Discovering Psychology Series

Behavioral Disorders of Childhood

3rd edition (5-TR)

Kristy McRaney, Ph.D.

Alexis Bridley, Ph.D.

Lee Daffin, Ph.D.

Washington State University

Version 3.00

August 2022

Contact Information about this OER:

1. Dr. Lee Daffin, Associate Professor of Psychology – ldaffin@wsu.edu

Table of Contents

Record of Changes

Edition	As of Date	Changes Made
1.0	January 2019	Initial writing; feedback pending
2.0	February 2021	Revisions and addition of vignettes
3.00	December 2022	Additional round of text revisions but main changes were to update the book to the newly released DSM 5-TR in March 2022. Some references have been updated as well.

Tokens of Appreciation

August 2022

Kristy, Alexis and I want to offer a special thank you to Ms. Robin Valkren, undergraduate within the online Bachelor of Science degree in Psychology program, for her proofreading of the 3rd edition during the fall 2022. Her edits and feedback and were incredibly helpful in making the quality of this textbook higher.

And now to our reader. We hope you enjoy the book and please, if you see any issues whether typographical, factual, or just want to suggest some type of addition to the material or another way to describe a concept, general formatting suggestion, etc. please let us know. The beauty of Open Education Resources (OER) is that we can literally make a minor change immediately and without the need for expensive printings of a new edition. And it's available for everyone right away. If you have suggestions, please email them to either Alexis or myself (Lee Daffin) using the emails on the title page.

Enjoy the 3rd edition of Behavioral Disorder of Childhood.

Lee Daffin

On behalf of, Kristy McRaney and Alexis Bridley

Part I. Setting the Stage

Part I. Setting the Stage

Module 1:
What is Child Psychopathology?

Module 1: What is Child Psychopathology?

Module Overview

Ben is an 8-year-old boy who lives in suburban Chicago, IL. He makes good grades and is one of the brightest kids in his class. However, he is constantly moving, gets distracted, talks out of turn and to his peers during quiet time, and makes many careless mistakes on his tests. He often gets in trouble at school for his disruptive behavior. At home, his parents get frustrated because they have to repeat directions and remind him to complete chores frequently. Ben also loves baseball, but he keeps getting distracted while on the field and misses important opportunities during the game, leading his peers to become frustrated with him. His parents can tell that Ben wants to make good behavioral choices, but for some reason, before even thinking, he ends up making a poor choice. Out of concern, his parents take Ben to see a child psychologist.

Ben's story, though hypothetical, is true of many children. Although many young children have excessive energy and struggle at times to stay on task, Ben's difficulties seem to exceed that of his peers. His behavioral problems are causing him to experience impairments in more than one life domain 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 the way society views mental illness today and how it has been regarded throughout history. Then we will review 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 how mental health professionals classify mental disorders.
- Describe the effect of stigma on those who have a mental illness.
- Outline the history of mental illness.
- Describe the 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 first have to understand what normal behavior is. *Normal* 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 20th 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 viewpoint espoused the dominant worldview of the time – mechanism – which presented the world as a great machine explained through the principles of physics and chemistry. In it, human beings serve as 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 an improvement, 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.”

Starting in the 1960s, figures such as Abraham Maslow and Carl Rogers sought to overcome the limitations of psychoanalysis and behaviorism by establishing a "third force" psychology, also known as humanistic psychology. 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, their methodology was much different. While humanistic psychology generally relied on qualitative methods, positive psychology utilizes a quantitative approach and aims to help people make the most out of life's setbacks, relate well to others, find fulfillment in creativity, and find lasting meaning and satisfaction (https://www.positivepsychologyinstitute.com.au/what-is-positive-psychology).

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 Abnormal Behavior Is?

In the previous section we showed that what we might consider normal behavior is difficult to define. Equally challenging is understanding what abnormal behavior is, which may be surprising to you. A publication which you will become intimately familiar with throughout this book, the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental*

Disorders 5th edition, Text Revision (DSM-5-TR; 2022), states that, “Although no definition can capture all aspects of the range of disorders contained in DSM-5" (pg. 13) 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. 14). Abnormal behavior, therefore, has the capacity to make 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. 14). 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 exhibit abnormal behavior are generally positive while doing so.
- **Deviance** – Closer examination of the word *abnormal* 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 infrequently occurs (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. Thus, a person is said to be deviant when he or she fails to follow the stated and unstated rules of society, called **social norms**. Social norms change over time due to shifts in accepted values and expectations. For instance, homosexuality was 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. However, 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 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 imply a person is 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.

1.1.3. The Costs of Mental Illness

This leads us to wonder what the cost of mental illness is to society. The National Alliance on Mental Illness (NAMI) states that mental illness affects a person's life which then ripples out to the family, community, and world. For instance, people with serious mental illness are at increased risk for diabetes, cancer, and cardiometabolic disease while 18% of those with a mental illness also have a substance use disorder. Within the family, an estimated 8.4 million Americans provide care to an adult with an emotional or mental illness with caregivers spending about 32 hours a week providing unpaid care. At the community level 21% of the homeless also have a serious mental illness while 70% of youth in the juvenile justice system have at least one mental health condition. And finally, depression is a leading cause of disability worldwide and depression and anxiety disorders cost the global economy $1 trillion each year in lost productivity (Source: NAMI, The Ripple Effect of Mental Illness infographic; https://www.nami.org/Learn-More/Mental-Health-By-the-Numbers).

In terms of worldwide impact, data from 2010 estimates $2.5 trillion in global costs, with $1.7 trillion being indirect costs (i.e., invisible costs "associated with income losses due to mortality, disability, and care seeking, including lost production due to work absence or early retirement") and the remainder being direct (i.e., visible costs to include "medication, physician visits, psychotherapy sessions, hospitalization," etc.). It is now projected that mental illness costs will be around $16 trillion by 2030. The authors add, "It should be noted that these calculations did not include costs associated with mental disorders from outside the healthcare system, such

as legal costs caused by illicit drug abuse" (Trautmann, Rehm, & Wittchen, 2016). The costs for mental illness have also been found to be greater than the combined costs of somatic diseases such as cancer, diabetes, and respiratory disorders (Whiteford et al., 2013).

Christensen et al. (2020) did a review of 143 cost-of-illness studies that covered 48 countries and several types of mental illness. Their results showed that mental disorders are a substantial economic burden for societies and that certain groups of mental disorders are more costly than others. At the higher cost end were developmental disorders to include autism spectrum disorders followed by schizophrenia and intellectual disabilities. They write, "However, it is important to note that while disorders such as mood, neurotic and substance use disorders were less costly according to societal cost per patient, these disorders are much more prevalent and thus would contribute substantially to the total national cost in a country." And much like Trautmann, Rehm, & Wittchen (2016) other studies show that indirect costs are higher than direct costs (Jin & Mosweu, 2017; Chong et al., 2016).

1.1.4. Defining Key Terms

Our discussion so far has concerned what normal and abnormal behavior is. 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 predict reliably, explain, diagnose, identify the causes of, and treat maladaptive behavior, is what we refer to as **abnormal psychology**. Abnormal behavior can become pathological and has led to the scientific study of psychological disorders, or **psychopathology**. From our previous discussion we can fashion the following definition of a psychological or mental disorder: **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. **Child psychopathology** refers to mental disorders that present during childhood.

You should have learned the following in this section:

- Abnormal behavior is a combination of personal distress, psychological dysfunction, deviance from social norms, dangerousness to self and others, and costliness to society.
- Abnormal psychology is the scientific study of abnormal behavior, with the intent to be able to predict reliably, explain, diagnose, identify the causes of, and treat maladaptive behavior.
- The study of psychological disorders is called psychopathology.
- 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.
- Child psychopathology refers to mental disorders that present during childhood.

Section 1.1 Review Questions

1. What is the disease model and what problems existed with it? What was to overcome its limitations?
2. Can we adequately define normal behavior? What about abnormal behavior?
3. What aspects are part of the American Psychiatric Association's definition of abnormal behavior?
4. How costly is mental illness?
5. What is abnormal psychology?
6. What is psychopathology?
7. How do we define mental disorders?
8. What is child psychopathology?

1.2. Classifying Mental Disorders

Section Learning Objectives

- Define and exemplify classification.
- Define nomenclature.
- Define epidemiology.
- Define the presenting problem and clinical description.
- Differentiate prevalence, incidence, and any subtypes.
- Define comorbidity.
- Define etiology.
- Define course.
- Define prognosis.
- Define treatment.

1.2.1. Classification

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

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

1.2.2. Determining Occurrence of a Disorder

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 divided by the total number of people in the sample. 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 a 2020 infographic by the National Alliance on Mental Illness (NAMI), for U.S. adults, 1 in 5 experienced a mental illness, 1 in 20 had a serious mental illness, 1 in 15 experienced both a substance use disorder and mental disorder, and over 12 million had serious thoughts of suicide (2020 Mental Health By the Numbers: US Adults infographic). In terms of adolescents aged 12-17, in 2020 1 in 6 experienced a major depressive episode, 3 million had serious thoughts of suicide, and there was a 31% increase in mental health-related emergency department visits. Among U.S. young adults aged 18-25, 1 in 3 experienced a mental illness, 1 in 10 had a serious mental illness, and 3.8 had serious thoughts of suicide (2020 Mental Health By the Numbers: Youth and Young Adults infographic). These numbers would represent period prevalence rates during the pandemic, and for the year 2020. In the, You are Not Alone infographic, NAMI reported the following 12-month prevalence rates for U.S. Adults: 19% having an anxiety disorder, 8% having depression, 4% having PTSD, 3% having bipolar disorder, and 1% having schizophrenia.

Source: https://www.nami.org/mhstats

Incidence indicates the number of new cases in a population over a specific period. 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. The first study, conducted from 1980 to 1985, 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 terms of disability, is strongly related to comorbidity, and that substance use disorders often result from disorders such as anxiety and bipolar disorders. The implications of this are significant as services to treat substance abuse and mental disorders are often separate, despite the disorders appearing together.

1.2.3. Other Key Factors Related to Mental Disorders

The **etiology** is the cause of the disorder. There may be social, biological, or psychological explanations for the disorder 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 in Module 2.

The **course** of the disorder is its particular pattern. A disorder may be *acute*, meaning that it lasts a short time, or *chronic,* meaning it persists for a long time. It can also be classified as *time-limited*, meaning that recovery will occur after some 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 facility, or through inpatient care or hospitalization at a mental hospital or psychiatric unit of a general hospital. According to NAMI, the average delay between symptom onset and treatment is 11 years with 45% of adults with mental illness, 66% of adults with serious mental illness, and 51% of youth with a mental health condition seeking treatment in a given year. They also report that 50% of white, 49% of lesbian/gay and bisexual, 43% of mixed/multiracial, 34% of Hispanic or Latinx, 33% of black, and 23% of Asian adults with a mental health diagnosis received treatment or counseling in the past year (Source: Mental Health Care Matters infographic, https://www.nami.org/mhstats).

You should have learned the following in this section:

- Classification, or how we organize or categorize things, provides us with a nomenclature, or naming system, to structure our understanding of mental disorders in a meaningful way.
- Epidemiology is the scientific study of the frequency and causes of diseases and other health-related states in specific populations.
- Prevalence is the percentage of people in a population that has a mental disorder or can be viewed as the number of cases divided by the total number of people in the sample.
- Incidence indicates the number of new cases in a population over a specific period.
- Comorbidity describes when two or more mental disorders are occurring at the same time and in the same person.
- The etiology is the cause of a disorder while the course is its particular pattern and can be acute, chronic, or time-limited.
- Prognosis is the anticipated course the mental disorder will take.

Section 1.2 Review Questions

1. What is the importance of classification for the study of mental disorders?
2. What information does a clinical description include?
3. In what ways is occurrence investigated?
4. What is the etiology of a mental illness?
5. What is the relationship of course and prognosis to one another?
6. What is treatment and who seeks it?

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 phenomenon 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 them, assign the person to a category for which we have a schema, and then use that to affect how we interact with them. 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 tend to imply things about an 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 an outside group 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. For example, when a company requires that an applicant have a certain education level or be able to lift 80 pounds as part of typical 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 of a specific 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 views within our conscious awareness.

We have spent quite a lot of space and time understanding 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 avoiding a romantic relationship, 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 altogether, resulting in social isolation. An example is when an employer intentionally does not hire a person because their mental illness is discovered.
- *Label avoidance* –To avoid being labeled as "crazy" or "nuts" people needing care may avoid seeking it altogether 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 worthy 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 a person who has 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 a difficult time had disappeared, resulting in greater levels of stress. To cope, some families concealed their relative's illness, and some parents struggled to decide whether it was their place to disclose their child's condition. Others fought with the issue of confronting the stigma through attempts at education versus just ignoring it due to not having enough energy or desiring to maintain personal boundaries. There was also a need to understand the 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 (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 university 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 also important to 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 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 was less stigmatized; their workplaces were in the county council where they were more likely to encounter patients who recover and return to normal life in society, rather than in 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 curriculum included two methods requiring contact with people 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 a psychiatrist mentored students while they interacted with patients in either inpatient or outpatient settings. Results showed that medical students held a stigma towards mental illness and that comprehensive medical education reduced 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 the 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. Conversely, 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 effectiveness increased among the public.

Self-stigma has also been shown to affect self-esteem, which then affects hope, which then affects the quality of life among people with severe mental illness. 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 illness and recovery, which leads to hopefulness and higher levels of self-esteem in clients. This may then reduce susceptibility to internalized stigma.

Stigma leads to health inequities (Hatzenbuehler, Phelan, & Link, 2013), prompting calls for stigma change. Targeting stigma involves two different agendas: The *services agenda* attempts to remove stigma so people can seek mental health services, and 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. The latter is successful when there is an increase in the number of people with mental illnesses in the workforce who are receiving reasonable accommodations. The federal government has tackled this issue with landmark legislation such as the Patient Protection and Affordable Care Act of 2010, Mental Health Parity and Addiction Equity Act of 2008, and the Americans with Disabilities Act of 1990. However, protections are not uniform across all subgroups due to "1) explicit language about inclusion and exclusion criteria in the statute or implementation rule, 2) vague statutory language that yields variation in the interpretation about which groups qualify for protection, and 3) incentives created by the legislation that affect specific groups differently" (Cummings, Lucas, and Druss, 2013). More on this in Module 15.

You should have learned the following in this section:

- Stigma is when negative stereotyping, labeling, rejection, and loss of status occur and take the form of public or self-stigma, and label avoidance.

Section 1.3 Review Questions

1. How does social cognition help us to understand why stigmatization occurs?
2. Define stigma and describe its three forms. What is courtesy stigma?
3. What are the effects of stigma on the afflicted?
4. Is stigmatization prevalent in the mental health community? If so, what can be done about it?
5. How can we reduce stigmatization?

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 noisemaking.

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 ailments 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 noteworthy was the Greek philosopher Plato (429-347 B.C.), who said that the mentally ill were not responsible for their actions and should not be punished. It was the

responsibility of the community and their families to care for them. The Greek physician Galen (A.D. 129-199) said mental disorders had either physical or psychological causes, including 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 practiced the concept of *contrariis contrarius*, meaning opposite by opposite, and introduced contrasting stimuli to bring about balance in the physical and mental domains. An example would be consuming a cold drink while in a warm bath.

1.4.3. 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 demonic 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 when 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 (bubonic plague) killed up to a third, or according to other estimates almost half, of the population. Famine, war, social oppression, and pestilence were also factors. The constant presence of death 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 again 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 perspective helped continue the decline of supernatural views of mental illness. In the mid to late 1500s, German physician Johann Weyer (1515-1588) 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 vehemently protested and banned by 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, the Salem Witch Trials of 1692, resulted in more than 200 people accused of practicing witchcraft and 20 deaths.

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 the facilities overcrowded, the 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. The earliest proponent was Francis Pinel (1745-1826), the superintendent of la Bicetre, a hospital for mentally ill men in Paris. Pinel stressed respectful treatment and moral guidance for the mentally ill while considering their individual, social, and occupational needs. Arguing that the mentally ill were sick people, Pinel ordered that chains be removed, outside exercise be allowed, sunny and well-ventilated rooms replace dungeons, and patients be extended kindness and support. This approach led to considerable improvement for many of the patients, so much so, that several were released.

Following Pinel's lead, 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 are 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 sentiment 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 reliance on astrology, showing that even he could not escape from the beliefs of the time.

Due to the rise of the moral treatment movement in both Europe and the United States, asylums became habitable places where those afflicted with mental illness could recover. Regrettably, 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 patients and other treatments were needed, though they had not been developed yet. Staff 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 overwhelmed the facilities, and patient counts soared to 1,000 or more. Prejudice against the new arrivals led to discriminatory practices in which immigrants were not

afforded the same 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 leading 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 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 straitjacket for 21 consecutive nights" (https://www.mhanational.org/our-history). His story aroused sympathy from 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: https://www.mhanational.org/

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 a distinct cause, course, and prognosis. In 1883 he published his textbook, *Compendium der Psychiatrie* (Textbook 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 Text Revision (published in 2022).

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 found. 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 the 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 nutritive 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, he 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 procedure removed the illness from her body and provided a near-instantaneous recovery. In reality, the patient was placed in a trancelike state which made her highly suggestible. With other patients, Mesmer would have them sit in a darkened room filled with soothing music, into which he would enter dressed in a colorful robe and pass from person to person touching the afflicted area of their body with his hand or a 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. However, 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**, better known today 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 Prevalence and Treatment of Mental Illness Today" presented the results of the National Comorbidity Study Replication of 2001-2003, which included 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, including the use of psychiatric drugs and deinstitutionalization, managed health care, private psychotherapy, positive psychology and prevention science, multicultural psychology, and prescription rights for psychologists.

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 anti-depressants used to treat depression and anxiety, mood-stabilizing medications to treat bipolar disorder, anti-psychotic drugs to treat schizophrenia, and anti-anxiety drugs to treat generalized anxiety disorder or panic disorder

Frank (2006) found that by 1996, psychotropic drugs were used in 77% of mental health cases and spending on these drugs grew from $2.8 billion in 1987 to about $18 billion in 2001 (Coffey et al., 2000; Mark et al., 2005), representing over a sixfold increase. The largest classes of psychotropic drugs are anti-psychotics and anti-depressants, followed closely by anti-anxiety medications. Frank, Conti, and Goldman (2005) point out, “The expansion of insurance coverage for prescription drugs, the introduction and diffusion of managed behavioral health care techniques, and the conduct of the pharmaceutical industry in promoting their products all have influenced how psychotropic drugs are used and how much is spent on them.” Is it possible then that we are overprescribing these mediations? Davey (2014) provides ten reasons why this may be so, including leading suffers from believing that recovery is in 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 survival; side effects; and a failure to change 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. He 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, possible providers, 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 more flexibility, the higher 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 most 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 cover part of the cost.

- *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.

Regarding the treatment needed for mental illness, managed care programs regulate the pre-approval of treatment via referrals from the PCP, determine which mental health providers can be seen, and oversee 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 responsibility away from 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 must 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 the 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 vital because bias based on 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) state, "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. 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 utilize both therapy and medication, 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, then scramble to treat it. More recently, medicine and science has taken a **prevention** stance, 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 it is the ideal time 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.

You should have learned the following in this section:

- Some of the earliest views of mental illness saw it as the work of evil spirts, demons, gods, or witches who took control of the person, and in the Middle Ages it was seen 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 demonic influence.
- During the Renaissance, humanism was on the rise which emphasized human welfare and the uniqueness of the individual and led to an increase in the number of asylums as places of refuge for the mentally ill.
- The 18th to 19th centuries saw the rise of the moral treatment movement followed by the mental hygiene movement.
- 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 which said that mental disorders were akin to physical disorders and had natural causes.
- Psychiatric or psychotropic drugs used to treat mental illness became popular beginning in the 1950s and led to deinstitutionalization or a shift from inpatient to outpatient care.

Section 1.4 Review Questions

1. How has mental illness been viewed across time?
2. Contrast the moral treatment and mental hygiene movements.
3. Contrast the biological or somatogenic perspective with that of the psychological or psychogenic perspective.
4. Discuss contemporary trends in relation to the use of drugs to treat mental illness, deinstitutionalization, managed health care, multicultural psychology, prescription rights for psychologists, and prevention science.

1.5. Research Methods in Psychopathology

Section Learning Objectives

- Define the 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. Still, before we proceed, it is prudent to further elaborate on what makes psychology scientific. It is safe to say that most people outside of 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 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 keyword here 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 —** 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 a requirement 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 that 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 essential to know that statistics have 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 mean or average, median, and mode. Along with standard deviation and variance, these are ways to describe our data. Second, there are **inferential statistics** that 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 interpret our results accurately 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 broader scientific community.	Once we have decided on whether our 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 occurs 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* – To know about the world around us, we have to be able to see it firsthand. When a mental disorder afflicts an individual, we can see it through their overt behavior. 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 leave major decisions to trusted companions. In these examples and numerous others, 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 must 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 do not 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? In terms of a reduction of symptoms, maybe not, but it could cost less money than the older drug and 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 disorder symptoms before any treatment was implemented, and then again once the treatment has run 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 happens, and the experimenter does not taint the data. 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 time. The advantage of this method over the naturalistic method is that the experimenter can use sophisticated equipment to record the session and examine it later. 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; we will talk about it more throughout this book.

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 founder of psychoanalysis, Sigmund Freud, took to develop his theories. The advantage of this method is that you arrive at a detailed description of the investigated behavior, but the disadvantage is that the findings may be unrepresentative of the larger population, and thus, lacking **generalizability**. Again, bear in mind that you are studying one person or a tiny group. Can you possibly make conclusions about all people from just one person, or even five or ten? The other issue is that the case study is subject to researcher bias in terms of what is included in the final narrative 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 analyze 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 questions used to 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 they are 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*. It can range from -1.00, a perfect inverse relationship in which one variable goes up as the other goes down, to 0 indicating 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, including variables that do not have any relationship with one another. 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 form 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 manipulated variable is called the **independent variable (IV)**, and the one that is measured is called the **dependent variable (DV)**. In the example under Experimentation in Section 1.5.1, the treatment for bipolar disorder was the IV, while the actual intensity or number of symptoms serve as the DV. A

common feature of experiments is 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 and make a 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 initial hypothesis made before starting the experiment.

In a drug study, to ensure the participants' expectations do not affect the final results by giving the researcher what he/she is looking for (in our example, symptoms improve whether the participant is receiving treatment or not), we might 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, so we have to focus on one individual using a **single-subject experimental design**. This differs from a case study in the sheer number of strategies available 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, 2017) also established the utility of social stories as a social learning tool for children with autism spectrum disorder (ASD) using an ABAB design. During the baseline phase (A), the four student participants were observed, and data recorded on an observation form. During the treatment phase (B), they listened to the social story and data was recorded in the same manner. Upon completion of the first B, the students 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 the outcome. All students showed improvement during the treatment phases in terms of the number of positive peer interactions, but the number of interactions reduced in the absence of social stories. 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 strengths and limitations. As such, for the psychologist to provide the most precise 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.**

You should have learned the following in this section:

- The scientific method is a systematic method for gathering knowledge about the world around us.
- A systematic explanation of a phenomenon is a theory and our specific, testable prediction is the hypothesis**.**
- Replication is when we repeat the study to confirm its results.
- Psychology's five main research designs are observation, case studies, surveys, correlation, and experimentation.
- No single research method alone is perfect - all have strengths and limitations.

Section 1.5 Review Questions

1. What is the scientific method and what steps make it up?
2. Differentiate theory and hypothesis.
3. What are the three cardinal features of science and how do they relate to the study of mental disorders?
4. What are the five main research designs used by psychologists? Define each and then state its strengths and limitations.
5. What is the advantage of 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	No

		settings.	
Psychiatrist	M.D.	Has specialized training in the diagnosis and treatment of mental disorders	Yes
Psychiatric Nurse Practitioner	M.S.N.	Has specialized treatment in the care and treatment of psychiatric patients	Yes
Occupational Therapist	B.S.	Trained to assist individuals suffering from physical or psychological handicaps and help them acquire needed resources	No
Pastoral Counselor	Clergy	Trained 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:

https://www.mhanational.org/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 sound conclusions are

important, but you must tell others what you have done too. This is accomplished by joining professional societies and submitting articles to peer-reviewed journals. Below are some of the organizations and journals relevant to applied behavior analysis.

1.6.2.1. Professional Societies

- **Society of Clinical Psychology – Division 12 of the American Psychological Association**
  - Website – https://div12.org/
  - 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 sections such as clinical emergencies and crises, clinical psychology of women, assessment psychology, and clinical geropsychology
- **Society of Clinical Child and Adolescent Psychology – Division 53 of the American Psychological Association**
  - Website – https://www.clinicalchildpsychology.org/
  - Mission Statement – "Our mission is to serve children, adolescents and families with the best possible clinical care based on psychological science. SCCAP strives to integrate scientific and professional aspects of

clinical child and adolescent psychology, in that it promotes scientific inquiry, training, and clinical practice related to serving children and their families."

- 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 seeks to "recognize and promote advanced competence within Professional Psychology," "provide a professional community that encourages communication between and among Members and Fellows of the Academy," "provide opportunities for advanced education in Professional Psychology," and "expand awareness and availability of AACP Members and Fellows to the public through promotion and education."
  - 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 – "To provide and encourage education programs to further, in every ethical way, the knowledge, understanding, and application of hypnosis in health care; to encourage research and scientific publication in the field of hypnosis; to promote the further recognition and acceptance of hypnosis as an important tool in clinical health care and focus for scientific research; to cooperate with other professional societies that share mutual goals, ethics and interests; and to provide a professional community for those clinicians and researchers who use hypnosis in their work."
  - 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 and related mental health fields by publishing scholarly articles, primarily involving narrative and systematic reviews as well as meta-analyses related to assessment, intervention, and service delivery."
- **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 socio-demographic 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."

You should have learned the following in this section:

- Mental health professionals take on many different forms with different degree requirements, training, and the ability to prescribe mediations.
- Telling others what we have done is achieved by joining professional societies and submitting articles to peer-reviewed journals.

Section 1.6 Review Questions

1. Provide a general overview of the types of mental professionals and the degree, training, and ability to prescribe medications that they have.
2. Briefly outline professional societies and journals related to clinical psychology and related disciplines.

Module Recap

In Module 1, we undertook a relatively 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. Then 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. Various mental health professionals use these designs, 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.

Part I. Setting the Stage

Module 2:
Models of Abnormal Psychology

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 single model can account for all aspects of abnormality. Hence, we advocate for a multi-dimensional and not a uni-dimensional model.

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

To effectively treat a mental disorder, we must 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 a person's life, but as they arise, the factors become part of the individual. In time, the cause of the person's psychopathology is due to all these individual factors.

2.1.2. Multi-Dimensional

So, 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 explain the etiology of mental disorders fully.

Before introducing the current main models, it is crucial 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. For example, to be diagnosed with separation anxiety disorder, you must present with three of eight symptoms for criteria A whereas for a major depressive episode as part of Bipolar II disorder, you have to display five (or more) symptoms for criteria A. There will be some variability in terms of what symptoms are displayed, 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 this information to better understand the person's condition and potential causes. Models aid us with doing all of this. Still, we must remember that the model is a starting point for the researcher, and due to this, it determines what causes might be investigated at the exclusion of other causes. Often, proponents of a given model find themselves in disagreement with proponents of other models. All forget that there is no individual model that completely explains human behavior, or in this case, abnormal behavior, and so each model contributes in its own way. Here 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.
- **Sociocultural** – includes factors such as one's gender, religious orientation, race, ethnicity, and culture.

You should have learned the following in this section:

- The uni-dimensional model proposes a single factor as the cause of psychopathology while the multi-dimensional model integrates multiple causes of psychopathology and affirms that each cause comes to affect other causes over time.
- There is no individual model that completely explains human behavior and so each model contributes in its own way.

Section 2.1 Review Questions

1. What is the problem with a uni-dimensional model of psychopathology?
2. Discuss the concept of a model and identify those important to understanding psychopathology.

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.

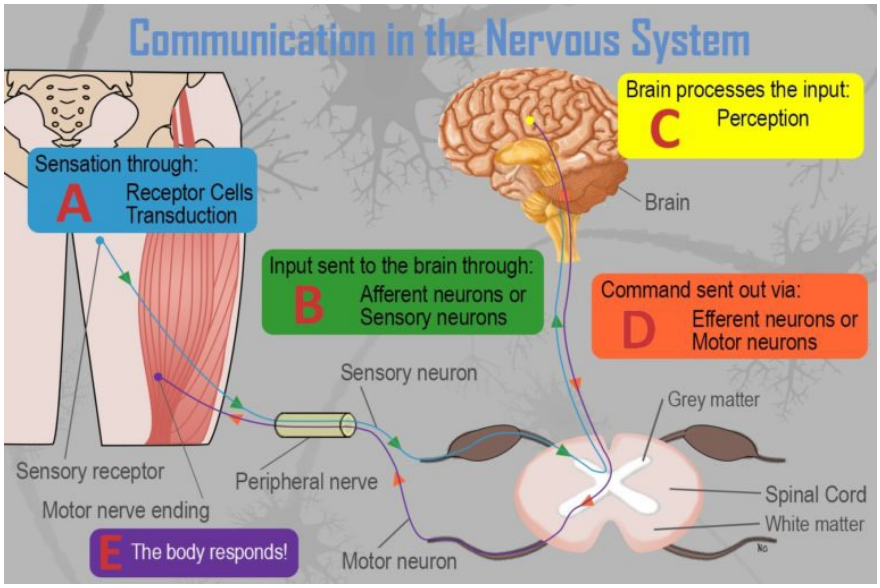
Proponents of the biological model view mental illness as being a result of a malfunction in the body to include issues with brain anatomy or chemistry. As such, we will need to establish a foundation for how communication in the nervous system occurs, what the parts of the nervous system are, what a 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 truly 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

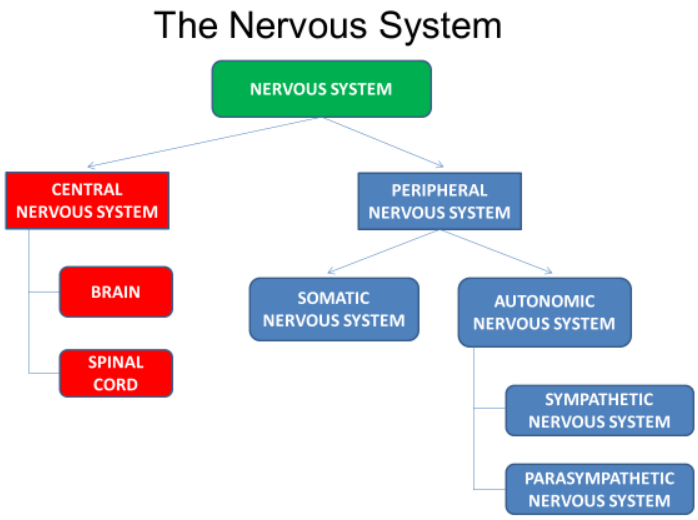


Please note that we will not cover this process in full, but just the parts relevant to our topic of psychopathology.

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 the functioning of blood vessels, glands, and internal organs such as the bladder, stomach, and heart. It consists of sympathetic and parasympathetic nervous systems. The **sympathetic nervous system** is involved when a person is intensely aroused. It provides the strength to fight back or to flee (fight-or-flight 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



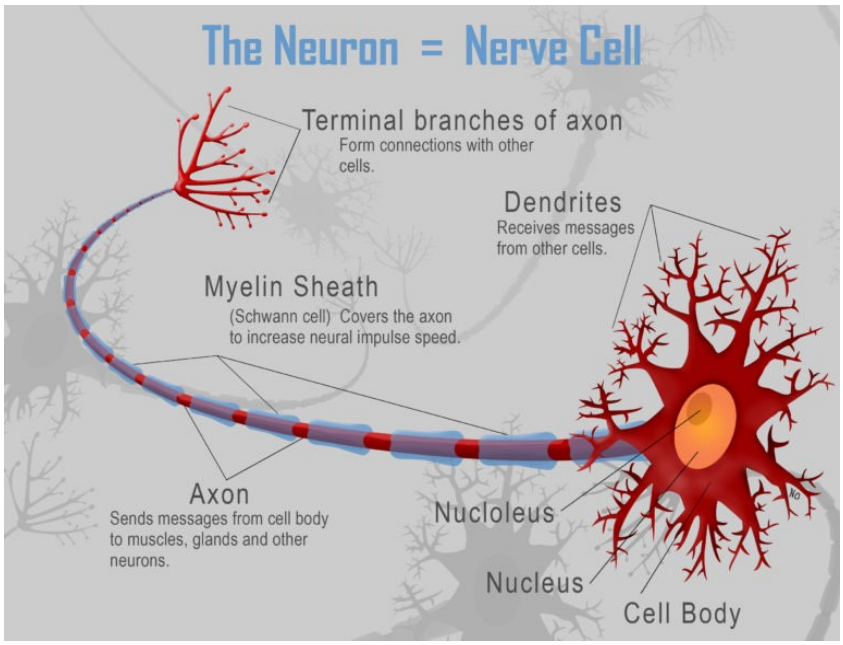
2.2.1.3. The neuron. The fundamental unit of the nervous system is the neuron, or nerve cell (See Figure 2.3). It has several structures in common with all cells in the body. The **nucleus** is the control center of the neuron, and the **soma** is the cell body. In terms of distinctive structures, these focus on the ability of a neuron to send and receive information. The **axon** sends signals/information to neighboring neurons while the **dendrites**, which resemble little trees, receive information from neighboring neurons. Note the plural form of dendrite and the singular form of axon; there are many 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 passes 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

The neural impulse proceeds across the following steps:

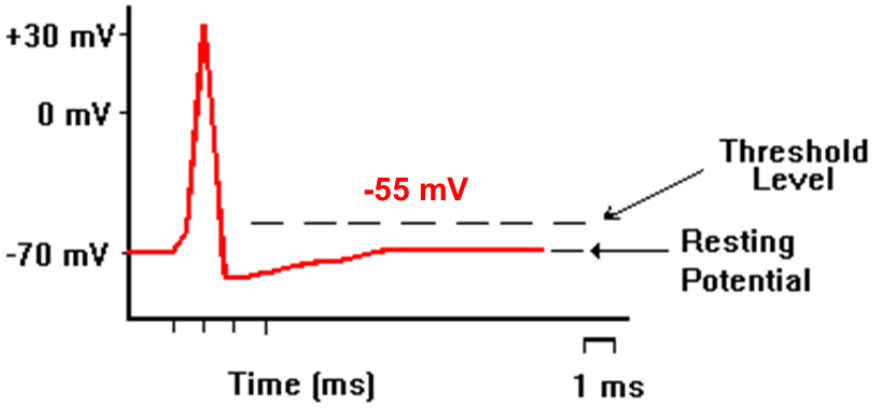
- Step 1 – Neurons waiting to fire are said to be in **resting potential** and **polarized**, or having a negative charge inside the neuron and a positive charge outside.
- Step 2 – If adequately stimulated, the neuron experiences an **action potential** and becomes **depolarized**. When this occurs, voltage-gated ion channels open, allowing positively charged sodium ions (Na+) to enter. This shifts the polarity to positive on the inside and negative outside. Note that **ions** are charged particles found both inside and outside the neuron.
- Step 3 – Once the action potential passes from one segment of the axon to the next, the previous segment begins to **repolarize**. This occurs because the Na channels close and potassium (K) channels open. K+ has a positive charge, 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 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 this process is cyclical. We started at resting potential in Step 1 and end at resting potential in Step 6.

Part 2. The Action Potential

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

Figure 2.4. The Action Potential



- Recall that a neuron is usually at resting potential and polarized. The charge inside is -70mV at rest.
- If it receives sufficient stimulation, causing the polarity inside the neuron to rise from -70 mV to -55mV (**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 fire at all. 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 the absolute refractory period.
- The neuron next moves into a relative refractory period, meaning it can fire but needs higher 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

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 destroy excess neurotransmitters in the synaptic space.

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

- **Dopamine** – controls voluntary movements and is associated with the reward mechanism in the brain
- **Serotonin** – regulates pain, sleep cycle, and digestion; leads to a stable mood, so low levels lead 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** – blocks 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 except smell

- **Hypothalamus** – involved in drives associated with the survival of both the individual and the species. It regulates temperature by triggering sweating or shivering and controls the complex operations of the autonomic nervous system
- **Amygdala** – responsible for evaluating sensory information and quickly determining its emotional importance
- **Hippocampus** – our "gateway" to memory. Allows us to form spatial memories so that we can accurately navigate through our environment and helps us to form new memories 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 for receiving and processing 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 some disorders involve specific areas of the brain. For instance, Parkinson's disease is a brain disorder that 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. It exists 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, one 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." Likewise, twin and family studies have shown that people with first-degree relatives suffering from OCD are at higher risk 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, the nervous system and the endocrine system. The main difference between these two systems is 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 manage 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, and 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 energy levels by controlling metabolism and the basal metabolic rate (BMR). It 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 several problems, including increased weight gain, interference with learning and memory, reduced bone density, high cholesterol, and an increased risk of depression. Similarly, the 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. Bacterial and viral infections. Infections can cause brain damage and lead to the development of mental illness or exacerbate existing symptoms. For instance, evidence suggests that contracting strep throat, "an infection in the throat and tonsils caused by bacteria called group A *Streptococcus"* (for more on strep throat, please visit https://www.cdc.gov/groupastrep/diseases-public/strep-throat.html), 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, caused by viral infections (for more on influenza, please see the following: https://www.mayoclinic.org/diseases-conditions/flu/symptoms-causes/syc-20351719), 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. *Antidepressants* are used to treat depression, but also anxiety, insomnia, and 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 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 include loss of appetite, headache, motor 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; 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 undergo ECT to treat conditions such as 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" (https://www.mhanational.org/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 trephination 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. However, 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 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 fundamentally biological. More on that over the next two sections.

You should have learned the following in this section:

- 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.
- Neurotransmitter imbalances and problems with brain structures/areas can result in mental disorders.
- Many disorders have genetic roots, are a result of hormonal imbalances, or caused by viral infections such as strep.
- Treatments related to the biological model include drugs, ECT, and psychosurgery.

Section 2.2 Review Questions

1. Briefly outline how communication in the nervous system occurs.
2. What happens at the synapse during neural transmission? Why is this important to a discussion of psychopathology?
3. How is the anatomy of the brain important to a discussion of psychopathology?
4. What is the effect of genes, hormones, and viruses on the development of mental disorders?
5. What treatments are available to clinicians courtesy of the biological model of psychopathology?
6. What are some issues facing the biological model?

2.3. Psychological Perspectives

Section Learning Objectives

- Describe the 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 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 who were considered millionaires at the time. Bertha, known in published case studies as Anna O., was expected to complete the formal education typical of upper-middle-class girls, 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, including symptoms such 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 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, Bertha was diagnosed with a tumor, and in 1936, she was summoned by the Gestapo 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 major schools of thought did; rather, it emerged from medicine and psychiatry to address psychopathology and examine 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.

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 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 the 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 and experience 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 five distinct stages in which the libido focuses 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**, 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 that 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. 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 falling in love with his mother while fearing that his father will find out and castrate him. Meanwhile, girls fall in love with the father and fear that their mother will find out, called the *Electra complex*. A fixation at this stage may result in low self-esteem, feelings of worthlessness, and shyness.
4. **Latency Stage** – From 6-12 years of age, children lose interest in sexual behavior, so 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 by 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 the repressed memory can cause problems later in life.
- **Reaction formation** – When an impulse is repressed and then expressed by its opposite. For example, you are angry with your boss but cannot lash out at him, so you are super friendly instead. Another example is having lustful thoughts about a coworker than you cannot express because you are married, 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 you lash out at your boss, you 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 that all we can do is deny how bad it is. An example is denying a diagnosis of lung cancer given by your doctor.
- **Identification** – 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. If your significant other is nagging you, you might regress by putting your hands over your ears and saying, "La la la la la la la…"
- **Rationalization** – When we offer well-thought-out reasons for why we did what we did, but 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 the 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—free association, transference, and dream analysis—as part of **psychoanalysis**, or psychoanalytic therapy, to understand the personalities of his patients and expose repressed material. 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 **resistance** revealed where issues persisted.

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

Finally, Freud used **dream analysis** to understand a person's innermost wishes. The content of dreams includes 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 made a lasting impact on the field of psychology but also has been criticized heavily. First, Freud made most of his observations in an unsystematic, uncontrolled way, and he relied on the case study method. Second, the participants in his studies were not representative of the broader population. Despite Freud's generalization, his theory was based on only a few patients. Third, he relied solely on the reports of his patients and sought no observer reports. Fourth, it is difficult to empirically study psychodynamic principles since most operate unconsciously. This begs the question of how we can 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, Sigmund Freud developed useful therapeutic tools for clinicians and raised awareness about the role the unconscious plays in both normal and abnormal behavior.

2.3.2. The Behavioral Model

2.3.2.1. What is learning? The behavioral model concerns the cognitive process of **learning**, which is any relatively permanent change in behavior due to experience and practice. Learning 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 when two separate events become connected. There are 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). Watson believed that the subject matter of psychology was to be observable behavior, and to that end, 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 classical/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 some stimuli automatically elicited responses (such as salivating to meat powder) and other stimuli 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, your leg extends out automatically. Another example is how a baby will root for a food source if the mother's breast is placed near their mouth. And if a nipple is placed in their mouth, they will also automatically suck via the sucking

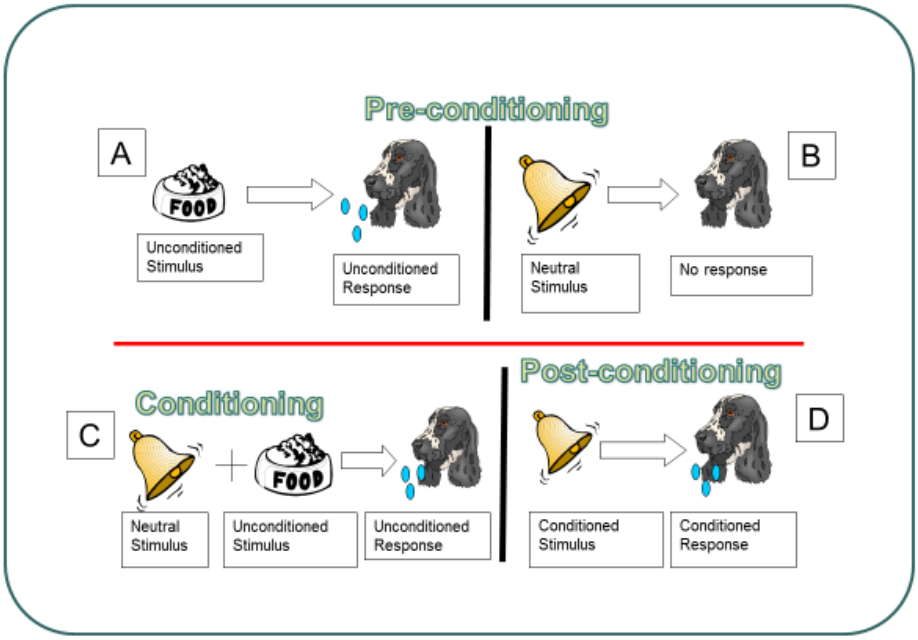
reflex. Humans have several of these reflexes, though not as many as other animals due to our more complicated nervous system.

