

- Describe how clinical psychology interacts with law.
- Describe issues related to civil commitment.
- Describe issues related to criminal commitment.
- Outline patient's rights.
- Clarify concerns related to the therapist-client relationship.

15.1. Legal Issues Related to Mental Illness

Section Learning Objectives

- Define forensic psychology/psychiatry.
- Describe potential roles a forensic psychologist might have.
- Define civil commitment.
- Identify criteria for civil commitment.
- Describe dangerousness.
- Outline procedures in civil commitment.
- Define criminal commitment.
- Define NGRI.
- Describe pivotal rules/acts/etc. in relation to the concept of insanity.
- Define GBMI.
- Clarify what it means to be competent to stand trial.

15.1.1. Forensic Psychology/Psychiatry

According to the American Psychological Association, **forensic psychology/psychiatry** is when clinical psychology is applied to legal arena in terms of assessment, treatment, and evaluation. Forensic psychology can also include the application of research from other subfields in psychology to include cognitive and social psychology. Training includes law and forensic psychology, and solid clinical skills are a must. According to APA, a forensic psychologist might, "perform such tasks as threat assessment for schools, child custody evaluations, competency evaluations of criminal defendants and of the elderly, counseling services to victims of crime, death notification procedures, screening and selection of law enforcement applicants, the assessment of post-traumatic stress disorder and the delivery and evaluation of intervention and treatment programs for juvenile and adult offenders." A key issue investigated by forensic psychologists includes "*mens rea*" or the insanity plea. We will discuss this shortly.

To learn more about forensic psychology, or to investigate the article mentioned above, please visit:

http://www.apa.org/ed/precollege/psn/2013/09/forensic-psychology.aspx

15.1.2. Civil Commitment

15.1.2.1. What is civil commitment? When individuals with a mental illness behave in erratic or potentially dangerous ways, to either themselves or others, then something must be done. The responsibility to act falls on the government through what is called *parens patriae* or "father of the country" or "country as parent." Action in this case involves involuntary commitment in a hospital or mental health facility and is done to protect the individual and express concern over their well-being; much like a parent would do for their child. It should be noted that an individual can voluntarily admit themselves to a mental health facility and upon doing so, staff will determine whether or not treatment and extended stay, are needed.

15.1.2.2. Criteria for civil commitment. Though states vary in the criteria used to establish the need for civil commitment, some criteria are common across states. First, the individual must present a clear danger to either themselves or others. Second, the individual demonstrates that he or she is unable to care for him or herself or make decisions about whether treatment or hospitalization is necessary. Finally, the individual believes he/she is about to lose control and so is in need of treatment or care in a mental health facility.

15.1.2.3. Assessment of "dangerousness." Dangerousness can best be defined as the person's capacity or likelihood for harming self or others. Most people believe that those who are mentally ill are more dangerous than those free of mental illness, especially when espousing self-reported conservatism and RWA (Right-Wing Authoritarianism; Gonzales, Chan, and Yanos, 2017; DeLuca and Yanos, 2015) or after tragic events such as a mass shooting (Metzl & MacLeish, 2015). The media plays a role in this and as McGinty et al. (2014) found, 70% of news coverage of serious mental illness (SMI) and gun violence over a 16 year period (1997 to 2012) was event focused and described specific shooting events by persons with SMI. The authors wrote, "Even in thematic news coverage focused on describing the general problem of SMI and gun violence, the majority of news stories did not

mention that most people with SMI are not violent or that we lack tools capable of accurately identifying persons with SMI who are at heightened risk of committing future violence." They concluded that media coverage of persons with SMI as violent may contribute to negative public attitudes.

Rozel & Mulvey (2017) showed that mental illness is a weak risk factor for violence though this is not to say that the mentally ill do not commit violent acts. The authors write, "…it has been documented repeatedly that people who report diagnosable levels of psychiatric symptoms also report more involvement in acts of violence toward others than the general population reports." It is estimated that roughly 4% of criminal violence can be attributed to the mentally ill (Metzl & MacLeish, 2015) while those with mental illness are three times more likely to be targets and not perpetrators of violence (Choe et al, 2008).