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 happens in three phases: preconditioning, conditioning, and postconditioning. See Figure 2.5 for an overview of Pavlov's classic experiment.

Preconditioning. Notice that preconditioning has both an A and a B panel. 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 response does not need to be learned and shows the relationship between an unconditioned stimulus (UCS) yielding an unconditioned response (UCR). Unconditioned means unlearned. In Panel B, we see that a neutral stimulus (NS) produces no response. Dogs do not enter the world knowing to respond to the ringing of a bell (which it hears).

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

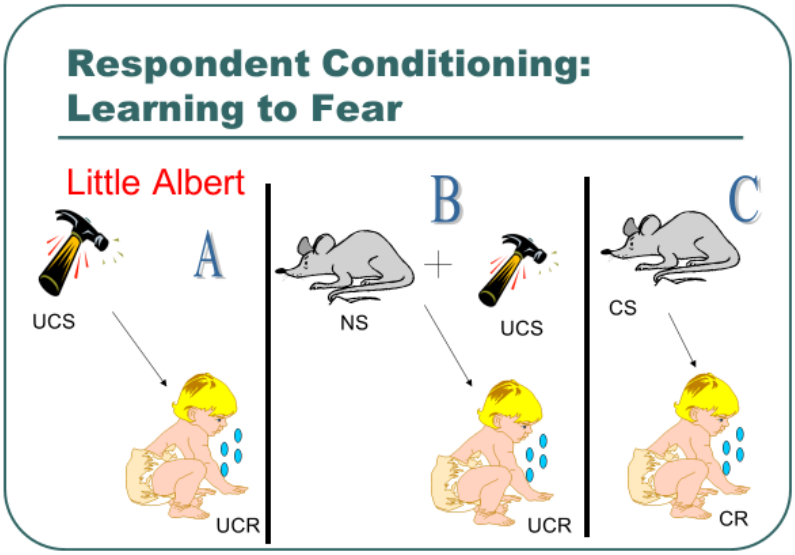
Figure 2.5. Pavlov's Classic Experiment



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.

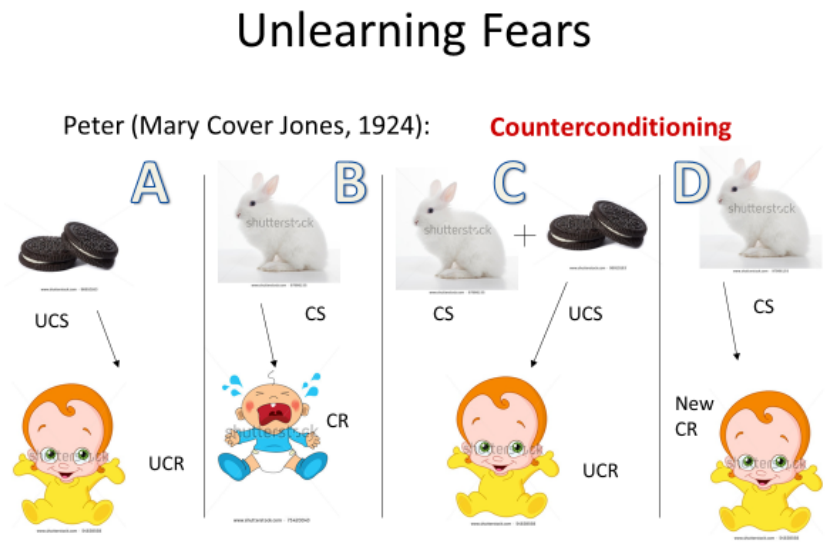
Watson and Rayner (1920) conducted one of the most famous studies in psychology. Essentially, they wanted to explore "the possibility of conditioning various types of emotional response(s)." The researchers ran a series of trials in which they exposed a 9-month-old child, known as Little Albert, to a white rat. Little Albert made no response outside of curiosity (NS–NR not shown). Panel A of Figure 2.6 shows 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 2.6. 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 2.7) 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 not to 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 a bit closer each time until, eventually, the child did not respond with distress to the rabbit (Panel D).

Figure 2.7. Unlearning Fears



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

Another respondent conditioning way to unlearn a fear is called **flooding** or exposing the person to the maximum level of stimulus and as nothing aversive occurs, the link between CS and UCS producing the CR of fear should break, leaving the person unafraid. That is the idea, at least. So, if you were afraid of clowns, you would be thrown into a room full of clowns. Hmm….

Finally, respondent conditioning has several properties:

- **Respondent Generalization** – When many similar CSs or a broad range of CSs elicit the same CR. An example is the sound of a whistle eliciting salivation much the same as a ringing bell, both detected via audition.
- **Respondent Discrimination** – When a single CS or a narrow range of CSs elicits a CR, i.e., teaching the dog to respond to a specific bell and ignore the whistle. The whistle 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. If food appears, the salivation response will be re-established.

2.3.2.3. Operant conditioning. Influential on the development of Skinner's operant conditioning, Thorndike (1905) proposed the **law of effect** 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 a puzzle box. Cats were food deprived the night before the experimental procedure was to occur. The next morning, researchers placed a hungry cat in the puzzle box and set a small amount of food outside the box, just close enough to be smelled. The cat could escape the box and reach the food by manipulating a series of levers. Once free, the cat was allowed to eat some food before being promptly returned to the box. With each subsequent escape and re-insertion into the box, the cat became faster at correctly manipulating the levers. This scenario demonstrates **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 led 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 or say) 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 strengthened and more likely to occur in the future.
- **Punishment** – Due to the consequence, a behavior/response is weakened and less likely to occur in the future.

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 2.8. 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 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 receive 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 seem counterintuitive, even though we experience NR all the time. NR 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 stimulus presents itself. For instance, what do you do if you have a headache? If you take Tylenol and the pain goes away, you will likely 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 crucial. Some are naturally occurring, while others 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 eight 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 grew 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 that did not have to be learned, and 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, ridicule, 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 the desired behavior is called the **reinforcement schedule.** Reinforcement can either occur *continuously* meaning every time the desired behavior is made the subject will receive some reinforcer, or *intermittently/partially* meaning reinforcement does not occur with every behavior. Our focus will be on partial/intermittent reinforcement.

Figure 2.9. Key Components of Reinforcement Schedules

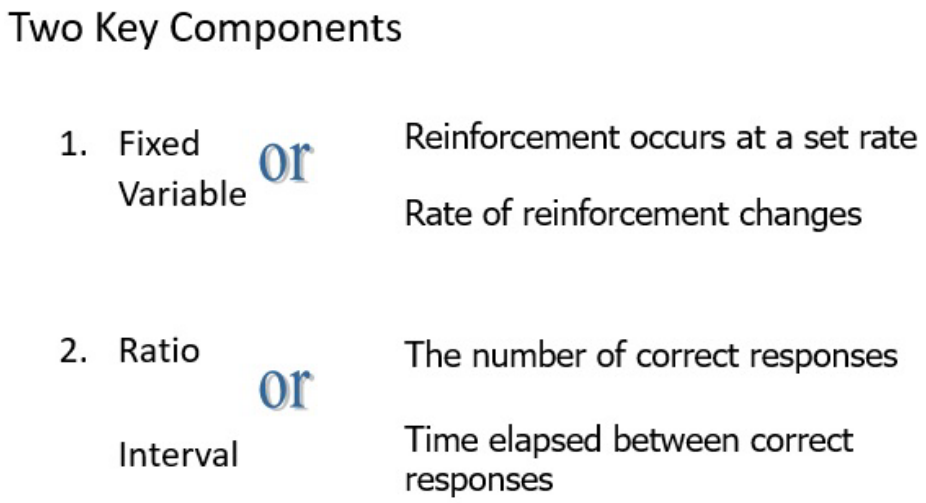


Figure 2.9 shows that that are two main components that make up a reinforcement schedule – when you will reinforce and what is being reinforced. In the case of when, it will be either fixed or at a set rate, or variable and at a rate that changes. In terms of what is being reinforced, we will either 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 an 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 approach is useful if the student is 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. 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 happen as soon as the anticipated reinforcer is removed? The answer is yes and no, depending on whether we are talking about continuous or partial reinforcement. With which type of schedule would you expect a person to stop responding to immediately if reinforcement is not there? Continuous or partial?

The answer is continuous. If a person is used to receiving reinforcement every time they perform a particular behavior, 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 endurance, we say that intermittent or partial reinforcement shows *resistance to extinction*, meaning the behavior does weaken, but gradually.

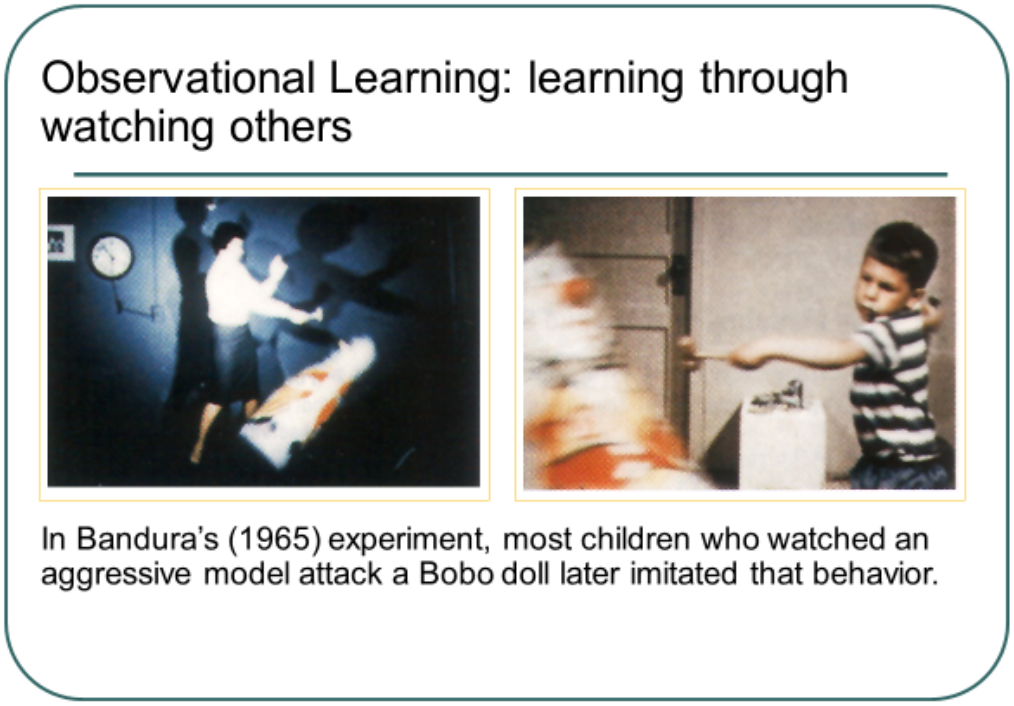
As you might expect, if reinforcement occurs after extinction has started, the behavior will re-emerge. Consider your parents for a minute. To stop some undesirable behavior you made in the past, they likely took away some privilege. I bet the bad behavior ended too. But did you ever go to your grandparent's house and grandma or grandpa—or worse, BOTH—took pity on you and let you play your video games (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?

Additionally, 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 trained to push a lever to receive a food pellet. If we stop providing the food pellets, in time, the rat will stop pushing the lever. If the rat pushes the lever again sometime in the future and food is delivered, the behavior spontaneously recovers. Hence, 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 stressors life presents, then you also might do the same. The critical part is what happens to the person modeling the behavior. 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 pivotal research on observational learning, and you likely already know all about it. Check out Figure 2.10 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, which provided a model. Next, the children are placed in a room with several toys in it. The room contains a highly prized toy, but they are told they cannot play with it. All other toys are allowed, including a Bobo doll. Children who watched the aggressive model behaved aggressively with the Bobo doll while those who saw the gentle model, played nice. Both groups were frustrated when deprived of the coveted toy.

Figure 2.10. Bandura's Classic Experiment



According to Bandura, all behaviors are learned by observing others, and we model our actions after theirs, so undesirable behaviors can be altered or relearned in the same way. **Modeling** techniques 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.

However, 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 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 psychopathology, the behavioral perspective is useful because explains maladaptive behavior in terms of learning gone awry. The good thing is that what is learned can be unlearned or relearned through **behavior modification**, the process of changing behavior. To begin, an applied behavior analyst identifies a target behavior, or behavior to be changed, defines it, works with the client to develop goals, conducts a functional assessment to understand what the undesirable behavior is, what causes it, and what maintains it. With this knowledge, a plan is developed and consists of numerous strategies to act on one or all 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 and 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 led to a cognitive-behavioral perspective that 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, many 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 the individual pairings of a feared object or situation and relaxation. 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 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 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 who 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 tends to spit when lecturing. The person in the back could also be shy and prefer sitting there so that s/he does not need to chat with others as much. Or, they are easily distracted and sits in the back so that all stimuli are in front of him/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 social psychology topic **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 external causes (situational). 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 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 adverse 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 improving now.
- Dichotomous thinking – Viewing people or events in all-or-nothing terms.

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 identify 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/About-Mental-Illness/Treatments/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 four 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." Figure out what is the worst thing that could happen and what alternative outcomes are possible.
2. Track the accuracy of the thought. If you believe focusing on a problem generates a solution, then write down each time you ruminate and 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. Try figuring out if you genuinely do not have time to go to the gym by recording what you do each day and then look at open times of the day. Add them up and see if making some minor, or major, adjustments to your schedule will free an hour to get in some 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, including mindfulness meditation and self-compassion. For more on these, visit:

https://www.psychologytoday.com/blog/in-practice/201301/cognitive-restructuring

The second major CBT strategy is called **cognitive coping skills training**. This strategy teaches social skills, communication, assertiveness through direct instruction, role playing, and modeling. For social skills training, identify the appropriate social behavior such as making eye contact, saying no to a request, or starting up a conversation with a stranger and determine whether the client is inhibited from making this behavior due to anxiety. For communication, decide if the problem is related to speaking, listening, or both and then develop a plan for use in various interpersonal situations. Finally, assertiveness training aids the client in protecting their rights and obtaining what they want from others. Those who are not assertive are often overly passive and never get what they want or are unreasonably aggressive and only get what they want. Treatment starts with determining situations in which assertiveness is lacking and developing 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** help reduce a client's worry and anxiety. Life involves a degree of uncertainty, and at times we must accept this. Techniques might include weighing the pros and cons of fighting uncertainty or change. The disadvantages should outweigh the advantages 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 apply this in more difficult areas. Finally, does uncertainty always lead to a negative end? We may think so, but a 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 apparent 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 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 know anything about controversial matters? The person's true intentions or thoughts and feelings are not readily available to us, or are covert, and do not make for useful 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 becomes distorted, now seen as having worth only when these significant others approve, leading to a disharmonious state and psychopathology. Individuals in this situation are unsure of 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 re-create themselves continually 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. Rogers did try to investigate his propositions scientifically, but 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 destiny and can make our own choices.

You should have learned the following in this section:

- According to Freud, consciousness had three levels (consciousness, preconscious, and the unconscious), personality had three parts (the id, ego, and superego), personality developed over five stages (oral, anal, phallic, latency, and genital), there are ten defense mechanisms to protect the ego such as repression and sublimation, and finally three assessment techniques (free association, transference, and dream analysis) could be used to understand the personalities of his patients and expose repressed material.
- The behavioral model concerns the cognitive process of learning, which is any relatively permanent change in behavior due to experience and practice and has two main forms – associative learning to include classical and operant conditioning and observational learning.
- 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.
- 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.
- Observational learning is learning by watching others and modeling techniques change behavior by having subjects observe a model in a situation that usually causes them some anxiety.
- The cognitive model focuses on schemas, cognitive errors, attributions, and maladaptive cognitions and offers strategies such as CBT, cognitive restructuring, cognitive coping skills training, and acceptance.
- The humanistic perspective focuses on positive regard, conditions of worth, and the fully functioning person while the existential perspective stresses the need for people to re-create themselves continually 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.

Section 2.3 Review Questions

1. What are the three parts of personality according to Freud?
2. What are the five psychosexual stages according to Freud?
3. List and define the ten defense mechanisms proposed by Freud.
4. What are the three assessment techniques used by Freud?
5. What is learning and what forms does it take?
6. Describe respondent conditioning.
7. Describe operant conditioning.
8. Describe observational learning and modeling.
9. How does the cognitive model approach psychopathology?
10. How does the humanistic perspective approach psychopathology?
11. How does the existential perspective approach psychopathology?

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 dependency 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. Gender is not a 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 higher 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: "While mental illnesses affect both men and women, the prevalence of mental illnesses in men is often lower than women. Men with mental illnesses are also less likely to have received mental health treatment than women in the past year. However, men are more likely to die by suicide than women, according to the Centers for Disease Control and Prevention. Recognizing the signs that you or someone you love may have a mental disorder is the first step toward getting treatment. The earlier that treatment begins, the more effective it can be."

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

In relation to women: "Some disorders are more common in women such as depression and anxiety. There are also certain types of disorders that are unique to women. For example, 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 the rates at which men and women experience these illnesses. But women may experience these illnesses differently – certain symptoms may be more common in women than in men, and the course of the illness can be affected by the sex of the individual. Researchers are only now beginning to tease apart the various biological and psychosocial factors that may impact the mental health of both women and men."

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 the context of 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 significantly 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-TR (Diagnostic and Statistical Manual of Mental Disorders, 5th edition, Text-Revision). Important here is that specific culture- and gender-related diagnostic issues are discussed for each disorder, demonstrating increased awareness of the impact of these factors. Still, the sociocultural model suffers from unclear findings and not allowing for the establishment of causal relationships, 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.

You should have learned the following in this section:

- The sociocultural model asserts that race, ethnicity, gender, religious orientation, socioeconomic status, sexual orientation all play a role in the development and treatment of mental illness.

Section 2.4 Review Questions

1. How do socioeconomic, gender, environmental, and multicultural factors affect mental illness and its treatment?
2. How effective is the sociocultural model at explaining psychopathology and its treatment?

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 identified as potential causes of mental illness and three treatment options were given. In terms of psychological perspectives, Freud's psychodynamic theory; the learning-related research 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.

Part I. Setting the Stage

Module 3:

Clinical Assessment, Diagnosis, and Treatment

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-TR and ICD-11. Finally, we discuss the reasons why people may seek treatment and what to expect when doing so.

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 diagnose 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?

For a mental health professional to be able to effectively help treat a client and know that the treatment selected worked (or is working), they 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 the person's problem and the presenting symptoms. This collection of information involves learning about the client's skills, abilities, personality characteristics, cognitive and emotional functioning, the social context in terms of environmental stressors that are faced, and cultural factors particular to them such as their language or ethnicity. Clinical assessment is not just conducted at the beginning of the process of seeking help but 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 decide 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 desired outcome occurs at the end. It should be clear from this discussion that clinical assessment is an *ongoing* process.

3.1.2. Key Concepts in Assessment

The assessment process involves three critical concepts – reliability, validity, and standardization. These three are important to science in general. First, we want the 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. 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, which is called *test-retest reliability*. For example, let's say 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. Therefore, it seems to measure personality, or we have an overall feeling that it measures what we expect it to measure.

Predictive validity is when a tool accurately predicts what will happen in the future. 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 later. 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 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 form of validity is called **cross-sectional validity.** We also need our raters 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 can 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. The latter includes 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 assess the client's personality, social skills, cognitive abilities, emotions, behavioral responses, or interests. They can be administered either individually or to groups in paper or oral fashion. **Projective tests** consist of simple ambiguous stimuli that can elicit an unlimited number of responses. They include the Rorschach 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 assesses 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 can be completed electronically or by hand. 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 used to diagnose cognitive impairments caused by brain damage due to tumors, infections, or head injuries; or changes in

brain activity. *Positron Emission Tomography or PET* is used to study the brain's chemistry. It begins by injecting the patient with a radionuclide that 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* provides 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 is used to diagnose brain damage caused 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 like mental disorders. Ruling out such conditions 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 is the measurement of a target behavior. The **target behavior** is whatever behavior we want to change, and it can be in *excess* and needing to be reduced, or in a *deficit* state and needing to be increased. During the 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. 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 determines 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 assesses 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?

You should have learned the following in this section:

- Clinical assessment is the collecting of information and drawing conclusions through the use of observation, psychological tests, neurological tests, and interviews.
- Reliability refers to consistency in measurement and can take the form of interrater and test-retest reliability.
- Validity is when we ensure the test measures what it says it measures and takes the forms of concurrent or descriptive, face, and predictive validity.
- Standardization is all the clearly laid out rules, norms, and/or procedures to ensure the experience each participant has is the same.
- Patients are assessed through observation, psychological tests, neurological tests, and the clinical interview, all with their own strengths and limitations.

Section 3.1 Review Questions

1. What does it mean that clinical assessment is an ongoing process?
2. Define and exemplify reliability, validity, and standardization.
3. For each assessment method, define it and then state its strengths and limitations.

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-TR.
- Describe the ICD-11.
- Clarify why the DSM-5-TR and ICD-11 need to be harmonized.

3.2.1. Clinical Diagnosis and Classification Systems

Before starting 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 outlined in an established classification system such as the DSM-5-TR or ICD-11 (both will be described shortly). Any diagnosis should have *clinical utility*, meaning it aids the mental health professional in determining prognosis, the treatment plan, and possible outcomes of treatment (APA, 2022). 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, 2022). Likewise, a patient may not meet the full criteria for a diagnosis but demonstrate a clear need for treatment or care, nonetheless. As stated in the DSM, "The fact that some individuals do not show all symptoms indicative of a diagnosis should not be used to justify limiting their access to appropriate care" (APA, 2022).

Symptoms that cluster together regularly 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 into distinct categories for which there are clear descriptions and criteria for making a diagnosis. Distinct is the keyword here. People suffering from delusions, hallucinations, disorganized thinking (speech), grossly disorganized or abnormal motor behavior, and/or negative symptoms are different from people presenting with a primary clinical deficit in cognitive functioning that is not developmental but acquired (i.e., they have shown a decline in cognitive functioning over time). The former suffers from a schizophrenia spectrum disorder while the latter suffers from a neurocognitive disorder (NCD). The latter can be further distinguished from neurodevelopmental disorders which manifest early in development and involve developmental deficits that cause impairments in social, personal, academic, or occupational functioning (APA, 2022). These three disorder groups or categories can be clearly distinguished from one another. Classification systems also permit the gathering of statistics to determine 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* (DSM) which is a "medical classification of disorders

and as such serves as a historically determined cognitive schema imposed on clinical and scientific information to increase its comprehensibility and utility. The classification of disorders (the way in which disorders are grouped) provides a high-level organization for the manual" (APA, 2022, pg. 11). The DSM is currently in its 5th edition Text-Revision (DSM-5-TR) and is produced by the American Psychiatric Association (APA, 2022). Alternatively, the World Health Organization (WHO) publishes the *International Statistical Classification of Diseases and Related Health Problems (ICD)* currently in its 11th edition. 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). In March 2022, a Text-Revision was published for the DSM-5, making it the DSM-5-TR.

The history of the DSM goes back to 1952 when the American Psychiatric Association published the first edition of the DSM which was "…the first official manual of mental disorders to contain a glossary of descriptions of the diagnostic categories" (APA, 2022, p. 5). The DSM evolved through four major editions after World War II into a diagnostic classification system to be used by psychiatrists and physicians, but also other mental health professionals. The Herculean task of revising the DSM 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 collaboration 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, 2022, pg. 6).

After the naming of a DSM-5 Task Force Chair and Vice-Chair in 2006, task force members were selected and approved by 2007, and workgroup members were approved in 2008. An intensive 6-year 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 website for public comment, presenting preliminary findings at professional meetings, performing field trials, and revisiting criteria and text" was undertaken (APA, 2022, pg. 7). The process involved physicians, psychologists, social workers, epidemiologists, neuroscientists, nurses, counselors, and statisticians, all who aided in the development and testing of DSM-5 while individuals with mental disorders, families of those with a mental disorder, consumer groups, lawyers, and advocacy groups provided feedback on the mental disorders contained in the book. Additionally, disorders with low clinical utility and weak validity were considered for deletion while "Conditions for Future Study" were placed in Section 3 and "contingent on the amount of empirical evidence generated on the proposed diagnosis, diagnostic reliability or validity, presence of clear clinical need, and potential benefit in advancing research" (APA, 2022, pg. 7).

3.2.2.2. The DSM-5 text revision process. In the spring 2019, APA started work on the Text-Revision for the DSM-5. This involved more than 200 experts who were asked to conduct literature reviews of the past 10 years and to review the text to identify any material that was out-of-date. Experts were divided into 20 disorder review groups, each with its own section editor.

Four cross-cutting review groups to include Culture, Sex and Gender, Suicide, and Forensic, reviewed each chapter and focused on material involving their specific expertise. The text was also reviewed by an Ethnoracial Equity and Inclusion work group whose task was to "ensure appropriate attention to risk factors such as racism and discrimination and the use of nonstigmatizing language" (APA, 2022, pg. 11).

As such, the DSM-5-TR "is committed to the use of language that challenges the view that races are discrete and natural entities" (APA, 2022, pg. 18). Some of changes include:

- Use of *racialized* instead of *racial* to indicate the socially constructed nature of race
- *Ethnoracial* is used to denote U.S. Census categories such as Hispanic, African American, or White
- *Latinx* is used in place of *Latino or Latina* to promote gender-inclusive terminology
- The term *Caucasian* is omitted since it is "based on obsolete and erroneous views about the geographic origin of a prototypical pan-European ethnicity" (pg. 18)
- To avoid perpetuating social hierarchies, the terms *minority* and *non-White* are avoided since they describe social groups in relation to a racialized "majority"
- The terms *cultural contexts* and *cultural backgrounds* are preferred to *culture* which is only used to refer to a "heterogeneity of cultural views and practices within societies" (pg. 18)
- The inclusion of data on specific ethnoracial groups only when "existing research documented reliable estimates based on representative samples."

This led to limited inclusion of data on Native Americans since data from nonrepresentative samples may be misleading.

- The use of *gender differences* or "women and men" or "boys and girls" since much of the information on the expressions of mental disorders in women and men is based on self-identified gender.
- Inclusion of a new section for each diagnosis providing information about suicidal thoughts or behavior associated with that diagnosis.

3.2.2.3. Elements of a diagnosis. The DSM-5-TR states that the following make up the key elements of a diagnosis (APA, 2022):

- Diagnostic Criteria and Descriptors – Diagnostic criteria are the guidelines for making a diagnosis and should be informed by clinical judgment. 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, 2022, pg. 22). For example, non-rapid eye movement (NREM) sleep arousal disorders can have either a sleepwalking or sleep terror type. Enuresis is nocturnal-only, diurnal-only, or both. *Specifiers* are not mutually exclusive or jointly exhaustive and so more than one

specifier can be given. For instance, binge eating disorder has remission and severity specifiers. 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. As the DSM-5-TR says, "Specifiers and subtypes provide an opportunity to define a more homogeneous subgrouping of individuals with the disorder who share certain features… and to convey information that is relevant to the management of the individual's disorder" (pg. 22).

- 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 basis for a visit resulting in ambulatory care medical services in outpatient settings. The principal diagnosis is generally the focus of attention or 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.

3.2.2.4. 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, specific learning disorder, motor disorders, and ADHD	7, 8, 9, & 10
Schizophrenia Spectrum	Disorders characterized by one or more of the following: delusions, hallucinations, disorganized thinking and speech, disorganized motor behavior, and negative symptoms	Not covered
Bipolar and Related	Characterized by mania or hypomania and possibly depressed mood; includes Bipolar I and II and cyclothymic disorder	12
Depressive	Characterized by sad, empty, or irritable mood, as well as somatic and cognitive changes that affect functioning; includes major depressive, persistent depressive disorder, mood dysregulation disorder, and premenstrual dysphoric disorder	12
Anxiety	Characterized by excessive fear and anxiety and related behavioral disturbances; Includes phobias, separation anxiety, panic disorder, generalized anxiety disorder, social anxiety disorder, agoraphobia	13
Obsessive-Compulsive	Characterized by obsessions and compulsions and includes OCD, hoarding, body dysmorphic disorder, trichotillomania, and excoriation	14
Trauma- and Stressor- Related	Characterized by exposure to a traumatic or stressful event; PTSD, acute stress disorder, adjustment disorders, and prolonged grief disorder	4 & 15
Dissociative	Characterized by a disruption or discontinuity in memory, identity, emotion, perception, body representation,	Not covered

	consciousness, motor control, or behavior; dissociative identity disorder, dissociative amnesia, and depersonalization/derealization disorder	
Somatic Symptom	Characterized by prominent somatic symptoms and/or illness anxiety associated with significant distress and impairment; includes illness anxiety disorder, somatic symptom disorder, and conversion disorder	Not covered
Feeding and Eating	Characterized by a persistent disturbance of eating or eating-related behavior to include bingeing and purging; Includes pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia, bulimia, and binge-eating disorder	5 & 16
Elimination	Characterized by the inappropriate elimination of urine or feces; usually first diagnosed in childhood or adolescence; Includes enuresis and encopresis	6
Sleep-Wake	Characterized by sleep-wake complaints about the quality, timing, and amount of sleep; includes insomnia, sleep terrors, narcolepsy, sleep apnea, hypersomnolence disorder, restless leg syndrome, and circadian-rhythm sleep-wake disorders	Not covered
Sexual Dysfunctions	Characterized by sexual difficulties and include premature or delayed ejaculation, female orgasmic disorder, and erectile disorder (to name a few)	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 the self-control of emotions and behavior and involve the violation of the rights of others and cause the individual to violate societal norms; includes oppositional defiant disorder, antisocial personality disorder, kleptomania, intermittent explosive disorder, conduct disorder, and pyromania	11
Substance-Related and Addictive	Characterized by the continued use of a substance despite significant problems related to its use	17
Neurocognitive	Characterized by a decline in cognitive functioning over time	Not

	and the NCD has not been present since birth or early in life; Includes delirium, major and mild neurocognitive disorder, and Alzheimer's disease	covered
Personality	Characterized by a pattern of stable traits which are inflexible, pervasive, and leads to distress or impairment; Includes paranoid, schizoid, borderline, obsessive-compulsive, narcissistic, histrionic, dependent, schizotypal, antisocial, and avoidant personality disorder	Not covered
Paraphilic	Characterized by recurrent and intense sexual fantasies that can cause harm to the individual or others; includes exhibitionism, voyeurism, sexual sadism, sexual masochism, pedophilic, and fetishistic disorders	Not covered

3.2.3. The ICD-11

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-11 went into effect January 1, 2022, though it was adopted in May 2019. The WHO states:

> ICD serves a broad range of uses globally and provides critical knowledge on the extent, causes and consequences of human disease and death worldwide via data that is reported and coded with the ICD. Clinical terms coded with ICD are the main basis for health recording and statistics on disease in primary, secondary and tertiary care, as well as on cause of death certificates. These data and statistics support payment systems, service planning, administration of quality and safety,

and health services research. Diagnostic guidance linked to categories of ICD also standardizes data collection and enables large scale research.

As a classification system, it "allows the systematic recording, analysis, interpretation and comparison of mortality and morbidity data collected in different countries or regions and at different times." As well, it "ensures semantic interoperability and reusability of recorded data for the different use cases beyond mere health statistics, including decision support, resource allocation, reimbursement, guidelines and more."

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

The ICD lists many types of diseases and disorders to include Chapter 06: Mental, Behavioral, or Neurodevelopmental Disorders. The list of mental disorders is broken down as follows:

- Neurodevelopmental disorders
- Schizophrenia or other primary psychotic disorders
- Catatonia
- Mood disorders
- Anxiety or fear-related disorders
- Obsessive-compulsive or related disorders
- Disorders specifically associated with stress
- Dissociative disorders
- Feeding or eating disorders
- Elimination disorders
- Disorders of bodily distress or bodily experience
- Disorders due to substance use or addictive behaviours

- Impulse control disorders
- Disruptive behaviour or dissocial disorders
- Personality disorders and related traits
- Paraphilic disorders
- Factitious disorders
- Neurocognitive disorders
- Mental or behavioural disorders associated with pregnancy, childbirth or the puerperium

It should be noted that Sleep-Wake Disorders are listed in Chapter 07.

To access Chapter 06 of the ICD-11, please visit the following:

https://icd.who.int/browse11/l-m/en#/http%3a%2f%2fid.who.int%2ficd%2fentity%2f334423054

3.2.4. Harmonization of DSM-5-TR and ICD-11

According to the DSM-5-TR, there is an effort to harmonize the two classification systems: 1) for a more accurate collection of national health statistics and design of clinical trials aimed at developing new treatments, 2) to increase the ability to replicate scientific findings across national boundaries, and 3) to rectify the issue of DSM-IV and ICD-10 diagnoses not agreeing (APA, 2022, pg. 13). Complete harmonization of the DSM-5 diagnostic criteria with the ICD-11 disorder definitions has not occurred due to differences in timing. The DSM-5 developmental effort was several years ahead of the ICD-11 revision process. Despite this, some improvement in harmonization did occur as many ICD-11 working group members had

participated in the development of the DSM-5 diagnostic criteria and all ICD-11 work groups were given instructions to review the DSM-5 criteria sets and make them as similar as possible (unless there was a legitimate reason not to). This has led to the ICD and DSM being closer than at any time since DSM-II and ICD-8 (APA, 2022).

You should have learned the following in this section:

- 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 outlined in an established classification system such as the DSM-5-TR or ICD-11.
- Classification systems provide mental health professionals with an agreed-upon list of disorders falling into distinct categories for which there are clear descriptions and criteria for making a diagnosis.
- Elements of a diagnosis in the DSM include the diagnostic criteria and descriptors, subtypes and specifiers, the principle diagnosis, and a provisional diagnosis.

Section 3.2 Review Questions

1. What is clinical diagnosis?
2. What is a classification system and what are the two main ones used today?
3. Outline the diagnostic categories used in the DSM-5-TR.

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 the article *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 though the signs you need to seek help are obvious at times, we often try "to sustain [our] 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 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." 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% 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 a failure to admit you need help, and there could be a biological issue that makes it almost impossible to heal yourself.

3.3.1.3. What exactly is psychotherapy? According to the APA, 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 treatments.

3.3.1.4. The client-therapist relationship. What is the ideal 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 help you cope better 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 take depends on the reason for the visit.

In terms of what you should expect, you and your therapist 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 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

You should have learned the following in this section:

- Anyone can seek treatment and we all can benefit from it at some point in our lives.
- Psychotherapy is when psychologists apply scientifically validated procedures to help a person feel better and develop healthy habits.

Section 3.3 Review Questions

1. When should you seek help?
2. Why should you seek professional help over the advice dispensed by family and friends?
3. How do you find a therapist and what should you bring to your appointment?
4. Does psychotherapy work?

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 17.

In Module 3 we reviewed 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-TR and ICD-11. For treatment, we discussed the reasons why someone may seek treatment, self-treatment, psychotherapy, the client-centered relationship, and how well psychotherapy works.

3rd edition as of December 2022

Part II. Disorders of Infancy and Early Childhood

Part II. Disorders of Infancy and Early Childhood

Module 4:
Disinhibited Social Engagement Disorder and Reactive Attachment Disorder

Module 4: Disinhibited Social Engagement Disorder and Reactive Attachment Disorder

Module Overview

In Module 4, we will discuss matters related to disinhibited social engagement disorder and reactive attachment disorder to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. We will also describe attachment and how its disruption affects the development of the two disorders. Be sure you refer to 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
- 4.2. Prevalence and Comorbidity
- 4.3. Etiology
- 4.4. Assessment and Treatment

Module Learning Outcomes

- Describe how disinhibited social engagement disorder and reactive attachment disorder present.
- Describe the prevalence of disinhibited social engagement disorder and reactive attachment disorder.

- Describe the etiology of disinhibited social engagement disorder and reactive attachment disorder.
- Describe how disinhibited social engagement disorder and reactive attachment disorder are assessed, diagnosed, and treated.

4.1. Clinical Presentation

Section Learning Objectives

- Describe the presentation and associated features of disinhibited social engagement disorder.
- Describe the presentation and associated features of reactive attachment disorder.
- Describe attachment and the impact attachment disruption has on the development of disinhibited social engagement disorder and reactive attachment disorder.

Although we are covering disinhibited social engagement disorder and reactive attachment disorder under the label of "early childhood disorders," both are technically trauma-related disorders. The reason we are covering them here is because the disorders emerge due to traumatic experiences during early childhood. They do not spontaneously present in late childhood or adulthood.

4.1.1. Clinical Presentation of Disinhibited Social Engagement Disorder

We will explore disinhibited social engagement disorder in terms of: (1) how the child presents and (2) the history of the child.

Let's start with how the child presents. These children, unlike children with reactive attachment disorder described below, tend to be overly social and interact with complete strangers. The child may walk up to someone in a store they have never met and hug them or even walk away with them. They may do this without ever hesitating when separating from their caregiver and might not even look to check back with their caregiver.

In terms of the history of the child, these children often experience *impaired caregiving,* which means that the caregiver does not sufficiently care for the child on a consistent basis. This could result from a child simply not being attended by their caregivers. Perhaps the parent does not interact with them regularly, or the basic needs of the child are not appropriately cared for, such as food, hygiene, and shelter. The child may be in a setting in which there are not enough caregivers to care for them consistently (e.g., an orphanage in which there are only a few caretakers with several infants), or they may not have had one consistent caregiver (e.g., they moved from one foster care setting to another regularly). These situations interrupt the attachment and security of a developing child. It should be noted that the child will have a developmental age of at least 9 months.

Disinhibited social engagement disorder may co-occur with developmental delays such as cognitive and language delays, stereotypies, and other signs of severe neglect. Signs of the disorder often persist even after these other signs of neglect are absent. As such, it is not uncommon for children with disinhibited social engagement disorder to present with no current signs of neglect. The DSM adds that the condition can present in children who show no signs of

disordered attachment. “Thus, disinhibited social engagement disorder may be seen in children with a history of neglect who lack attachments or whose attachments to their caregivers range from disturbed to secure” (APA, 2022, pg. 299).

The functional consequences of disinhibited social engagement disorder include impairment of a young child’s abilities to relate interpersonally to adults and peers. Social functioning and social competence may be impaired and there may be increased risk for peer conflicts and victimization.

4.1.2. Clinical Presentation of Reactive Attachment Disorder

We will discuss reactive attachment disorder in much the same way as disinhibited social engagement disorder starting with how the child presents. Children with this disorder typically present as detached from others or emotionally withdrawn. They do not seek comfort from caregivers or respond to physical touch when distressed. These children typically have low levels of expressed emotions, particularly positive emotion. They may experience unexplained irritability, sadness, and fearfulness.

In terms of the history of the child, similarly to disinhibited social engagement disorder, the child will have experienced a pattern of extremes of insufficient care manifested as one of the following: social neglect or deprivation; repeated changes of primary caregivers that restrict their ability to form stable attachments, and/or rearing in unusual settings that do not allow for the formation of attachments. The clinical features of the disorder manifests between the ages of 9 months and 5 years.

Associated features include developmental delays, especially in cognition and language. Reactive attachment disorder impairs a young child's ability to relate interpersonally to adults or peers and causes functional impairment across many domains of early childhood.

Making Sense of the Disorders

According to the information above, both disorders share the same or very similar history. The disorders can be distinguished as follows:

- Diagnose **disinhibited social engagement disorder** if the child displays culturally inappropriate, overly familiar behavior with relative strangers without checking back with the adult caregiver.
- Diagnose **reactive attachment disorder** if the child rarely or minimally goes to an attachment figure for comfort, support, protection, or nurturance when distressed.

4.1.3. Attachment

Zeanah, Chesher, Boris, and the AACAP CQI (2016) define attachment as a "biologically driven process that results in organization of behaviors in the young child, especially behavior designed to achieve physical proximity to a preferred caregiver when the child is in need of comfort, support, nurturance, or protection (p. 991)." Attachment begins to develop early in infancy, which is recognizable in an infant as early as 7 to 9 months of age. At this age, infants will often show hesitation around unfamiliar adults and become distressed when separated from their caregiver – this is a sign of attachment. If this does not occur, caregivers may become concerned. A child can form attachments with more than one caregiver, however, they have a maximum number of individuals they can do this with. Although attachment begins to form

around 7 to 9 months, attachment can occur after this time. Thus, if a child is removed from an impaired caregiving situation and placed in a situation in which he or she receives sensitive, responsive, and consistent care from a caregiver, they may be able to form an attachment appropriately (Zeanah, et al., 2016). However, if they have tried to attach to multiple individuals, perhaps their ability to fully attach may be compromised.

Mary Salter Ainsworth created the Strange Situation procedure in 1969 to assess attachment. In this procedure, the child experiences separating and reuniting with their primary caregiver. Strangers are also introduced during this procedure. During all trials and interactions, the infant's reaction is monitored and recorded (Krapp, 2005). In this procedure, the caregiver, baby, and observer first enter a room, then after 30 seconds, the observer leaves, allowing the caregiver and baby to explore the room together. Next, a stranger enters the room quietly, interacts with the caregiver, and approaches the infant as the caregiver leaves. The caregiver will be absent and the stranger will stay with the infant for three minutes. After three minutes, the caregiver comes back, engages with the child, and the stranger leaves. Next, the caregiver says goodbye to the child and leaves the room again for three minutes. At this point, the baby is alone in the room but observed for behaviors and safety through a one-way mirror. The baby is alone for three minutes before the stranger, but not the caregiver, enters the room. After another three minutes, the caregiver returns and the stranger leaves. During each of these variations, the infant's behaviors are monitored and recorded. Observers will note things such as whether the child moves close to their caregiver, cling to their caregiver, if they ignore either their caregiver or the stranger, if they avoid or reject contact from an adult, if they look around for their caregiver, or if they vocalize or interact across the room with their caregiver or the stranger. The

presence or absence of a combination of these behaviors helps determine the child's attachment style (Krapp, 2005).

When we talk about attachment, we often talk about different attachment styles (Zeanah, Chesher, Boris, and AACP CQI, 2016). Avoidant or resistant attachment is a risk factor for later psychopathology and negative trajectories, whereas secure attachment is a protective factor. During the Strange Situation procedure, a child that is **securely attached** is likely to explore a room while the caregiver is present, feeling confident that their caregiver will be there to help or support them, if needed. They are also easily calmed by their caregiver when distressed. A child that is **avoidant** does not seek their caregiver out or utilize their caregiver for soothing. A **resistant attachment** style may be represented by a child never moving away from their caregiver to explore the room, or a child that is difficult to sooth. **Disorganized attachment** typically involves patterns of interactions that are not fully described above or are significantly inconsistent. Attachment is considered either secure, avoidant, resistant, or disorganized (Zeanah, et al., 2016).

You should have learned the following in this section:

- A child with disinhibited social engagement disorder tend to be overly social and interact with complete strangers and have experienced *impaired caregiving,* which means that the caregiver does not sufficiently care for the child on a consistent basis.
- A child with reactive attachment disorder do not seek comfort from caregivers or respond to physical touch when distressed and has experienced a pattern of extremes of insufficient care.
- A child's attachment style is either secure, avoidant, resistant, or disorganized. Avoidant or resistant attachment is a risk factor for later psychopathology and negative trajectories whereas secure attachment is a protective factor.

Section 4.1 Review Questions

1. In what ways are disinhibited social engagement disorder and reactive attachment disorder similar and different from one another?
2. What is attachment and what styles can a child/person display to a caregiver

4.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence of disinhibited social engagement disorder and reactive attachment disorder.
- Describe common disorders that are comorbid with disinhibited social engagement disorder and reactive attachment disorder.

4.2.1. Disinhibited Social Engagement Disorder

4.2.1.1. Prevalence. The prevalence of disinhibited social engagement disorder is largely unknown (APA, 2022) and considered to be extremely rare. The DSM notes that prevalence is up to 2% in low-income community populations in the United Kingdom.

4.2.1.2. Differential diagnosis. Disinhibited social engagement disorder must be differentiated from ADHD. Unlike ADHD, children with disinhibited social engagement disorder do not show difficulties with either attention or hyperactivity (APA, 2022).

Additionally, the medical condition of Williams syndrome, caused by a partial chromosomal deletion, may mimic disinhibited social engagement disorder symptoms (Zeanah, et al., 2016). These children struggle with social discrimination and tend to be overly approaching to strangers. However, they display this behavior despite not having a history of neglect/trauma symptoms (Zeanah, et. al, 2016). As such, if a child is socially disinhibited, but has no history of neglect, disinhibited social engagement disorder is not the likely diagnosis. In fact, it may be that the behavior is caused by a more medically-based etiology such as Williams syndrome.

4.2.1.3. Comorbidity. Cognitive and language delays, autism spectrum disorder, as well as stereotypies are often comorbid with disinhibited social engagement disorder. ADHD and externalizing disorders are often comorbid in younger children and in middle childhood (APA, 2022).

4.2.2 Reactive Attachment Disorder

4.2.2.1. Prevalence. The prevalence of reactive attachment disorder is largely unknown and considered extremely rare. Young children exposed to severe neglect often present with reactive attachment disorder before being placed in foster care or institutions. Despite this, the disorder is uncommon and occurs in less than 10% of neglected children, even when severe neglect has occurred (APA, 2022).

4.2.2.2. Differential diagnosis. Due to the significant deficit in emotional-social reciprocity, autism spectrum disorder must be differentiated from reactive attachment disorder. Whether or not a child was neglected, and the presence or absence of restricted or repetitive behaviors/interests, help differentiate the two disorders (APA, 2022). If a child has no history of severe neglect or abuse, the diagnosis of autism spectrum disorder is more likely than reactive attachment disorder. If the child does not have restricted/repetitive behaviors/interests, the child is not likely to be diagnosed with autism spectrum disorder, and as such, deficits in social/emotional reciprocity are more likely to be explained by reactive attachment disorder, particularly if there is a significant neglect/abuse history.

4.2.2.3. Comorbidity. Cognitive and language delays, severe malnutrition, as well as stereotypies are often comorbid with reactive attachment disorder. There may be a comorbidity with ADHD, though this is not clearly established.

You should have learned the following in this section:

- The prevalence of both reactive attachment disorder and disinhibited social engagement disorder is largely unknown, though both are considered to be extremely rare.
- Disinhibited social engagement disorder must be differentiated from ADHD and Williams syndrome while reactive attachment disorder must be differentiated from autism spectrum disorder.
- Cognitive and language delays and stereotypies are often comorbid with both disorders.

Section 4.2 Review Questions

1. How prevalent are reactive attachment disorder and disinhibited social engagement disorder?
2. What other disorders must each be distinguished from?
3. Are there any common comorbid conditions or disorders with reactive attachment disorder and disinhibited social engagement disorder?

4.3. Etiology

Section Learning Objectives

- Describe environmental causes of disinhibited social engagement disorder and reactive attachment disorder.
- Clarify if there are any genetic causes of either disorder.

4.3.1. Environmental

4.3.1.1. General. For both disinhibited social engagement disorder and reactive attachment disorder, severe social neglect and impaired caregiving is the overall cause. These experiences disrupt the attachment process during the critical developmental period for a child. This disruption results in behavioral patterns that are problematic, not only in the short-term, but long-term, particularly if no interventions are implemented.

4.3.1.2. Causes specific to disinhibited social engagement disorder. Some evidence has been presented suggesting that both blunted reward sensitivity and decreased inhibitory control are associated with indiscriminate social behavior. Multiple placement disruptions, a mother with borderline personality disorder, aberrant caregiving behaviors, and low quality of care are also implicated as causes. It should be noted that the disorder may still persist even if the child's caregiving environment greatly improves.

4.3.2. Genetics and Disinhibited Social engagement Disorder

Some genetic vulnerabilities involving the brain-derived neurotrophic factor and serotonin transporter genes, combined with history of neglect/caregiving, may result in more

significant difficulties with social disinhibition (Zeanah, et al., 2016). For example, a child in a foster-care setting that also has a genetic vulnerability may have more significant symptoms of disinhibited social engagement disorder than a child in the same setting without the genetic vulnerability.

You should have learned the following in this section:

- Severe social neglect and impaired caregiving is the overall cause for both disinhibited social engagement disorder and reactive attachment disorder.
- In the case of disinhibited social engagement disorder, blunted reward sensitivity, decreased inhibitory control, multiple placement disruptions, a mother with borderline personality disorder, and aberrant caregiving behaviors and low quality of care are also cited as potential causes.
- For disinhibited social engagement disorder, some genetic vulnerabilities, combined with history of neglect/caregiving, may result in more significant difficulties with social disinhibition.

Section 4.3 Review Questions

1. What are the most common causes of disinhibited social engagement disorder and reactive attachment disorder?
2. Are there any other unique factors for the two disorders?

4.4. Assessment and Treatment

Section Learning Objectives

- Describe assessment tools commonly used to diagnose disinhibited social engagement disorder and reactive attachment disorder.
- Describe treatment options for disinhibited social engagement disorder and reactive attachment disorder.

4.4.1. Assessment

Assessment will include a thorough interview with a caregiver. This caregiver may be the biological parent of the child, or a foster parent, social worker, or other relative. An understanding of, not only the child's history, such as trauma, access to care and nurturing, etc., but also developmental progress and social interactions, are important. An in-depth exploration of how the child responds to support, calming, and nurturing is needed. Moreover, understanding how the child typically reacts to strangers is imperative. Much of this is gained through interviews. However, observation is also important.

Observations may be largely informal. A psychologist may note several behaviors as they occur, such as how the child approaches the psychologist and interacts with them. If the child immediately runs up to the psychologist and wants the psychologist to hold them, that is notable. If the child gets upset in the room, and they do not seek their caregiver out for comfort, or if they reject the caregiver's attempts to comfort them, that is important. Also, if the caregiver or psychologist praise the child for doing something well, and the child seems to not react or be impacted by the praise, that is also notable.

The psychologist may also choose to implement a more formal observational assessment. Although the most known and validated observational procedure to assess attachment is the Strange Situation procedure, this is typically only conducted in a research setting, and is less commonly used in clinical settings. However, Zeanah, et al. (2016) developed an informal procedure that can be used to obtain qualitative information that does not result in an objective score, necessarily. The authors note that the safety and appropriateness of the child's current care, living situation, and caregiver relationship should also be examined.

4.4.2. Treatment

Because it is theorized that, due to significant impairment in caregiving, the relationship and attachment between the child and caregiver is damaged, therapies focus on repairing that relationship or establishing a bond between the child and a new caregiver if the offending caregiver is no longer involved in the child's life. However, it should be noted that little research directly investigates the impact of therapies on these disorders.

A significant goal of therapy is to improve sensitive caregiving from the caregiver. This involves increasing the caregiver's ability to 'tune in' to the child so that they can be particularly responsive and sensitive to the child's needs. This can be achieved by either (1) working only with the caregiver or (2) by working with the caregiver *and* the child. Treatment is not typically conducted with only the child because that does not allow a clinician to appropriately address the core concern – attachment (Zeanah, et al., 2016).

4.4.2.1. Caregiver only treatment. One of the first things that may occur in therapy is an attempt to (1) understand the relationship between the caregiver and infant and (2) provide support to the caregiver. To help the child and caregiver attach, which is the primary goal of the

intervention, the caregiver must be emotionally ready to do so (Zeanah, et al., 2016). In other words, if a caregiver is overwhelmed, frustrated, and defeated, they may not be able to respond consistently and calmly to the child. If they cannot do this, attachment cannot be fostered. As such, the caregiver's own feelings and reactions must be acknowledged and supported. The therapist may also ask the caregiver to talk about their relationship with the child so that the therapist can examine the parent-child relationship in detail. This will allow the therapist to identify interactions between the parent and child that can be improved. Once the caregiver is emotionally ready, and the therapist understands the relationship between the child and parent, then work on their attachment can occur. Video review or group therapy may occur as well (Zeanah, et al., 2016).

4.4.2.2. Child-parent dyad treatment. When working with both the child and the caregiver, focus is on the dyad and the emotional interchanges (Zeanah, et al., 2016). Therapy typically starts by focusing on strengths in the relationship and parenting skills. This allows the parent to trust the therapist, for the therapist to build rapport with the caregiver, and to lower defensiveness and feelings of low efficacy. Following this, coaching the caregiver through moments of disengagement or frustration occurs. This may occur through either child-parent psychotherapy or Attachment and Biobehavioral Catch Up (ABC) videotape review and the clinician shaping the caregiver's responses. ABC targets not only the attachment but the environment the child lives in. The intervention, in addition to building sensitive caregiving, also works to increase the predictability in the child's environment and to decrease caregiver behaviors that may distress the child (CEBC, 2018, September).

You should have learned the following in this section:

- Assessment will include a thorough interview with a caregiver as well as observation. This will include how the child responds to the psychologist.
- Therapies focus on repairing the caregiver-child relationship or establishing a bond between the child and a new caregiver.
- A primary goal of therapy is to improve sensitive caregiving from the caregiver.
- Caregiver only treatment includes an attempt to (1) understand the relationship between the caregiver and infant and (2) provide support to the caregiver.
- With child-parent dyad treatment, the focus is on the dyad and the emotional interchanges.

Section 4.4 Review Questions

1. What methods are used to assess the presence of either disinhibited social engagement disorder or reactive attachment disorder?
2. What treatment approaches exist for disinhibited social engagement disorder and reactive attachment disorder?

Apply Your Knowledge

CASE VIGNETTE

Cindy, five years old, and Marcus, five years old, have lived with their biological mother for most of their life. Their biological mother experienced significant mental illnesses leading to debilitating depression. Their mother also has a history of serious substance abuse problems leading to an inability to function and carry out necessary daily life tasks. Throughout their life, their mother often had strangers in and out of the home, and unfortunately, their mother also experienced domestic violence, much of which, Cindy and Marcus witnessed. The caregiving that their mother provided was often impaired, and Cindy and Marcus found themselves having to find their own food and means of safety. In the past year, both Cindy and Marcus were placed in foster care due to concerns of neglect and abuse.

Cindy will often reject her foster parents attempts to provide comfort. For example, when Cindy's foster mother tries to hug her, Cindy tenses her whole body. Cindy is often looking around in her environment attempting to predict any danger. She does not readily engage with other children, and Cindy also has significant difficulties with severe emotional meltdowns.

Marcus, on the other hand, is not withdrawn. He tends to go up to everyone and will even hug strangers in the grocery store. He does not seem to look out for danger and often places himself in safety-compromising situations. His foster mother has read reports in his file that he has engaged in this type of behavior for many years.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What disorders, if any, might be present for Cindy?
2. Do you think Marcus meets the criteria for a disorder? If so, what is it? If not, why not?
3. Why are two children coming from the same home and experiences displaying drastically different behaviors?
4. What protective factors would help both Cindy and Marcus have the healthiest trajectory in the future?

Module Recap

In this module, we learned about disinhibited social engagement disorder and reactive attachment disorder. We discussed the various behaviors and symptoms of both disorders and how they relate to the various presentations. We also learned about attachment styles and how they related to disinhibited social engagement disorder and reactive attachment disorder. We then discussed the prevalence of these disorders, frequently comorbid disorders, and possible causes. We ended on a discussion of how disinhibited social engagement disorder and reactive attachment disorder are assessed and treated.

In our next module we will discuss another category of disorders that appear in infancy and early childhood – feeding disorders.

Part II. Disorders of Infancy and Early Childhood

Module 5:
Feeding Disorders

Module 5: Feeding Disorders

Module Overview

In Module 5, we will discuss matters related to feeding disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include pica, rumination disorder, and avoidant/restrictive food intake disorder. Be sure you refer to 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

- 5.1. Clinical Presentation and DSM-5-TR Criteria
- 5.2. Prevalence and Comorbidity
- 5.3. Etiology
- 5.4. Assessment and Treatment

Module Learning Outcomes

- Describe how pica, rumination disorder, and avoidant/restrictive food intake disorder present.
- Describe the prevalence of the feeding disorders.
- Describe the etiology of the feeding disorders.
- Describe how the feeding disorders are assessed, diagnosed, and treated.

5.1. Clinical Presentation and DSM-5-TR Criteria

Section Learning Objectives

- Contrast feeding and eating disorders.
- Describe the presentation and associated features of pica.
- Describe the presentation and associated features of rumination disorder.
- Describe the presentation and associated features of avoidant/restrictive food intake disorder.

5.1.1. Overview of Feeding and Eating Disorders

According to the DSM-5-TR, feeding and eating disorders, "…are characterized by a persistent disturbance of eating or eating-related behavior that results in altered consumption or absorption of food and that significantly impairs physical health or psychosocial functioning" (APA, 2022, pg. 371). These disorders used to be considered two different areas of concern in previous editions of the DSM, but with the DSM-5 (and now 5-TR), they have been combined into one category (APA, 2013 and 2022). For our purposes, we will separate them out into two separate modules – one on feeding disorders and one on eating disorders.

So, what makes them different from one another? First, **feeding disorders** are simply that, concern with how one feeds and consumes food, typically in young children and/or individuals with other developmental concerns (e.g., intellectual delays). Second, an **eating disorder** is related more to eating habits, rather than feeding concerns, whether it be under or overeating, and occurs more frequently in typically developing (meaning no developmental or cognitive delays) individuals from childhood or adolescence to adulthood. As noted earlier, our

discussion in this module will focus on pica, rumination disorder, and avoidant/restrictive food intake disorder (the feeding disorders) and later we will discuss anorexia, bulimia, and binge eating disorders (the eating disorders; in Module 16).

5.1.2. Pica

Pica is the act of eating items that are not food on a regular or recurring basis over a period of at least one month. These nonnutritive, nonfood substances could include grass, chalk, dirt, paper, hair, soap, wool, paint, gum, pebbles, coal, ash, or starch, to name a few. Pica can impair physical functioning but any impairment in social functioning usually occurs in conjunction with other disorders. The behavior is inappropriate to the developmental level of the child, so it should not be diagnosed before age 2 to exclude developmentally normal mouthing of objects leading to ingestion, typical of infants. Also, the behavior should not be part of a culturally supported or socially normative practice such as ingesting a particular grass for medicinal or spiritual purposes (APA, 2022). Pica is not often associated with specific biological abnormalities, though deficiencies in vitamins and minerals such as iron and zinc, have been reported.

Pica may occur more frequently in disorders such as autism spectrum disorder (or other mental health disorders); however, a clinician must be careful to ensure that the act of eating nonfood items is to a higher degree than would be expected if another disorder is present. For example, if a child regularly attempts to eat grass because they like the oral stimulation of it, and are diagnosed with autism spectrum disorder, a clinician must determine if the individual is attempting to ingest a nonfood item, or if they are trying to orally stimulate. If the individual is only trying to orally stimulate, they would not be diagnosed with pica.

5.1.3. Rumination Disorder

Rumination disorder is the frequent act of regurgitating food over a period of at least 1 month with no medical explanation such as gastroesophageal reflux and in the absence of a body-image/weight-related reason such as anorexia, bulimia, and binge-eating disorder. An individual may rechew and then eject the food from the mouth or re-swallow (APA, 2022).

Functional consequences of rumination disorder include growth delay due to malnutrition secondary to repeated regurgitation. Though the onset of the disorder can occur at any time during life, when it begins in infancy, the age at onset is between ages 3 and 12 months, and it can be fatal.

5.1.4. Avoidant/Restrictive Food Intake Disorder

Avoidant/restrictive food intake disorder is a new disorder to the DSM 5 (APA, 2013) and replaces and extends the DSM-IV diagnosis of feeding disorder of infancy or early childhood (Norris & Katzman, 2015). It is an eating or feeding disturbance associated with at least one of the following: 1) significant weight loss, 2) significant nutritional deficiency, 3) dependence on enteral feeding or oral nutrition supplements, and 4) marked interference with psychosocial functioning. The disturbance is not better explained due to a lack of available food or a culturally sanctioned practice.

The food avoidance or restriction may occur due to the sensory characteristics of qualities of food such as its appearance, color, smell, texture, taste, or temperature. It may occur due to a conditioned negative response associated with food intake following, or in anticipation of, an aversive event such as choking, repeated vomiting, or a traumatic procedure. And for some, the food avoidance or restriction occurs due to a lack of interest in eating or food.

You should have learned the following in this section:

- Pica is the act of eating items that are not food on a regular or recurring basis and over a period of at least one month.
- Rumination disorder is the frequent act of regurgitating food over a period of at least 1 month with no medical explanation such as gastroesophageal reflux and in the absence of a body-image/weight-related reason.
- Avoidant/restrictive food intake disorder is associated with at least one of the following: 1) significant weight loss, 2) significant nutritional deficiency, 3) dependence on enteral feeding or oral nutrition supplements, and 4) marked interference with psychosocial functioning.

Section 5.1 Review Questions

1. How do eating and feeding disorders differ?
2. Outline the history of feeding and eating disorders in the DSM.
3. What is pica?
4. What is rumination disorder?
5. What is avoidant/restrictive food intake disorder? How does food avoidance or restriction manifest?

5.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence of pica, rumination disorder, and avoidant/restrictive food intake disorder.
- Describe comorbid disorders with pica, rumination disorder, and avoidant/restrictive food intake disorder.

5.2.1. Pica

Limited data suggests that the prevalence of pica is around 5% of school-age children and a third of pregnant women engage in pica, especially if they have food insecurity (APA, 2022; Murray, Thomas, Hinz, Munsch, & Hilbert, 2018). Some studies indicate that pica is more common in males than females (El-Nemer, Alian, Salah-Eldin, Khalil; 2014) whereas others do not show a significant difference between genders (APA, 2022; Murray et al., 2018). A worldwide meta-analysis showed a prevalence of 28% during pregnancy and/or the postpartum period (APA, 2022).

Autism spectrum disorder and intellectual developmental disorder (intellectual disability) are the two most common comorbid diagnoses. Less common are schizophrenia and OCD. Pica may also be comorbid with trichotillomania (hair-pulling disorder), excoriation (skin-picking) disorder, and avoidant/restrictive food intake disorder.

5.2.2. Rumination Disorder

Limited European data suggests a prevalence of 1% to 2% of grade-school-age children. Rumination disorder also occurs more frequently in individuals with an intellectual disability (Olden, 2001). It can occur in the context of a concurrent medical condition or another mental disorder such as generalized anxiety disorder.

5.2.3 Avoidant/Restrictive Food Intake Disorder

Again, prevalence rates are largely unknown. A study in Australia reported a frequency of 0.3% among individuals aged 15 years and up. Avoidant/restrictive food intake disorder is more common in children with autism spectrum disorder and when so, has a male predominance. This is possibly due to sensory concerns and rigidity that leads to children having very specific preferences for foods. Children may actively refuse to eat many foods due to texture aversions. When autism spectrum disorder is not comorbid, the disorder occurs about equally between males and females.

As mentioned, autism spectrum disorder is a common comorbid disorder. Other neurodevelopmental disorders may be highly comorbid such as ADHD, intellectual development disorder (intellectual disability), anxiety disorders, and OCD (APA, 2022).

You should have learned the following in this section:

- There is limited data on the prevalence of the feeding disorders discussed in this module. This data suggests pica has a prevalence of 5% in school-age children and a third of pregnant women engage in pica, rumination disorder has a prevalence of 1% to 2% of grade-school-age children, and avoidant/restrictive food intake disorder may occur in 0.3% of individuals aged 15 years and up.
- Pica and avoidant/restrictive food intake disorder are comorbid with autism spectrum disorder and intellectual developmental disorder, as well as each other. As well, they occur about equally in men and women.

Section 5.2 Review Questions

1. How common are the three feeding disorders?
2. Are the disorders any more common in men or women?
3. What are the most frequently comorbid disorders?

5.3. Etiology

Section Learning Objectives

- Describe various biological, behavioral, and sociocultural causes of feeding disorders.

5.3.1. Pica

Pica is commonly associated with an iron deficiency. It is theorized that extremely low iron levels lead to individuals consuming non-perishable foods that, although not nutritious, may contain high iron content. El-Nnemer and colleagues (2014) confirmed that a large portion of children exhibiting pica in their study had low levels of zinc, hemoglobin, iron, and ferritin. They also found that other factors such as low nourishment and low socioeconomic status were risk factors for pica for the individuals in their study. Neglectful caregiving, lack of supervision, and developmental delay may also be risk factors for the development of pica (APA, 2022).

5.3.2. Rumination Disorder

Information related to rumination disorder etiology is limited. However, like pica, neglectful caregiving, a difficult parent-child relationship, lack of stimulation, and early life stress may be important risk factors to consider (APA, 2022).

5.3.3 Avoidant/Restrictive Food Intake Disorder

Various factors may contribute to the development of avoidant/restrictive food intake disorder. Parent-child interactions may be particularly important to consider. How a parent approaches feeding the child is important. It may be that they do not present and feed the child 'properly.' Additionally, when the child rejects the food, the parent may become frustrated and discouraged. This may further increase tension during feeding and lead to increased restriction and avoidance. Moreover, parental psychopathology, such as mothers with eating disorders, (APA, 2022) and neglect/abuse may exacerbate the above interactions as well.

Children with avoidant/restrictive food intake disorder often have families with high rates of anxiety (Cooper et al., 20014). Gastrointestinal or gastroesophageal disorders may lead to children highly restricting their food as well (Burklow et al., 1998).

You should have learned the following in this section:

- Pica is commonly associated with an iron deficiency. Low nourishment, low socioeconomic status, and neglectful caregiving are risk factors.
- A lack of stimulation, neglectful caregiving, a difficult parent-child relationship, and early life stress may be important risk factors to consider where rumination disorder is concerned.
- Parent-child interactions and families with high rates of anxiety are risk factors for avoidant/restrictive food intake disorder.

Section 5.3 Review Questions

1. What are the risk factors for pica?
2. What are the risk factors for rumination disorder?
3. What are the risk factors for avoidant/restrictive food intake disorder?

5.4. Assessment and Treatment

Section Learning Objectives

- Describe assessment methods for feeding disorders.
- Describe treatment options for feeding disorders.

5.4.1. General Assessment of Feeding-related Concerns

Often, assessment begins with parent/caregiver reports during interviews. A psychologist will likely conduct a thorough interview to obtain detailed information about what the child eats or does not eat, if the behaviors improve or worsen at any point, and any other related concerns. They may also ask questions related to the parent's feeding practices with the child. Additionally, a thorough medical examination to rule out medical conditions causing these atypical feeding concerns is necessary. Given the need for both a psychological and medical screening, assessments for these disorders often occur in specialized feeding clinics where a multidisciplinary team can conduct thorough screenings. These screenings may include blood draws for nutrition checks, gastrointestinal and gastroesophageal scopes, swallow studies (to rule out any structural issues with feeding movements), and observation of parent-child feeding sessions.

5.4.2. Therapy for Feeding-related Concerns

Similar to assessment, treatment frequently, but not always, occurs in a feeding clinic. Because these disorders occur more often in the developmentally delayed, treatment is very likely to take place in other settings. It is important to point out that because avoidant/restrictive

food intake disorder is highly comorbid with autism spectrum disorder due to sensory concerns, feeding therapy may also be incorporated into a child's ABA goals (Applied Behavior Analysis (ABA); this will be discussed in the module on autism spectrum disorder), and thus, be conducted outside of a feeding clinic setting.

In general, nutritional supplementation may be used to protect a child's health as well as potentially mitigate the need for certain behaviors. For example, if an individual has extremely low levels of iron, iron supplements may be given to increase the individual's iron levels, and thus, reduce pica behaviors. If there are structural or gastro-related reasons for the disorders, interventions related to medical, occupational (in the sense of occupational therapy, not vocational terms), or other specialized fields of expertise will be utilized.

5.4.2.1 Pica. Behavioral interventions are the primary modality of treatment for pica (Call, Simmons, Lomas Mevers, & Alvarez, 2015; Sturmey & Williams, 2016). For example, a technique within behavioral interventions that appears beneficial is differential reinforcement (Slocum, Mehrkam, Peters, & Vollmer, 2017). Briefly, **differential reinforcement** is when we attempt to get rid of undesirable or problem behaviors (in this case, pica) by using positive reinforcement (e.g., providing a reward of some sort) of desirable behaviors. For example, Differential Reinforcement of Alternative Behavior (DRA) is useful in reducing pica. DRA is when we reinforce the desired behavior and do not reinforce undesirable behavior. Hence, the desired behavior increases, and the undesirable behavior decreases to the point of extinction. The main goal of DRA is to increase a desired behavior such as eating an edible food item or even discarding the non-edible item. The therapist might praise the individual, offer a tangible reward, etc. when an individual selects an edible food item or discards a non-edible item.

5.4.2.2 Rumination disorder. Again, behavioral interventions are heavily utilized. A functional behavioral assessment is often implemented first to help understand the reason for the behaviors. A **functional behavioral assessment** is when we closely scrutinize the antecedents and consequences of behaviors to see what affects the occurrence or nonoccurrence of a desired or problem behavior. Through functional behavioral assessments, research indicates that rumination is automatically reinforced by sensory stimulation; that is, rumination provides a sensory stimulus that is reinforcing (Luiselli, 2015). This was more common than rumination occurring due to an attempt to gain attention, gain a desirable toy/object, or to escape from something. As such, interventions may attempt to replace the sensory stimulation of regurgitation of food/rumination by introducing either food or liquid continuously, on a fixed schedule, for a period of time following the target meal (Luiselli, 2015). This provides a replacement sensory stimulation (through foods or liquids) for an extended period of time. Because the sensory stimulation is considered to be desirable by the individual, it serves as reinforcement. Since the reinforcement (the food or liquid) is provided for a period, and does not require a particular behavior from the individual for the individual to receive the reinforcer, it is considered *noncontingent*. As such, **noncontingent reinforcers** are being utilized. It should be noted that, the foods and liquids that are used as noncontingent reinforcers must be food or liquids that are desired or preferred by the individual. The idea of this type of therapy is to break the automatic reinforcement of rumination by providing expected reinforcing experiences in an alternative way.

5.4.2.3 Avoidant/restrictive food intake disorder. Interventions may focus on similar behavioral principals outlined above (Sharp, Burrell, & Jaquess, 2014). Children may be rewarded with contingent attention, such positive reinforcement, when the child eats a previously

rejected food (Werle, Murphy, & Budd, 1993). This may occur in a clinic and/or at home. Essentially, behavioral principals are applied to increase food tolerance. Efforts for parents to learn how to implement these behavioral strategies around meals shows promising improvement for the child (Sharp, Burrell, & Jaquess, 2014; Najdowski, Wallace, Reagon, Penrod, Higbee, & Tarbox, 2010). The parent is providing food exposure for the child and rewarding the child for success. Again, strategies such as DRA, previously described, become useful here. These interventions may occur in the context of a feeding clinic; however, they may also occur at home, or in the context of a child's ongoing ABA therapy.

Consider that child-parent interactions may also strongly impact the development and maintenance of avoidant/restrictive food intake disorder. As such, carefully exploring the feeding patterns during mealtime between the parent and child is imperative. If irritability or frustration perpetuate the feeding difficulty, interventions to help increase the parent's awareness and provide support for the parent may be utilized. The parent might even observe a feeding therapist implement a feeding session with the child. In this situation, the therapist can model helpful strategies, how to deal with irritability from the child, etc.

You should have learned the following in this section:

- Parent/caregiver reports during interviews and a thorough medical examination are used to assess feeding disorders. The assessments often occur in specialized feeding clinics where a multidisciplinary team can conduct thorough screenings, but it not limited to these clinics.
- Behavioral interventions are the primary modality of treatment for pica, rumination disorder, and avoidant/restrictive food intake disorder.

Section 5.4 Review Questions

1. What types of assessment are used for feeding disorders?
2. What treatment approaches are used for feeding disorders?

Apply Your Knowledge

CASE VIGNETTE

Claudia is a 6-year-old girl. Her mother reported that Claudia is complaining of stomach aches and is somewhat lethargic. Upon further questioning, her physician learns that Claudia is very picky. Her mother explains that Claudia does not like certain foods and will not eat any foods that are orange in color. However, despite her pickiness, her mother stated that Claudia often attempts to eat paper and chalk. Her mother is not sure why and tries to keep Claudia from engaging in such behaviors but is often unsuccessful.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What disorder(s) would you consider for Claudia? What other information would you want?
2. How do you think her physician should proceed? What assessments and answers do we need to understand?
3. What treatments may be helpful for Claudia?

Module Recap

In Module 5, we discussed feeding disorders including pica, rumination disorder, and avoidant/restrictive food intake disorder. We discussed how these disorders present. In addition, we clarified the epidemiology, comorbidity, and etiology of each disorder. Finally, we discussed how these disorders are assessed and potential treatment options for each.

In our next module, we will continue to discuss disorders of infancy and early childhood by reviewing elimination disorders.

Part II. Disorders of Infancy and Early Childhood

Module 6:
Elimination Disorders

Module 6: Elimination Disorders

Module Overview

In Module 6, we will discuss matters related to elimination disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include enuresis and encopresis. Be sure you refer to 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 and DSM-5-TR Criteria
- 6.2. Prevalence and Comorbidity
- 6.3. Etiology
- 6.4. Assessment and Treatment

Module Learning Outcomes

- Describe the presentation of enuresis.
- Describe the presentation of encopresis.
- Describe the prevalence of enuresis and encopresis.
- Describe the etiology of enuresis and encopresis.
- Describe how enuresis and encopresis are assessed, diagnosed, and treated.

6.1. Clinical Presentation and DSM-5-TR Criteria

Section Learning Objectives

- Describe the presentation and associated features of enuresis.
- Describe the presentation and associated features of encopresis.

6.1.1. Enuresis

Enuresis is the repeated voiding of urine into bed or clothes and can be involuntary or intentional. It has occurred at least two times per week for the past three consecutive months. The child must also be at least five years of age. Mental health professionals can specify whether the enuresis is nocturnal only (i.e., urinary incontinence only while sleeping), diurnal only (i.e., urinary incontinence only while awake), or nocturnal and diurnal (i.e., both while asleep and awake; APA, 2022). The nocturnal only type is most common and occurs during the first one-third of the night. Diurnal enuresis most commonly occurs in the early afternoon on school days or after coming home from school. The degree of impairment is a function of, "…the limitation on the child's social activities (e.g., ineligibility for sleep-away camp) or its effect on the child's self-esteem, the degree of social ostracism by peers, and the anger, punishment, and rejection on the part of caregivers" (APA, 2022, pg. 401).

6.1.2. Encopresis

Encopresis is defined as the repeated passage of feces into inappropriate places such as clothing or onto the floor, whether involuntary or intentional. There has been at least one such event each month for the past three months, and the individual must be at least four years of age. Mental health professionals will specify whether encopresis is with or without constipation and overflow incontinence (APA, 2022). The child often feels ashamed and may wish to avoid situations, such as going to camp, that might lead to embarrassment. Encopresis is associated with, "a significant decrease in health-related quality of life and family functioning, particularly in older children" (APA, 2022, pg. 404). When the incontinence is clearly deliberate, oppositional defiant disorder or conduct disorder may also be present.

You should have learned the following in this section:

- Enuresis is the repeated voiding of urine into bed or clothes and can be involuntary or intentional. It has occurred at least two times a week for the past three consecutive months and the child must be at least 5 years of age.
- Encopresis is defined as the repeated passage of feces into inappropriate places such as clothing or onto the floor, whether involuntary or intentional. There has been at least one such event each month for the past three months and the individual must be at least four years of age.

Section 6.1 Review Questions

1. What is enuresis?
2. What is encopresis?
3. What are the functional consequences of both elimination disorders?

6.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence of encopresis and enuresis.
- Describe comorbid disorders of encopresis and enuresis.

6.2.1. Enuresis

Daytime incontinence has a prevalence between 3.2% and 9.0% in children aged 7 years, from 1.1.% to 4.2% in youth ages 11-13 years, and from 1.2% to 3.0% in adolescents ages 15-17 years of age. Nocturnal enuresis is more common in males than females.

Most children with enuresis do not have a comorbid mental disorder. That said, the prevalence of comorbid behavioral and developmental symptoms does appear to be higher in children with both diurnal and nocturnal enuresis than those without incontinence.

6.2.2. Encopresis

Most children older than four years of age diagnosed with encopresis have the subtype "with constipation and overflow incontinence." The disorder affects 1% to 4% of children in high-income countries, while in some Asian countries a prevalence rate of 2% to 8% has been reported. It also affects children aged 4-6 years more than children aged 10-12 years. Encopresis is also higher among children who were low-income youth or were abused or neglected early.

The gender ratio appears to be about equal in children younger than 5 years but is more common in boys than in girls among older children.

Enuresis is often reported in children with encopresis, especially in children not presenting with constipation and overflow incontinence.

You should have learned the following in this section:

- Daytime incontinence is more prevalent in children aged 7 years.
- Enuresis is generally more common than encopresis.
- Nocturnal enuresis is more common in males than females.
- The gender ratio for encopresis appears to be about equal in children younger than 5 years but is more common in boys than in girls among older children.
- Enuresis is often reported in children with encopresis.

Section 6.2 Review Questions

1. What are the prevalence rates of encopresis and enuresis?
2. In which gender are encopresis and enuresis most common?
3. What disorders are comorbid with encopresis and enuresis?

6.3. Etiology

Section Learning Objectives

- Describe the biological/genetic basis/causes of elimination disorders.
- Describe environmental/psychosocial causes of elimination disorders.

6.3.1. Biological/Genetic

6.3.1.1. Enuresis. Because medical conditions can explain urinary incontinence, careful consideration for specific bladder conditions or medical conditions that can impact urinary continence (e.g., neurogenic bladder or untreated diabetes) must be made. Additionally, nocturnal enuresis has been associated with a mismatch between nocturnal urine production, nocturnal bladder storage capacity, and the ability to arouse from sleep (APA, 2022).

Finally, there appears to be a heritability factor in enuresis, with children being anywhere from 3.6 (for enuretic mothers) to 10.1 times (for enuretic fathers) more likely to develop enuresis if their parents had enuresis themselves in childhood (APA, 2022; von Gontard et al., 2011).

6.3.1.2. Encopresis. Similar to enuresis, fecal incontinence can be caused by other medical conditions. For example, spina bifida and chronic diarrhea can lead to fecal incontinence. Because of this, a medical examination and/or consideration for specific medical conditions must be considered. This is because encopresis is not diagnosed if incontinence is explained better by a medical condition.

6.3.2. Environmental/Psychological

Ineffective toilet training procedures, or toilet-training procedures that occur later than necessary, may contribute to enuresis and encopresis. Moreover, high levels of stress may also impact these disorders. Additionally, if a child experiences chronic constipation, and experiences painful bowel movements, they may become extremely fearful of defecating and avoid doing so. This perpetuates concerns with encopresis. Thus, they may avoid defecating for long enough that they can physically no longer do so, resulting in accidents. The DSM also notes that for encopresis, "…anxiety, depression, behavioral disorders, psychological stressors (e.g., bullying, poor school performance), and lower socioeconomic status" (APA, 2022, pg. 404) may contribute to the development of fecal incontinence.

You should have learned the following in this section:

- Other medical conditions should be checked when diagnosing enuresis or encopresis.
- Ineffective toilet training procedures, or toilet-training procedures that occur later than necessary, may contribute to enuresis and encopresis.

Section 6.3 Review Questions

1. What are biological or genetic factors for enuresis or encopresis?
2. What are environmental/psychological factors for enuresis or encopresis?

6.4. Assessment and Treatment

Section Learning Objectives

- Describe how elimination disorders are assessed and diagnosed.
- Describe treatment options for elimination disorders.

6.4.1. General Assessment of Elimination Disorders

Similar to feeding-related disorders, assessments focus on parent/caregiver reports during interviews, as well as a thorough medical examination, to rule out medical conditions causing the elimination concerns. Utilizing a voiding diary may help parents recognize when and how frequently accidents occur. They may be asked to note the volume of waste/urine and frequency of accidents in addition to successful occurrences of voiding in the toilet. (Reiner & Kratochvil, 2008).

6.4.2. Treatment of Enuresis

The use of *urine alarm therapy* is helpful. The basic principal behind this therapy is that an alarm activates when moisture is detected. These systems often utilize a pad that is placed on a child's mattress, although some more advanced systems may be incorporated into clothing. For examples of these alarms, you can check out https://www.pottymd.com. The alarm will either vibrate or sound (or both) when it becomes wet. This awakens the child to prompt them to go to the bathroom. The idea is, eventually, behavioral conditioning occurs, and the child slowly

begins to awaken on their own to use the bathroom (Shepard, Poler, & Grabman, 2017). The alarm system is often utilized for several months, typically a minimum of 3 (Reiner & Kratochvil, 2008). This method can be used independently; however, it is often combined with dry bed training as well (Shepard, Poler, & Grabman, 2017).

Dry bed training utilizes several strategies. For example, scheduling wakeup times throughout the night to check for dryness and/or go to the bathroom may be implemented. If an accident occurs, **overcorrection** may be used. Overcorrection requires that a child become responsible for changing sheets, changing clothes, etc. when an accident occurs, rather than the parent doing it for them. Combining dry-bed training with urine alarm therapy is more effective than only utilizing urine alarm therapy (Shepard, Poler, & Grabman, 2017).

Some children may simply hold their urine for so long that accidents occur. This may be especially true for diurnal enuresis. As such, a small watch that reminds a child to go to the bathroom at a set frequency may be used.

6.4.3. Treatment of Encopresis

Treatment for encopresis may incorporate *biofeedback.* The goal is to teach the individual to recognize their own muscular movements by using probes that send signals to the individual physically or visually. This may help the child learn to relax and contract anal muscles, further allowing them to control bowel movements. Another option may include *enhanced toilet training* which teaches and trains individuals to relax and contract muscles through the use of simple strategies such as breathing exercises, relaxation, etc. (Shepard, Poler, & Grabman, 2017).

6.4.4. General Treatment Considerations

For many of the interventions discussed, use of rewards can be very helpful when implementing the behavioral therapies. It is imperative to stress that punishment and shame should not occur when a child has an accident.

Some medicinal interventions may be utilized. For example, desmopressin, imipramine, oxybutynin, tolterodine, and propantheline (Reiner & Kratochvil, 2008) or nortriptyline (Ghanizadeh & Haghighat, 2012) may be useful. However, when medication is withdrawn, chance of encopresis or enuresis reoccurring is high (Ghanizadeh & Haghighat, 2012).

Dietary supplementation may also be helpful and necessary, particularly with encopresis. For example, naturally increasing fiber in a child's diet or introducing polyethylene glycol-3350 (over-the-counter laxative) may prove helpful (Reiner & Kratochvil, 2008).

You should have learned the following in this section:

- Assessments focus on parent/caregiver reports during interviews, a thorough medical examination to rule out medical conditions causing the elimination concerns, and a voiding diary.
- Treatments for enuresis include urine alarm therapy, dry bed training with overcorrection, and using a small watch during the day to remind the child to urinate.
- Treatments for encopresis include biofeedback and enhanced toilet training.
- Rewards (per operant conditioning), medicinal interventions, and dietary supplementation may be helpful.

Section 6.4 Review Questions

1. How does a mental health professional assess for elimination disorders?
2. What treatments are possible for enuresis?
3. What treatments are possible for encopresis?
4. What general strategies can be used to aid with elimination disorders?

Apply Your Knowledge

CASE VIGNETTE

7-year-old Jamar is a healthy boy with no social concerns. He is developmentally on-track in all areas except one – he has never been able to achieve nighttime dryness. He still has to wear pullups at night. He has no difficulty staying dry during the day, and never has bowel accidents (during the day or at night). Jamar wants to have sleepovers like his other friends, and he is becoming increasingly embarrassed that he has to turn down frequent offers by his friends to spend the night.

Annie is a 10-year-old girl that is healthy by all respects and has no significant intellectual impairments or other delays. However, she presented as extremely shy and embarrassed because she has frequent bowel movement accidents. Recently, she even had an accident on a bus ride to a field trip, and this led significant distress for Annie, as her peers were keenly aware of her accident which led to teasing and bullying. She does not experience significant or persistent constipation.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What disorder is Jamar likely to be diagnosed with? What treatment options might his family be offered to try?
2. What disorder is Annie likely to be diagnosed with? What treatment options might her family be offered to try?

Module Recap

In this module, we learned about enuresis and encopresis. We discussed the various symptoms of these elimination disorders. We then discussed the prevalence of elimination disorders and identified potential comorbid disorders. We then looked at the etiology of elimination disorders. Finally, we discussed the process of assessing and treating these disorders. This concludes our discussion of disorders in infancy and early childhood.

Our next module starts our discussion of developmental delays and motor disorders. We will start by reviewing intellectual development disorder (intellectual disability) and specific learning disorder.