Regardless of this, we do attempt to identify the level of dangerousness a person may exhibit or have the potential to exhibit. How easy is it to make this prediction? As you might think, it can be very difficult. First, the definition of dangerousness is vague. It implies physical harm, but what about psychological abuse or the destruction of property? Second, past criminal activity is a good predictor of future dangerousness but is often not admissible in court. Third, context is critical and in some situations the person is perfectly fine, but in other circumstances, like having to wait in line at the DMV, the person experiences considerable frustration and eventually anger or rage.

15.1.2.4. Procedures in civil commitment. The procedure for civil commitment does vary a bit from state to state, but some procedures are held in common. First, a family member, mental health professional, or primary care practitioner, may request that the court order an examination of an individual. If the judge agrees, two professionals, such as a mental health professional or physician, are appointed to examine the person in terms of their ability for self-care, need for treatment, psychological condition, and likelihood to inflict harm on self or others. Next, a formal hearing is held which gives the examiners a chance to testify as to what they found. Testimonials may also be given by family and friends, or by the individual him/herself. Once this is completed, the judge renders judgment about whether confinement is necessary and if so, for how long. Typical confinements last from 6 months to 1 year, but an indefinite period can be specified too. In the latter case, the individual has periodic reviews and assessment. In emergency situations, the process stated above can be skipped and short-term commitment made, especially if the person is an imminent threat to him/herself or others.

Before we move on, consider for a minute that a person who is accused of a crime is innocent until proven guilty, has a trial, and if found guilty beyond a reasonable doubt (or almost complete certainty) is only then incarcerated. This is not true of the mentally ill who may be committed to a facility without ever having committed a crime or having a trial, but simply because he or she was judged as having the *potential* to do so (or was seen as dangerous). This *potential* means that there has to be "clear and convincing" proof which the Supreme Court defines as 75% certainty. Obviously, the standard to commit is much different for those accused of criminal acts and those who are mentally ill.

15.1.3. 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. This is called **criminal commitment.** Individuals may plead **not guilty by reason of insanity (NGRI)** or as it is also called, the **insanity plea**. When a defendant pleads NGRI they are acknowledging their guilt for the crime (*actus rea)* but wish to be seen as not guilty since they were mentally ill at the time (*mens rea)*.

The origins of the modern definition of insanity go back to Daniel M'Naghten in 1843 England. He murdered the secretary to British Prime Minister, Robert Peel, during an attempted assassination of the Prime Minister. He was found to be not guilty due to delusions of persecution which outraged the public and led to calls for a more clear definition of insanity. The **M'Naghten rule** states that the having a mental disorder at the time of a crime does not mean the person was insane. The individual also had to be unable to know right from wrong or comprehend the act as wrong. But how do you know what the person's level of awareness was when the crime was committed?

Dissatisfaction with the M'Haghten rule led some state and federal courts in the U.S. to instead adopt the **irresistible impulse test** (1887) which focused on the inability of a person to control their behaviors. The issue with this rule is in distinguishing when a person is unable to maintain control rather than choosing not to exert control over their behavior. This meant there were two choices in the U.S. in terms of how insanity was defined – the M'Haghten rule and the irresistible impulse test. A third test emerged in 1954 from the Durham v. United States case, though it was short lived. The **Durham test, or products test,** stated that a person was not criminally responsible if their crime was a *product* of a mental illness or defect. It offered some degree of flexibility for the courts, but was viewed as too flexible. Since almost anything can cause something else, the term product is too vague.

In 1962, the American Law Institute (ALI) offered a compromise to the three tests/rules in use at the time. Called the **American Law Institute standard**, it stated that people are not criminally responsible for their actions if at the time of their crime they had a mental disorder or defect that did not allow them to distinguish right from wrong and to obey the law. Though this became the standard, it also became controversial when defense attorneys used it as the basis to have John Hinckley, accused of attempting to assassinate President Ronald Regan, found not guilty by reason of insanity in 1982.