Part III. Developmental and Motor-related Disorders

Part III. Developmental and Motor-related Disorders

Module 7:
Intellectual Developmental Disorder & Specific Learning Disorder

Module 7: Intellectual Developmental Disorder & Specific Learning Disorder

Module Overview

In Module 7, we will discuss matters related to intellectual developmental and learning disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include intellectual developmental disorder (intellectual disability) and specific learning 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. Prevalence and Comorbidity

7.3. Etiology

7.4. Assessment

7.5. Treatment

Module Learning Outcomes

- Describe how intellectual developmental disorder and specific learning disorder present.
- Describe the prevalence of intellectual developmental disorder and specific learning disorder.

- Describe the etiology of intellectual developmental disorder and specific learning disorder.
- Describe how intellectual developmental disorder and specific learning disorder are assessed, diagnosed, and treated.

7.1. Clinical Presentation

Section Learning Objectives

- Describe the presentation and associated features of intellectual developmental disorder.
- Describe the presentation and associated features of specific learning disorder.
- Clarify the differences and similarities between intellectual developmental disorder and specific learning disorder.

7.1.1. Intellectual Developmental Disorder (Intellectual Disability)

At the core of an intellectual disability is a deficit in cognitive or intellectual functioning. Historically, we labeled individuals with this presentation of deficits as having mental retardation, but this term was changed to intellectual disability with the passage of Public Law 111-256, also called Rosa's law, to combat stigmatization and misuse of the term. While the terms intellectual disability and intellectual developmental disorder are considered interchangeable, we will use intellectual developmental disorder in this book.

When considering intellectual developmental disorder there are two primary areas of major deficits – intellectual functioning (Criterion A) and adaptive functioning (Criterion B; APA, 2022). See Section 7.1.1.3 for a third criterion.

7.1.1.1. Intellectual functioning (Criterion A). Intellectual or cognitive functioning refers to our ability to problem solve, understand and analyze complex material, think abstractly, absorb information from our environment, learn from experience, plan, judge, and reason. Critical components include working memory, verbal comprehension, quantitative reasoning, cognitive efficacy, and perceptual reasoning. An individual with intellectual developmental disorder has a significant deficit in this area as confirmed by clinical assessment and individualized, standardized, culturally appropriate intelligence testing. The DSM-5-TR states that those with intellectual developmental disorder have scores approximately two standard deviations or more below the population mean. If a test has a standard deviation of 15 and a mean of 100, their scores will fall in the 65-75 range (70 ± 5; APA, 2022).

7.1.1.2. Adaptive functioning (Criterion B). Adaptive skills are those that help us successfully navigate our daily lives. Our ability to understand safety signs in our environment, make appointments, interact with others, complete hygiene routines, etc. are examples of adaptive functioning. These are the skills one needs to live independently and be socially responsible. Individuals with intellectual developmental disorder typically have adaptive skills that are far below what is expected given their chronological age.

According to the DSM-5-TR (APA, 2022) adaptive functioning involves adaptive reasoning in three main domains: conceptual, social, and practical. First, the *conceptual domain* (also called the academic domain) involves competence in memory, language, math reasoning, problem-solving, etc. Second, the *social domain* involves being aware of the thoughts and

feelings of other people, showing empathy, interpersonal communication skills, and social judgment, for example. Finally, the *practical domain* involves learning and self-management across life settings such as job responsibilities, personal care, and recreation. As you will see later, adaptive functioning is typically measured by standardized measures using knowledgeable informants and the individual, if possible.

7.1.1.3. Onset of intellectual developmental disorder (Criterion C). It should be noted that a third criterion must also be met– the onset of deficits described in criteria A and B must be present early in the neurodevelopmental period. As such, it is most frequently diagnosed in children. Intellectual developmental disorder is not something one would "acquire" in adulthood. If an individual experiences cognitive and adaptive function decline in later years, this is not considered intellectual developmental disorder (a neurodevelopmental disorder) but is more likely a neurocognitive disorder that may be due to a number of things such as traumatic brain injury or dementia. As such, although an individual can go undiagnosed until adulthood, and then as an adult be diagnosed with intellectual developmental disorder, there must be significant and undoubtable evidence of cognitive delay and adaptive functioning delay in the early developmental period. Otherwise, an adult would not be diagnosed with intellectual developmental disorder.

7.1.1.4. Severity specifiers. Rather than IQ scores, intellectual developmental disorder is assigned a severity specifier based on the level of delays related to adaptive functioning. Essentially, the more support someone needs, the more severe the intellectual disability. Severity ranges from Mild (least severe), to Moderate, to Severe, and Profound (most severe; APA, 2022). Severity is considered in relation to the three domains of conceptual, social, and practical. For instance, a specifier of severe would result the child having little understanding of written

language or concepts involving numbers, quantity, and money (conceptual domain), speech and communication being focused on the here and now within everyday events (social domain), and not being able to make responsible decisions regarding the well-being of self or others, necessitating supervision at all times (practical domain).

7.1.1.5. Associated features. Individuals with intellectual developmental disorder have difficulties with social judgment, assessing risk, emotions, are gullible, and lack awareness. This can lead to increased rates of accidental injury, being exploited by others, possible victimization or physical and sexual abuse, and unintentional criminal involvement. They may also become distressed about their intellectual limitations (APA, 2022).

7.1.1.6. Clarification on nomenclature. The DSM-5-TR uses the term *intellectual development disorder* to clarify its relationship with the ICD-11 classification system which uses the term *Disorders of Intellectual Development*. The equivalent term of *intellectual disability* is placed in parentheses for continued use. It should be noted that both terms (i.e., intellectual developmental disorder and intellectual disability) are used in the medical and research literature, while the term intellectual disability is more commonly used by educators, advocacy groups, and the lay public.

7.1.2. Specific Learning Disorder

A specific learning disorder is characterized by persistent difficulties learning critical academic skills during the years of formal schooling such as reading of single words accurately and fluently or arithmetic calculation; performance of the affected academic skills being well below expected for age; learning difficulties being apparent in the early school years for most individuals, and that the learning difficulties are considered “specific” for four reasons. First, the

learning difficulties are not better explained by intellectual developmental disorder, global developmental delay, hearing or vision disorders, or neurological or motor disorders. Second, they cannot be attributed to more general external factors such as economic or environmental disadvantage. Third, they cannot be attributed to neurological disorders such as a pediatric stroke, motor disorders, or to vision or hearing disorders. Fourth, a learning difficulty can be restricted to one academic skill or domain.

Historically, learning disorders were diagnosed when there was a significant discrepancy between an individual's intellectual/cognitive ability (as measured by an intelligence test) and their academic achievement (as measured by a standardized achievement test) as this was required by DSM-IV-TR criteria. This method is referred to as the *discrepancy model.* While many still use this model, and nothing in the DSM-5-TR disallows it, the DSM-5 criteria were rewritten to allow more flexibility. Ultimately, a discrepancy between one's IQ and academic achievement is no longer required; however, there must be specific data indicating an individual is preforming significantly below what would be expected given their chronological age.

In addition to significant academic deficits, there must be evidence that efforts (e.g., tutoring, increased and specialized instruction) to improve abilities within the specific area have been made before diagnosing a specific learning disorder. This is to ensure that an individual has had full access to educational material and supports before a professional assigns a learning disorder diagnosis to them. In school systems, this is where tiered interventions have come into play (more on this in Section 7.5).

7.1.2.1. Domain/subskill specific specifiers. Once an individual has been diagnosed with a learning disorder, all academic domains and subskills that have been impaired should be noted as follows:

- **With impairment in reading –** The individual has trouble comprehending material, reading fluently and quickly, or reading words accurately.
- **With impairment in mathematics –** The individual has trouble with number sense, memorization of arithmetic facts, math reasoning, and calculation.
- **With impairment in written expression –** The individual has trouble with accurately spelling words, using correct punctuation and grammar, or with writing clearly and organized.

7.1.2.1. Matters of dyslexia and dyscalculia. Technically, dyslexia and dyscalculia are not diagnoses in the DSM-5-TR, but are alternative terms used to describe learning disorders in reading (dyslexia) and math (dyscalculia). **Dyslexia** refers to a pattern of learning difficulties characterized by problems with accurate or fluent word recognition, decoding, and spelling (APA, 2022). **Dyscalculia** refers to a pattern of learning difficulties characterized by "problems processing numerical information, learning arithmetic facts, and performing accurate or fluent calculations" (APA, 2022, pg. 78).

Although these two terms are used very frequently in school systems and by professionals such as Speech/Language Pathologists, they are not diagnoses and are considered alternative terms in the DSM-5-TR. Instead, a mental health professional will diagnose a s*pecific learning disorder with impairment in reading* (for dyslexia) and a *specific learning disorder with impairment in mathematics (*for dyscalculia). This is an excellent example of how professionals will sometimes discuss the same phenomenon but use different terminology.

7.1.3. Differences and Similarities between the Disorders

Although intellectual developmental disorder and specific learning disorder may seem very similar, it is important not to confuse the two, as they are different. When thinking about both disorders, we have three distinct core areas to consider: adaptive functioning, cognitive/intellectual ability (IQ), and academic achievement. A rudimentarily way to think about this is with intellectual developmental disorder we are concerned with adaptive functioning and IQ, and with specific learning disorder we are concerned with IQ (sort of) and academic achievement. Although IQ matters (sort of) in both disorders, the reason it is important varies slightly. Because IQ is considered in both disorders, people often intertwine and confuse the two.

Let's take a minute and think about this: IQ is what we are cognitively able to do – what we *can* do. Adaptive skills and academic achievement refer to what we *are* doing.

7.1.3.1 Intellectual developmental disorder. If we *cannot* perform in the average range on an IQ test and we *are not* performing daily living tasks appropriately (for our chronological age –we would not expect a 7-year-old to make their own doctor's appointment but would expect them to know to dial 911 in an emergency), then this is indicative of intellectual developmental disorder (intellectual disability).

7.1.3.2. Specific learning disorder. To differentiate between a specific learning disorder and intellectual developmental disorder, it is useful to consider the discrepancy between what is expected of an individual (what they *can* do) and what they *are* doing. If an individual cannot perform averagely because their IQ is substantially below average, we could not expect them to perform at an average level on academic tasks. For example, if a person's IQ is 70 and they cannot function typically on cognitive tasks, we would not expect them to achieve an academic score of 100, a 30-point jump from what they can do to what they are doing. If an individual has

an IQ of 70, we would expect their academic score to be around 70, which would not necessarily indicate a specific learning disorder, even though the score is low. In this case, they would be performing as expected, so the low score achievement score would reflect low cognitive abilities resulting from intellectual development disorder. However, if there is a large discrepancy between what a person can do and what they are doing, for example, someone with an IQ of 100 scoring only a 70 in an academic achievement task, this could indicate a specific learning disorder.

7.1.3.3. Specific learning disorder in the cognitively delayed and in the cognitively gifted. Individuals with extreme cognitive functioning abilities are often overlooked. For example, children that are gifted, but have a reading disorder, often go undiagnosed because their deficits look like average abilities to others. Here is an example to illustrate this:

A 2nd grader with a high cognitive ability earns all As. She excels in math and writing. In fact, she is far past her peers in these areas. She has long learned her multiplication and division facts and is even working on some basic geometry skills. She can write and has been drafting paragraphs with ease and has even started learning to write essays. She loves math and writing, but she dislikes reading. When in class, she reads just like her peers, no more advanced, but right on 2nd grade level expectations. She finds reading to be more difficult, though, and it doesn't come nearly as easy as math and writing. However, because she is on track compared to her peers, her teachers and parents do not recognize any issues – her grades are fine and her school standardized testing is not a problem.

What if you learned that her standardized math and writing scores matched her intellectual ability (meaning her *can do* and *is doing* matched) but her reading score (*is doing*), although average, is well below what would be expected given her IQ (*can do*) and is much

lower than her math and writing scores, despite still being an acceptable score? Would you say she may have a reading disorder? If you said yes, you are right. If you said no, you may be right too. The fact is, this is a gray area. Previous versions of the DSM would have made it easy to diagnose this child with a learning disorder in reading. The DSM-5-TR makes it a bit tougher. If this reading deficit, compared to her own abilities, caused apparent impairment (internal distress, preventing her from advancing in math and writing because her reading abilities were lagging behind her other abilities), one would be inclined to diagnose her with a specific learning disorder in reading. However, one can see how this child could be overlooked and undiagnosed for years.

Now let's reverse the scenario. A 2nd grade girl has a diagnosis of an intellectual developmental disorder (intellectual disability). She struggles in all areas of academics. However, her math abilities are even more behind than her reading and writing. Do you think one could make a case for a specific learning disorder in math? Theoretically, they could. But it takes a lot of careful documentation of intervention attempts (see RTI discussion) and standardized testing that makes it undoubtedly clear that this is true (similar to the above example).

When an individual has an IQ that lands in an extreme (low or high), their weaknesses are often missed. As such, providers and educators must be careful not overlook potential specific learning disorders in these individuals.

You should have learned the following in this section:

- Intellectual developmental disorder is characterized by intellectual functioning (Criterion A) and adaptive functioning (Criterion B) deficits and they must occur during the developmental period.
- Specifiers for intellectual developmental disorder indicate severity – mild, moderate, severe, or profound.
- A specific learning disorder is characterized by persistent difficulties learning critical academic skills during the years of formal schooling such as reading of single words accurately and fluently or arithmetic calculation; performance of the affected academic skills being well below expected for age; learning difficulties being apparent in the early school years for most individuals, and that the learning difficulties are considered "specific" (for four reasons).
- Specifiers for a specific learning disorder indicate if there is impairment in reading, mathematics, or written expression.
- Dyslexia and dyscalculia are not diagnoses in the DSM-5-TR but rather are alternative terms used to describe learning disorders in reading (dyslexia) and math (dyscalculia).

Section 7.1 Review Questions

1. How does intellectual development disorder present and what specifiers are used?
2. Why was the name intellectual development disorder chosen and what other name does it go by currently? What term was used previously and why has it been removed?
3. How does specific learning disorder present and what specifiers are used?
4. How are the two disorders both similar and different from one another?

7.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence and course of intellectual developmental disorder and specific learning disorder.
- Describe comorbid disorders of intellectual developmental disorder and specific learning disorder.

7.2.1. Intellectual Developmental Disorder

Intellectual development disorder occurs in approximately 1% of the overall general population while the global prevalence varies by country and level of development. Prevalence is 16 per 1,000 in middle-income countries but 9 per 1,000 in high-income countries (APA, 2022). Intellectual development disorder is more common in males than females, although sex ratios are inconsistent in the literature (APA, 2022; Einfeld & Emerson, 2008). It is hypothesized that the reason there is a higher occurrence of intellectual development disorder in males is due to general genetic vulnerability, often linked to X chromosome issues that males experience (Harris, 2006). Prevalence is higher in youth than in adults and there are no significant differences between ethnoracial groups.

Onset of intellectual development disorder is in the developmental period, though etiology and severity of brain dysfunction affect exact age and characteristic features at onset. For individuals with more severe intellectual development disorder, delayed motor, language, and social milestones are typical within the first 2 years of life. For individuals with mild

intellectual development disorder, impairments may not be identifiable until school age when problems with academic learning are evident.

Intellectual development disorder is often comorbid with other medical and physical conditions as well as other neurodevelopmental conditions including autism and ADHD. Moreover, depression, bipolar, and anxiety disorders are often comorbid with intellectual development disorder. Impulse-control disorders, major neurocognitive disorder, and stereotypic movement disorder are frequently comorbid (APA, 2022)

7.2.2. Specific Learning Disorder

Specific learning disorder occurs in approximately 5 to 15% of school-age children in Brazil, Northern Ireland, and the United States. It is more common in males than females and suicidal thoughts and behavior were found in U.S. adolescents aged 15 years in public school presenting with poor reading ability (APA, 2022).

Onset, recognition, and diagnosis of specific learning disorder typically occur during the elementary school years as this is when children are required to read, spell, write, and learn mathematics. During early childhood and before the child starts school, there may be warning signs to include delays or deficits in language, problems with rhyming or counting, and issues related to fine motor skills needed for writing. Specific learning disorder is lifelong, though an individual may experience a persistent shifting array of learning difficulties across the lifespan. According to APA (2022), negative functional consequences occur across the lifespan and can include, "…lower academic attainment, higher rates of high school dropout, lower rates of postsecondary education, high levels of psychological distress and poorer overall mental health, higher rates of unemployment and underemployment, and lower incomes" (DSM-5-TR, pg. 84).

The different types of specific learning disorder are comorbid with one another (i.e., impairment in mathematics with reading), other neurodevelopmental disorders (e.g., ADHD, ASD, developmental coordination disorder, and communication disorders), anxiety disorders, behavioral problems, and depressive disorders.

You should have learned the following in this section:

- Intellectual development disorder occurs in approximately 1% of the overall general population while specific learning disorder occurs in approximately 5 to 15% of school-age children.
- Both disorders are more common in males than females.
- Onset of intellectual development disorder is in the developmental period, though etiology and severity of brain dysfunction affect exact age and characteristic features at onset.
- Onset, recognition, and diagnosis of specific learning disorder typically occur during the elementary school years.
- Intellectual development disorder is often comorbid with other medical and physical conditions, other neurodevelopmental conditions including autism and ADHD, and depression, bipolar, and anxiety disorders.
- Specific learning disorder is often comorbid with the different types of specific learning disorder, other neurodevelopmental disorders, developmental coordination disorder, and communication disorders, anxiety disorders, behavioral problems, and depressive disorders.

Section 7.2 Review Questions

1. Are specific learning disorder or intellectual developmental disorder more common and which gender displays it at greater rates?
2. When is the onset of both disorders?
3. What other disorders are intellectual developmental disorder comorbid with?
4. What other disorders are specific learning disorder comorbid with?

7.3. Etiology

Section Learning Objectives

- Describe biological basis/causes of intellectual developmental disorder and specific learning disorder.
- Describe environmental causes of intellectual developmental disorder and specific learning disorder.

7.3.1. Biological

7.3.1.1. Intellectual developmental disorder (intellectual disability). Biological factors heavily influence the development of intellectual developmental disorder. Genetic conditions (Kaufmann, Capone, Carter, & Lieberman, 2008) or brain malformations (Michelson et al., 2011) are important factors. Chromosomal differences and abnormalities, such as Fragile X syndrome and Downs syndrome, are heavily linked to intellectual developmental disorder (Harris, 2006). Additionally, brain and central nervous system malformations such as spina bifida are risk factors and correlated with intellectual developmental disorder (Harris, 2006).

7.3.1.2. Specific learning disorder. Heritability estimates have reached 50% in twin studies (Goldstein, Naglieri, & DeVries, 2011). Individuals are up to 10 times more likely to have specific learning disorder if one of their first relatives has a specific learning disorder as well (Shalev et al., 2001). In general, specific learning disorder development appears to be linked to neurological differences in the brain. However, specific information on the areas of the brain which are most impacted, and the specific central nervous system differences, are not well documented (Goldstein, et al., 2011).

7.3.2. Environmental

7.3.2.1. Intellectual developmental disorder. Risk factors include malnutrition of the mother during pregnancy or medical conditions preventing nutrition absorption of the fetus. Maternal illness or disease, such as diabetes or varicella (chickenpox), during pregnancy increases the risk of intellectual developmental disorder. Fetal exposure to alcohol, drugs, toxins, etc. also impacts potential development of intellectual developmental disorder. Events during labor and delivery or soon after delivery such as infant seizures, traumatic brain injuries, or infections (e.g., herpes simplex, measles, meningitis, malaria, or rubella) are related to the development of intellectual developmental disorder. Other events such as severe social deprivation, abuse, or exposure to high levels of lead or mercury raise the risk.

7.3.2.2. Specific learning disorder. Low birth weight (Aarnoudse-Moens, Weisglas-Kuperus, van Goudoever, & Oosterlaan, 2009) and fetal exposure to nicotine (Piper, Gray, & Birkett, 2012) are risk factors for developing a specific learning disorder. Early educational experiences may also heavily impact an individual's neural development and neural connections (Goldstein, et al., 2011). For example, if an individual does not have proper exposure to educational materials early on, this may negatively impact the neural connections that are established, and thus, lead to a higher risk for developing specific learning disorder.

You should have learned the following in this section:

- Biological factors heavily influence the development of intellectual developmental disorder and include genetic conditions, brain malformations, chromosomal differences, and brain and central nervous system malformations.
- For specific learning disorder, heritability estimates have reached 50% in twin studies and neurological differences in the brain are linked to higher risk.
- Environmental risk factors for intellectual developmental disorder include malnutrition of the mother during pregnancy; medical conditions preventing nutrition absorption of the fetus; maternal illness or disease; fetal exposure to alcohol, drugs, and toxins; and events during labor and delivery or soon after delivery.
- Environmental risk factors for specific learning disorder include low birth weight, fetal exposure to nicotine, and early educational experiences.

Section 7.3 Review Questions

1. What factors lead to a greater risk of developing intellectual developmental disorder?
2. What factors lead to a greater risk of developing specific learning disorder?

7.4. Assessment

Section Learning Objectives

- Describe commonly used assessment tools.

7.4.1. Observations and Interviews

Although observation may be used for assessment of these disorders, it is the least clinically utilized. A school observation may be conducted to ensure other disorders are unnecessary to investigate (for example, ADHD) that may have explained some of the concerns from parents and educators. However, from a diagnostic standpoint, the most helpful information will be objective data (with some supplementation of information from interviews). Interviews are utilized to discover basic developmental information, history of learning skills and academic performance, developmental milestone achievement (e.g., when a child first walked, talked, etc.), and current adaptive functioning skills.

7.4.2. Objective Measures

7.4.2.1. Adaptive measures. We rely on standardized forms to assess individual's adaptive functioning for many reasons. First, I (the first author) cannot begin to tell you how many first-time parents have looked at me and said, "My child isn't doing X, Y, or Z, but she is my first child, so I'm not sure if she should be doing that yet or not." Or, "I didn't realize my (first) child was behind on certain skills until I had his sister, and his sister achieved these skills quicker/earlier than him (or has even passed him on some skills)." This is so common. Many parents are not sure how to compare their child's abilities, because they simply do not have a

comparison to use. Because of this, a simple verbal report of concerns of adaptive functioning is not reliable and always useful. Additionally, it is imperative to obtain information from multiple individuals to determine if the child is *clinically* behind. Objective data allows us to do this reliably and in a standardized manner. This is helpful because adaptive functioning can be very subjective. Common objective measures of adaptive functioning include the Adaptive Behavior Assessment System, 3rd edition (ABAS-3) and the Vineland Adaptive Behavior Scales, 3rd edition (Vineland-3). These are both questionnaires and can be administered to parents as well as teachers.

7.4.2.2. Intellectual tests. Intellectual tests assess an individual's cognitive functioning, also known as intelligence. We obtain an intelligence quotient (IQ) from these tests. IQ tests typically take anywhere between 1 to 1.5 hours. Although abbreviated forms of IQ tests exist, when examining an individual's IQ for the purpose of understanding if cognitive or learning deficits are present, we want to utilize a full IQ battery and not an abbreviated version. Common IQ tests that are used include the Stanford-Binet Intelligence Scales, 5th edition (SB-5) Wechsler Intelligence Scale for Children, 5th edition (WISC-V), Woodcock Johnson Tests of Cognitive Abilities, 4th edition (WJ-IV), and the Kaufman Assessment Battery for Children, 2nd edition, Normative Update (KABC-II NU). Remember, this score tells us when an individual *can* do.

7.4.2.3. Academic achievement tests. Achievement tests assess academic achievement in an individual. We use these, instead of *only* looking at one's grades, because it allows for a more consistent comparison. For example, if we only relied on grades, we would not be able to control for Teacher A's grading or curriculum being more strict than Teacher B's grading or curriculum. Using a standardize assessment gives us not only a more reliable, but also a more valid, assessment of where an individual is functioning, academically. Common tests include the

Wechsler Individual Achievement Test, 3rd edition (WIAT-III), Woodcock Johnson Tests of Achievement, 4th edition (WJ-IV), and Kaufman Test of Educational Achievement, 3rd edition (KTEA-III). Remember, this score tells us what the individual *is* doing.

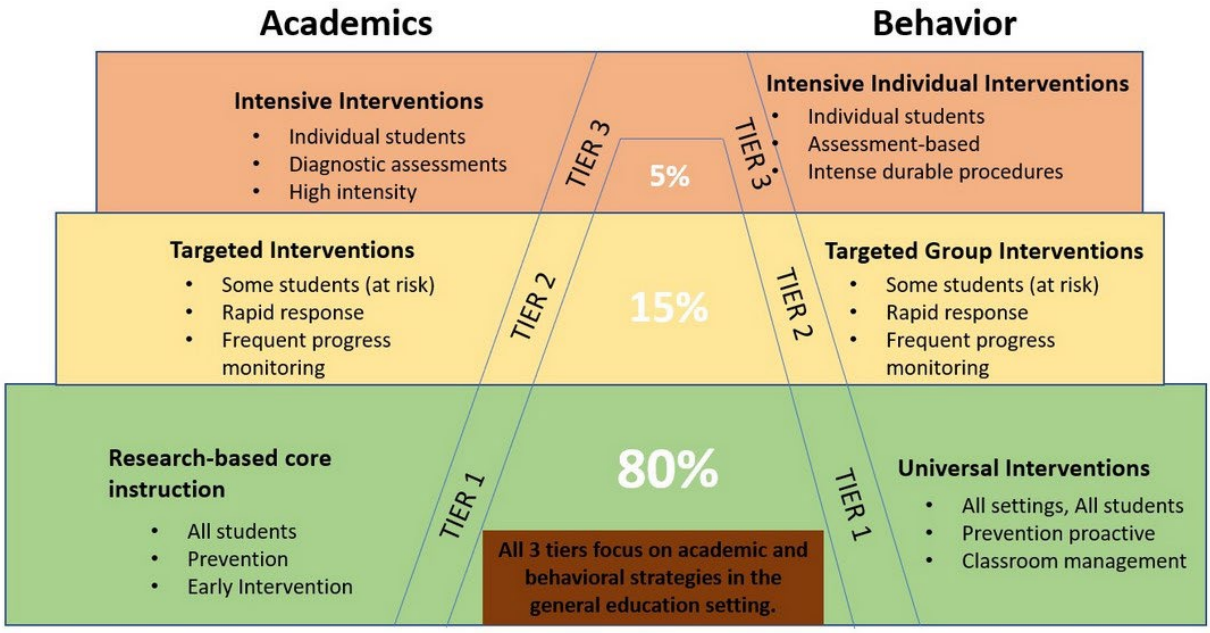
7.4.2.4. Records. Educational records including test grades, report cards, and standardized testing scores are utilized as well, but never on their own. Essentially, while this information is helpful, they cannot be used independently to determine if there is a clinical cognitive or learning deficit.

7.4.3. Response to Intervention (RTI)

Response to Intervention, often referred to as RTI, is a systematic approach to assessing an individual's ability to learn. This occurs in the school setting. The basic, general instruction all students receive is often referred to as Tier 1. If children begin to fall behind in academics in any area, they are identified and placed into a Tier 1 intervention group, or Tier 2 in the case that all students are already considered the Tier 1 group. If their learning and performance is remediated, they are transitioned out of this group. If their learning and performance is not mediated, they are transitioned into a Tier 2 intervention group. Again, if remediated, they transition down or out of tiered programing, and if they are not remediated, they transition to a Tier 3 group. If children are not remediated in a Tier 3 group, this is strong evidence of a learning disorder. Essentially, the child has been provided extensive, intense, and prolonged academic intervention, yet academic deficits are still notable. Tiered interventions involve very targeted interventions to improve academic performance. Tier 1 is the lowest level of intensity with Tier 3 being the highest. Curriculum-based measures of performance are used to screen all students, and then continually used with students who are channeled into the Tiered system. In

Tier 2, small group instruction is typically utilized whereas in Tier 3 one-on-one instruction is commonly used. Figure 7.1 provides a helpful visualization of Tiered programming.

Figure 7.1. Tiered Programming



Note. Image adapted from Livingston Parish Publish Schools: http://lpsb.org/parents/curriculum/r_t_i-_response_to_intervention

You should have learned the following in this section:

- A school observation may be conducted to ensure other disorders do not need to be investigated.
- Objective measures include adaptive measures, intellectual tests, academic achievement tests, and records.
- Responses to intervention (RTI) is a systematic approach to assessing an individual's ability to learn and proceeds through a series of tiers.

Section 7.4 Review Questions

1. Why is observation the least clinically utilized assessment tool?
2. Why are objective measures the most useful assessment tool?
3. What is Response to Intervention and how is it used?

7.5. Treatment

Section Learning Objectives

- Describe treatment options for intellectual developmental disorder and specific learning disorder.

7.5.1. Intellectual Developmental Disorder

7.5.1.1. Community supports and programs. For individuals with intellectual developmental disorder, community supports may be critical during childhood, and even more so as the individual transitions to adulthood. Community supports may include organizations devoted to socialization and family support. For example, The Arc is an incredible organization that is devoted to servicing individuals with developmental delays, including, but not limited to, intellectual developmental disorder. They often engage in advocacy efforts, offer training for the community and professionals, and employment services for individuals with intellectual developmental disorder or other developmental delays. Local chapters will often host social gatherings and events for individuals and their families (The Arc, 2018). Typically, there is an Arc chapter in most major cities and areas. Other community supports may involve government funded programming for living arrangements, supplemental income, etc.

As individuals transition to adulthood, some programming that may need to be considered is home/living arrangements. Historically, individuals with intellectual developmental disorder were often institutionalized. However, in recent years, a strong push to deinstitutionalize care, and provide group and community home options has occurred. As such, a more common and inclusive living option for individuals may be a group home in which multiple individuals

live in a home-like setting with constant supervision, medical care access, and transportation. Another option, often referred to as supported independent living, is a situation in which fewer, such as four, individuals live in an apartment or similar setting, and are provided constant supervision by one individual. This is a less restrictive environment than a group home, as only one supervising staff is present, where nurses and other medical staff are not readily available. Moreover, individuals with intellectual developmental disorder are often capable of successful employment, and these opportunities are provided in group and independent living home arrangements. Individuals with intellectual developmental disorder, depending on the severity of their intellectual impairment, may work in settings with routine tasks (e.g., assembling plasticware packets, bussing tables) in independent settings (e.g., employed independently within the community) or in 'supervised workshops' (i.e., settings where multiple individuals with disabilities are employed and provided significant help and supervision while working).

7.5.1.2. Education. Individuals with intellectual developmental disorder receive an Individualized Education Plan (IEP) at their school, which is federally regulated and implemented at the state level, through the Individuals with Disabilities Education Act (IDEA) established in 2004 (IDEA, n.d.). This was enacted to ensure fair and equal access to public education for all children. An IEP outlines particular accommodations and supports things to which a child is entitled in the educational setting so they are able to access educational material to the fullest degree. Children with intellectual developmental disorder may receive typical academic instruction in an inclusion classroom, meaning they are in a general educational class. However, the more severe the disability, the more supports they may require. As such, this may mean the child is pulled out at periods of time to receive specialized instructions. If the child's disability is severe, they may be placed in a self-contained classroom, which is a class with a

small number of children who also have a severe disability, often with several teachers/teacher aids. Supports and accommodations may include reduced workloads, extended time to master material, increased instructional aid, etc. Additionally, supports may also extend beyond academic specific areas. For example, social skills may be a focus of intervention.

Eventually, a determination about whether or not to place an individual with severe deficits on a diploma track will be made. If an individual is not placed in a diploma track, they will receive a "certificate of completion" from high school, rather than a high school diploma. Non-diploma track supports might focus heavily on functional skills rather than traditional academics. For example, rather than worrying about mastering algebra, the individual's education may focus on learning functional mathematics so that they will be able to successfully manage a grocery shopping trip/purchase.

Some **college programs** have been designed to allow individuals with developmental delays such as intellectual developmental disorder to access the college experience and receive specialized vocational instruction. For example, Mississippi State University's ACCESS program (which is an acronym for Academics, Campus Life, Community Involvement, Employment Opportunities, Socialization, and Self-Awareness) is 4-year, *non-degree* program designed for individuals with a developmental delay, including intellectual developmental disorder. Students live on campus where they participate in the full college experience and receive a "Certification of Completion" within a specific vocational area when they finish the program (MSU, n.d.).

7.5.1.3. Psychotherapy. Although research has demonstrated the benefits of combined use of behavioral and cognitive-behavioral therapies, they are often underutilized in individuals with intellectual developmental disorder (Harris, 2006). Therapy often focuses on the emotional

and behavioral impacts of intellectual developmental disorder, normalizing the individual's experiences, and treating comorbid depression, anxiety, or other mental health conditions (Harris, 2006). Another strong area of focus may be increasing adaptive functioning skills. For example, helping the individual learn and regularly implement daily hygiene, chores, etc. and learning to navigate within their home and community safely and successfully may be a focus of therapy.

7.5.1.4. Medication. Medications to manage emotional or behavioral concerns occurring comorbid with an individual's intellectual developmental disorder diagnosis may be beneficial. For example, if an individual has intellectual developmental disorder and depression, an antidepressant may be beneficial in helping to resolve some symptoms of depression. However, medications are not used to "treat" intellectual developmental disorder.

7.5.2. Specific Learning Disorder

7.5.2.1. Education. Individuals with specific learning disorder receive an Individualized Education Plan (IEP) as well. Focus is placed on increasing instructional aids for the child. The child will often be taken aside for additional, one-on-one interventions in the academic areas of concern. Additionally, the child may receive additional supports such as extended time on tests and assignments, partial credit (when partial credit is not typically given in a particular class), and early access to study guides or access to study guides even if one is not regularly given in a class. A child with a reading impairment may also be allowed to have tests read to them, especially on nonreading-related tests, such as history. The reason for doing this is so that the child's performance in the nonreading-subject (e.g., science, history) is not negatively impacted by their reading deficit. The child may also be able to verbally respond to test items and have a

teacher write their answers. The child may receive opportunities to correct errors on a test for additional credit, etc. These are some examples of accommodations and are not an exhaustive list. The accommodations and supports that are implemented should be specific to the child, their deficits, and their current needs.

Tutoring, whether in school or privately, is often useful as well. This increases exposure to material and provides additional support and intervention. Empirically based tutoring methods are sometimes used, particularly for children with dyslexia.

7.5.2.2. Medication. Like intellectual developmental disorder, medicine is not used to 'treat' specific learning disorder. However, given that ADHD is highly comorbid with specific learning disorder, ADHD-related medications may be beneficially utilized, when this comorbidity is present for a child. Moreover, as chronic underachievement in an academic area may lead to anxiety and depressive states for some children, medicinal intervention (or psychotherapy) may also be helpful.

You should have learned the following in this section:

- Treatment options for intellectual developmental disorder include community supports and programs, educational interventions such as IEPs, and psychotherapy.
- Treatment options for specific learning disorder include educational interventions such as IEPs and tutoring.
- Medicine is not utilized to 'treat' either disorder, but to manage emotional or behavioral concerns that are occurring comorbid with the two disorders.

Section 7.5 Review Questions

1. What treatments exist for intellectual developmental disorder?
2. What treatments exist for specific learning disorder?
3. How is medicine used to treat both disorders?

Apply Your Knowledge

CASE VIGNETTE
Review these two cases:

https://sites.google.com/site/kellyannelare/home/case-study-on-intellectual-disabilities/introducing-janetta

https://ohioemploymentfirst.org/up_doc/Case_Study_Intellectual_Disability_accessible.pdf

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What ways were Janetta and Kesha's experiences similar?
2. What was the most notable take always from either Janetta or Kesha's cases as they relate to our text?
3. What are the key take-aways from how these disorders are addressed in the education setting?

Module Recap

In this module, we learned about intellectual developmental disorder (intellectual disability) and specific learning disorder. We discussed the various symptoms of both disorders and how they relate to the various presentations. We carefully examined the similarities and differences between intellectual developmental disorder and specific learning disorder as well. We then discussed the prevalence of these disorders, frequently comorbid disorders, and their etiology. We ended on a discussion of how intellectual developmental disorder and specific learning disorder are assessed and treated.

Next, we will learn about autism spectrum disorder. This is the second of three chapters in Part III: Developmental and Motor-related Disorders.

Part III. Developmental and Motor-related Disorders

Module 8:
Autism Spectrum Disorder (ASD)

Module 8: Autism Spectrum Disorder

Module Overview

In Module 8, we will discuss matters related to autism spectrum disorder to include its clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include autism spectrum disorder and social (pragmatic) communication disorder. Be sure you refer to 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

- Describe how autism spectrum disorder (ASD) presents.
- Describe the prevalence and comorbidity of ASD.
- Describe the etiology of ASD.
- Describe how ASD is assessed, diagnosed, and treated.

8.1. Clinical Presentation

Section Learning Objectives

- Outline the history of autism spectrum disorder and describe misconceptions.
- Describe the common symptoms and associated features of autism spectrum disorder.
- Describe social (pragmatic) communication disorder.

8.1.1. History of Autism Spectrum Disorder

Autism spectrum disorder (ASD) is a newly added disorder to the DSM-5. In prior versions of the DSM, it was split into two different disorders – Asperger's syndrome and autistic disorder. For autistic disorder, the child had to present with developmental, cognitive, and language delays, while for Asperger's syndrome, this was not needed. The restrictive behaviors of autistic disorder that presented as motor movements most often presented as restrictive and circumscribed interests in Asperger's syndrome (APA, 2000).

Historically, these two disorders were differentiated by developmental history. However, research indicated that distinguishing between a child with high functioning autism and Asperger's syndrome was difficult (Barahona-Correa & Filipe, 2015; Happe, 2011). With the publication of the DSM-5 in 2013, the disorders were combined into one spectrum disorder – autism spectrum disorder (ASD; APA, 2013; Barahona-Correa & Filipe, 2015). Individuals that were previously diagnosed with Asperger's syndrome or autistic disorder (as well as pervasive developmental disorder not otherwise specified) were 'grandfathered' into the diagnosis of ASD.

8.1.2. Clinical Presentation of Autism Spectrum Disorder

The essential features of ASD are twofold and include persistent impairment in reciprocal social communication and social interaction (Criterion A) and restricted, repetitive patterns of behavior, interests, or activities (Criterion B). It is described as a spectrum because of the varying manifestations of the disorder due to severity of the autistic condition, developmental level, chronological level, and gender (APA, 2022).

8.1.2.1. Criterion A: Social communication and social interaction. The child must present with all of the following symptoms. These include behaviors such as poor eye contact, dominating a conversation/or lacking ability to maintain conversation due to absent or limited **reciprocity** (i.e., "to and fro" conversation in the exchange of information), trouble with integrating verbal communication with nonverbal communication (e.g., for example using gestures, body language, or visual guiding), and struggles with maintaining friendships or relationships in general. Individuals with these difficulties have trouble understanding others' perspectives, reading emotions, and inferring minor and subtle social cues.

8.1.2.2. Criterion B: Restricted and repetitive behaviors, interests, or activities. The child must present with at least two of the following. First, they engage in stereotyped movement, frequently called **stereotypy**, such as hand flapping, spinning, or any repetitive movement that does not have an obvious function. They might display **restricted or repetitive play** such as lining up toys or fixating on a part of a toy or button. Individuals may have excessive and **restricted interests** such as being overly interested in history, dinosaurs, robotics, etc. Their interests may be so intense that, if discussing their interests, it is difficult to get them to move on to a new topic, or it may be the only thing about which they will engage in a conversation.

Restricted behaviors might include strict adherence to routines or schedules. Individuals may become very dysregulated if there is a new routine introduced or their routine is changed in any way. Moreover, **sensory concerns** may be of particular relevance. Some individuals may seek out sensory stimulation (sensory seeking behavior which is often referred to as "*stimming*") as a soothing method. In fact, many repetitive movements can be seen as sensory stimulating. Examples, although not an exhaustive list, of "stimming" may include rubbing hands on a rough material repetitively (tactile), putting objects on their mouth/lips (oral), grunting or making nonfunctional vocalizations (vocal), or looking at lights or visual lines in odd ways (visual). Others may avoid certain sensory input which is known as a *sensory aversion*. For example, being very averse to certain textures, heightened sensitivity to pitch or volume of noise, or bright lights.

8.1.2.3. Diagnosing ASD. Individuals must exhibit both social communication concerns *and* restricted/repetitive behaviors/interests/activities to be diagnosed with ASD. Furthermore, these symptoms must have been present very early in development (APA, 2022).

Although language delays are common in children with ASD, they are not necessarily required. Moreover, there is often a misconception that if a child has severe ASD, they are nonverbal, meaning they do not have language. This is not accurate. Children with ASD present very differently from each other. Regarding ASD, Dr. Stephen Shore said, "If you've met one person with autism, you've met one person with autism." In other words, one child with ASD may have symptoms that present very differently from another child with ASD, but both children have ASD.

8.1.2.4. Specifiers for ASD. ASD is diagnosed within the context of *language development and intellectual development* given that these factors may indicate prognosis. As

such, clinicians will assign specifiers that identify if there is any evidence of language impairment or intellectual impairment. For intellectual impairment, separate estimates of verbal and nonverbal skill are necessary. For language impairment, the clinician should consider receptive and expressive language skills separately since receptive may lag behind expressive language development in ASD.

Moreover, ASD is diagnosed in the context of current *severity* for both social communication impairments and restricted, repetitive patterns of behavior, interests, or activities. The clinician will assign a severity level to each of the two main criteria indicating how much support the individual needs. Level 3 indicates "Requiring very substantial support," Level 2 means "Requiring substantial support, and Level 1 states, "Requiring support."

8.1.2.5. Associated features. Intellectual and/or language impairment is common in many individuals with ASD. They may have difficulty seeing the world from another person's perspective, called a *theory-of-mind deficit*. Motor deficits are also present and can include odd gait, being clumsy, and walking on tiptoes. Self-injury such as banging one's head or biting one's wrist are also common.

8.1.2.6. Development and course. The behavioral features of ASD first become noticeable in early childhood as some children will present with a lack of interest in social interaction during their first year of life. For children in which skills have been lost, parents and other caregivers may provide a history showing gradual or relatively rapid deterioration in social behaviors or language skills occurring between 12 and 24 months.

In terms of symptoms, the first symptoms of ASD typically involve delayed language development, often accompanied by lack of social interest or unusual social interactions, odd play patterns such as carrying a toy around but never playing with it, and unusual communication

patterns such as knowing the alphabet but not responding when their name is said. During the second year, odd and repetitive behaviors, and the absence of typical play become more apparent (APA, 2022).

8.1.3. Social (Pragmatic) Communication Disorder

Social (pragmatic) communication disorder is similar to autism spectrum disorder in that social communication, whether verbal or nonverbal, is impacted. Pragmatics refers to the social use of language and communication. To receive a diagnosis, all of the following must be present: 1) problems with using communication for social purposes such as greeting or exchanging information, 2) difficulty with changing communication to match context or needs of the listener such as recognizing that one speaks softer in a classroom but louder at a football game, 3) difficultly following the rules for conversation or storytelling such as understanding that individuals engaged in a conversation take turns speaking, and 4) problems understanding what is not explicitly stated and nonliteral or ambiguous meanings of language such as idioms, humor, or metaphors.

Although, in many ways this may seem very similar to ASD, one of the biggest differences is that restricted or repetitive behaviors/interests are not present. It should be noted that, as individuals with ASD age, restricted/repetitive behaviors tend to decline. If this occurs, but the individual had a history of the restricted/repetitive behaviors, they are still diagnosed with ASD, even if those behaviors are not currently present, rather than social (pragmatic) communication disorder.

8.1.3.1. Development and course. Social (pragmatic) communication disorder is not typically diagnosed in children under four years of age. By age 4 or 5 years, most children have

obtained adequate speech and language capabilities to allow for the identification of specific deficits in social communication, though milder forms of the disorder may go undetected until early adolescence, when language and social interactions become more complex.

Making Sense of the Disorders

- Autism spectrum disorder is distinguished from social (pragmatic) communication disorder by the presence in ASD of restricted/repetitive patterns of behavior, interests, or activities

…..

- and their absence in social (pragmatic) communication disorder.

Note to the Reader: Social (pragmatic) communication disorder was presented in this module only so that it can be distinguished from ASD in terms of a differential diagnosis. It will not be discussed going further, and to be candid, the DSM does not really have much more to say about it either.

You should have learned the following in this section:

- The current autism spectrum disorder was previously separated into two disorders – autistic disorder and Asperger's syndrome – before DSM-5.
- The essential features of ASD are twofold and include persistent impairment in reciprocal social communication and social interaction (Criterion A) and restricted, repetitive patterns of behavior, interests, or activities (Criterion B).
- Social (pragmatic) communication disorder is characterized by difficulty with pragmatics or the social use of language and communication.
- The two disorders are distinguished by the presence of restricted/repetitive patterns of behavior, interests, and activities in ASD but not social (pragmatic) communication disorder.

Section 8.1 Review Questions

1. Has there always been an autism spectrum disorder?
2. What are the essential features of ASD?
3. What specifiers are used with ASD?
4. When are the symptoms of ASD first noticed and what are they?
5. How is an individual diagnosed with social (pragmatic) communication disorder?
6. What distinguishes the two disorders discussed in this module?

8.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence of ASD.
- Describe common disorders that are comorbid with ASD.

8.2.1. Prevalence of ASD

ASD has been reported in 1% to 2% of the U.S. population. Prevalence is lower among African American (1.1%) and Latinx (0.8%) children compared to Caucasian children (1.3%). In non-U.S. countries prevalence has approached 1% of the population. The male:female ratio globally is 3:1. Individuals with ASD are at greater risk for suicide death and children with ASD who had impaired social communication had a higher risk for self-harm with suicidal intent, suicidal thoughts, and suicide plans by age 16 compared to those without the impaired social communication (APA, 2022).

8.2.2. Comorbidity of ASD

Comorbid disorders are very common for children with autism. Specifically, 70% of children with autism have a comorbid diagnosis. Further, 40% of children with autism have two or more additional disorders. ASD is comorbid with intellectual developmental disorder and language disorder, specific learning difficulties, anxiety disorders, depression, ADHD, and avoidant/restrictive food intake disorder.

You should have learned the following in this section:

- ASD has been reported in 1% to 2% of the U.S. population.
- ASD is 3 to 4 times more common in males than females.
- ASD is comorbid with intellectual developmental disorder and language disorder, specific learning difficulties, anxiety disorders, depression, ADHD, and avoidant/restrictive food intake disorder.

Section 8.2 Review Questions

1. What percentage of the U.S. population has ASD?
2. How does this prevalence rate compare to other countries?
3. What disorders are comorbid with ASD?
4. What percentage of people with ASD have one comorbid disorder? What percentage have two or more?

8.3. Etiology

Section Learning Objectives

- Describe biological bases/causes of ASD.

It is largely considered that there is a strong interaction effect of environment and biology/genetics that lead to the development of autism. These causes will be discussed below.

8.3.1. Biological Basis

8.3.1.1. Brain structure/neurological risk. Studies have most consistently shown that children with ASD have atypical brain size/overgrowth of brain structures. Also, differences specifically related to amygdala functioning have been noted in children with ASD. Additionally, underactivity in the temporal lobe when engaging in a face perception test has been noted (Volkmar & Wiesner, 2017).

8.3.1.2. Family/genetic risk. Twin studies are often used to help understand genetic vulnerability of disorders. It is thought that there is a very strong genetic component to autism, but the roots of that are unknown. For example, research has indicated a 56% to 95% heritability of autism in twin studies; moreover, monozygotic twins (i.e., identical twins; developed from one embryo) displayed higher correlates than dizygotic twins (i.e., fraternal twins; developed from two different embryos; Colvert, Tick, McEwen, et al., 2015). Essentially, monozygotic twins which share more DNA makeup, evidenced stronger heritability estimates indicating likely genetic predispositions. However, as much as researchers have tried, they have not pinpointed a specific genetic marker that accounts for autism or predispositions of autism.

Spontaneous gene mutations may also be related to autism. According to the DSM-5-TR (APA, 2022) as many as 15% of cases of ASD appear to be associated with a known genetic mutation. Children that have a sibling with ASD have higher risk for later being diagnosed with ASD (CDC, 2018, May).

In terms of parental factors, older parental age and complicated childbirth are associated with a higher risk for developing ASD. However, no singular parental factor could predict autism in a study by Gardener, Spielgeman, & Buka (2009). The DSM-5-TR (APA, 2022) also suggests that extreme prematurity and in utero exposure to certain drugs or teratogens such as valproic acid can be causes of ASD.

8.3.1.3. Vaccines. For a long time, there was a large misconception that vaccines, particularly the MMR vaccine, caused ASD. Since that time, we have learned that is not true. To understand this, we must first understand the background of the misconception. The idea that vaccines caused autism began around the late 90s/early 2000s when a team of researchers, headed by Andrew Wakefield, published a study that did not causally link, but indicated a relation, between MMR vaccines and autism. Statements about the study were grossly overgeneralized and summarized, and bias in the funding of the study was also revealed. Parts of the paper were criticized as being incorrect and, overall, the study presented an ethical concern (e.g., cherry-picking data, misleading statements about the data). The study was later retracted, and Andrew Wakefield lost his license (Rao & Andrade, 2011). However, by the time this was discovered, the media and society had grown to believe that vaccines caused autism. Researchers have continued to study this extensively and continue to find that there is no relation between autism and vaccines (Dudley, et. Al, 2018; Uno, Uchiyama, Aleksic, & Ozaki, 2015; Taylor, Swerdfeger, & Eslick, 2014) to no avail. The general public still believes that vaccines cause

autism (Sheikh, Swetlik, & Wilson, 2018). Although this list listed under etiology, **vaccines do not cause autism, according to research, and as such are not considered an etiological pathway to autism.**

You should have learned the following in this section:

- In terms of biological causes of ASD, brain size/overgrowth of brain structures such as amygdala are an issue, as well as underactivity in the temporal lobe.
- Research has indicated a 56% to 95% heritability of autism in twin studies and monozygotic twins have higher correlates than dizygotic twins
- Spontaneous gene mutations may also be related to autism.
- It is a misconception that vaccines cause autism, as scientific research continues to show.

Section 8.3 Review Questions

1. What brain structures have been implicated as causes of ASD?
2. Is autism heritable? How do we know?
3. What is the role of vaccines in ASD?

8.4. Assessment

Section Learning Objectives

- Describe assessment tools commonly used to assess ASD.

When assessing for ASD, psychologists often rely heavily on observations. They also rely on parent-report, particularly for early development. Because children are often diagnosed very young, teacher-reports may not be relevant if they are not yet enrolled in preschool. Often, self-report is not used. However, if the child is older, and is higher functioning or will have a delayed diagnosis, self-report will certainly be obtained. To obtain parent and teacher reports (and when appropriate, self-report) of symptoms, a psychologist often utilizes two methods: an interview and objective measures. For *behavioral observations*, a psychologist will observe the child, in person, either in their office and/or at school. The observation is a bit more formal than observations of other disorders. A good assessment will include information from all three areas (i.e., observation, interview, and objective measures) to make an informed diagnostic decision.

Unfortunately, there is a serious issue in screening and diagnosing children with ASD. We have improved our ability to accurately diagnosis, but we are still far from meeting appropriate screening efforts. For example, only 17.2% of children in the state of Mississippi are regularly screened for developmental milestone achievement in a standardized way. In Oregon, the state with the best screening rate, is still only 58.8% (Hirai, Kogan, Kandasamy, Reuland, & Bethell, 2018). Because early detection of ASD is imperative given its implications for treatment prognosis, these numbers are startling.

8.4.1. Observations

One of the gold standard assessment tools to diagnose ASD is the ADOS-2 (Lord, Rutter, et al., 2012). This stands for the Autism Diagnostic Observation Schedule, Second Edition. The ADOS-2 is administered by a clinician directly with the child (or adult). It can be administered to children as young as 12 months and through adulthood. It consists of 5 modules (Toddler, Module 1, 2, 3, and 4). The age and verbal abilities of the child determines which module is to be used. The Toddler Module and Module 1 allow for a caregiver to also be in the room. Module 2 allows for a caregiver to be present, if needed. Modules 3 and 4 are ideally conducted without a caregiver in the room. The ADOS-2 is comprised of a series of activities that the examiner completes with the child or adult. The activities are designed to elicit certain interactions and behaviors and allows the clinician to assess those abilities. For example, we may want to see if the child points, or do they notice certain interactions in the room, etc. At the end of the administration, the clinician scores the interactions by utilizing a detailed scoring protocol which results in one final score. This score will classify how likely a child is to meet diagnostic criteria.

8.4.2. Interview

In general, a comprehensive clinical interview will be conducted with parents. An attempt to understand the child's current abilities, history of development and milestone progression, and current symptoms will be obtained. Although this is often done in an unstructured interview, the Autism Diagnostic Interview, Revised ADI-R (Rutter, LeCouteur, & Lord, 2003) was designed to thoroughly assess for developmental traits and related symptoms of autism. It is a structured interview that allows the clinician to thoroughly screen all relevant areas and results in a final score to use to indicate the likelihood of autism. While this is often considered to be the second

tool (with the ADOS-2 being the primary tool) to a gold-standard assessment of autism, it is often not utilized due to the extensive time it takes. To complete just the ADI-R alone, it can take approximately 90 to 150 minutes. Keep in mind, this would be on top of completing an ADOS-2 and any other objective measures and interviews a clinician requires. Because of limited resources and the extensive time the ADI-R takes, though an excellent tool, it is not as frequently utilized as the ADOS-2.

8.4.3. Objective Measures

Other standardized measures are often utilized to include the following:

- Autism Spectrum Rating Scales (Goldstein & Naglieri, 2010; ASRS). This tool includes several items that address various symptoms and behaviors related to autism such as repetitive behaviors, sensory concerns, communication skills, etc. It can be used for children as young as 2 and up to age 18 years old.
- The Social Responsiveness Scale (Constantino & Gruber, 2012; SRS) is similar to the ASRS but focuses more on the social aspects and social impacts of symptoms and behaviors.
- The Sensory Profile (Dunn, 2014; SR-2) may be included to understand in more detail various sensory experiences a child has.
- The Gilliam Autism Rating Scale: 3rd Edition (Gilliam, 2014; GARS) is commonly used to screen but should not be used exclusively to diagnose a child. This is a very helpful tool to understand how likely autism is in a child.
- The Modified Checklist for Autism in Toddlers, Revised with Follow-Up (Robins, Fein, & Barton, 2009; M-CHAT R/F) are excellent screening tools as well. They are most

helpful when used in a primary care office and are quick to administer and easy to score. This helps physicians recognize if a child should be referred to a psychologist for a more in-depth evaluation.

8.4.4. Medical Screening

Because language delay is one of the key features in children with very early signs of ASD, careful medical screening is also important. Although occasional ear infections (medically referred to as otitis media) is not particularly concerning, frequent or undetected ear infections that involve fluid buildup in their ears (medically referred to as effusion), may lead to some hearing impairments (O'Conner, Coggins, Gagnon, Rosenfeld, Shin, & Walsh, 2016). If a child is not able to fully hear properly due to muffling difficulties the fluid causes, the child may be at higher risk of experiencing a language delay or oddities in their speech development (Roberts, Hunter, & Gravel, et al., 2004; O'Conner, et al., 2016). Thus, hearing loss leading to language delays would be due to a medical explanation rather than a developmental delay related to autism. As such, it is important when assessing a child to ensure that their hearing has been screened. Hearing screenings can be done in extremely young children by specialized providers, often a pediatric ear, nose, and throat (pediatric ENT) specialist. Other related medical screenings may include assessment of neurological deficits that impaired gross or fine motor movement.

You should have learned the following in this section:

- When assessing for ASD, psychologists often rely heavily on observations. They also rely on parent-report, particularly for early development.
- A comprehensive clinical interview will be conducted with parents. An attempt to understand the child's current abilities, history of development and milestone progression, and current symptoms will be obtained.
- One of the gold standard assessment tools to diagnose ASD is the ADOS-2.
- Hearing loss leading to language delays would be due to a medical explanation rather than a developmental delay related to autism. As such, it is important when assessing a child to ensure that their hearing has been screened.

Section 8.4 Review Questions

1. What assessment measures are used to assess ASD?
2. Why does hearing loss have to be assessed?

8.5 Treatment

Section Learning Objectives

- Describe treatment options for autism.

8.5.1 Behavioral Interventions and Educational Supports

8.5.1.1. Early intervention. In recent years, the term *early intervention* has been used more loosely, but typically speaking, this intervention includes significant behavioral intervention (i.e., applied behavioral intervention), often conducted in the home, but may also include ancillary interventions such as parent training, speech, physical and occupational therapy, etc. Therapy often starts in the home but, as the child progresses, interventions transition to other settings, such as school, in the community, or in outpatient clinics. Interventions are intensive and include several hours a week (e.g., for some children 30-40 hours a week). Services typically start around age 3 or 4 and last for about 2 years (Reichow, 2012).

Early intensive behavioral intervention (EIBI) is one of the most effective, evidenced-based treatment options for children with ASD. Although there are some variations in this finding, an overwhelming amount of research indicates significant benefits from EIBI that is not matched by other interventions (Reichow, 2012; Eldevik, Hastings, Hughes, et al.; Makrygianni & Reed, 2010). Early intervention is considered to be potentially most beneficial because the younger our brains are, the more plasticity they have. **Plasticity** is the ability for our brain to modify its neural connections. As such, we are able to grow and change our brain connections and structures more easily when we are younger than when we are older. Therefore, the therapies and interventions applied in early development may most impactful.

8.5.1.2. Applied behavioral analysis. The most critical component of any treatment for autism is often considered to be Applied Behavioral Analysis (ABA). You most often hear people reference this therapy by its acronym, ABA. ABA is a large component of EIBI. ABA can take place in a child's home, which is common when early intervention is applied, as well as school and in outpatient clinics. Many children receive a large number of their ABA services in the outpatient clinic as well, because receiving early intervention services with in-home ABA is difficult to obtain in most states. For example, the first author of this text is a psychologist in south Mississippi and can attest to the fact that very few children get enough ABA services in early intervention programs in that area. As such, most families supplement or forgo any state-funded services and seek outpatient services for their children. With private services, children receive many more ABA hours, but unfortunately, a majority of it has to happen in the outpatient clinic setting. That is, unless families decide to self-pay when insurance will not cover in-home or in-school services (Note: In-school services are often offered by a school, but at a much lower number of hours than what a family may desire/require), these services happen in a clinic or office setting. Theoretically, ABA is applicable beyond treatment for autism; however, insurance companies will only pay for ABA (if they cover ABA at all) if the child is diagnosed with ASD. So, although ABA is not exclusive to autism, as far as receiving treatment covered through insurance, it is exclusive to autism.

So, what is ABA exactly? ABA is essentially the practice of changing behavior by understanding the function or absence of a behavior and manipulating components of the individual's environment or motivation to change that behavior. A therapist will assess what happens before the behavior (antecedent), what the behavior actually is (behavior), and what happens after the behavior (consequence). These pieces of information help a therapist

understand why a behavior occurs and what potentially maintains it. For example, many behaviors are maintained because a child wants to obtain *attention*, obtain a *tangible good*, or escape an *undesired task*. For example, if a child throws a tantrum every time his mother leaves a room, it may be that he is throwing a tantrum because he has been denied her attention or because he knows he will be expected to do work when she leaves and wants to escape an undesired task. The behavioral therapist's job is to assess the function of the behavior, and then manipulate that. The therapist will likely build in rewards to help motivate a behavioral change. For example, if the function is to escape an undesired task, they could work for 10 minutes and earn a 3-minute iPad break. Or, if the function is to gain the mother's attention, they could work for 10 minutes and then play with mom for 3 minutes. This is a basic example to illustrate ABA. A component that sets ABA apart is that nearly everything is tracked in ABA, where each behavior is monitored and noted. This results in incredible graphs and data that is used to inform treatment and planning for future goals and sessions.

Task Analysis in very generic terms is when we take a task and break it down to the smallest task possible. For example, when we discuss putting on a shirt, we might generally say you pick the shirt up and put the shirt on, but a task analysis would go into more detail. You might say Step 1 is to pick up the shirt by the bottom, Step 2 is to put your left arm in the left arm hole, Step 3 is to push your arm all the way through until you see your hand, and so on. After a therapist has identified the analysis of a task, they will use *chaining.* Chaining can occur from the start of a task and move through the task (forward chaining) or at the end and work backward (backward chaining). Essentially, each step of the task is achieved, and then the next step, forward or backward, is achieved until the entire task (e.g., putting on a shirt on) is accomplished independently.

Discrete trail training is also common in ABA and may also be used to help achieve chaining and task success with behaviors that are currently absent. For example, language use may be a primary goal for some children, initially. Essentially, a therapist presents a behavior (e.g., models/requests), waits for a child to display the desired behavior, then responds (typically with a reward for a successful behavioral trial), and then waits for a moment before moving on to the next trial. These happen relatively quickly, and again, each behavior is recorded (Anderson et al., 1996).

The only people qualified to fully implement ABA are BCBAs (or BCBA) or RBTs. A BCBA is a Board-Certified Behavioral Analyst that typically has at least a master's degree or higher. A BCBA is a Board Certified Assistant Behavioral Analyst that has at least a 4-year degree. An RBT is a Registered Behavior Technician that has at least a high school diploma. Typically speaking, RBTs implement a bulk of therapy and are supervised by BCBAs. BCBAs, typically handle initial appointments with clients, create treatment plans, analyze data of clients, and supervise RBTs and the interventions they are implementing with children. Each state regulates their own process and requirements to achieve formal licensure with these titles.

Other strategies that can be helpful in ABA and for parents are the use of timers, warnings, social stories, and visual schedules. Warnings and timers help children that struggle with transitions to prepare for an upcoming transition. Social stories are pictorial representations of a series of events that will occur in a situation. For example, going to a dentist office may be a perfect opportunity to use a social story with a child. The story should include all the steps involved (from getting in the car to checking out at the desk at the end of the visit). A visual schedule is a pictorial schedule that provides expectation about upcoming transitions.

8.5.1.3. Developmental preschools. Children with autism can access a developmental preschool. This access typically allows them to enter school prior to kindergarten and receive a variety of services. The services are not typically academic oriented, rather, they are focused on various therapies, support, and social-emotional development. Children might receive some academic instruction, occupational or physical therapy, speech/language therapy, hearing/vision services, or other necessary interventions. These services are offered through the public-school system, and children with an IEP, per the Individuals with Disabilities Education Act (IDEA), are able to access these settings.

8.5.1.4. Speech therapy. Children with speech delays benefit from enrolling in speech therapy with a speech/language pathologist (SLP). Typically, children will attend 30-minute sessions one to two times per week. While our conversation about speech therapy will be brief, it is important to note that many children that do not develop language, or those who still struggle to develop functional language, will end up using either the PECS system or an augmented communication device, which is a fancy way of saying a tablet with particular programming that helps facilitate communication. While there are differences in the two, it is important to understand that these are alternative ways for someone to communicate. The biggest difference, in very basic terms, is that the PECS system is 'hard copy' in which a child takes a picture and moves it on a surface, whereas an augmented communication device implements a similar concept, except the child presses the picture on a screen. Though this is a brief explanation, the important thing to know is that when children do not develop language, they may be fitted with an alternative way to communicate that heavily relies on pictures.

8.5.1.5. Occupational/physical therapy. Gross and fine motor delays are common in children with autism. As such, many children will work with an occupational therapist (OT) or

physical therapist (PT) to improve motor skills. The individual they work with likely will depend on their deficits. For example, an OT will tend to focus more on fine motor skills (e.g., holding an eating utensil, pinching finger food) whereas a PT will focus more on gross motor skills (e.g., walking, throwing a ball). At times, children with significant sensory symptoms benefit from working with an OT to reduce sensory concerns as well.

8.5.1.6. Social skills training. Some children benefit from social skills training in addition to their other therapies. Typically speaking, these therapies occur in group settings with children elementary school age and older. Social skills groups focus on teaching very basic social skills and then having the group members practice. At times, groups will have volunteer peers that are typically developing participate in the group. This is to allow the children that are working on social skills to practice the skills they are learning with typically developed peers.

8.5.1.7. Psychotherapy. Older children may begin to develop insight that they are perceived differently than their peers. They may desire friendships but find it difficult to develop them due to social skill deficits, and feel frustrated by their behaviors and symptoms. As a result, internal distress may develop due to their experiences related to autism. As such, psychotherapy to address associated anxiety, depression, or general distress may be helpful. Further, research indicates empirical benefits for cognitive-behavioral therapy (in the individual or group setting; Sizoo & Kuiper, 2017 and McGillivray & Evert, 2014, respectively; CBT will be discussed further in future chapters) and well as mindfulness (process of focusing on the moment and fully appreciating it; Sizoo & Kuiper, 2017).

8.5.1.8. Family support. Parent support groups are often beneficial for families. Additionally, parent training is often a large component of treatment to help parents employ similar concepts that are being used in ABA. Moreover, siblings of children with ABA may need

some explanation about some of their siblings' behaviors. For example, a child may ask, "Why doesn't my sister want to talk to me?" This can be confusing to siblings, particularly young ones. Children's books to help facilitate this can be helpful for parents. Moreover, Sesame Street also came out with an episode, "Meet Julia" that does a nice job of helping children understand autism.

8.5.1.9. Psychopharmacological. Psychotropic medications do not "treat" autism, but some medications may help with associated features or common comorbid disorders. For example, children with autism may be likely to experience high anxiety. In this case, anti-anxiety medications may be beneficial. Moreover, stimulants to help reduce hyperactivity, either due to ASD or comorbid ADHD may also be helpful. Additionally, antidepressants may be helpful in decreasing repetitive behaviors and may also help taper irritability and tantrums. Anti-psychotic medications may decrease irritability, hyperactivity, stereotyped behavior, and aggression (NICHD, 2017, January).

You should have learned the following in this section:

- Treatment approaches include early intensive behavioral intervention, ABA, developmental preschools, speech therapy, occupational and physical therapy, social skills training, psychotherapy, and family support.
- Psychotropic medications do not "treat" autism, but some medications may help with associated features or common comorbid disorders.

Section 8.5 Review Questions

1. Describe the various treatment approaches used for ASD.
2. How are psychotropic medications used to "treat" autism?

Apply Your Knowledge

CASE VIGNETTES

Howard is 7 years old and presents at his GP with his mother as she is concerned about his challenging behavior in school. He is very noncompliant and has hit staff and pupils. Howard had early language delay but now uses fluent sentences. His school reports indicate that he has moderately impaired intellectual ability with above average reading skills and a marked failure to develop any peer relationships. His parents report that his language is stereotyped and repetitive and that he repeats videos and DVDs. He is very limited in terms initiating social communication and has a restricted pattern of interests, currently an over-focus on DVDs. He has stereotyped repetitive motor mannerisms and seeks to feel people‟s clothes. Howard does use eye gaze, facial expression and gesture but is an infrequent initiator of communication. Howard shows some appropriate responses to other people‟s emotions but also often shows an odd response, for example smiles if distress shown. He is unconcerned about modulating behavior according to the social context and has some fixed routines, for example reading through all the notices at the swimming pool every time.

Susan is a 15 year old girl referred by the GP because of poor school attendance and low mood. At assessment, Susan says that she has been feeling sad most of the time for 6 months. It takes her 4 hours to get to sleep and she feels tired all day. Her appetite has gone down and she has lost about a stone. She thinks she is stupid and ugly. She is finding it hard to concentrate on her schoolwork. She does not want to go to school because she is worried other students will make fun of her. However, she manages to go shopping without problems. She does not want to be dead, and hopes that life will get better. Susan says that she has always been bullied at school and that people have always called her „Oddball“. People at school laugh at the way she speaks and make fun of what she says. They tease her because she has a big collection of dolls and dolls houses and likes to talk about it a lot. She has never had a true friend. She would like to have friends but never knows how to act around people. Susan’s parents confirm that Susan has never had friends. She did not have any interest in other children before she started school and just wanted to play dolls. has never had friends. She did not have any interest in other children before she started school and just wanted to play dolls. There was no imaginative play with the dolls – she just liked collecting them and lining them up. When she started at school she was happy to just wander around on her own. From the age of 10 she started saying that she wished she had friends, but never talked about friends she had and never wanted to invite other children round. She has always spent a lot of time doing her schoolwork and has always been top of her year, which her parents are very proud of. Susan has always liked to keep to herself at home and has never been that bothered by what other family members have wanted to do, and has

Apply Your Knowledge Continued

never shown concern towards other family members. Susan started talking before her 1st birthday. Her speech has always been flat without variation. She would sometimes speak at length about her dolls, which she has always been obsessed with. She has never shown good eye contact and would never point, wave or clap as a child.

Vignettes taken directly from NICE and replicated here.

National Collaborating Centre for Women's and Children's Health (UK). (2011). Autism: Recognition, Referral and Diagnosis of Children and Young People on the Autism Spectrum. NICE Clinical Guidelines, No. 128. Londong: RCOG Press. Available from: https://www.ncbi.nlm.nih.gov/books/NBK92985/

Direct PDF of full document can be accessed at: https://www.nice.org.uk/guidance/cg128/resources/clinical-case-scenarios-pdf-183180493.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What symptoms of ASD do you notice for Howard? Are there symptoms that are inconsistent for Howard? Are there things about Howard's presentation that would lead to more or less of a likelihood that Howard is diagnosed with ASD?
2. What symptoms of ASD do you notice for Susan? Are there symptoms that are inconsistent for Susan? Are there things about Susan's presentation that would lead to more or less of a likelihood that Susan is diagnosed with ASD?
3. What procedures would you like to see followed to assess Howard? Susan?
4. What treatments might be a good fit for Howard? Susan? Why?

Module Recap

In this module, we learned about autism spectrum disorder. We discussed the various behaviors and symptoms of ASD and how they relate to the various presentations. Then we discussed the prevalence of ASD and frequently comorbid disorders. We also learned about the etiology of ASD. We ended on a discussion of how ASD is assessed and treated.

In our next module, we will discuss motor disorders such as Tourette's and Stereotypic Movement Disorder.

Part III. Developmental and Motor-related Disorders

Module 9:
Motor-Related Disorders

Module 9: Motor-Related Disorders

Module Overview

In Module 9, we will discuss matters related to motor-related disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include stereotypic movement and tic disorders. Be sure you refer to 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. Prevalence and Comorbidity
- 9.3. Etiology
- 9.4. Assessment and Treatment

Module Learning Outcomes

- Describe how stereotypic movement disorder and tic disorders present.
- Describe the prevalence of stereotypic movement disorder and tic disorders.
- Describe the etiology of stereotypic movement disorder and tic disorders.
- Describe how stereotypic movement disorder and tic disorders are assessed, diagnosed, and treated.

9.1. Clinical Presentation

Section Learning Objectives

- Describe the presentation and associated features of stereotypic movement disorder.
- Describe the presentation and associated features of tic disorders.

9.1.1. Stereotypic Movement Disorder

Stereotypic movement disorder involves "repetitive, seemingly driven, and apparently purposeless motor behavior" such as hand flapping, body rocking, or hitting one's own body (APA, 2022, pg. 89). Whether efforts to stop such movements are successful is questionable. For children that are developing typically, directing attention to the movements, or distracting them from performing the movements, are successful. For children with neurodevelopmental disorders such efforts are less successful. What behavior is displayed varies, but each child has his or her own signature or individually patterned behavior. Duration of the behavior is a few seconds to several minutes or longer while the frequency can vary from many occurrences during a single day to several weeks between episodes. The behaviors can occur during moments of boredom, excitement, stress, fatigue, or when the child is engrossed in other activities.

The stereotypic movements cause impairment in social, academic, or other activities and can result in self-injury. In fact, the mental health professional should specify if stereotypic movement disorder is with or without self-injurious behavior and what the severity of the disorder is in terms of mild, moderate, or severe. A mild presentation is one in which the stereotypic movement is easily suppressed by a sensory stimulus or distraction. Severe would be characterized by continuous movements that interfere with daily living. The onset of the disorder

is during the early developmental period, typically within the first three years of life (APA, 2022).

9.1.2. Tic Disorders

Tic disorders consist of three separate diagnoses. The most commonly known of the three is Tourette's disorder. The second diagnosis is persistent (chronic) motor or vocal tic disorder. The third is provisional tic disorder. We will start by discussing the tic disorders generally and then move to specifics of each.

9.1.2.1. General symptoms. These disorders present before adulthood (i.e., 18 years old). Typically speaking, the time in which they present is between the ages of 4 and 6 and are most severe in symptomology between the ages of 10 and 12.

For all three disorders, tics are present. The DSM defines a tic as, "a sudden, rapid, recurrent, nonrhythmic motor movement or vocalization (APA, 2022, pg. 93). Tics can be either *motor movements* (motor) or *vocalizations* (vocal). These can be *simple*, meaning they only involve one movement or vocalization, or they can be *complex*, meaning they involve multiple movements, vocalizations, or a combination of movements and vocalizations within the same tic. Tics are largely considered to be involuntary. It is common for tics to increase in severity for a period of time and then resolve or drastically reduce for a period of time (APA, 2022).

9.1.2.2. Tourette's disorder. Tourette's disorder occurs when *both* motor and vocal tics are present. More than one motor tic must be present and at least one vocal tic must occur (APA, 2022) to be classified as Tourette's disorder. The tics do not have to occur together and do not have to be complex tics. Though tics may wax and wane in frequency, they should have persisted for more than one year since first tic onset.

9.1.2.3. Persistent (chronic) motor or vocal tic disorder. This is when *either* one or more motor tic *or* one or more vocal tic is present. However, vocal and motor tics are *not* both present (APA, 2022). These again can be simple or complex and only the presence of one tic is required. To receive this diagnosis, the individual must never have been diagnosed with Tourette's disorder. The mental health professional will specify if with motor tics only or with vocal tics only and though tics may wax and wane in frequency, they should have persisted for more than one year since first tic onset.

9.1.2.4. Provisional tic disorder. This diagnosis is used if there are single or multiple motor and/or vocal tics but they have been present for less than one year since first tic onset. The criteria for Tourette's disorder or persistent (chronic) motor or vocal tic disorder have never been met.

Making Sense of the Disorders

In relation to motor disorders, note the following:

- Diagnose **Tourette's disorder** if there are more than one motor and at least one vocal tic (both types are present) and this condition has persisted for more than one year.
- Diagnose **persistent (chronic) motor or vocal tic disorder** if one or more motor or vocal tics are present (only one type is present) and this condition has persisted for more than one year.
- Diagnose **provisional tic disorder** if there is a single or multiple motor and/or vocal tic(s) (some type of tic is present) but the condition has been present for less than one year.

You should have learned the following in this section:

- Stereotypic movement disorder involves repetitive, seemingly driven, and apparently purposeless motor behavior such as hand flapping, body rocking, or hitting one's own body.
- According to the DSM, a tic is "a sudden, rapid, recurrent, nonrhythmic motor movement or vocalization." Tics can be either *motor movements* (motor) or *vocalizations* (vocal) and can be simple or complex.
- Diagnose Tourette's disorder if there are more than one motor and at least one vocal tic (both types are present) and this condition has persisted for more than one year.
- Diagnose Persistent (chronic) motor or vocal tic disorder if one or more motor or vocal tics are present (only one type is present) and this condition has persisted for more than one year.
- Diagnose provisional tic disorder if there is a single or multiple motor and/or vocal tic(s) (some type of tic is present) but the condition has been present for less than one year.

Section 9.1 Review Questions

1. What is stereotypic movement disorder? What specifiers are used with it?
2. What is a tic?
3. What are the three tic disorders and what makes them different from one another?

9.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence of stereotypic movement disorder and tic disorders.
- Describe common disorders that are comorbid with stereotypic movement disorder and tic disorders.

9.2.1. Stereotypic Movement Disorder

Stereotypic movement disorder is relatively rare, occurring in only about 3 to 4% of the general population. However, in individuals with intellectual developmental disorder (intellectual disability), the prevalence rate is higher and occurs in about 4 to 16% of individuals. The risk is greater in individuals with severe intellectual developmental disorder.

Stereotypic movement disorder is often comorbid with other genetic and biologically based disorders such fragile X syndrome, Rett syndrome, Lesch-Nyhan syndrome, as well as other related conditions (Oliver, Petty, Ruddick, & Bacarese-Hamilton, 2012). Additionally, the disorder is comorbid with ADHD, motor coordination problems, tics/Tourette's disorder, and anxiety (APA, 2022).

9.2.1.1. Differential diagnosis: autism spectrum disorder. Although the repetitive movements of stereotypic movement disorder may be reminiscent of behaviors seen in children with autism and may develop in the same developmental timeframe (e.g., around or before age 3) as autism spectrum disorder, children with stereotypic movement disorder do not display social communication and reciprocity deficits. Moreover, they do not have circumscribed interests, difficulty with transitions, delayed speech, etc. Essentially, the only symptoms related to autism

spectrum disorder that are displayed is the stereotypy related to behavior. As the DSM says, "When autism spectrum disorder is present, stereotypic movement disorder is diagnosed only when there is self-injury or when the stereotypic behaviors are sufficiently severe to become a focus of treatment" (APA, 2022, pg. 91).

9.2.1.2. Differential diagnosis: tic disorders. It may seem that distinguishing between a tic and stereotyped movement would be hard. However, there are a few factors that help differentiate the two. For example, tics tend to be variable, meaning they change over time. Stereotyped movements, although they may change, tend to be more fixed and consistent. Moreover, stereotyped movements related to stereotypic movement disorder tend to present earlier in development (before age 3 years) than tics related to tic disorders do (mean age at onset 4-6 years). Finally, tics typically are quick, brief, and fleeting whereas stereotypic movements tend to be more prolonged and repetitive (APA, 2022).

9.2.1.3. Differential diagnosis: OCD. Professionals can distinguish between obsessive-compulsive disorder and stereotypic movement disorder due to the absence of obsessions in the latter. As the DSM says, "In OCD the individual feels driven to perform repetitive behaviors in response to an obsession or according to rules that must be applied rigidly, whereas in stereotypic movement disorder the behaviors are seemingly driven but apparently purposeless" (APA, 2022, pg. 92).

OCD-related disorders of trichotillomania (e.g., pulling hair) and excoriation (e.g., picking at skin) may seem difficult to differentiate. However, in trichotillomania and excoriation, there tends to be a purpose for the behavior (e.g., extreme anxiety) and the behavior is not typically patterned or displayed in a rhythmic way whereas in stereotypic movement disorder,

the behaviors are more patterned or rhythmic. And stereotypic movement disorder has an earlier onset than OCD-related disorders which tends to be around puberty or later (APA, 2022).

9.2.2. Tic Disorders

Tic disorders are relatively rare. The CDC estimates that approximately 0.3% of children are diagnosed with Tourette's disorder (CDC, 2018) while the DSM reports an estimated 3 per 1,000 for the prevalence of clinically identified cases (APA, 2022). Tic disorders are more common in males and there are lower rates in African American and Latinx individuals. Epidemiological studies have shown tics to occur in children from all continents (APA, 2022).

Comorbid disorders are common, with 86% of individuals with Tourette's disorder also having another psychological disorder (CDC, 2018, October). ADHD, disruptive behavior, and OCD are commonly comorbid with tic disorders (APA, 2022) with about two-thirds of individuals with Tourette's disorder also having ADHD and one-third having OCD (CDC, 2018, October).

In terms of differential diagnosis, tic disorders must be distinguished from OCD and related disorders. The compulsions of OCD attempt to prevent or reduce anxiety or distress and are usually performed in response to an obsession while individuals with a tic disorder feel the need to perform the action in a particular fashion, equally on both sides of the body a specific number of times, or until a "just right" feeling is achieved. As for body-focused repetitive behavior disorders, they tend to be more goal directed and complex than tics (APA, 2022).

You should have learned the following in this section:

- Stereotypic movement disorder is relatively rare, occurring in only about 3 to 4% of the general population while tic disorders are even more rare, occurring in less than 1% of the general population.
- Stereotypic movement disorder is often comorbid with ADHD, motor coordination problems, tics/Tourette's disorder, and anxiety, but should be distinguished from ASD, tic disorders, and OCD.
- ADHD, disruptive behavior, and OCD are commonly comorbid with tic disorders and it should be distinguished from ADHD.

Section 9.2 Review Questions

1. How prevalent are stereotypic movement disorder and tic disorders?
2. What disorders are comorbid with stereotypic movement disorder and tic disorders?
3. What other disorders should be distinguished from stereotypic movement disorder?
4. How are tic disorders different from OCD?

9.3. Etiology

Section Learning Objectives

- Describe biological basis/causes of motor disorders.
- Describe environmental causes of motor disorders.

9.3.1. Biological

9.3.1.1. Stereotypic movement disorder. Individuals with lower cognitive functioning are at higher risk for stereotypic movement disorder and have poorer response to interventions. Some medical conditions and genetic syndromes such as Lesch-Nyhan syndrome or Rett syndrome are at higher risk for stereotypies (APA, 2022). Little is known about the genetic and biological vulnerabilities that lead to stereotypic movement disorder (Zinner & Mink, 2010) though some evidence suggests the disorder to be somewhat heritable due to the high frequency of cases that have a positive family history of motor stereotypies (APA, 2022).

9.3.1.2. Tic disorders. There is a strong heritability component for tic disorders. For example, research indicates there is up to a 50% chance that a parent with a genetic vulnerability for Tourette's disorder will pass the genetic susceptibility on to their child (CDC, 2018, April) and the DSM estimates that the heritability of tic disorder is 70%-85% (APA, 2022). Additionally, it is theorized that tics may be triggered due to atypicalities in an individual's ability to breakdown dopamine (CDC, 2018, April).

9.3.2. Environmental

9.3.2.1 Stereotypic movement disorder. For stereotypic movement disorder, social isolation and lack of nurturing may lead an individual to attempt to self-stimulate and repetitive, stereotyped behaviors may develop (APA, 2022). Thus, similar to what was discussed with rumination disorder, stereotypic movements may be automatically reinforced due to the internal stimulation it provides (Ricketts, 2013). Environmental stress may trigger stereotypic behavior and fear may alter the physiological state which results in increased frequency of stereotypic behaviors.

9.3.2.2. Tic disorders. In tic disorders, individuals may actually mimic others' behaviors (not as a way to mock them, but as a result of their disorder). Moreover, stressors may exacerbate symptoms of tics. As such, high levels of stress in an environment, increased excitement, or high levels of worry may lead to a higher frequency and intensity of tics. Some research also indicates that children whose mothers smoked while pregnant or experienced significant complications during pregnancy may have a higher risk for developing tics. Low birth weight might also be a risk factor for developing tics. Finally, some infections have been associated with later development of tics in children (CDC, 2018, April) and advanced parental age is a risk factor (APA, 2022).

You should have learned the following in this section:

- Biological risk factors for stereotypic movement disorder include lower cognitive functioning and some medical conditions and genetic syndromes while for tic disorders there is a strong genetic component.
- Environmental risk factors for stereotypic movement disorder include social isolation, lack of nurturing, and environmental stress while for tic disorders advanced parental age and complications during pregnancy are important to consider.

Section 9.3 Review Questions

1. What are key risk factors for stereotypic movement disorder?
2. What are key risk factors for tic disorders?

9.4. Assessment and Treatment

Section Learning Objectives

- Describe how motor disorders are assessed and diagnosed.
- Describe treatment options for motor disorders.

9.4.1. General Assessment for Motor Disorders

Assessing for a motor disorder is done with observation and interviewing. Information is gathered about frequency, context, and severity, as well as the presence of voluntary versus involuntary movements. Some tools may be used to rule out other disorders, such as measures to screen/assess for autism spectrum disorder and OCD-related concerns. Additionally, medical assessments may be conducted to ensure that behaviors are not better captured by a medical condition.

9.4.2. Treatment of Stereotypic Movement Disorder

9.4.2.1. Behavioral therapy. Less research has been conducted on treating stereotypic movement disorder. The most commonly researched and used intervention is behavioral therapy. Specifically, differential reinforcement or habit reversal therapy (described in CBITS intervention below) with modifications has shown promise (Rinker, 2013). When utilizing differential reinforcement, the specific method is often differential reinforcement of other behaviors (DRO) which is when delivery of a reinforcer is contingent on the absence of an undesirable behavior (a tic) for some period.

9.4.2.2. Psychopharmacology. Medications including fluoxetine (a selective serotonin reuptake inhibitor), clomipramine (a tricyclic antidepressant), and risperidone (an atypical neuroleptic) have been noted to positively impact repetitive behaviors in children with autism spectrum disorder, and thus, are used with children diagnosed with stereotypic movement disorder at times. However, there is no evidence that these medications are empirically efficacious and beneficial in reducing stereotypy in children with stereotypic movement disorder (Zinner & Mink, 2010).

9.4.3. Treatment of Tic Disorders

9.4.3.1. Psychotherapy. Comprehensive Behavioral Intervention for Tics (CBIT, Woods et al., 2008) is considered the most established and efficacious treatment for tics. The treatment utilizes habit reversal training which includes increasing awareness and then introducing an incompatible response to a behavior. CBIT treatment includes the core components of (1) increasing the individual's awareness of tics, (2) establishing competing behaviors to use when an urge or tic begins, (3) increasing relaxation strategies, and (4) making changes to reduce situations and events that increase tics. As such, the treatment starts with *awareness training*. This includes having a child fully describe and understand each tic as well as identify different areas in the body the individual may feel "urges" in just before a tic. Next, the clinician and child will come up with *competing responses* to use when an urge to tic occurs, rendering engaging of the tic difficult or even impossible. An example of a competing response may be to clench one's jaw and press their lips together for a tic that involves licking one's lips. Finally, attempts to help an individual relax as well as reduce situations in which their tics increase (e.g., high stress, change in routines, etc.) are then focused on (Woods et al., 2008).