Public uproar led the American Psychiatric Association to reiterate the stance of the M'Naghten test and assert people were only insane if they did not know right from wrong when they committed their crime. The **Federal Insanity Defense Reform ACT (IDRA)** of 1984, "was the first comprehensive Federal legislation governing the insanity defense and the disposition of individuals suffering from a mental disease or defect who are involved in the criminal justice system." The ACT included the following provisions:

- significantly modified the standard for insanity previously applied in the Federal courts
- placed the burden of proof on the defendant to establish the defense by clear and convincing evidence
- limited the scope of expert testimony on ultimate legal issues
- eliminated the defense of diminished capacity, created a special verdict of "not guilty only by reason of insanity," which triggers a commitment proceeding
- provided for Federal commitment of persons who become insane after having been found guilty or while serving a Federal prison sentence.

Source: https://www.justice.gov/usam/criminal-resource-manual-634-insanity-defense-reform-act-1984

This is the current standard in all federal courts and about half of all state courts, with Idaho, Kansas, Montana, and Utah choosing to get rid of the insanity plea all together.

For more on the insanity plea, please visit:

https://www.npr.org/sections/health-shots/2016/08/05/487909967/with-no-insanity-defense-seriously-ill-people-end-up-in-prison

Another possibility is for the jury to deliver a verdict of **guilty but mentally ill (GBMI)**, effectively acknowledging that the person did have a mental disorder when committing a crime, but the illness was not responsible for the crime itself. The jurors can then convict the accused and suggest he or she receive treatment. Though this looks like an excellent alternative, jurors are often confused by it (Melville & Naimark, 2002), NGRI verdicts have not been reduced, and all prisoners have access to mental health care anyway. Hence it differs from a guilty verdict in name only (Slovenko, 2011; 2009).

A final concept critical to this discussion is whether the defendant is **competent to stand trial** and refers to the accused's mental state at the time of mental examination after arrest and before going to trial. To be deemed competent, Federal law dictates that the defendant must have a rational and factual understanding of the proceedings and be able to rationally consult with counsel when presenting his/her defense (Mossman et al., 2007; Fitch, 2007). This condition guarantees criminal and civil rights and ensures the accused understands what is going on during the trial and can aid in his or her defense. If they are not fit or competent then they can be hospitalized until their mental state improves.

15.2. Patient's Rights

Section Learning Objectives

- Describe rights patients with mental illness have and identify key court cases.

The following are several rights that patients with mental illness have. They include:

- **Right to Treatment –** The 1966 case of *Rouse v.* Cameron, the D.C. District court said that the right to treatment is a constitutional right and failure to provide resources cannot be justified due to insufficient resources. In the 1972 case of *Wyatt v. Stickney*, a federal court ruled that the state of Alabama was constitutionally obligated to provide all people who were committed to institutions with adequate treatment and had to provide more therapists, privacy, exercise, social interactions, and better living conditions for patients. In the case of *O'Connor v. Donaldson* (1975), the court ruled that patient's cases had to be reviewed periodically to see if they could be released and if they are not a danger *and* are able to survive on their own or with help from family or friends, that they be released.
- **Right to Refuse Treatment** – As patients have the right to request treatment, they too have the right to refuse treatment such as biological treatment, psychotropic medications (*Riggins v. Nevada, 1992)*, and electroconvulsive therapy.
- **Right to Less Restrictive Treatment** – In *Dixon v. Weinberger* (1975), a U.S. District court ruled that individuals have a right to receive treatment in facilities less restrictive than mental

institutions. The only patients who can be committed to hospitals are those unable to care for themselves.
- **Right to Live in a Community** – The 1974 U.S. District court case, *Staff v. Miller,* ruled that state mental hospital patients had a right to live in live in adult homes in their communities.

15.3. The Therapist-Client Relationship

Section Learning Objectives

- Describe three concerns related to the therapist-client relationship.