9.4.3.2. Psychopharmacology. Commonly prescribed medications include older classes of antipsychotics known as typical neuroleptics, newer classes of antipsychotics, known as atypical neuroleptics, and alpha-2-adrenergic agonists. Often, medical professionals will prescribe alpha-2-adrenergic agonists as a first step in medicinal intervention. It should be noted that it can take a few months before medication shows any notable improvement in tics. The next option may include atypical neuroleptics. Finally, as a last resort, a typical neuroleptic may be utilized. However, there are serious negative side-effects with these medications - some of which are not reversible. As such, these are used infrequently and with caution (Zinner & Mink, 2010).

It should be noted that tic disorders are frequently comorbid with ADHD, and thus, the treatment of ADHD with medicine must be considered carefully. This is because the medications that are typically used to treat ADHD (i.e., stimulants), have a potential to have negative impacts on tics, partially due to the impact the stimulant medicine may have on dopamine. As such, professionals and families may choose to medicate ADHD with non-stimulant medicines if tics are also present. One non-stimulant option, alpha-2-adrenergic agonists, can be used to treat both ADHD *and* tics (Zinner & Mink, 2010).

You should have learned the following in this section:

- Assessing for a motor disorder is done with observation and interviewing and information is gathered about frequency, context, and severity, as well as the presence of voluntary versus involuntary movements.
- Behavior therapy and psychopharmacology are used to treat stereotypic movement disorder.
- Comprehensive Behavioral Intervention for Tics (CBIT) and psychopharmacology are used to treat tic disorders.

Section 9.4 Review Questions

1. How are motor disorders assessed?
2. What methods are used to treat motor disorders?

Apply Your Knowledge

CASE VIGNETTE

Amir, a 6-year-old boy, was brought to a psychologist because his parents are concerned with some of his behaviors. Amir appears very energetic upon entering the psychologist's office. He has trouble sitting still, but otherwise is compliant and socially interactive with the psychologist. Amir's parents report that he has a history of repetitive throat clearing. They explained that he also says the same words over and over and has done this since he was a young toddler. Amir also often grabs his crotch area. This behavior is particularly concerning to his parents due to the social implications of such behavior. His parents report that these behaviors increase when Amir is nervous. Amir has a hard time explaining why he engages in these behaviors but states he feels better after he does them. However, when the psychologist asked if it feels sort of like an itch or a sneeze, Amir shook his head quickly to communicate an emphatic "yes" to the psychologist.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What disorder(s) may you consider for Amir? Do you think there may be a need to consider non-motor related disorders? Do you need more information? If so what information and how can you get that information?
2. Do you think Amir's behaviors are somewhat typical? How do decipher typical from abnormal/atypical?
3. Do you think Amir is at risk for social impairments due to his behaviors?
4. What treatments may be beneficial for Amir?
5. What is Amir's likely trajectory?

Module Recap

In this module, we learned about stereotypic movement disorder and tic disorders. We discussed the various symptoms of motor disorders. We then discussed the prevalence of motor disorders and examined potential comorbid disorders. We then looked at the etiology of motor disorders. Finally, we discussed the process of assessing and treating these disorders.

This concludes our discussion of developmental delays and motor disorders. In our next part we will begin a discussion of behavior-related disorders, starting with attention-deficit/hyperactivity disorder.

3rd edition as of December 2022

Part IV. Behavior-related Disorders

Part IV. Behavior-Related Disorders

Module 10:
Attention-Deficit/Hyperactivity Disorder

Module 10: Attention-Deficit/Hyperactivity Disorder (ADHD)

Module Overview

In Module 10, we will discuss matters related to attention deficit/hyperactivity disorder, commonly referred to as ADHD, to include its clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Be sure you refer to 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. Prevalence and Comorbidity
- 10.3. Etiology
- 10.4. Assessment
- 10.5. Treatment

Module Learning Outcomes

- Describe how ADHD presents.
- Describe the prevalence and comorbidity of ADHD.
- Describe the etiology of ADHD.
- Describe how ADHD is assessed, diagnosed, and treated.

10.1. Clinical Presentation

Section Learning Objectives

- Provide a history of ADHD and clarify misconceptions.
- Describe common symptoms and associated features of ADHD.
- Outline the development and course of ADHD.

10.1.1. Brief Overview of the Recent History of ADHD

In previous editions of the DSM, a child could be diagnosed with either attention deficit disorder (ADD) or attention-deficit/hyperactivity disorder (ADHD). More recently, mental health practitioners have come to understand that both ADD, and ADHD are, in fact, the same phenomenon that simply present a little differently. ADD presents with more inattentive and distractibility symptoms whereas ADHD presents with more impulsive and hyperactive symptoms. While the symptoms seem different, mental health professionals realized they were related to a similar etiological and psychological phenomenon. Because of this, both disorders were combined into one disorder, ADHD, with specifications on the type: 1) Predominantly inattentive type, 2) Predominately hyperactive/impulsive type, and 3) Combined type. Since that time, we have come to understand ADHD even better, leading to a revision of the diagnostic criteria of the disorder.

With the publication of the DSM-5 (APA, 2013), and with its update, the DSM-5-TR (APA, 2022), the umbrella term ADHD was still used. However, instead of *types*, we now use the term "*presentations*." This change occurred because it was realized that, although someone may exhibit more hyperactive/impulsive symptoms at one point in life (e.g., childhood), at a later

point (e.g., adulthood), they may come to exhibit more inattentive/distractibility symptoms. With the use of *types*, the impression was that this change could not occur because the type was stagnant and stable. Use of the term *presentations* allows for a better understanding that symptom presentation can be fluid and change. Thus, we now have *one* disorder, ADHD, with three *presentations* (predominantly inattentive, predominantly hyperactive/impulsive, and combined). With some context now established in terms of how our understanding of attention-deficit/hyperactivity disorder has progressed over time, we now will examine its clinical presentation.

10.1.2. Clinical Presentation of Attention-Deficit/Hyperactivity Disorder

ADHD is a disorder in which individuals have difficulty with **executive functioning**, an individual's decision-making ability, which involves working memory, inhibition of inappropriate or unhelpful responses, and the ability to focus on relevant information while dismissing unimportant or irrelevant information (Barkley, 2015). Essentially, an individual's ability to regulate their cognitions, emotions, and behaviors, are impaired. Individuals may lose things frequently, talk excessively, forget assignments/appointments, fidget frequently, move constantly, get distracted, and struggle with organization. Symptoms also are required to be present in more than one setting. For example, if symptoms are only present at school, an individual would not be diagnosed with ADHD.

Symptoms are generally organized into two main categories: hyperactivity and impulsivity symptoms and inattention symptoms. An individual receives a diagnosis of ADHD with the needed specifier if there are: 1) at least six symptoms of inattention for this specifier, 2) at least six symptoms of hyperactivity and impulsivity for this specifier, or 3) six of each of the

preceding two for the combined specifier (APA, 2022). The exact symptoms an individual may experience are described below.

10.1.2.1. Inattentive symptoms (A1). Children who are inattentive tend to lose things necessary for tasks or activities, do not listen when spoken to directly, do not follow through on instructions and fail to finish tasks, do not give close attention to details or make careless mistakes, and are easily distracted by extraneous stimuli. They also are forgetful in daily activities; avoids, dislikes, or are reluctant to partake of activities requiring sustained mental effort; fail to sustain attention in tasks or play activities; and have problems organizing tasks and activities. Of these 9 symptoms, 6 or more must be displayed to receive the specifier and should have occurred for the past six months (APA, 2022).

10.1.2.2. Hyperactivity and impulsivity symptoms (A2). These symptoms are related to excessive energy and movement as well as impulsivity. Individuals with these symptoms are often described as high energy or "on the go," talkative, and fidgety. These children may have a hard time waiting their turn, standing still, remaining in their seat, or engaging in leisure activities quietly. They blurt out answers before the question has been completed and often interrupt or intrude on others. Of these 9 symptoms, 6 or more must be displayed to receive the specifier and should have occurred for the past six months (APA, 2022).

10.1.2.3. Combined presentation. To receive the combined presentation specifier, an individual must display at least six inattention symptoms and six hyperactivity and impulsivity symptoms for the past six months (APA, 2022).

10.1.2.4. Associated features. Individuals with ADHD (any specifier) display emotional dysregulation or emotional impulsivity, are often quick to anger, easily frustrated, and overreact

emotionally. They may exhibit neurocognitive deficits and often show delays in language, motor, or social development.

10.1.2.5. Development and course. Excessive motor activity is often observed by parents when the child is a toddler but, "…symptoms are difficult to distinguish from highly variable normative behavior before age 4 years" (APA, 2022, pg. 71). In preschool, hyperactivity is most common. ADHD is most often identified during the elementary school years as the inattention becomes more prominent and impairing. Hyperactivity becomes less prominent during adolescence and is confined to being fidgety, restless, or impatient. In adulthood, impulsivity, inattention, and restlessness may remain problematic, even if hyperactivity has diminished.

You should have learned the following in this section:

- In earlier editions of the DSM, what we call ADHD today was either ADD or ADHD.
- We now have one disorder, ADHD, with three *presentations* (predominantly inattentive, predominantly hyperactive/impulsive, and combined).
- Symptoms of ADHD are generally organized into two main categories: hyperactivity and impulsivity symptoms and inattention symptoms.
- To receive the combined presentation specifier, an individual must display at least six inattention symptoms and six hyperactivity and impulsivity symptoms for the past six months.
- ADHD is most often identified during the elementary school years as the inattention becomes more prominent and impairing.

Section 10.1 Review Questions

1. Describe the path to arrive at one disorder (ADHD) with three presentations.
2. What symptoms are included in the inattention category?
3. What symptoms are included in the hyperactive and impulsive category?
4. When is ADHD most often identified and diagnosed and why?

10.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence of ADHD.
- Describe common disorders that are comorbid with ADHD.
- Described disorders with similar presentations that must be differentiated from ADHD when diagnosing ADHD.

10.2.1. Prevalence of ADHD

ADHD occurs in about 7.2% of children worldwide, with cross-national prevalence rates ranging from 0.1% to 10.2% of children and adolescents and higher prevalence rates occurring in special populations, such as foster children and correctional settings. ADHD is more often diagnosed in males than in females. Females are more likely to present primarily with inattentive features. Because predominantly inattentive symptoms are not as disruptive and noticeable as hyperactive/impulsive symptoms, predominantly inattentive presentations of ADHD may go undiagnosed or be diagnosed much later than ADHD predominantly hyperactive/impulsive or combined presentations. Finally, ADHD is a risk factor for suicidal ideation and behavior in children (APA, 2022).

10.2.2. Comorbidity of ADHD

According to the CDC, approximately 60% of children with ADHD have another comorbid disorder (CDC, 2018a, September). About 50% of children with ADHD combined presentation, and about 25% of children with ADHD predominantly inattentive presentation, are

also diagnosed with Oppositional Defiant Disorder (ODD). Conduct Disorder is also highly comorbid with ADHD (i.e., about 25% of youth with ADHD combined presentation) and most children and adolescents with disruptive mood dysregulation disorder have symptoms that also meet the criteria for ADHD (APA, 2022).

Learning disorders are also commonly comorbid with ADHD. However, differentiating if a learning disorder is present, in addition to ADHD, requires thorough evaluation (see differential discussion below).

Mood and anxiety disorders, as well as OCD and intermittent explosive disorder, are less likely to be comorbid than other behavioral disorders (CDC, 2018a, September). However, they do occur at a higher rate in children with ADHD compared to children without ADHD (APA, 2022). Many individuals with ADHD report daytime sleepiness that could meet criteria for hypersomnolence disorder.

10.2.3. Differential Diagnosis

10.2.3.1. ODD. Because inattention and impulsivity can lead to noncompliance with rules, psychologists need to carefully assess behaviors and differentiate between ADHD and ODD. For example, a child may be told to clean their room. A child with ADHD may (1) not hear or fully attend to the instruction and then not comply or (2) may hear the instruction, begin to clean their room, get distracted mid-way, and start playing with a toy they found, perhaps impulsively, while they are supposed to be cleaning. Although *noncompliant* with the command, they are not actively being *defiant*. A child with ODD may be told to clean their room, and rather than comply, may actively defy the command. Because symptoms of ADHD can lead to a higher risk of noncompliance, we must be careful to not misperceive noncompliance with defiance.

However, both can occur together (see comorbidity section above), and as such, when both are present, both will be diagnosed.

10.2.3.2. Anxiety. Anxiety can lead to difficulty with concentration, fidgeting, and distractibility, which overlap with some symptoms of ADHD. It is not uncommon for a child to be referred for concerns related to ADHD, especially ADHD predominantly inattentive presentation, but may, in fact, be experiencing anxiety instead. Bear in mind that the inattention in anxiety disorders may be attributable to worry and rumination. Differential diagnosis of anxiety versus ADHD is important because treatment for the two disorders is different.

10.2.3.3. Learning disorders. Because symptoms of ADHD can impair school performance and learning, psychologists must differentiate between (1) general impairment in learning due to inattentive, impulsiveness, etc. or (2) a specific impairment in an identified learning area (i.e., math, reading, written expression).

10.2.3.4. Intermittent explosive disorder. Both disorders share high levels of impulsive behavior, but serious aggression toward others is common with intermittent explosive disorder and not ADHD. As well, those with intermittent explosive disorder do not experience issues with sustaining attention, characteristic of ADHD.

You should have learned the following in this section:

- ADHD occurs in about 7.2% of children worldwide.
- ADHD is more often diagnosed in males than in females.
- ODD, conduct disorder, and some learning disorders are comorbid with ADHD.

Section 10.2 Review Questions

1. How prevalent is ADHD in children?
2. Which gender is more likely to be diagnosed with ADHD? Are there differences in how the symptoms present?
3. Which disorders are comorbid with ADHD?
4. How do we distinguish ADHD from ODD, anxiety disorders, learning disorders, and intermittent explosive disorder?

10.3. Etiology

Section Learning Objectives

- Describe biological bases/causes of ADHD.
- Describe environmental causes of ADHD.

10.3.1. Biological

10.3.1.1. Genetic. ADHD is considered to be strongly influenced by genetics. Typically, ADHD has not been linked to chromosomal atypicalities. Rather, a general genetic susceptibility that has not fully been understood is at play. It is likely that ADHD susceptibility is polygeneic – involving more than one genetic trait. Twin studies have indicated that an average of 71-73% of ADHD symptom variance was explained by genetics factors (Barkley, 2015).

10.3.1.2. Structural abnormalities. Physiological structural and functional abnormalities in the frontal lobe area of the brain have also been linked to ADHD symptoms. Some research indicates that other areas involved may include the anterior cingulate, basal ganglia, cerebellum, and corpus callosum. Additionally, smaller anterior right frontal regions, caudate nucleus and globus pallidus have also been associated with ADHD. Delayed maturation in the prefrontal cortex, which is also highly connected to executive functioning, has been associated with ADHD (Barkley, 2015).

10.3.1.3. Functional differences. Slow wave activity in the frontal lobe and decreased beta activity has been noted in individuals with ADHD. Moreover, decreased blood flow in the prefrontal area of the brain have been indicated. Deficiencies in the availability of dopamine and norepinephrine (neurotransmitters) have been found in individuals with ADHD (Barkley, 2015).

10.3.2. Environmental

Very low birth weight is one of the strongest and most consistently noted environmental risk factors for ADHD, with more extreme low weight associated with greater risk (APA, 2022). Moreover, premature delivery is also associated with ADHD. Prenatal exposure to toxins, especially smoking, but also alcohol and other drugs, is associated with higher rates of ADHD. Additionally, environmental toxins, such as heavy exposure to lead or pesticides, is linked to ADHD symptoms (APA, 2022; Barkley, 2015).

Streptococcal infection has also been mildly linked to later development of ADHD. This typically only occurs when, following the infection, an individual's body has an autoimmune response to the production of the infection antibodies that results in the destruction of the basal ganglia (Barkley, 2015).

In general, there is very weak evidence for psychosocial factors impacting the development of ADHD (Barkley, 2015).

You should have learned the following in this section:

- Genetics, structural abnormalities, and functional differences are biological causes of ADHD.
- Environmental causes of ADHD include very low birth weight, prenatal exposure to toxins, premature delivery, and streptococcal infection.

Section 10.3 Review Questions

1. What are biological causes of ADHD?
2. What are environmental causes of ADHD?

10.4. Assessment

Section Learning Objectives

- Describe assessment tools commonly used.

When assessing for ADHD, psychologists often rely on parent-report, teacher-report, and observations. Occasionally, when the child is old enough, a psychologist will also incorporate the child's own self-report of symptoms. To obtain parent and teacher reports (and when appropriate, self-report) of symptoms, a psychologist often utilizes two things: an interview and objective measures. For behavioral observations, a psychologist will often observe the child in person, either in their office and/or at school. A good assessment will include information from all three areas (i.e., observation, interview, and objective measures) to make an informed diagnostic decision. Unfortunately, there has been a growing issue in the field of children being quickly diagnosed based on a short, 15-minute visit with a pediatrician/primary care provider. This has led to a great deal of discussion about concerns of overinflated prevalence rates due to misdiagnosis. As such, there has been a big push in the field to have children properly assessed and diagnosed for ADHD, particularly before initiating medicinal intervention/psychopharmacology.

10.4.1. Observations

Observations can be completed in various ways. This is often determined by the setting in which an assessment is taking place as well as the resources available to a psychologist. For example, if the assessment is taking place within the school setting, a psychologist will often find

a time to sit in a classroom with the child to observe him or her. The psychologist will attempt to do this with as little attention drawn to themselves as possible in an effort to observe the child without impacting their behavior. This is because children have typically been sent the message to "be on your best behavior" when a visitor comes to their classroom. As such, it is best that the teacher does not draw attention to the psychologist's presence. This stage of the assessment often takes place before the child has ever met the psychologist as well. This is so the child is not aware that they are being observed. If the child were to meet the psychologist beforehand, the child would be more inclined to recognize that the psychologist was there for them and may attempt to monitor their own behavior. As such, the psychologist would not be able to obtain a valid observation of their behavior. (See Module 1, Section 1.5.2.1 about naturalistic observation for a more detailed discussion about the strengths and weaknesses of this research method.)

Although observation in a classroom or similar setting is ideal, this is not always feasible. This is more likely to be the case when an assessment is initiated in an outpatient setting (meaning a clinic, doctor's office, etc.). School observations are difficult to obtain for professionals in the outpatient setting for various reasons. One reason is that managed health care (insurance companies) often do not cover services conducted within the school. This means that a psychologist working in a clinic cannot get paid for their time observing a child in a school. Other times, there is simply not an opportunity because a child is homeschooled, etc. In these circumstances, providers often rely on observing the child within the clinic. For example, some providers may intentionally wait to call a child from the waiting room for an appointment. Instead, they may use the first portion of their appointment to observe the child playing in the waiting room. Other times, they may simply conduct informal observations during their appointment. For example, while talking with parents, they may also be watching and noting

various behaviors a child is engaging in. They may also spend time one-on-one with the child playing and talking. During this time, although it may seem like they are simply playing, the provider will be noting the various behaviors and interactions that are occurring. These are creative ways to obtain valid and important observations when sometimes more natural observations, such as a school observation, are not possible.

10.4.2. Interview

An assessment for ADHD should always include some version of an interview. This will likely start with a parent. The psychologist will sit with the parents and ask several questions. They will attempt to gain an understanding of when symptoms were first noticed, if the child is experiencing any impairment related to the symptoms, and so forth. While they will focus on understanding the presence or absence of ADHD-related symptoms, they will also screen for other potential disorders with common comorbidity and/or similar symptom presentations. For example, they may screen for ODD symptoms since it is commonly comorbid with ADHD. Moreover, they may also screen for anxiety symptoms since, often, anxiety and ADHD can present with similar symptoms and be misdiagnosed.

Because symptoms must be present in more than one setting, a secondary interview may be conducted. This often occurs with teachers. This is easily obtained in situations where an assessment is initiated in the school setting. However, in situations in which the assessment was initiated in an outpatient clinic, this is more difficult to obtain, even via phone. The reason for this is the same as outlined above in the difficulties with obtaining observations in outpatient assessments. As such, providers often rely on objective measures from a teacher if they are unable to obtain an interview. The focus of the teacher and parent interviews are often similar.

However, in the teacher interview, the focus is more on specific impairment and functioning within the classroom and with peers. For example, the psychologist will ask many questions related to ability to stay on task, careless mistakes in work, ability to socialize with peers, etc.

If a child can communicate appropriately, meaning they are verbal and have appropriately developed speech, the child will be interviewed. This may occur informally while drawing or playing with the child, particularly if they are very young. As children get older, this will resemble more of an interview. Questions will focus on current difficulties such as "Is it hard to remember to turn your homework in?" "Do you lose things a lot?" "Do people say you talk a lot?" Again, these questions will be worded in a way that is appropriate for the child, depending on their age.

10.4.3. Objective Measures

There are a variety of objective measures that can be used. These are typically questionnaires that are filled out by the parent, teacher, and the child themselves (when appropriate). Children can begin reporting on their own symptoms anywhere between the ages of 6-11, depending on the specific questionnaire being used. Assessments specific to ADHD symptoms include, but are not limited to, the Conners-3, Disruptive Behavior Rating Scales (DBRS), and the NICHQ (National Institute for Children's Health Quality) Vanderbilt Assessment Scales. The Conners-3 provides both overall scores as well as a symptom count. The DBRS and the Vanderbilt provide a symptom count number. Other questionnaires that may be used but are not specific for ADHD are the Behavior Assessment System for Children, Third Edition (BASC-3) and the Achenbach System of Empirically Based Assessment (ASEBA). These forms provide overall scores for scales related to hyperactivity, impulsivity, and

inattention. However, they do not provide symptom counts. As such, the BASC and Achenbach scales are often used in combination with a tool such as the DBRS, Vanderbilt, and/or Conners-3.

You should have learned the following in this section:

- When assessing for ADHD, psychologists often rely on parent-report, teacher-report, and observations.
- An assessment for ADHD should always include some version of an interview.
- Assessments specific to ADHD symptoms include, but are not limited to, the Conners-3, Disruptive Behavior Rating Scales (DBRS), and the NICHQ (National Institute for Children's Health Quality) Vanderbilt Assessment Scales.

Section 10.4 Review Questions

1. What assessment tools are commonly used to assess for ADHD?

10.5. Treatment

Section Learning Objectives

- Describe treatment options for ADHD.
- Examine efficacy of the varying treatment options.

Of children that are diagnosed with ADHD, about 30% are receiving medication only, 15% are receiving psychotherapy/behavioral therapy, 32% are receiving both medicine and psychotherapy/behavioral therapy, and 23% are receiving no treatment at all (CDC, 2018a, September). According to the CDC (2018a, September), 9 out of 10 children with ADHD receive some type of school support at some point in their education.

10.5.1. Psychopharmacological

10.5.1.1. Stimulants. Historically, central nervous system (CNS) stimulants have been used the longest to treat ADHD, medicinally (CDC, 2018a, September). According to the CDC (2018b, September), 70-80% of children exhibit fewer symptoms with the introduction of stimulant medication. These medications work quickly and have short and extended-release formulas. This classification of drug includes methylphenidate and amphetamine. Stimulants are a controlled substance drug. These drugs work by increasing the availability of dopamine and norepinephrine (Barkley, 2015). Some negative side effects may include decreased appetite and resulting weight loss, difficulty sleeping, stomachaches and headaches, and higher heart rates/blood pressure. These drugs may potentially increase tics in children as well, if tics are a current concern (Barkley, 2015)

10.5.1.2. Non-stimulants. These are considered a slightly newer generation of medicinal intervention for ADHD. These mediations do not typically work as quickly as stimulants, however, they may have longer lasting effects (CDC, 2018b, September). Because they are not a controlled substance, some parents prefer to attempt to alleviate symptoms using non-stimulant medications. Additionally, up to 30% of individuals may not respond, or only have a partial response, to stimulants (Barkley, 2015). As such, non-stimulants may be tried in lieu of stimulants or in addition to stimulants. Atomoxetine, guanfacine, and clonidine are examples of nonstimulants used for ADHD. Some negative side effects include headaches, decreased appetite, nausea/vomiting, sedation, and fatigue (Barkley, 2015).

10.5.2. Psychotherapy

Therapy to mediate symptoms of ADHD is typically behaviorally based. Therapies may be conducted with the child, parents, or both. Who the therapy is conducted with largely depends on the child's age. For example, parent training is more likely to be utilized for younger and middle-aged children. However, older children and adolescents may also benefit from direct behavioral therapy. In some situations, a child/adolescent may benefit from receiving direct behavioral therapy while their parents also receive parent training (e.g., parent management training, PMT). At times, some work in cognitive and emotional realms may be beneficial as well. For example, children with ADHD are more likely to have a negative attribution bias. Essentially, they may interpret benign situations (e.g., someone accidently bumped into me) as hostile or malicious (e.g., they bumped into me on purpose) and then react impulsively to this. Cognitive therapy strategies can help to correct this misinterpretation of events.

10.5.2.1. Parent training. The goal of parenting training is to help parents implement consistent parenting strategies to increase structure and predictability. For example, parents learn how to deliver instructions and commands to children in a way that they are more likely to be successful. This may mean breaking large chores down into more manageable pieces, etc. It also might focus on giving more attention and praise to positive behaviors while ignoring negative, minor misbehaviors. This is so that we see an increase in the behaviors we want to see (if we attend to a behavior, the behavior will increase because attention is a strong reinforcer) and a decrease in negative behaviors (when we ignore behavior, we remove attention which reduces the likelihood of it reoccurring since the strong reinforcer of attention has been withdrawn). There are various, evidenced-based and empirically supported, treatment protocols that target parent management training. Some examples include Incredible Years Parenting Program, Triple P, Parent-Child Interaction Therapy, Defiant Child, etc.

10.5.2.2. Child-focused therapy. When working with the child or adolescent, we may begin working on implementing behavioral strategies to increase success and reduce impairment. While these are behaviorally focused, components of cognitive and emotional work may be intermixed to address common biases (see description of negative attribution bias above) and difficulties (e.g., low frustration tolerance). Also, because organization and studying skills are often impaired, another focus may be in *Organizational Skills Training (OST).* This training focuses increasing the child's ability to organize materials, plan tasks, use checklists/timers/planners, and protect school and studying time (Gallagher, Abikoff, & Spira, 2014).

10.5.3. Academic Interventions

Children with ADHD may benefit from simple and common academic modifications. For example, because children with ADHD may be easily distracted, they may benefit from taking tests in an alternate location that is quiet and free from distractions. It is also likely that, because they are more prone to get off task or distracted, that it will take them longer to take a test. A common accommodation provided to reduce impairment in this area is offering extended time on tests. Other accommodations might include preferential seating (e.g., being able to sit in the front of the class where distractions are minimized and a teacher can prompt a child to be on task more readily) or alternative seating (e.g., ability to sit on a balance ball, quietly stand next to their chair rather than sit, etc.), and frequent breaks during assignments/tests. School-home notes and reward systems may also be implemented to (1) improve behavior in the classroom and (2) keep parents informed of the child's behaviors as well as learning objectives and assignments due.

You should have learned the following in this section:

- Psychopharmacological treatments for ADHD include stimulants and non-stimulants.
- Therapy to mediate symptoms of ADHD is typically behaviorally based and may be conducted with the child, parents, or both.
- The goal of parenting training is to help parents implement consistent parenting strategies to increase structure and predictability.
- When working with the child or adolescent, we may begin working on implementing behavioral strategies to increase success and reduce impairment.
- Children with ADHD may benefit from simple and common academic modifications.

Section 10.5 Review Questions

1. What approaches are used to treat ADHD?

Apply Your Knowledge

CASE VIGNETTES

Alex is an 8-year-old boy who lives with his mother, father, and sister. He has a family history of ADHD. His intellectual functioning is average, he has great friendships, and is active in extracurricular activities. Alex has always been a child with an excess of energy and has struggled to sit still often. For example, during dinner, he often gets out of his chair, wiggles in his seat, and interrupts his family members conversations. His teachers notice some of these behaviors at school as well. However, his grades and social interactions have not been impaired. Alex struggles to fall asleep at night and he still as some trouble with urinary accidents, mostly at night.

Shaunda is a 12-year-old girl who lives with her mother and father. Shaunda does not have a known family history of ADHD. Her intellectual functioning is average, she also has great friendships, and is active in extracurricular activities as well. Shaunda does not get into trouble often, but her parents get frustrated by Shaunda's frequent forgetfulness in tasks at home. For example, Shaunda will begin cleaning her room and get distracted mid-task. Her mother often comes in to find Shaunda playing with a toy she found while cleaning. Shaunda also often forgets to put things up. For example, when getting milk out to make cereal, she often leaves the milk on the counter. Shaunda gets frustrated with herself as well. She reports not meaning to do these things, but simply forgets or gets distracted. These behaviors are apparent at school, despite her teachers not reporting difficulties. For example, her teachers do not report concerns for her behaviors at school, and she is described as a pleasant and compliant child. However, Shaunda's grades are suffering. She has many incompletes for homework and often makes careless mistakes on tests.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. Do you think Alex may have ADHD? If so, what other symptoms would you want to screen for? Do you think it is possible that Alex may be missed and not diagnosed with ADHD, even if he has ADHD? If so, why – what contextual factors may contribute to this?
2. Do you think Shaunda may have ADHD? If so, what other symptoms would you want to screen for? Do you think it is possible that Shaunda may be missed and not diagnosed with ADHD, even if she has ADHD? If so, why – what contextual factors may contribute to this?
3. What are some other disorders you would want to be looking for Shaunda?

Module Recap

In this module, we learned about ADHD. We discussed the history of ADHD and how the field moved from two separate disorders (ADD and ADHD) to one disorder (ADHD) with three presentations (predominantly inattentive presentation, predominantly hyperactive/impulsive presentation, and combined presentation). We discussed the various behaviors and symptoms of ADHD and how they relate to the various presentations. We then discussed the prevalence of ADHD, disorders frequently comorbid with ADHD, and the etiology of ADHD. We ended on a discussion of how ADHD is assessed and treated.

In our next module, we will learn about oppositional defiant disorder (ODD) and conduct disorder (CD), two behavioral disorders.

Part IV. Behavior-Related Disorders

Module 11:

Disruptive, Impulse-Control, and Conduct Disorders

Module 11: Disruptive, Impulse-Control, and Conduct Disorders

Module Overview

In Module 11, we will discuss matters related to disruptive, impulse-control, and conduct disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. We will cover oppositional defiant disorder, conduct disorder, and intermittent explosive disorder. Be sure you refer to 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

- Describe how disruptive, impulse-control, and conduct disorders present.
- Describe the prevalence of disruptive, impulse-control, and conduct disorders.
- Describe the etiology of disruptive, impulse-control, and conduct disorders.
- Describe how disruptive, impulse-control, and conduct disorders are assessed, diagnosed, and treated.

11.1. Clinical Presentation

Section Learning Objectives

- Describe the presentation and associated features of oppositional defiant disorder.
- Describe the presentation and associated features of conduct disorder.
- Describe the presentation and associated features of intermittent explosive disorder.

11.1.1. Oppositional Defiant Disorder

Oppositional defiant disorder is characterized by a child that is defiant/argumentative, angry/irritable, and vindictive, and has shown this pattern of behavior for at least six months. Of the eight possible symptoms, the child must present with at least four of them. In terms of angry/irritable mood, they may lose their temper often, are easily annoyed or touchy, and are often angry and resentful. In terms of argumentative/defiant behavior the child argues with authority figures, actively defies or refuses to comply, deliberately annoys others, or blames others for their mistakes. Finally, they must have acted spiteful or vindictive at least twice within

the past six months. Distress occurs in the child's immediate social context or affects social, occupational, educational, or other important areas of functioning. Functional consequences of these behaviors include frequent conflicts with parents, teachers, supervisors, peers, and romantic partners (APA, 2022). The disorder typically appears during the preschool years and rarely later than early adolescence.

11.1.2. Conduct Disorder

Conduct disorder is a more severe behavioral disorder in which an individual displays a disregard, not only for rules and authority, but also the rights and conditions of humans and/or animals. Behaviors that may be exhibited are stealing, fighting, cruelty to people or animals, fire-setting, running away from home, bullying or threatening others, using a weapon that can cause harm, committing a mugging or armed robbery, forcing someone into sexual activity, deliberately destroying another person's property, lying to obtain goods or favors, stealing items of nontrivial value without confronting the victim, staying out at night in clear violation of parental rules, and being truant from school. The preceding represents 15 symptoms of which the person must present with at least three in the past 12 months, with at least one criterion present in the past 6 months.

There are three subtypes of conduct disorder focused on the age of onset. The *childhood-onset type* occurs prior to age 10 while the *adolescence-onset type* occurs after age 10. The *unspecified onset subtype* is used when age of onset is unknown. Males usually receive the childhood-onset subtype and have disturbed peer relationships, likely were diagnosed with oppositional defiant disorder in early childhood, and typically have symptoms that meet full criteria for conduct disorder before puberty.

Conduct disorder is often associated with limited prosocial emotions. To qualify for this specifier, at least two of the following characteristics must have been displayed persistently over the past 12 months and in multiple relationships and settings. These include: a lack of remorse or guilt, a lack of concern for the feelings of others (callous – lack of empathy), being unconcerned about performance, and having shallow or deficient affect.

Functional consequences of these behaviors include being suspended or expulsed from school, problems in work adjustment, legal problems, sexually transmitted diseases, physical injury from accidents or fights, and unplanned pregnancy. It is also associated with early onset of sexual behavior; alcohol, tobacco, and illegal substances use; and reckless and risk-taking behaviors.

The onset of conduct disorder occurs as early as the preschool years, but it is during middle childhood through middle adolescence that the first significant symptoms usually emerge. The DSM states, “Physically aggressive symptoms are more common than nonaggressive symptoms during childhood, but nonaggressive symptoms become more common than aggressive symptoms during adolescence” (APA, 2022, pg. 534).

11.1.3. Intermittent Explosive Disorder

Intermittent explosive disorder is characterized by recurrent behavioral outbursts which represent a failure to control aggressive impulses. It is manifested by one of the following: 1) verbal or physical aggression toward property, animals, or other individuals which occur twice a week on average, for up to three months; and 2) “…three behavioral outbursts involving damage or destruction of property and/or physical assault involving physical injury against animals or other individuals occurring within a 12-month period” (APA, 2022). The level of aggressiveness

displayed by the individual is out of proportion with the experienced provocation or stressors and is not for the purpose of achieving a tangible objective such as money, power, or intimidation. The disorder should not be diagnosed in individuals younger than 6 years.

Functional consequences of these behaviors include loss of friends, relatives, or marital instability in the social domain, demotion or loss of employment in the occupational domain, or civil suits due to the aggressive behavior against person or property in the legal domain. There could also be criminal charges and financial loss due to the destruction of objects.

You should have learned the following in this section:

- Oppositional defiant disorder is characterized by a child that is defiant/argumentative, angry/irritable, and vindictive and has shown this pattern of behavior for at least six months. At least 4 of 8 symptoms must be present.
- Conduct disorder is a more severe behavioral disorder in which an individual displays a disregard not only for rules and authority, but also the rights and conditions of humans and/or animals. The individual must display at least 3 of the 15 symptoms.
- Conduct disorder is often associated with limited prosocial emotions.
- Intermittent explosive disorder is characterized by recurrent behavioral outbursts which represent a failure to control aggressive impulses.

Section 11.1 Review Questions

1. Which of the three disorders discussed in this section is the most severe?
2. Which of the three disorders is associated with limited prosocial emotions?
3. Which of the disorders is not diagnosed in children under 6 years of age?
4. Which disorder is characterized by being irritable, argumentative, and vindictive?

11.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence and course of oppositional defiant disorder, conduct disorder, and intermittent explosive disorder.
- Describe comorbid disorders with oppositional defiant disorder, conduct disorder, and intermittent explosive disorder.
- Describe disorders with similar presentations that must be differentiated from oppositional defiant disorder, conduct disorder, and intermittent explosive disorder.

11.2.1. Oppositional Defiant Disorder

11.2.1.1. Prevalence. According to the DSM-5-TR, the cross-national prevalence of oppositional defiant disorder ranges from 1% to 11% with an average prevalence estimate of around 3.3%. The disorder is more common in boys than girls prior to adolescence (APA, 2022).

11.2.1.2. Comorbidity. Oppositional defiant disorder occurs more often in children, adolescents, and adults also diagnosed with ADHD and often precedes conduct disorder. Other comorbid disorders are anxiety disorders and major depressive disorder. Rates of substance use disorders are higher in adolescents and adults diagnosed with oppositional defiant disorder.

11.2.1.3. Differential diagnosis. Oppositional defiant disorder should be distinguished from *conduct disorder.* Both disorders bring the individual in conflict with adults and authority figures, but the behaviors of oppositional defiant disorder are usually less severe than conduct disorder and do not include aggression toward people or animals, destruction of property, or a pattern of theft or deceit. However, the impairment associated with oppositional defiant disorder

may be equivalent or greater than that of conduct disorder. Finally, oppositional defiant disorder includes problems of emotional dysregulation which are absent from conduct disorder.

Oppositional defiant disorder shares with *intermittent explosive disorder* high rates of anger. However, those with intermittent explosive disorder often show serious aggression toward others that is not characteristic of oppositional defiant disorder.

Finally, stressors may lead to emotional dysregulation, which can present as tantrums and oppositional behavior in children, or aggressive behaviors in adolescents. The DSM says, "Temporal association with a stressor and symptom duration of less than 6 months after the resolution of the stressor may help distinguish *adjustment disorder* from oppositional defiant disorder" (APA, 2022).

11.2.2. Conduct Disorder

11.2.2.1. Prevalence. In the United States and other largely high-income countries, one-year population prevalence estimates range from 2% to more than 10%, with a median of 4%. In the United States, the lifetime prevalence was 12% among men and 7.1% among women. For those with conduct disorder, suicidal thoughts, suicidal attempts, and suicide occur at a higher-than-expected rate.

In relation to sex and gender-related diagnostic issues, girls and women diagnosed with conduct disorder are more likely to display lying, truancy, running away, and prostitution while boys and men with the disorder exhibit fighting, stealing, vandalism, and school discipline problems. Both boys and men and girls and women display relational aggression, however, girls and women show less physical aggression than boys and men (APA, 2022).

11.2.2.2. Comorbidity. Conduct disorder has been found to be comorbid with ADHD and oppositional defiant disorder, and this comorbid presentation predicts the poorest outcomes. Other comorbid disorders include specific learning disorder, anxiety disorders, depressive or bipolar disorders, and substance-related disorders.

11.2.2.3. Differential diagnosis. Conduct disorder and intermittent explosive disorder share the feature of high rates of aggression, but the aggression in intermittent explosive disorder is limited to impulsive aggression that is not premeditated and does not seek to accomplish an aim such as money, power, or intimidation. Additionally, nonaggressive symptoms of conduct disorder are not present in intermittent explosive disorder.

11.2.3. Intermittent Explosive Disorder

11.2.3.1. Prevalence. The 1-year prevalence in the United States is about 2.6% with a lifetime prevalence of 4.0%. When intermittent explosive disorder is comorbid with PTSD, the rate of lifetime suicide attempts increases (41%; APA, 2022).

11.2.3.2. Comorbidity. Disorders comorbid with intermittent explosive disorder include depressive disorders, anxiety disorders, PTSD, bulimia, binge-eating disorder, and substance use disorders. Additionally, antisocial personality disorder, borderline personality disorder, ADHD, conduct disorder, and oppositional defiant disorder are comorbid.

11.2.3.3. Differential diagnosis. Both intermittent explosive disorder and ADHD share high levels of impulsive behavior, but serious aggression toward others is common with intermittent explosive disorder and not ADHD. As well, those with intermittent explosive disorder do not experience issues with sustaining attention, characteristic of ADHD.

Antisocial and borderline personality disorders share the feature of recurrent, problematic impulsive aggressive outbursts but the level of impulsive aggression is higher with intermittent explosive disorder.

You should have learned the following in this section:

- The prevalence of oppositional defiant disorder and conduct disorder go up to about 10% while intermittent explosive disorder is below 3%.
- When diagnosing any of these disruptive, impulse-control, and conduct disorders it is imperative that they be distinguished from each other to avoid misdiagnosis.
- Oppositional defiant disorder is usually less severe than conduct disorder and though high rates of anger occur with oppositional defiant disorder and intermittent explosive disorder, the serious aggressive behavior toward others is not part of oppositional defiant disorder.
- The three disorders are all comorbid with ADHD, anxiety disorders, depressive disorders, and substance use disorders.

Section 11.2 Review Questions

1. Describe the prevalence of the disruptive, impulse-control, and conduct disorders. Which disorder is most common and which is least common?
2. Describe patterns of comorbidity across the three disorders and which disorders they are uniquely comorbid with.
3. Distinguish the three disruptive, impulse-control, and conduct disorders from one another. What other disorders share features with each?
4. Are there any gender differences worth noting with the three disorders?

11.3. Etiology

Section Learning Objectives

- Describe the environmental causes of disruptive, impulse-control, and conduct disorders.
- Describe genetic and physiological causes of disruptive, impulse-control, and conduct disorders.
- Explain the General Coercive Cycle.

11.3.1. Oppositional Defiant Disorder

11.3.1.1. Environmental. Harsh, inconsistent, or neglectful child-rearing practices predict increases in symptoms, and oppositional symptoms predict increases in these types of parenting. Furthermore, when childcare is disrupted by a succession of caregivers, oppositional defiant disorder becomes more prevalent. Finally, children with the disorder are at greater risk of bullying peers as well as being bullied by peers (APA, 2022).

11.3.1.2. Genetic and physiological. Neurobiological markers such as a slower resting heart rate, skin conductance reactivity, reduced basal cortisol reactivity, and abnormalities in the amygdala and prefrontal cortex are associated with oppositional defiant disorder.

11.3.2. Conduct Disorder

11.3.2.1. Environmental. "Parental rejection and neglect, inconsistent child-rearing practices, harsh discipline, physical or sexual abuse, lack of supervision, early institutional living, frequent changes of caregivers, large family size, parental criminality, and certain kinds

of familial psychopathology" are all risk factors for conduct disorder at the family level (APA, 2022, pgs. 534-5). Peer rejection, being part of a delinquent peer group, exposure to violence, and neighborhood disadvantage are cited as community-level risk factors.

11.3.2.2. Genetic and physiological. Having a caregiver or close relative that has been diagnosed with conduct disorder leads to a higher risk of a child developing the disorder. Children of parents with severe alcohol use disorder, depressive and bipolar disorders, schizophrenia, ADHD, or conduct disorder are also at higher risk (APA, 2022). Family history may be particularly predictive of a child developing *childhood onset*, which is considered to have the worst prognosis. Slower resting heart rate, reduced autonomic fear conditioning, and low skin conductance is also noted in individuals with conduct disorder.

11.3.3. Intermittent Explosive Disorder

11.3.3.1. Environmental. Those with a history of physical and emotional trauma during the first 20 years of life are at greater risk of developing the disorder. Within some refugee population settings, long-term displacement from home and separation from family are risk factors (APA, 2022).

11.3.3.2. Genetic and physiological. Genetic factors are a major risk factor, with first-degree relatives of those with intermittent explosive disorder being at increased risk.

11.3.4. The General Coercive Cycle

Although there are various developmental pathway theories on how these disorders develop, such as Multiple Pathways (Loeber & Stouthamer-Loeber, 1998), Gerald Patterson's Coercive Family Process Model (1982) is one of the most referenced and utilized theories. The

model describes a pattern of interactions that occur within a family. When families engage in negative interactions, children learn and model aggressive behaviors. This is largely grounded in social-learning theory. Ultimately, children learn through negative reinforcement (see Module 3 for a discussion) of coercive parent-child interactions. Most of the parent training protocols to treat behavioral problems are based on Patterson's model.

Patterson (1982) theorized that various family factors impact a parent's traits. Those parental traits then disrupt family dynamics which eventually leads to child antisocial behavior. The interactions occurring within these families tend to present as depicted in the figures below. Figure 11.1 gives a general overview of the process and Figure 11.2 gives a more specific example/application of the process.

Figure 11.1 General Coercive Cycle

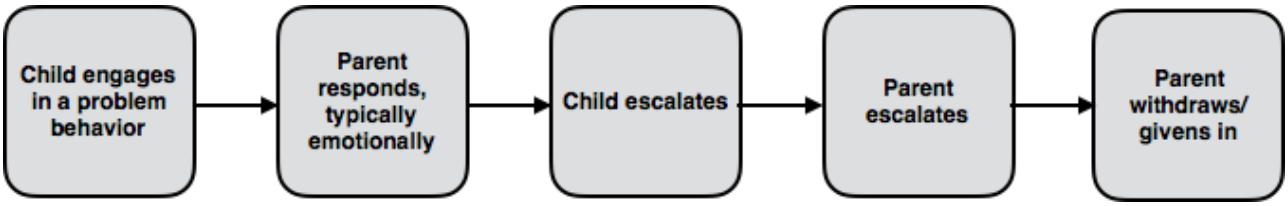
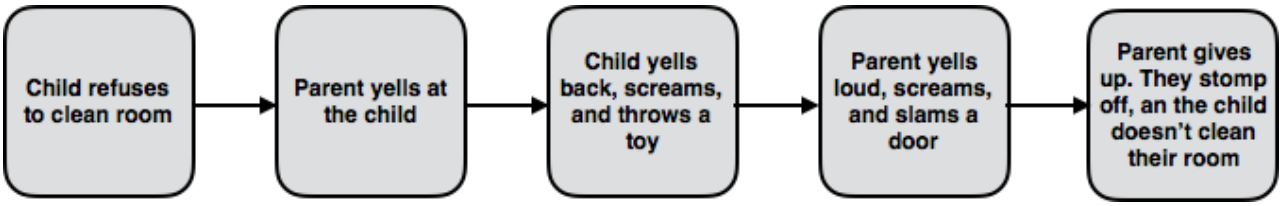


Figure 11.2 Specific Example of the Coercive Cycle



You should have learned the following in this section:

- The disruptive, impulse-control, and conduct disorders are thought to be caused by a series of environmental risk factors such as harsh, inconsistent, or neglectful child-rearing practices.
- Genetic and physiological risk factors are also present for all three disorders.
- The General Coercive Cycle helps to explain socio-cultural causes of these disorders.

Section 11.3 Review Questions

1. What are environmental causes of each of the disruptive, impulse-control, and conduct disorders?
2. What are genetic/physiological causes of each of the disruptive, impulse-control, and conduct disorders?
3. What is the General Coercive Cycle?

11.4. Assessment

Section Learning Objectives

- Describe assessment tools commonly used to diagnosis disruptive, impulse-control, and conduct disorders.

11.4.1. Assessment Tools

When assessing for disruptive, impulse-control, and conduct disorders, the process is very similar to that outlined in the previous chapter on ADHD. Psychologists, again, rely on parent-report, teacher-report, and observations. If the child is old enough, a psychologist will also incorporate the child's own self-report of symptoms. However, this must be done carefully, because the behaviors being assessed are ones that an individual may be more inclined to under-report or deny. For example, even though an individual may challenge authority frequently, the child may be inclined to answer in a more socially desirable way, denying that they challenge authority. As such, a psychologist must be careful to assess the validity of reports, particularly from self-reports of the child/adolescent.

11.4.2. Observations

Observations can be completed in various ways, similar to observations described for ADHD. However, formal observation may be less necessary. Although formal and inconspicuous observation of a child in the classroom can be valuable, the behaviors in disruptive, impulse-control, and conduct disorders are highly pronounced and noticeable. As such, we tend to rely on parent and teacher reports more heavily. Nonetheless, a psychologist

may obtain a good sample of observations when intervening and talking with the child. For example, when interviewing a child, if the psychologist requests the child sit in a particular area, but the child refuses, this may be evidence of defiance. This does not require the psychologist to observe the child in the waiting room or school to gain this information. However, some symptoms, although severe, may occur infrequently. For example, fire setting, although severe, is not likely to occur during an observation period. Though observations are sometimes useful when obtained, this is why they are less critical for diagnosing disruptive, impulse-control, and conduct disorders than they are for a disorder such as ADHD.

11.4.3. Interview

An assessment for disruptive behaviors of disruptive, impulse-control, and conduct disorders should always include some version of an interview which will likely start with a parent. The interview will focus on gaining an understanding of current symptoms and behaviors. Additionally, the time in which symptoms first began will be a critical area of focus, especially when there is concern of conduct disorder. The age at which symptoms were initially noticed is important, due to the impact this has on the child's prognosis and trajectory. Another important step in the interview process is to assess and understand family and parenting practices, because these factors closely relate to etiology of these disorders as well as treatment implications.

Child interviews will usually be attempted. Sometimes with particularly defiant children, interviews are difficult. However, an attempt should be made. During the interview, a psychologist will note displayed affect and attempt to document the presence of irritability, empathy, prosocial emotions, etc. This information is gained, not only through answers the

individual provides, but their tone, body language, and facial expressions. In a sense, a mini observation may occur within the interview.

11.4.4. Objective Measures

A variety of objective measures can be used. These are typically questionnaires that are completed by the parent, teacher, and the child themselves, when appropriate. Children can begin to report on their own symptoms anywhere between the ages of 6-11, depending on the specific questionnaire being used. The mental health professional will generally utilize similar assessments noted in the ADHD chapter, since ADHD has a high comorbidity with oppositional defiant disorder, intermittent explosive disorder, and conduct disorder. Because of this, scales that were designed to assess ADHD also include subscales that measure disruptive, impulse-control, and conduct disorders symptoms and behaviors. As such, scales used include, but are not limited to, the Conners-3, Disruptive Behavior Rating Scales (DBRS), and the NICHQ (National Institute for Children's Health Quality) Vanderbilt Assessment Scales. The Conners-3 provides both a T-score as well as a symptom count. The DBRS and the Vanderbilt provide a symptom count number. Other questionnaires that may be used, but are not specific for ADHD, are the Behavior Assessment System for Children, Third Edition (BASC-3) and the Achenbach System of Empirically Based Assessment (ASEBA). These forms provide T-scores for scales related to anger/aggression and conduct problems. However, these do not provide symptom counts. As such, the BASC and Achenbach scales are often used in combination with a tool such as the DBRS, Vanderbilt, and/or Conners-3.

You should have learned the following in this section:

- We can assess the disruptive, impulse-control, and conduct disorders in a manner very similar to those used for ADHD given the fact that ADHD is comorbid with all three disorders discussed in this module.
- Psychologists rely on parent-report, teacher-report, and observations.
- There are a variety of objective measures that can be used such as questionnaires.

Section 11.4 Review Questions

1. Why are many of the same tools used to assess ADHD used to assess the disruptive, impulse-control, and conduct disorders?
2. Why are parent and teacher reports used more than observation by the psychologist?
3. What information does a psychologist use during an interview?
4. When are children able to report their own symptoms on a questionnaire?

11.5 Treatment

Section Learning Objectives

- Describe treatment options for disruptive, impulse-control, and conduct disorders.
- Examine efficacy of the treatment options.

11.5.1. Oppositional Defiant Disorder

11.5.1.1. Psychotherapy. A common treatment option is *parent management training (PMT).* The goal of parenting training is to help parents implement consistent parenting strategies to increase structure and predictability. For example, parents learn how to deliver instructions and commands to children in a way in which they are more likely to succeed. For example, this could mean breaking large chores down into more manageable pieces. It also requires parents to specifically outline the goal behavior (e.g., put your shoes in your closet) they want to see. This results in gaining the child's attention (which includes establishing eye contact and may require moving closer to the child or physically directing their attention), saying their child's name, stating the expectation clearly, and remaining firm with the directive.

PMT also focuses on giving more attention and praise to positive behaviors while ignoring minor misbehaviors. This is in order to increase desired behaviors (if we attend to a behavior, the behavior will increase because attention is a strong reinforcer) and decrease negative behaviors (when we ignore the behavior, we remove attention which reduces the likelihood of it reoccurring since the reinforcer of attention has been withdrawn). It can be challenging for parents to reward or praise behavior that is 'expected.' For example, a parent may say "Why am I praising them for brushing their teeth? They should be doing that." This is a

valid and common reaction. However, because the behavior is currently absent, it is necessary to give positive attention to increase that behavior. Think about it, if you get praised by your boss at work, are you going to work harder to get recognized again? Yup, I bet you are! It is the same principal here. In fact, mental health practitioners often use this very analogy to help parents understand this.

PMT also involves teaching parents to systematically implement consequences that reduce elevated emotion. This typically involves removal of privileges or the introduction of undesired activity as well as a time out, when appropriate. There are various, evidenced-based and empirically supported, treatment protocols that target parent management training. The following are examples of such but are not an exhaustive list: Incredible Years Parenting Program, Triple P, Parent-Child Interaction Therapy, Defiant Child, etc. Overall, the goal of these intervention programs is to reduce the likelihood of the parent-child coercive cycle discussed earlier in this chapter.

11.5.2. Conduct Disorder

11.5.2.1. Psychotherapy. *Multisystemic therapy (MST*) is an intensive treatment option that has demonstrated efficacious results for the treatment of conduct disorder, especially in cases of more extreme conduct problems. MST is a therapy that takes place in the child/adolescent's home, school, and overall community, in which the therapist works with the child, their family, and other community members. Therapists can be accessed more readily in MST than in other treatment modalities, meet with the child/family multiple times a week and follow a family for several months. This allows more opportunity for meaningful and intensive interventions at the individual, familial, and neighborhood/community level. A recent

metanalysis (Tan & Fajarado, 2017) confirmed that, overall, research indicates that MST can lead to improved functioning for children with severe behavioral and conduct problems. Although MST is a preferred treatment modality for youth with conduct disorder, it is costly and difficult to obtain in some areas of the country, due to lack of resources. However, MST may be less costly than typical services in the short-term due to costs saved from a reduction in crime and incarceration (Tan & Fajarado, 2017). Other studies (e.g., Dopp, Borduin, Wagner, & Sawyer, 2014) have shown similar benefits as those found in Tan and Fajarado's (2017) study.

11.5.3. Intermittent Explosive Disorder

Psychology Today notes that the treatment of intermittent explosive disorder can be highly effective if started as early as possible. They write, "School-based violence prevention programs, for example, may lead to early identification of *intermittent explosive disorder* cases, leading to treatment that could prevent associated psychopathology." Treatment involves a combination of medication and psychotherapy. In terms of the latter, cognitive behavioral therapy can aid individuals in developing coping mechanisms, such as relaxation techniques, to deal with their impulses. Group counseling and anger management programs are also used.

In terms of medication, no specific medications exist for treating the disorder. That said, medications such as anti-depressants, anti-anxiety agents, anticonvulsants, and mood stabilizers can be used by reducing the impulsive behavior or aggression. They conclude, "Since *intermittent explosive disorder* can be comorbid with conditions such as anxiety or depression, clinicians need to factor that into their treatment plan, especially if medication is used."

To see the article for yourself, please visit:

https://www.psychologytoday.com/us/conditions/intermittent-explosive-disorder#treatment

11.5.4. Psychopharmacology

Medications are not used to address symptoms of behavioral disorders. However, if a child has a behavioral disorder and comorbid impulsivity or mood concerns (see relevant modules for more information), medications may be used to address those concerns as they may exacerbate disruptive, impulse-control, and conduct disorders symptoms.

You should have learned the following in this section:

- Psychotherapy can be used to treat the disruptive, impulse-control, and conduct disorders discussed in this chapter.
- Generally speaking, medications are not used to address symptoms of behavioral disorders.

Section 11.5 Review Questions

1. What is parent management training and what disorder is it used for?
2. What is multisystemic therapy and what disorder is it used for?
3. How is CBT used to treat intermittent explosive disorder?
4. Are medications effective for treating disruptive, impulse-control, and conduct disorders? If so, how?

Apply Your Knowledge

CASE VIGNETTE

William is a 16-year-old boy that lives with his mother. He has never met his father, but his father and his paternal family have a long history of incarceration. William's mother has worked hard to provide a safe home for William and meet all of his needs. However, to do that, she has to work two jobs, and that means William is often at home alone or unattended. He has been involved in school sports; however, his grades dropped which got him kicked off the school sports teams. He has a group of friends that he gets along with and considers to be a strong support system. He and his friends often skip class and William has smoked marijuana with his friends at times, although he reports that he does not do this regularly. William has never stolen anything, he's never had contact with the legal system, and he has a part-time job that he has kept for 8 months now. William and his baseball coach have a strong connection, and his coach has been working with William to get his grades up so that he can rejoin the baseball team.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What risk factors are present for William?
2. What protective factors are present for William?
3. Does he meet criteria for one of the disorders discussed in this module? Why or why not?

Module Recap

In this module, we learned about disruptive, impulse-control, and conduct disorders. We discussed the various behaviors and symptoms of oppositional defiant disorder, conduct disorder, and intermittent explosive disorder and how they relate to the various presentations. We then discussed the prevalence of these disorders, frequently comorbid disorders, and the etiology of the disruptive, impulse-control, and conduct disorders. In our discussion of etiology, we also learned about the coercive cycle. We ended on a discussion of how the disruptive, impulse-control, and conduct disorders are assessed and treated. Note that the DSM also includes descriptions of pyromania and kleptomania, both of which are thought to start in adolescence.

This concludes our discussion of behavior-related disorders. In the next unit we will discuss mood-related disorders which includes depressive and bipolar-related disorders.

Part V. Mood and Anxiety-related Disorders

Part V. Mood and Anxiety-Related Disorders

Module 12:

Mood Disorders

Module 12: Mood Disorders

Module Overview

In Module 12, we will discuss matters related to mood disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include depressive disorders, disruptive mood dysregulation, and bipolar disorders as well as suicide. 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. Prevalence and Comorbidity
- 12.3. Etiology
- 12.4. Assessment
- 12.5. Treatment of Mood Disorders

Module Learning Outcomes

- Describe how mood disorders present.
- Describe prevalence and comorbidity of mood disorders.
- Describe the etiology of mood disorders.
- Describe how mood disorders are assessed.
- Describe treatment options for mood disorders.

12.1. Clinical Presentation

Section Learning Objectives

- Distinguish the two distinct groups of mood disorders.
- Identify and describe the two most common types of depressive disorders.
- Clarify why disruptive mood dysregulation disorder was added as a new diagnosis.
- Classify symptoms of depression.
- Describe what disruptive mood dysregulation disorder is and its diagnostic features.
- Distinguish the forms bipolar disorder takes.
- Contrast a manic episode with a hypomanic episode.
- Define cyclothymic disorder.
- Describe suicidality and self-harm.

12.1.1. Distinguishing Mood Disorders

Within mood disorders are two distinct groups—individuals with depressive disorders and individuals with bipolar disorders. **The key difference between the two mood disorder groups is episodes of mania/hypomania.** More specifically, in bipolar I disorder, the individual experiences a manic episode that "may have been preceded by and may be followed by hypomanic or major depressive episodes" (APA, 2022, pg. 139) whereas for bipolar II disorder, the individual has experienced in the past or is currently experiencing a hypomanic episode and has experienced in the past or is currently experiencing a major depressive episode. In contrast, individuals presenting with a depressive disorder have never experienced a manic or hypomanic episode.

12.1.2. Types of Depressive Disorders

The two most common types of depressive disorders are major depressive disorder (MDD) and persistent depressive disorder (PDD). **Persistent depressive disorder**, which in the DSM-5 now includes the diagnostic categories of dysthymia and chronic major depression, is a continuous and chronic form of depression. While the symptoms of PDD are very similar to MDD, they are usually less acute, as symptoms tend to ebb and flow over a long period (i.e., more than two years). **Major depressive disorder**, on the other hand, has discrete episodes lasting at least two weeks in which there are substantial changes in affect, cognition, and neurovegetative functions (APA, 2022, pg. 177).

In addition to major depressive disorder and persistent depressive disorder, **disruptive mood dysregulation disorder** will be discussed as it is a newly created diagnosis that is specific to childhood and has an age of onset before 10 years and is not diagnosed after before age 6 or after age 18. It was added to the DSM 5, "…to address the considerable concern about the appropriate classification and treatment of children who present with chronic, persistent irritability relative to children who present with classic (i.e., episodic) bipolar disorder" (APA, 2022, pg. 179). More on this disorder in Section 12.1.4.3.

12.1.3. Symptoms Associated with Depressive Disorders

When making a diagnosis of depression, there are a wide range of symptoms that may be present. These symptoms can generally be grouped into four categories: mood, behavioral, cognitive, and physical symptoms.

12.1.3.1. 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. It is important to note that in children and adolescents, an irritable or cranky mood, and not a sad or dejected mood, may develop. The DSM-5-TR adds that this presentation should be distinguished from a pattern of irritability when the individual is frustrated (APA, 2022).

12.1.3.2. Behavioral. Behavioral issues such as decreased physical activity and reduced productivity—both at home and work—are often observed in individuals with depression. This is typically where a disruption in daily functioning occurs as individuals with depressive disorders are unable to maintain their social interactions and employment responsibilities. Children that were once heavily involved in their church youth group or sports team may begin missing meetings/practices, withdraw their involvement in the group/team, or underperform when present with the group. They may not find pleasure in the activities or may just not have the energy to carry out the activities.

12.1.3.3. 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. Individuals with depressive disorders often feel worthless, which creates a negative feedback loop by reinforcing their overall depressed mood. They also report difficulty concentrating on tasks, as they are easily distracted from outside stimuli. This assertion 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). This poor concentration may lead to a precipitous drop in grades in

children. Finally, thoughts of suicide and self-harm do occasionally occur in those with depressive disorders (*Note - this will be discussed in more detail in Section 12.1.6*).

12.1.3.4. Physical. Changes in sleep patterns are common in those experiencing depression with reports of both hypersomnia and insomnia. **Hypersomnia**, or excessive sleeping, often impacts an individual's daily functioning as they spend the majority of their time sleeping as opposed to participating in daily activities (i.e., meeting up with friends or getting to work on time). Reports of **insomnia** are also frequent and can occur at various points throughout the night to include difficulty falling asleep, staying asleep, or waking too early with the inability 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 suggests that these symptoms are a component of the disorder rather than a secondary symptom of sleep disturbance.

Additional physical symptoms, such as a 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 overeat, often seeking "comfort foods," such as those high in carbohydrates. Due to these changes in eating behaviors, there may be associated changes in 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.

12.1.4. Diagnostic Criteria and Features for Depressive Disorders

12.1.4.1. Major depressive disorder (MDD). According to the DSM-5-TR (APA, 2022), to meet the criteria for a diagnosis of major depressive disorder, an individual must experience at least *five* symptoms across the four categories discussed above, and at least one of the symptoms is either 1) a depressed mood most of the day, almost every day, or 2) loss of interest or pleasure in all, or most, activities, most of the day, almost every day. These symptoms must be present for *at least two weeks* and cause clinically significant distress or impairment in important areas of functioning such as social and occupational. The DSM-5 cautions that responses to a significant loss (such as the death of a loved one, financial ruin, and discovery of a serious medical illness or disability), can lead to many of the symptoms described above (i.e., intense sadness, rumination about the loss, insomnia, etc.) but this may be the normal response to such a loss. Though the individual's response resembles a major depressive episode, clinical judgment should be utilized in making any diagnosis and be based on the clinician's understanding of the individual's personal history and cultural norms related to how members should express distress in the context of loss.

12.1.4.2. Persistent depressive disorder (PDD). For a diagnosis of persistent depressive disorder, an individual must experience a depressed mood for most of the day, for more days than not, for at least two years (1 year for children or adolescents; APA, 2022). This feeling of a depressed mood is also accompanied by *two* or more additional symptoms, to include changes in appetite, insomnia or hypersomnia, low energy or fatigue, low self-esteem, feelings of hopelessness, and poor concentration or difficulty with decision making. The symptoms taken together cause clinically significant distress or impairment in important areas of functioning such as social and occupational and these impacts can be as great as or greater than MDD. The

individual may experience a temporary relief of symptoms; however, the individual will not be without symptoms for more than two months during this two-year period (or 1 year for children and adolescents).

Making Sense of the Disorders

In relation to depressive disorders, note the following:

Diagnosis MDD ….. if symptoms have been experienced for at least two weeks and can be regarded as severe

Diagnosis PDD … if the symptoms have been experienced for at least two years and are not severe

12.1.4.3. Disruptive mood dysregulation disorder. Disruptive mood dysregulation disorder is characterized by chronic severe, persistent irritability that has two prominent clinical manifestations – first, frequent temper outbursts and second, chronic, persistently irritable or angry mood that is present between these outbursts. The outbursts are verbal such as rages or behavioral such as physical aggression toward people or property and "…are grossly out of proportion in intensity or duration to the situation or provocation" (APA, 2022, pg. 178). They occur about three or more times per week and are inconsistent with developmental level. This all has occurred for at least 12 months and there has not been a period lasting three or more consecutive months without symptoms. The symptoms are present in in two of three settings to include home, school, or with peers and are severe in at least one of these. As noted earlier, the age of onset is before age 10 and disruptive mood dysregulation disorder should not be diagnosed for the first time before age 6 or after age 18 (APA, 2022).

Functional consequences of disruptive mood dysregulation disorder include a disruption in a child's family and peer relationships, as well as their performance in school. They have

difficulty participating in the activities often enjoyed by healthy children. Finally, they have trouble initiating and sustaining friendships.

12.1.5. Bipolar Disorders

12.1.5.1. Distinguishing bipolar I and II disorders. According to the DSM-5-TR (APA, 2022), 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 and/or followed by a hypomanic or major depressive episode, however, diagnostic criteria for a manic episode is the *only* criteria that needs to be met for a bipolar I diagnosis. 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. Descriptions of both manic and hypomanic episodes follow below.

Making Sense of the Disorders

In relation to bipolar I and II disorders, note the following:

Diagnosis bipolar I disorder …. if an individual has ever experienced a **manic** episode

Diagnosis bipolar II disorder … if the criteria has only been met for a **hypomanic** episode

12.1.5.2. Manic episode. The key feature of a **manic episode** is a specific period in which an individual reports abnormal, persistent, or expansive irritable mood for nearly all day, every day, for *at least one week* (APA, 2022). 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 interpersonal interactions. They also display rapid shifts in mood, also known as **mood lability**, ranging from happy, neutral, to irritable. At least three of the symptoms described below (four if the mood is only irritable) must be present and represent a noticeable change in the individual's typical behavior.

Inflated self-esteem or grandiosity (Criterion B1) is present during a manic episode. Occasionally these inflated self-esteem levels can appear delusional. For example, individuals may believe they are friends with a celebrity, do not need to abide by laws, or even perceive themselves as God. They also engage in multiple overlapping new projects (Criteria B6 and 7), often initiated with no prior knowledge about the topic, and engaged in at unusual hours of the day.

Despite the increased activity level, individuals experiencing a manic episode also require a decreased need for sleep (Criterion B2), sleeping as little as a few hours a night yet still feeling rested. Reduced need for sleep may also be a precursor to a manic episode, suggesting that a manic episode is to begin imminently. It is not uncommon for those experiencing a manic episode to be more talkative than usual. It can be difficult to follow their conversation due to the quick pace of their talking, as well as tangential storytelling. Additionally, they can be difficult to interrupt in conversation, often disregarding the reciprocal nature of communication (Criterion B3). If the individual is more irritable than expansive, speech can become hostile and they engage in tirades, particularly if they are interrupted or not allowed to engage in an activity they are seeking out (APA, 2022).

Based on their speech pattern, it should not be a surprise that racing thoughts and flights of ideas (Criterion B4) also present during manic episodes. Because of these rapid thoughts,

speech may become disorganized or incoherent. Finally, individuals experiencing a manic episode are distractable (Criterion B5).

12.1.5.3. Hypomanic episode. As mentioned above, for a bipolar II diagnosis, an individual must report symptoms consistent with a major depressive episode *and* at least one hypomanic episode. An individual with bipolar II disorder must not have a history of a manic episode—if there is a history of mania, the diagnosis will be diagnosed with bipolar I. A **hypomanic episode** is like a manic episode in that the individual will experience abnormally and persistently elevated, expansive, or irritable mood and energy levels, however, the behaviors are not as extreme as in mania. Additionally, behaviors consistent with a hypomanic episode must be present for at least four days, compared to the one week in a manic episode.

Making Sense of the Disorders

Take note of the following in relation to manic and hypomanic episodes:

- A manic episode is severe enough to cause impairments in social or occupational functioning and can lead to hospitalization to prevent harm to self or others.
- A hypomanic episode is NOT severe enough to cause such impairments or hospitalization.

12.1.5.4. Cyclothymic disorder. Notably, there is a subclass of individuals who experience numerous periods with hypomanic symptoms that do not meet the criteria for a hypomanic episode and *mild* depressive symptoms (i.e., do not fully meet criteria for a major depressive episode). These individuals are diagnosed with **cyclothymic disorder** (APA, 2022). Presentation of these symptoms occur for two or more years and are typically interrupted by

periods of normal mood not lasting more than two months at a time. The symptoms cause clinically significant distress or impairment in important areas of functioning, such as social and occupational. While only a small percentage of the population develops cyclothymic disorder, it can eventually progress into bipolar I or bipolar II disorder (Zeschel et al., 2015).

12.1.6. Suicide and Self-Harm

12.1.6.1. Suicide. **Suicidal ideation** is the act of thinking, considering, or planning suicide. **Suicide** is death that is caused by an intentional, self-directed injurious behavior with the purpose to end one's life (NIH, n.d.). **Suicidal attempts** or **actions** are actions that are engaged in with the intent to end one's life without the result of death. It is important to know that there is a strong push to remove the terminology of "committed suicide," "completed suicide," perhaps even "suicide attempt" from our day to day, and empirical, language. The reason for this is that it has a negative connotation. How else do we use the word committed? Consider this: "the person *committed* a crime." We use committed, frequently, when discussing criminal activity. Moreover, when we use the term, completed suicide or attempted suicide, both indicate a "pass" or "fail," "successful" or "unsuccessful" judgment. Negative connotations with both of these scenarios are unnecessary and can be particularly painful for family members that have had a loved one die by suicide or for other individuals that have themselves engaged in suicidal actions. As such, for the purpose of this book (and we hope for any conversations you have moving forward), we will use terms such as ***died by suicide*** and *suicidal actions,* rather than *committed suicide, suicide attempt, failed suicide attempt,* etc..

While there are many theories concerning suicide, Joiner's Interpersonal-Psychological Theory of Suicidal Behavior has become a commonly endorsed theory to help (1) explain

suicidal actions and (2) implement preventative measures and treatment. His theory includes several important components. First, **thwarted belongingness**, or not feeling connected to others or feeling isolated, and **perceived burdensomeness**, the idea that an individual cannot meaningfully contribute to one's own life, other's life, or society (e.g., physical impairment, unemployment), are very important factors that contribute to an individual's likelihood to engage in suicidal actions. The third component is an individual's **acquired capability for suicidality** or the idea that, over time, an individual who has been exposed to pain or life-threatening danger are desensitized, to a degree, to death or bodily harm. For example, an individual that has previously engaged in suicidal actions or a soldier that has been exposed to combat has been exposed to painful and life-threatening events. Thus, to some degree, their sensitization to bodily harm or death may be lower than an individual that has not had these experiences. The combination of the presence of all three of these concepts (i.e., thwarted belongingness, perceived burdensomeness, and acquired capability for suicidality) contributes to an individual's desire for suicide (Joiner et al., 2009; Van Orden et al.; 2008).

12.1.6.2. Nonsuicidal Self-Injury (NSSI). NSSI, also frequently referred to as self-harm, includes any self-injurious actions that an individual engages in without the intent to end one's life. For example, an individual with NSSI may have no suicidal ideation but regularly engages in NSSI. Examples of NSSI may include cutting, burning, or picking one's skin. Essentially, any action that injures oneself, without the intent to end one's life, can be viewed as NSSI. Individuals that self-harm often report doing so because (1) they want a release from their emotions or a distraction from their internal pain or (2) they feel so numb they want to feel *something/anything.* As such, just because someone engages in self-harm does not mean they have suicidal ideation. Because (1) an individual engaging in self-harm is likely highly

emotionally distressed, (2) self-harm may increase one's acquired capacity for suicide (see above), and (3) self-harm can lead to accidental lethality. As such, self-harm should be seriously addressed and treated.

You should have learned the following in this section:

- Mood disorder fall into one of two groups – depressive or bipolar disorders – with the key distinction between the two being episodes of mania/hypomania.
- Symptoms of depression fall into one of four categories – mood, behavioral, cognitive, and physical.
- Persistent depressive disorder shares symptoms with major depressive disorder though they are usually not as severe and ebb and flow over a period of at least two years.
- Disruptive mood dysregulation disorder is characterized by chronic severe, persistent irritability that has two prominent clinical manifestations – first, frequent temper outbursts and second, chronic, persistently irritable or angry mood that is present between these outbursts.
- An individual is diagnosed with bipolar I disorder if they have ever experienced a manic episode and are diagnosed with bipolar II disorder if the criteria has only been met for a hypomanic episode.
- A manic episode is characterized by a specific period in which an individual reports abnormal, persistent, or expansive irritable mood for nearly all day, every day, for at least one week.
- A hypomanic episode is characterized by abnormally and persistently elevated, expansive, or irritable mood and energy levels, though not as extreme as in mania, and must be present for at least four days. It is also not severe enough to cause impairments or hospitalization.
- Joiner's Interpersonal-Psychological Theory of Suicidal Behavior has become a commonly endorsed theory to help (1) explain suicidal actions and (2) implement preventative measures and treatment.
- NSSI, also frequently referred to as self-harm, includes any self-injurious actions that an individual engages in without the intent to end one's life.

Continued below……

Section 12.1 Review Questions

1. What are the different categories of mood disorder symptoms? Identify the symptoms within each category.
2. What are the key differences in a major depression and a persistent depressive disorder diagnosis?
3. What is disruptive mood dysregulation disorder?
4. What is the difference between bipolar I and II disorder?
5. What are the key diagnostic differences between a hypomanic and manic episode?
6. What is an integral theory to help explain and prevent suicidal actions?
7. What is NSSI?

12.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence and comorbidity of depressive disorders.
- Describe the prevalence and comorbidity of disruptive mood dysregulation disorder.
- Describe the prevalence and comorbidity of bipolar disorders.
- Describe the prevalence of suicidality.

12.2.1. Depressive Disorders

12.2.1.1. Prevalence. According to the DSM-5-TR (APA, 2022), the 12-month prevalence rate for major depressive disorder is approximately 7% within the United States. Recall that DSM-5 persistent depressive disorder is a blend of DSM-IV dysthymic disorder and chronic major depressive disorder. The prevalence rate for DSM-IV dysthymic disorder is much lower than MDD, with a 0.5% rate among adults in the United States, while DSM-IV chronic major depressive disorder is 1.5%.

As well, individuals in the 18- to 29- year-old age bracket report the highest rates of MDD than any other age group. Women experience about twofold higher rates than men of MDD, especially between menarche and menopause (APA, 2022). The estimated lifetime prevalence for major depressive disorder in women is 21.3% compared to 12.7% in men (Nolen-Hoeksema, 2001). Regrading DSM-IV dysthymic disorder and chronic major depressive disorder, the prevalence among women is 1.5 and 2 times greater than the prevalence for men for each of these diagnoses, respectively (APA, 2022).

12.2.1.2. Comorbidity. 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). Nearly three-fourths of participants with lifetime MDD in a large-scale research study also met the criteria for at least one other DSM disorder (Kessler, Berglund, et al., 2003). MDD has been found to co-occur with substance-related disorders, panic disorder, generalized anxiety disorder, PTSD, OCD, anorexia, bulimia, and borderline personality disorder. Gender differences do exist within comorbidities such that women report comorbid anxiety disorders, bulimia, and somatoform disorders while men report comorbid alcohol and substance abuse. In contrast, those with PDD are at higher risk for psychiatric comorbidity in general and for anxiety disorders, substance use disorders, and personality disorders in particular (APA, 2022).

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

12.2.2. Disruptive Mood Dysregulation Disorder

12.2.2.1. Prevalence. Disruptive mood dysregulation disorder is common among children presenting to pediatric mental health clinics but prevalence estimates in the community are unclear. Clinical samples generally show that disruptive mood dysregulation disorder is more common in boys and younger age groups (APA, 2022).

12.2.2.2. Comorbidity. The strongest overlap is with oppositional defiant disorder though children presenting to a pediatric mental health clinic have a wide range of disruptive behavior, mood, anxiety, and autism spectrum symptoms and diagnoses.

It is worth noting that in terms of differential diagnosis, children with symptoms consistent with intermittent explosive disorder present with severe temper outbursts much like children with disruptive mood dysregulation disorder, but unlike these children, their mood is not persistently irritable or angry between outbursts.

12.2.3. Bipolar Disorders

12.2.3.1. Prevalence. The 12-month prevalence of bipolar I disorder in the United States is 1.5% and did not differ statistically between men and women. In contrast, bipolar II disorder has a prevalence rate of 0.8% in the United States and 0.3% internationally (APA, 2022) and some clinical samples suggest it is more common in women, with approximately 80-90% of individuals with rapid-cycling episodes being women (Bauer & Pfenning, 2005). Childbirth may be a specific trigger for a hypomanic episode, occurring in 10-20% of women in nonclinical settings and most often in the early postpartum period.

12.2.3.2. Comorbidity. Those with bipolar I disorder typically have a history of three or more mental disorders. The most frequent comorbid disorders include anxiety disorders, alcohol use disorder, other substance use disorder, and ADHD, along with borderline, schizotypal, and antisocial personality disorder.

Bipolar II disorder is more often than not associated with one or more comorbid mental disorders, with anxiety disorders being the most common (38% with social anxiety, 36% with specific phobia, and 30% having generalized anxiety). As with bipolar I, substance use disorders

are common with alcohol use (42%) leading the way, followed by cannabis use (20%). Premenstrual syndrome and premenstrual dysphoric disorder are common in women with bipolar II disorder especially (APA, 2022).

Finally, cyclothymic disorder has been found to be comorbid with substance-related disorders and sleep disorders.

12.2.4. Suicidality

Individuals with a depressive disorder have a 17-fold increased risk for suicide over the age- and sex-adjusted general population rate. Features associated with an increased risk for death by suicide include anhedonia, living alone, being single, disconnecting socially, having access to a firearm, early life adversity, sleep disturbance, feelings of hopelessness, and problems with decision making. Women attempt suicide at a higher rate though men are more likely to complete suicide. Finally, the premenstrual phase is considered a risk period for suicide by some (APA, 2022).

In terms of bipolar disorders, the lifetime risk of suicide is estimated to be 20- to 30- fold greater than in the general population and 5-6% of individuals with bipolar disorder die by suicide. Like depressive disorders, women attempt suicide at a higher rate though lethal suicide is more common in men with bipolar disorder. About 1/3 of individuals with bipolar II disorder report a lifetime history of suicide attempt, which is similar in bipolar I disorder, though lethality of attempts is higher in individuals with bipolar II (APA, 2022).

You should have learned the following in this section:

- Major depressive disorder is experienced by about 7% of the population in the United States, afflicting young adults and women the most.
- Bipolar I disorder afflicts 1.5% and bipolar II disorder afflicts 0.8% of the U.S. population with bipolar II affecting women more than men and no gender difference being apparent for bipolar I.
- Individuals with a depressive disorder have a 17-fold increased risk for suicide while the lifetime risk of suicide for an individual with a bipolar disorder is estimated to be 20- to 30- fold greater than in the general population and 5-6% of individuals with bipolar disorder die by suicide.
- Depressive disorders have a high comorbidity with substance use disorders, anxiety disorders, and some personality disorders.
- Bipolar disorders have a high comorbidity with anxiety disorders and substance abuse disorders while cyclothymic disorder is comorbid with substance-related disorders and sleep disorders.
- Prevalence of disruptive mood dysregulation disorder is unclear, though it is more common in boys and younger age groups. It is also common with oppositional defiant disorder and disruptive behavior, mood, anxiety, and autism spectrum symptoms and diagnoses.

Section 12.2 Review Questions

1. What are the prevalence rates of the mood disorders?
2. What gender differences exist in the rate of occurrence of mood disorders?
3. How do depressive and bipolar disorders compare in terms of suicidality (attempts and lethality)?
4. What are common comorbidities for the depressive disorders?
5. What are common comorbidities for bipolar disorders?
6. What is the prevalence of disruptive mood dysregulation disorder and what other disorders is it comorbid with?

12.3. 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.

12.3.1. 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 developing a depressive disorder. Among the biological factors are genetic factors, biochemical factors, and brain structure.

12.3.1.1. Genetics. Like with any disorder, researchers often explore the prevalence rate of depressive disorders among family members 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. Research supports this with regards to depressive disorders as there is nearly a 30% increase in relatives diagnosed with depression compared to 10% of the general population (Levinson & Nichols, 2014). Similarly, there is 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 roughly 50%, similar to that of siblings. A large-scale study found that if one identical twin was diagnosed with depression, there was a 46% chance their identical twin was diagnosed with depression. In contrast, the rate of a depression diagnosis in fraternal twins was only 20%. Despite the fraternal twin rate still being higher than that of a first-degree relative, this study provided enough evidence that there is a strong genetic link in the development of depression (McGuffin et al., 1996).

More recently, scientists have been studying depression at a molecular level, exploring possibilities of gene abnormalities as a cause for 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 that of major depressive disorder. Twin studies within bipolar disorder yielded concordance rates for identical twins at as high as 72%, yet the range for fraternal twins, siblings, and other close relatives ranged from 5-15%. It is important to note that both percentages are significantly higher than that of the general population, suggesting a strong genetic component within bipolar disorder (Edvardsen et al., 2008). The DSM-5-TR more recently reports heritability estimates around 90% in some twin studies and the risk of bipolar disorder being around 1% in the general population compared to 5-10% in a first-degree relative (APA, 2022).

12.3.1.2. 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 relationship was discovered accidentally in the 1950s when MAOIs 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 affected the 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 implicated in the development of bipolar disorder; however, the idea was that there was a drastic *increase* in serotonin during mania episodes. Unfortunately, research supports the opposite. It is believed that low levels of serotonin and *high levels* of norepinephrine may explain mania episodes (Soreff & McInnes, 2014). Despite these findings, additional research within this area is needed to conclusively determine what is responsible for the manic episodes within bipolar disorder.

12.3.1.3. 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.

12.3.1.4. Brain anatomy. Seeing as neurotransmitters have been implicated in the development of depressive disorders, it should not be a surprise that various brain structures have also been identified as contributors to mood disorders. While exact anatomy and pathways are yet to be determined, research studies implicate the **prefrontal cortex**, the **hippocampus**, and the **amygdala**. More specifically, drastic changes in blood flow throughout the prefrontal cortex have been linked with depressive symptoms. Similarly, a smaller hippocampus, and consequently, fewer neurons, has also been linked to depressive symptoms. Finally, heightened activity and blood flow in the amygdala, the brain area responsible for our fight or flight response, is also consistently found in individuals with depressive symptoms.

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

12.3.2. 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 them regardless of their behavior. The following day, the dogs were placed in a similar apparatus; however, this time they 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 preventing 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 research on **attributional style** (Nolen-Hoeksema, Girgus & Seligman, 1992). There are two types of attributional styles—positive and negative. A negative attributional style focuses on the *internal, stable*, and *global* 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 would 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 oneself, 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. Individuals with depressive symptoms often develop these maladaptive attitudes regarding everything in their life, indirectly isolating themselves from others. The **cognitive triad** also plays into the maladaptive attitudes in that the individual interprets these negative thoughts about 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 the end of the module). Among the most common are catastrophizing, jumping to conclusions, and overgeneralization. I always like to use my dad (first author's 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 line. Does this happen *every* time he is at the store? I'm doubtful, but his error in thinking leads to him believing this is 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 negatively, Beck stated that there is another set of negative thoughts that occur automatically. Research studies have continually supported Beck's maladaptive thoughts, attitudes, and errors in thinking as fundamental issues in those with depressive disorders (Lai et al., 2014; Possel & Black, 2014). Furthermore, as you will see in the treatment section (Section 4.5), cognitive strategies are among the most effective forms of treatment for depressive disorders.

12.3.3. Behavioral

The behavioral model explains depression as a result of a change in the number of rewards and punishments one receives throughout their life. This change can come from work, intimate relationships, family, or even the environment in general. Among the most influential in the field of depression is Peter Lewinsohn. He stated depression occurred in most people due to reduced positive rewards in their life. Because they were not 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 keeps receiving 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.

12.3.4. Sociocultural

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

12.3.4.1. 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 by 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 many factors lead a couple to separate or 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 oppositely, with stress and marital 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. Studies have shown that women who had three or more young children, and also lacked a close confidante and outside employment, were more likely than other mothers to become depressed (Brown, 2002).