Three concerns are of paramount important in terms of the therapist-client relationship. These include the following:

- **Confidentiality –** As you might have learned in your introduction to psychology course, **confidentiality** guarantees that information about you is not disseminated without your consent. This applies to students in participating in research studies as well as patients seeing a therapist.
- **Privileged communication –** Confidentiality is an ethical principle while **privileged communication** is a legal one, and states that confidential communications cannot be disseminated without the patient's permission. There are a few exceptions to this which include the client being younger than 16, when they are a dependently elderly person and a victim of a crime, or when the patient is a danger to him or herself or others, to name a few.
- **Duty to Warn** – In the 1976, *Tarasoff v. the Board of Regents of the University of California* ruling, the California Supreme Court said that a patient's right to confidentiality ends when there is a danger to the public, and that if a therapist determines that such a danger exists, he/she is obligated to warn the potential victim. Tatiana Tarasoff, a student at UC, was stabbed to death by graduate student, Prosenjit Poddar in 1969, when she rejected his romantic overtures, and despite warnings by Poddar's therapist that he was an imminent threat. The case highlights the fact that therapists have a legal and ethical obligation to their client but at the same time a legal obligation to society. How exactly should they balance these competing obligations, especially when they are vague? The 1980 case of *Thompson v. County of Alameda* ruled that a therapist does not have a duty to warn if the threat is nonspecific.

Check This Out:

Can you play video games so much, that it becomes addictive? Does this mean that it is a diagnosable mental illness to be listed in the DSM 5? Currently, the disorder is only listed in the DSM 5 as a *condition for further study* and has called it **internet gaming disorder**. Primarily affecting adolescent males aged 12 to 20, it is thought to include symptoms such as:

- Preoccupation or obsession with Internet games

- Withdrawal symptoms when not playing Internet games
- The person has tried to stop or curb playing Internet games, but has failed to do so
- A person has had continued overuse of Internet games even with the knowledge of how much they impact a person's life
- The person uses Internet games to relieve anxiety or guilt or to escape

Psychology Today writes, "Again, while Internet Gaming Disorder is not an "official" disorder in the *DSM-5*, the APA is encouraging further research on the disorder for possible inclusion in future editions of the *DSM*."

(See: https://www.psychologytoday.com/us/blog/here-there-and-everywhere/201407/internet-gaming-disorder-in-dsm-5)

Did the World Health Organization (WHO) already make a decision for themselves about this? In the draft of ICD 11 the WHO list the disorder as a mental health condition and defines it as: "a "persistent or recurrent" behavior pattern of "sufficient severity to result in significant impairment in personal, family, social, educational, occupational or other important areas of functioning." For more on this "disorder," check out the following articles:

- The Cognitive Psychology of Internet Gaming Disorder (2014 article in Clinical Psychology Review) – https://www.sciencedirect.com/science/article/pii/S0272735814000658
- CNN – https://www.cnn.com/2017/12/27/health/video-game-disorder-who/index.html
- Huffington Post – https://www.huffingtonpost.com/christopher-j-ferguson/the-muddled-science-of-internet-gaming-disorder_b_9405478.html
- WHO – http://www.who.int/features/qa/gaming-disorder/en/

What do you think?

Module Recap

And that's it. Our final module explored some concepts that transcend any one mental disorder but affect people with mental illness in general. This included civil and criminal commitment and issues such as NGRI or the insanity plea, what makes someone dangerous and what we should do about it, and determining competency to stand trial. We then moved to patient's rights such as the right to treatment

and conversely, the right to refuse treatment. Finally, we ended by discussing the patient-therapist relationship and specifically, when the patient's right ro confidentiality and privileged communication ends, and the therapist has a moral and legal obligation to warn. We hope you find these topics interesting and explore the issues further through the links that were provided and peer reviewed articles that were cited.

1

References

For a complete list of references, please access the link below:

References – Abnormal Psychology 1st Edition

2

Index

#

A

B

C

D

E

F

G

H

I

J

K

L

M

N

O

P

Q

R

S

T

U

V

W

X

Y

Z