12.3.4.2. 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 a 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.

Within the United States, many researchers have explored potential differences across ethnic or racial groups in both rates of depression, as well as presenting symptoms of those diagnosed with depression. These studies continually fail to identify any significant differences between ethnic and racial groups; however, one major study has identified a difference in the rate of recurrence of depression in Hispanic and African Americans (Gonzalez et al., 2010). While the exact reason for this is unclear, researchers propose a lack of treatment opportunities as a possible explanation. According to Gonzalez and colleagues (2010), approximately 54% of depressed white Americans seek out treatment, compared to the 34% and 40% Hispanic and African Americans, respectively. The fact that there is a large discrepancy in the use of treatment between 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.

12.3.4.3. 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 men (Schuch et al., 2014). There are a few speculations as to 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 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 face increased risk for 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 in depression symptoms (Hankin, 2009).

While many theories try to explain the gender discrepancy in depressive episodes, no 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 topics within the subject of depression, while simultaneously being the least understood phenomena within clinical psychology.

You should have learned the following in this section:

- In terms of biological explanations for depressive disorders, there is evidence that rates of depression are higher among identical twins (the same is true for bipolar disorders), that the 5-HTT gene on chromosome 17 may be involved in depressive disorders, that norepinephrine and serotonin affect depressive (both being low) and bipolar disorders (low serotonin and high norepinephrine), the hormones cortisol and melatonin affect depression, and several brain structures are implicated in depression (prefrontal cortex, hippocampus, and amygdala) and bipolar disorder (basal ganglia and cerebellum).
- In terms of cognitive explanations, learned helplessness, attributional style, and maladaptive attitudes to include the cognitive triad, errors in thinking, and automatic thoughts, help to explain depressive disorders.
- Behavioral explanations center on changes in the rewards and punishments received throughout life.
- Sociocultural explanations include the family-social perspective and multi-cultural perspective.
- Women are twice as likely to experience depression and this could be due to women being more likely to be diagnosed than men (called the artifact theory), variations in hormone levels in women (hormone theory), women being more likely to experience chronic stressors (life stress theory), the fostering of an interdependent functioning in women (gender roles theory), and that women are more likely to intently focus on their symptoms (rumination theory).

Section 12.3 Review Questions

1. How do twin studies explain the biological causes of mood disorders?
2. What brain structures are implicated in the development of mood disorders? Discuss their role.
3. What is learned helplessness? How has this concept been used to study the development and maintenance of mood disorders?
4. What is the cognitive triad?
5. What are common cognitive distortions observed in individuals with mood disorders?
6. What are the identified theories that are used to explain the gender differences in mood disorder development?

12.4. Assessment

Section Learning Objectives

- Describe assessment tools commonly used to diagnose mood disorders.

In general, assessment includes a thorough interview with a caregiver and the child/adolescent. The Diagnostic Interview Schedule for Children (DISC) may be utilized which is a structured diagnostic interview that is administered to caregivers to help inform diagnostic decisions for children. The DISC screens for relevant disorders in childhood, and helps the clinician ensure that they have screened for full diagnostic criteria of disorders, and fully assessed for, not only the child's presenting concern, but comorbid disorders as well. Another option, the Kiddie-Schedule for Affective Disorders and Schizophrenia or K-SADS is a semi-structured interview, that can even be implemented with a child/adolescent, that also screens and assesses for childhood psychological disorders.

Observations may be largely informal. Although observations may be helpful at times, most of the diagnostic decision making will be reliant on interview reports and objective measures.

Objective measures are heavily utilized. Measures may be completed by the child, depending on their age, teachers, and parents. General emotional and behavioral measures, such as the BASC-3 (discussed in previous chapters) as well as narrow-band measures that directly assess depression and mania are utilized. The Children's Depression Inventory (CDI-2), Revised Children's Anxiety and Depression Scale (RCADS), Child Bipolar Questionnaire (CBQ),

Pediatric Behavior Rating Scale (PBRS), and Parent-Young Mania Rating Scale (P-YMRS) can be used to assess for depressive- and bipolar-related symptoms in children.

You should have learned the following in this section:

- Assessment includes a thorough interview with a caregiver and the child/adolescent, such as the Diagnostic Interview Schedule for Children (DISC).
- Objective measures are heavily utilized such as the CDI-2, RCADS, CBQ, PBRS, and P-YMRS.
- Observations can be used, but are not the primary assessment tool.

Section 12.4 Review Questions

1. What assessment tools that are used to assess mood disorders in children and adolescents?

12.5. 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.

12.5.1. 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 ones 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).

12.5.1.1. 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.

12.5.1.2. Psychopharmacology - Selective serotonin reuptake inhibitors (SSRIs). SSRIs 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 SSRIs include but are not limited to nausea, insomnia, and reduced sex drive.

SSRIs 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 neurons. 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 small differences are beneficial to patients in that there are a few treatment options to maximize medication benefits and minimize side effects.

12.5.1.3. 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 like SSRIs in that they work by affecting 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 postsynaptic neurons. While effective, tricyclic antidepressants have been increasingly replaced by SSRIs due to their reduced side effects. However, tricyclic antidepressants have been shown to be more effective in treating depressive symptoms in individuals who have not been able to achieve symptom reduction via other pharmacological approaches.

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

12.5.1.4. Psychopharmacology - Monoamine oxidase inhibitors (MAOIs). The use of MAOIs as a treatment for depression began serendipitously as patients in the early 1950s reported reduced depression symptoms while on the medication to treat tuberculosis. Research studies confirmed that MAOIs were effective in treating depression in adults outside the treatment of tuberculosis. Although still prescribed, they are not typically first-line medications due to their safety concerns with hypertensive crises. Because of this, individuals on MAOIs have strict diet restrictions to reduce their risk of hypertensive crises (Shulman, Herrman & Walker, 2013).

How do MAOIs work? In basic terms, monoamine oxidase is released in the brain to remove excess neurotransmitters norepinephrine, serotonin, and dopamine. MAOIs essentially prevent the monoamine oxidase (hence the name monoamine oxidase *inhibitors*) from removing these neurotransmitters, thus resulting in 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, weight gain, etc. (APA, 2010). Despite these side effects, studies have shown that individuals prescribed MAOIs for depression have a treatment response rate of 50-70% (Krishnan, 2007). Overall, despite their effectiveness, MAOIs are likely the best treatment for late-stage, 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.

12.5.1.5. Psychotherapy - Cognitive behavioral therapy (CBT). CBT was founded by Aaron Beck in the 1960s 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 triangle**—cognitions (thoughts), behaviors, and emotions. Beck believed that these three components are interconnected, and therefore, affect one another. It is believed that CBT can improve emotions in depressed patients by changing both cognitions (thoughts) and behaviors, which in return enhances 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. CBT generally follows four phases of treatment:

- **Phase 1: Increasing pleasurable activities.** Similar to behavioral activation (see below), the clinician encourages the patient to identify and engage in activities that are pleasurable to the individual. The clinician can help the patient to select the activity, as well as help them plan 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 can 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.

12.5.1.6. 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 Ferster (1973), as well as Lewinsohn

and colleagues (Lewinsohn, 1974; Lewinsohn, Biglan, & Zeiss, 1976), the goal of BA is to increase the frequency of behaviors so that individuals have opportunities to experience greater contact with sources of reward in their lives. 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 they do not impact the outcome of their pleasurable activities. Finally, the clinician works with the patient on effective social skills. By minimizing negative behaviors and maximizing pleasurable activities, the individual will receive more positive reward and reinforcement from others and their environment, thus improving their overall mood.

12.5.1.7. Psychotherapy - Interpersonal therapy (IPT). IPT was developed by Klerman, Weissman, and colleagues in the 1970s as a treatment arm for a pharmacotherapy study of depression (Weissman, 1995). The treatment was created based on data from post-World War II individuals who expressed a substantial 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 is that depressive episodes compromise interpersonal functioning, which 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 related to the depressive symptoms and solve this crisis so the patient can improve their life situation while relieving depressive symptoms.

12.5.1.8. 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: concurrently, sequentially, or within a stepped manner (McGorry et al., 2010). With a stepped manner 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 preferences may produce better outcomes than clinician-driven treatment decisions.

12.5.1.9. A note about treating disruptive mood dysregulation disorder. Specific treatment guidelines have not been established for disruptive mood dysregulation disorder. Given its relatively recent emergence, we still have a long way to go before we fully understand treatment options. However, some research that examined clinical presentations closely related to disruptive mood dysregulation disorder have been conducted. For example, a randomized control trial indicated that children with severe mood disturbances did not benefit from lithium (as cited by Rao, 2014). Other research indicates children with similar presentations may also benefit from some atypical antipsychotics (e.g., risperidone), combined treatment of behavioral

therapy plus divalproex sodium, or combined treatment of atypical antipsychotics and stimulants (as cited by Rao, 2014). Again, these studies did not specifically examining children with disruptive mood dysregulation disorder; and as such, we cannot necessarily translate these findings directly to children with disruptive mood dysregulation disorder.

Like psychopharmacological options, psychotherapy options are largely unknown. It is hypothesized that some behavioral interventions, that also target parenting strategies, may be helpful (Rao, 2014).

12.5.2. Bipolar Disorder

12.5.2.1. Psychopharmacology. Unlike treatment for MDD, there is some controversy regarding 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 potent in treating depressive symptoms, and therefore, the combination approach is believed to help manage 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). Antidepressants often 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.

12.5.2.2. 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.

Moreover, **Interpersonal Social Rhythm Therapy (IPSRT)** is an evidenced-based treatment for bipolar disorder. The goal is to help an individual regulate their daily routines through consistent, daily routines and sleeping schedules (*social rhythm*) and help the individual recognize how these routines impact mood. Another goal is to help the individual recognize how their mood is impacted by major life events (*interpersonal*; SAMHSA, 2018, March). The treatment has four phases:

1. Initial: Begin to understand the child's routines
2. Intermediate: Implement helpful social rhythms.
3. Maintenance: Reinforce rhythms that have been established. Build child and parent's self-efficacy by using strategies learned.
4. Final: Reduce treatment frequency until treatment is fully terminated.

IPSRT was originally designed and implemented with adults. As such, some adaptations may need to be made when implementing the treatment with adolescents. However, overall, with adaptations (e.g., including parents in therapy, depending on age of the adolescent; additional targets of school functioning added, etc.), IPSRT appears helpful and beneficial for youth with bipolar disorder (Hlastala, Kotler, McClellan, & McCauley, 2010)

Family-focused treatment (**FFT**) for adolescents with bipolar disorder has also been found to be beneficial (Mikolwitz et al., 2000). This therapy, originally designed for schizophrenia, includes all immediate family members in therapy. It also focuses on several areas including psychoeducation about bipolar disorder and its symptoms, the causes of bipolar disorder, and the importance of medication compliance. Families learn how to recognize and respond to symptoms and how to implement helpful coping strategies. Moreover, given that negative expressed emotions can increase symptoms of bipolar disorder, efforts to limit and reduce problematic interactions with the family are made (Society of Clinical Psychology, 2016).

12.5.3. Suicide

Jobes, Rudd, Overholser, & Joiner (2007) and Rudd, Mandrusiak & Joiner (2006) have outlined and provided many issues and recommendations for the prevention of suicide. One of the problems they highlighted was the use of "No Suicide Contracts." A no suicide contract is essentially when a clinician "contracts" with the client to not engage in suicide. This method is **not** effective. It essentially highlights what *not to* do and does not give an individual alternative choice of *what to do.* This strategy has not been shown to be effective whereas a similar alternative, the *coping card*, which gives individuals an outline of behaviors/actions to engage in rather than an outline of what not to do has been shown to be beneficial.

There are also several phone applications, such as Virtual Hope Box, that can be downloaded that facilitate creating a coping card. A helpful number and agency to utilize is the 988 Suicide and Crisis Lifeline, formerly called the National Suicide Prevention Lifeline. As their website states, "The Lifeline's network of over 200 crisis centers has been in operation since 2005, and has been proven to be effective. It's the counselors at these local crisis centers

who answer the contacts the Lifeline receives every day. Numerous studies have shown that callers feel less suicidal, less depressed, less overwhelmed and more hopeful after speaking with a Lifeline counselor" (https://988lifeline.org/).

An effective intervention and prevention strategy for suicide prevention is firearm restriction, or means restriction, in general. For example, it has been shown that individuals that die by suicide more often have firearms in their home and that having a firearm unlocked and loaded is a higher risk factor for suicide (Conwell, et al., 2002). Although this seems like a straightforward intervention, it does have some barriers. However, efforts to restrict means from an individual that is experiencing suicidal ideation is our most effective and concrete method of prevention. As such, all steps to ensure means are restricted as much as possible should be taken.

12.5.4. Outcome of Treatment

12.5.4.1. Depressive treatment. As we have discussed, major depressive disorder has a variety of treatment options, all found to be efficacious. However, research suggests that while psychopharmacological interventions are more effective in rapidly reducing symptoms, psychotherapy, or even a combined 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, 2022).

12.5.4.2. 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. Bipolar patients often desire the euphoric highs that are associated with manic and hypomanic episodes, leading them to forgo their medication. A combination of psychopharmacology and psychotherapy aimed at increasing the rate of adherence to medical treatment may be the most effective treatment option for bipolar I and II disorder.

You should have learned the following in this section:

- Treatment of depressive disorders include psychopharmacological options such as anti-depressant mediations, SSRIs, tricyclic antidepressants, and MAOIs and/or psychotherapy options to include CBT, behavioral activation (BA), and interpersonal therapy (IPT). A combination of the two main approaches often works best, especially in relation to maintenance of wellness.
- Treatment of bipolar disorder involves mood stabilizers such as Lithium and psychological interventions with the goal of medication adherence, as well as social skills training and problem-solving skills.
- Regarding depression, psychopharmacological interventions are more effective in rapidly reducing symptoms, while psychotherapy, or even a combined treatment approach, is more effective in establishing long-term relief of symptoms.
- A combination of psychopharmacology and psychotherapy aimed at increasing the rate of adherence to medical treatment may be the most effective treatment option for bipolar I and II disorder.

Section 12.5 Review Questions

1. Discuss the effectiveness of the different pharmacological treatments for mood disorders.
2. What are the four phases of CBT? How do they address symptoms of mood disorder?
3. What is ITP and what are its main treatment strategies?
4. What are the effective treatment options for bipolar disorder?

Apply Your Knowledge

CASE VIGNETTE

George, a 6-year-old boy, was brought to a psychologist because his parents are concerned by his frequent irritable and angry mood. He often gets extremely upset by small things. For example, when at the grocery store, his mother could not find his favorite cereal, so she picked up a different cereal instead. When she got home and told George she couldn't find his favorite cereal, he cried and screamed and kicked for 30 minutes. George's mother explained that he does this frequently. His teachers have expressed concerns and he has been sent to the principal's office several times due to outbursts. George no long gets invited for play dates with his friends either. George doesn't sleep well, and his mother wonders if this is part of the problem. George doesn't seem sad, but he is certainly irritable. She hasn't necessarily noticed a change in George's appetite or activities he enjoys. George's mother and father separated a year ago and George's mother reported she noticed a change in George when this happened.

George's mother is unsure of family history of mood disorders because her family and George's father's family did not discuss mental health openly while growing up.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. Do you think George is experiencing a mood disorder or behavioral disorder? Why?
2. If you were to suspect a mood disorder, which disorder may be the most fitting? Why? Do you need more information? If so, what information?
3. What treatment options may be best for George?
4. Are there family and social factors that should be considered? If so, what can be done to address these factors?

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. We also learned about disruptive mood dysregulation disorder. Be sure you are clear on what makes them different from one another in terms of their clinical presentation, diagnostic criteria, epidemiology, comorbidity, and etiology. This will help you with understanding what treatment options there are and their efficacy.

In the next module we will discuss anxiety-related disorders.

Part V. Mood and Anxiety-Related Disorders

Module 13:
Anxiety Disorders

Module 13: Anxiety Disorders

Module Overview

In Module 13, we will discuss matters related to anxiety disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include separation anxiety disorder, selective mutism, generalized anxiety disorder, specific phobia, agoraphobia, social anxiety disorder, and panic disorder. Be sure you refer to 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. Prevalence and Comorbidity
- 13.3. Etiology
- 13.4. Assessment
- 13.5. Treatment

Module Learning Outcomes

- Describe how anxiety disorders present.
- Describe the prevalence and comorbidity of anxiety disorders.
- Describe the etiology of anxiety disorders.
- Describe assessment and treatment options for anxiety disorders.

13.1. Clinical Presentation

Section Learning Objectives

- Describe how separation anxiety disorder presents.
- Describe how selective mutism presents.
- Describe how generalized anxiety disorder presents.
- Describe how specific phobia presents.
- Describe how agoraphobia presents.
- Describe how social anxiety disorder presents.
- Describe how panic disorder presents.

The hallmark symptoms of anxiety-related disorders are excessive fear and anxiety and related behavioral disturbances. How do we distinguish fear from anxiety? The DSM says that fear is an emotional response to a real or perceived imminent threat which leads to "…surges of autonomic arousal necessary for flight or flight, thoughts of immediate danger, and escape behaviors." Anxiety, on the other hand, is the anticipation of a future threat leading to, "…muscle tension and vigilance in preparation for future danger and cautious or avoidant behaviors" (APA, 2022, pg. 215). The anxiety disorders differ from one another in the types of objects or situations that lead to fear, anxiety, or avoidance behavior. We will cover separation anxiety disorder, selective mutism, generalized anxiety disorder, specific phobia, agoraphobia, social anxiety disorder, and panic disorder.

13.1.1. Separation Anxiety Disorder

Separation anxiety disorder is characterized by excessive fear or anxiety concerning being separated from those to whom the individual is attached. A child may worry about the caregiver becoming seriously ill, dying, or being permanently separated from them, and show persistent and excessive fear of or reluctance about being alone or without this figure at home or other settings. They may refuse to go to school or other places and worry about experiencing an untoward event such as getting lost or being kidnapped that causes separation from a major attachment figure. They may refuse to sleep in their own bed or have recurring nightmares that involve separation from their attachment figure. These children may also have several physical manifestations of the anxiety including headaches and stomachaches when they are separated from, or anticipate separation from, the major attachment figure. This fear, anxiety, or avoidance is persistent and has lasted for at least four weeks in children and adolescents. For adults, it must last 6 months or more.

Adults or children with the disorder may exhibit social withdrawal, apathy, difficult concentrating or playing, and sadness and for children, the school refusal can lead to academic difficulties and social isolation. Children may also display anger and act aggressively toward the person forcing separation from their attachment figure and can become homesick. Children diagnosed with separation anxiety disorder are seen as demanding, intrusive, and in need of constant attention (APA, 2022).

13.1.2. Selective Mutism

Selective mutism is characterized by an absence of speech in particular social situations in which a person is expected to speak, such as at school or work, despite speaking in other situations. This lack of speech is not due to a communication disorder; does not occur exclusively during the course of autism spectrum disorder, schizophrenia, or another psychotic disorder; or due to a lack of knowledge of, or comfort with, the spoken language required in a specific social situation (APA, 2022). For example, a child with selective mutism may speak fluently and freely with a trusted caregiver in the privacy of their home (providing evidence that the child does not have a speech, language, or communication disorder) but produces no speech at school with peers or a teacher. The disturbance must have lasted for at least one month and is often marked by high social anxiety. According to the DSM, associated features include, "excessive shyness, fear of social embarrassment, social isolation and withdrawal, clinging, compulsive traits, negativism, temper tantrums, or mild oppositional behavior" (APA, 2022, pg. 222). The onset of the disorder is usually before 5 years but may not become apparent until entry into school.

13.1.3. Generalized Anxiety Disorder

Generalized anxiety disorder is characterized by an underlying excessive anxiety and worry related to a wide range of events or activities and lasting for more days than not for at least six months. While many individuals experience some degree of worry throughout the day, individuals with generalized anxiety disorder experience worry of greater intensity and for longer periods than the average person (APA, 2022). 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. To receive a diagnosis of generalized anxiety disorder, three or more of the following somatic symptoms must be present in adults as well: restlessness, fatigue, difficultly concentrating, irritability, muscle tension, and problems sleeping (APA, 2022).

13.1.4. Specific Phobia

Specific phobia is distinguished by 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 or situation. When individuals are face-to-face with their specific phobia, immediate fear is present, and the phobic object or situation is actively avoided or endured. It should also be noted that these fears are excessive and irrational, often severely impacting one's daily functioning. The fear, anxiety, or avoidance is persistent, lasting at least six months (APA, 2022).

Individuals can experience multiple specific phobias at the same time. In fact, nearly 75% of individuals with a specific phobia report fear of more than one object and the average individual fears three or more objects or situations (APA, 2022). When making a diagnosis of specific phobia, it is important to identify the stimulus. Among the most diagnosed specific phobias are animals, natural environment (height, storms, water), blood-injection-injury (needles, invasive medical procedures), or situational (airplanes, elevators, enclosed places). In terms of gender differences, women predominantly experience animal, natural environment, and situational specific phobias while blood-injection-injury phobia is experienced by both men and women equally (APA, 2022).

13.1.5. Agoraphobia

Agoraphobia is defined as intense fear or anxiety triggered by two or more of the following: 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 crowd or standing in line; or being outside of the home alone. The individual fears or avoids these situations because they believe something terrible may occur and due to concern over not being able to escape or help not being available (APA, 2022). Active avoidance of the situations occurs and can be behavioral such as changing daily routines or using delivery to avoid entering a restaurant or cognitive such as using distraction to bear with an agoraphobic situation. An example of a behavioral avoidance would be a child refusing to join a sport steam or only engaging in online gaming for social interaction. The avoidance can result in the person being homebound. The fear or anxiety is out of proportion to the actual danger they pose and has been present for at least six months.

13.1.6. Social Anxiety Disorder

For **social anxiety disorder**, the anxiety or fear relates to 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, or unlikeable, 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 and have occurred for six months or more.

To explain social anxiety in greater detail, let's review the story of Mary. Mary reported the onset of her social anxiety disorder in early elementary school when teachers would call on students to read parts of their textbook aloud. Mary stated that she was fearful of making mistakes while reading and to alleviate this anxiety, she would read several sections ahead of the class to prepare for her turn to read aloud. Despite her preparedness, one day in 5th grade, Mary was called to read, and she stumbled on a few words. While none of her classmates realized her mistake, Mary was extremely embarrassed and reported higher levels of anxiety during future *read aloud* moments in school. In fact, when she was called upon, Mary stated she would completely freeze up and not talk at all. After a few moments of not speaking, her teacher would skip Mary and ask another student to read her section. It took several years and a very supportive teacher for Mary to begin reading aloud in class again.

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

It is important to note that the cognitive interpretation of these social events is often excessive and out of proportion to the actual risk of being negatively evaluated. As we saw in Mary's case, when she stumbled upon her words while reading to the class, none of her peers even noticed her mistake. Situations in which individuals experience anxiety toward a real threat, such as bullying or ostracizing, would not be diagnosed with social anxiety disorder as the negative evaluation and threat are real.

13.1.7. 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 of fear or impending doom along with at least four physical or cognitive symptoms. Physical symptoms include heart palpitations, sweating, trembling or shaking, shortness of breath, feeling as though they are being choked, chest pain, nausea, dizziness, chills or heat sensations, and numbness/tingling. Cognitive symptoms may consist of feelings of derealization (feelings of unreality) or depersonalization (feelings of being detached from oneself), the fear of losing control or 'going crazy,' or the fear of dying (APA, 2022). While symptoms generally peak within a few minutes, 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 are primarily "out of the blue," they cause significant worry or anxiety in the individual as they are unsure of when the next attack will happen. In contrast to unexpected there are also expected panic attacks, or those that have an obvious trigger. The DSM-5-TR states that presence of expected panic attacks does not rule out the diagnosis of panic disorder as about half of individuals diagnosed with the disorder in the United States and Europe have both types of attacks (APA, 2022).

In some individuals, significant behavioral changes such as fear of leaving their home or attending large events occur as the individual is fearful an attack will happen in one of these situations, causing embarrassment. Additionally, individuals report worry that others 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.

The frequency and intensity of these panic attacks vary widely among individuals. Some people report panic attacks occurring once a week for months on end, others report more frequent attacks multiple times a day, but then experience weeks or months without any attacks. The intensity of symptoms also varies among individuals, with some patients experiencing four or more symptoms (full-symptom) or less than four (limited-symptom. Furthermore, individuals report variability within their panic attack symptoms, with some panic attacks presenting with more symptoms than others. To be diagnosed with panic disorder, the individual must present with more than one unexpected full-symptom panic attack (APA, 2022).

You should have learned the following in this section:

- All anxiety disorders share the hallmark symptoms of excessive fear or worry related to behavioral disturbances.
- Separation anxiety disorder is characterized by excessive fear or anxiety concerning being separated from those to whom the individual is attached.
- Selective mutism is characterized by an absence of speech in particular social situations in which a person is expected to speak, such as at school or work, despite speaking in other situations.
- Generalized anxiety disorder is characterized by an underlying excessive worry related to a wide range of events or activities and an inability to control their worry through coping strategies.
- Specific phobia is characterized by fear or anxiety specific to an object or a situation and individuals can experience fear of more than one object.
- Agoraphobia is characterized by intense fear related to situations in which the individual is in public situations where escape may be difficult and help may not be able to come.
- Social anxiety disorder is characterized by fear or anxiety related to social situations, especially when evaluation by others is possible.
- Panic disorder is characterized by a series of recurrent, unexpected panic attacks coupled with the fear of future panic attacks.

Section 13.1 Review Questions

1. What is the difference between fear and anxiety?
2. What is separation anxiety disorder?
3. What is selective mutism and how does it differ from social anxiety disorder?
4. What are the key differences between generalized anxiety disorder and agoraphobia?
5. Individuals with social anxiety disorder will experience both physical and cognitive symptoms, particularly when presented with social interactions. What are these symptoms?
6. What are the common types of specific phobias?
7. What are the physical and cognitive symptoms observed during panic disorder?
8. What are the key components of panic disorder?

13.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe prevalence and comorbidity of separation anxiety disorder.
- Describe prevalence and comorbidity of selective mutism.
- Describe prevalence and comorbidity of generalized anxiety disorder.
- Describe prevalence and comorbidity of specific phobia.
- Describe prevalence and comorbidity of agoraphobia.
- Describe prevalence and comorbidity of social anxiety disorder.
- Describe prevalence and comorbidity of panic disorder.

13.2.1. Separation Anxiety Disorder

Separation anxiety disorder occurs in approximately 4% of children and decreases in prevalence from childhood through adolescence and adulthood. For instance, only 1.6% of adolescents in the United States have the disorder and for adults, 12-month prevalence ranges from 0.9% to 1.9%. In toddlers, separation anxiety disorder occurs about equally between girls and boys but in school-aged children, it is more prevalent for girls than boys. Separation anxiety disorder is highly comorbid with other anxiety disorders, such as generalized anxiety disorder and specific phobia (APA, 2022).

13.2.2. Selective Mutism

Selective mutism is relatively rare, occurring in only 0.03% to 1.9% of the population and is more common in young children than adolescents and adults. There is conflicting evidence about gender, with some studies/samples showing equal distribution and others showing a higher prevalence in girls. Prevalence does not vary by race/ethnicity. Selective mutism is highly comorbid with other anxiety disorders, particularly social anxiety disorder followed by separation anxiety disorder and specific phobia. Communication disorders or delays also appear in some children with selective mutism (APA, 2022).

13.2.3. Generalized Anxiety Disorder

The 12-month prevalence for generalized anxiety disorder is estimated to be 0.9% of adolescents and 2.9% of the adult general population of the United States while the mean 12-month prevalence around the world is 1.3% (with a range of 0.2% to 4.3%). The disorder occurs more frequently in women and adolescent girls, those of European descent, and those living in high-income countries (APA, 2022).

There is a high comorbidity between generalized anxiety disorder and the other anxiety-related disorders, as well as unipolar depressive disorders. Comorbidity with substance use, neurodevelopmental, neurocognitive, psychotic, and conduct disorders is less common for those afflicted with generalized anxiety disorder. Generalized anxiety disorder is associated with higher levels of suicidal ideation and behavior and psychological autopsy studies reveal it is the most frequent anxiety disorder diagnosed in suicides (APA, 2022).

13.2.4. Specific Phobia

The prevalence rate for specific phobia is 8-12% in the United States and about 6% in European countries. Prevalence rates in children average about 5% across varies countries, with a range of 3% to 9% and are approximately 16% in adolescents ages 13-17 years in the United States. There is a 2:1 ratio of females to males diagnosed with specific phobia. Prevalence rates are lower in older individuals and those from Asia, Africa, and Latin America.

Seeing as the onset of specific phobia occurs at a younger age than most other anxiety disorders, it is generally the primary diagnosis. Children/teens diagnosed with a specific phobia are at an increased risk for additional psychopathology later in life such as other anxiety disorders, depressive and bipolar disorders, substance-related disorders, and somatic symptom disorder. Additionally, personality disorders, in particular dependent personality disorder, are comorbid. Specific phobia is associated with the transition from suicidal ideation to attempt (APA, 2022).

13.2.5. Agoraphobia

The prevalence rate of agoraphobia worldwide for adolescents and adults is 1% to 1.7%. It may occur in childhood, but incidence peaks in late adolescence and early adulthood. As with other anxiety disorders, women are twice as likely to be diagnosed with it.

As with other anxiety disorders, common comorbid mental disorders include other anxiety disorders and depressive disorders. Agoraphobia is also comorbid with PTSD and alcohol use disorder. For those with comorbid major depressive disorder, the agoraphobia is more treatment-resistant compared to those with agoraphobia alone. About 15% of patients diagnosed with agoraphobia report suicidal thoughts or behavior (APA, 2022).

13.2.6. Social Anxiety Disorder

The overall prevalence rate of social anxiety disorder is significantly higher in the United States than in other countries, with an estimated 7% of the U.S. population diagnosed with social anxiety disorder, compared to 0.5% to 2.0% worldwide (median prevalence in Europe is 2.3%). Twelve-month prevalence rates in young adolescents are about half those in adults. Regarding gender, there is a higher diagnosis rate in females than males. This gender discrepancy is greater among adolescents and young adults. Finally, non-Hispanic whites in the United States have a higher prevalence rate than Asian, Latinx, African American, and Caribbean Black descent (APA, 2022).

Among the most common comorbid diagnoses with a social anxiety disorder are other anxiety-related disorders, major depressive disorder, and substance-related disorders. The high comorbidity rate among anxiety-related disorders and substance-related disorders is likely connected to the efforts of self-medicating to deal with social fears. For example, an individual with social anxiety disorder may consume more alcohol in social settings in efforts to alleviate the anxiety of the social situation. The comorbidity with major depressive disorder may be due to the chronic social isolation associated with social anxiety disorder. Comorbidity has also been found with body dysmorphic disorder and avoidant personality disorder. In children, social anxiety disorder is comorbid with high-functioning autism spectrum disorder and selective mutism.

13.2.7. Panic disorder

The 12-month prevalence for panic disorder in the general population is estimated at around 2-3% in adults and adolescents across the United States and several European countries. Higher rates of panic disorder are found in American Indians and non-Latinx whites. Females are more commonly diagnosed than males with a 2:1 diagnosis rate. Prevalence declines from about 1.2% in adults older than 55 to 0.7% in adults aged 64 and up.

Panic disorder rarely occurs in isolation, as 80% of individuals report symptoms of other anxiety disorders, major depressive disorder, bipolar I and bipolar II disorder, and possibly mild alcohol use disorder. Some individuals diagnosed with panic disorder also develop a substance-related disorder, likely as an attempt to treat their anxiety with alcohol or other substances. About 25% of patients report suicidal thoughts and the disorder may increase the risk for future suicidal behaviors but not deaths. (APA, 2022).

Unlike some of the other anxiety disorders, there is a high comorbidity with general medical symptoms. More specifically, individuals with panic disorder are more likely to report somatic symptoms such as dizziness, cardiac arrhythmias, COPD, asthma, irritable bowel syndrome, and hyperthyroidism (APA, 2022). The relationship between panic symptoms and these conditions is unclear.

You should have learned the following in this section:

- Prevalence rates for anxiety disorders range from 0.03% for selective mutism up to 12% for specific phobia.
- For most anxiety disorders, females are twice as likely to be diagnosed.
- Many anxiety disorders are comorbid with one another.
- Other common comorbid disorders include depressive disorders and substance-related disorders.
- Agoraphobia has a high comorbidity with PTSD and panic disorder with general medical symptoms.
- Most anxiety disorders are associated with suicidal thoughts and behaviors, but not always deaths.

Section 13.2 Review Questions

1. Create a table of the prevalence rates across the various anxiety related disorders. What are the differences between the disorders?
2. How do prevalence rates vary as a function of gender, race, nationality, and age?
3. What other disorders commonly occur with specific anxiety related disorders and why?
4. What anxiety-related disorder has a high comorbidity with medical symptoms?
5. What is the relationship of the disorders with suicidal ideation and attempts/behaviors? Be specific.

13.3. 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.

13.3.1. Biological

13.3.1.1. 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 account for more anxiety disorders than genetics alone (Bienvenu, Davydow, & Kendler, 2011). The quest to identify specific genes that may predispose individuals to develop anxiety disorders has led researchers to the serotonin transporter gene (5-HTTLPR). Mutation of the 5-HTTLPR gene is related to a reduction in serotonin activity and an increase in anxiety-related personality traits (Munafo, Brown, & Hairiri, 2008). Evidence also exists that separation anxiety disorder is heritable, with heritability estimated at 73% in a community sample of 6-year-old twins.

13.3.1.2. 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 to ready the body for a response. First, the amygdala triggers the hypothalamic-pituitary-adrenal (HPA) axis to prepare for immediate action— either

to fight or flight. The second pathway is activated by the feared stimulus itself, by sending a sensory signal to the **hippocampus** and **prefrontal cortex**, to determine if the threat is real or imagined. If it is determined that no threat is present, the amygdala sends a calming response to the HPA axis, thus reducing the level of fear. If a threat is 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 the 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 responsible for 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.

13.3.2. Psychological

13.3.2.1. Psychological - Cognitive. The cognitive perspective on the development of anxiety related disorders centers around dysfunctional thought patterns. 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 potentially stressful events, which contributes to an overall heightened 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 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 threat. This misevaluation of symptoms as impending disaster likely maintain symptoms as the cognitive misinterpretations to physiological arousal creates a negative feedback loop, leading to more physiological changes.

Social anxiety is also primarily 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.

13.3.2.2. Psychological – Behavioral. The behavioral explanation for the development of anxiety disorders is mainly reserved for phobias—both specific and social phobia. More precisely, behavioral theorists focus on **respondent conditioning** - when two events that occur close together become strongly associated with one another, despite their lack of causal relationship (see Module 2 for an explanation of respondent conditioning). Watson and Rayner's (1920) infamous Little Albert experiment is an example of how respondent conditioning can be used to induce fear through associations. In this study, Little Albert developed a fear of white rats by pairing a white rat with a loud sound. This experiment, although lacking ethical standards, was groundbreaking in the development of learned behaviors. Over time, researchers have been able to replicate these findings (in more ethically sound ways) to provide further evidence of the role of respondent conditioning in the development of phobias.

13.3.2.3. Psychological – Modeling is another behavioral explanation of the development of specific and social phobias. In modeling, an individual acquires a fear though observation and imitation (Bandura & Rosenthal, 1966). For example, when a young child observes their parent display irrational fear of an animal, the child may then begin to display similar behavior. Similarly, seeing another individual being ridiculed in a social setting may

increase the chances of developing 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 object or situation is not something that should be feared.

While modeling and respondent conditioning largely explain the development of phobias, there is some speculation that the accumulation of many these learned fears will develop into generalized anxiety disorder. Through **stimulus generalization,** or the tendency for the conditioned stimulus to evoke similar responses to other stimuli, a fear of one stimulus (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.

13.3.3. 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 significant contributors to anxiety disorders, additional sociocultural influences such as gender and discrimination have also received considerable attention, mainly due to the epidemiological nature of the disorder.

Gender has largely been researched within anxiety disorders due to the consistent discrepancy in the 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 minorities 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.

For separation anxiety disorder, life stressors, especially those related to a loss (e.g., death of a relative or pet, change in schools, parental divorce, or move to a new neighborhood) lead to its development. Additionally, being bullied during childhood and a history of parental overprotection and intrusiveness have been shown to be risk factors.

In the case of selective mutism, parental shyness, social isolation, and social anxiety can be risk factors, as well as negative activity (neuroticism) and behavioral inhibition. That said, temperamental risk factors are not well identified (APA, 2022).

You should have learned the following in this section:

- Biological causes of anxiety disorders include the serotonin transporter gene (5-HTTLPR); brain structures to include the amygdala, hippocampus, and prefrontal cortex; and the locus coeruleus and corticostriatal-thalamocortical (CSTC) circuit in relation to panic disorder.
- Psychological causes of anxiety disorders include maladaptive assumptions, the linking of events through respondent conditioning, modeling, and stimulus generalization as it relates to generalized anxiety disorder.
- Sociocultural causes of anxiety disorders include social pressures leading to a higher rate of diagnosis for women and discrimination and prejudice which affects ethnic minorities and other marginalized groups. Life stressors are a risk factor for social anxiety disorder.

Section 13.3 Review Questions

1. Discuss the biological etiology of panic disorders. What brain structures and neurotransmitters are involved?
2. How does the cognitive model explain the development and maintenance of anxiety related disorders?
3. What is the difference between emotion-focused and problem-focused coping strategies? How do these two coping strategies explain differences in anxiety related disorders?
4. What are the effects of prejudice and discrimination on the development of anxiety disorders?

13.4. Assessment

Section Learning Objectives

- Describe assessment tools commonly used for anxiety disorders.

Similar to mood disorders, assessment will include a thorough *interview* with a caregiver and the child/adolescent. Tools include the Diagnostic Interview Schedule for Children (DISC) and Kiddie-Schedule for Affective Disorders and Schizophrenia or K-SADS.

Observations may be largely informal. Although observations may be helpful at times, most of the diagnostic decision making will rely on interview reports and objective measures.

Objective measures are also heavily utilized. Measures may be completed by teachers, parents, and the child, depending on their age. General emotional and behavioral measures, such as the BASC-3 (discussed in previous chapters) as well as narrow-band measures that directly assess anxiety such as the Revised Children's Manifest Anxiety Scale (RCMAS) and Multidimensional Anxiety Scale for Children, 2nd Edition (MASC 2), are used.

You should have learned the following in this section:

- Assessment of anxiety disorders occurs using interview tools, observations, and objective measures.

Section 13.4 Review Questions

1. What tools are used in assessing anxiety disorders?
2. Are some assessment tools better than others? How so?

13.5 Treatment

Section Learning Objectives

- Describe treatment options separation anxiety disorder.
- Describe treatment options selective mutism.
- Describe treatment options for generalized anxiety disorder.
- Describe treatment options for specific phobia.
- Describe treatment options for agoraphobia.
- Describe treatment options for social anxiety disorder.
- Describe treatment options for panic disorder.

13.5.1. Separation Anxiety Disorder

13.5.1.1. Exposure. Exposure therapy is often utilized to reduce anxiety. With separation anxiety disorder, the child may be encouraged to gradually separate from their caregiver (gradual exposure). They may begin by imagining this separation, work toward increasing separation within the therapy setting, and then progress to separating in real-world situations (e.g., school).

13.5.1.2. Relaxation training. Prior to engaging in exposure training, the individual must learn a relaxation technique to apply during the onset of anxiety related to being separated from a primary attachment figure. Deep breathing (control, slow, and purposeful breathing) and distraction (focusing on alternative things, grounding oneself to their senses) are commonly used strategies.

13.5.1.3. Cognitive restructuring. The clinician can then help the patient establish new, positive thoughts to replace the maladaptive cognitions. Research indicates that implementing

cognitive restructuring techniques before, during, and after exposure sessions enhances the overall effect of treatment of social anxiety disorder (Heimberg & Becker, 2002).

13.5.2. Selective Mutism

Children with selective mutism also benefit from exposure and relaxation training. Their exposure will likely consist of increasing exposure to speaking with imaginal exercise, progressing to actual increased speech within the therapy setting, and ending by increasing speech in the real-world setting. In addition, social skills training may help children with selective mutism, especially given the high comorbidity of social anxiety disorder. Social skills training is discussed in Section 13.5.5.2.

13.5.3. Generalized Anxiety Disorder

13.5.3.1. Psychopharmacology. Benzodiazepines, a class of sedative-hypnotic drugs that will be discussed in more detail in the substance abuse module, 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 generalized anxiety disorder. 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 effective treatment options such as

CBT, relaxation training, and biofeedback are often encouraged before the use of pharmacological interventions.

13.5.3.2. Rational-Emotive therapy. Albert Ellis developed rational emotive therapy 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, established a treatment to address these thoughts and 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. By identifying and replacing these assumptions, the individual will experience relief of generalized anxiety disorder symptoms (Ellis, 2014).

13.5.3.3. Cognitive Behavioral Therapy (CBT). CBT is discussed in detail in the Mood Disorder Module; however, it is also among the most effective treatment options for a variety of anxiety disorders, including generalized anxiety disorder. 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).

13.5.3.4. 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 on their physiological states. There are several ways a patient can connect 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, usually 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 can 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).

13.5.4. Specific Phobias

13.5.4.1. Exposure treatments. While there are many treatment options for specific phobias, research routinely supports the behavioral techniques as the most effective treatment strategies. Seeing as the behavioral theory suggests phobias develop via respondent conditioning, the treatment approach revolves around breaking the maladaptive association 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 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 learns 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 teaching several different types of 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 can effectively employ relaxation techniques to reduce their 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.

Flooding is another exposure technique in which the clinician does not utilize a fear hierarchy, but rather repeatedly exposes the individual to their most feared object or situation. Similar to systematic desensitization, flooding can be done in either in vivo or imaginal exposure. Clearly, this technique is more intensive than 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.

13.5.4.2. Modeling is another common technique used to treat phobias (Kelly, Barker, Field, Wilson, & Reynolds, 2010). In this technique, the clinician approaches the feared object/subject while the patient observes. As the name implies, the clinician models appropriate behaviors when exposed to the feared stimulus, showing 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.

13.5.5. Agoraphobia

Similar to the treatment approaches for specific phobias, exposure-based techniques are among the most effective treatment options for individuals with agoraphobia. However, unlike the high success rate in specific phobias, exposure treatment for agoraphobia has been less effective in providing complete relief from 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 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 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 type of treatment provides improvement for 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 should continue for several years at minimum (Craske & Barlow, 2014).

13.5.6. Social Anxiety Disorder

13.5.6.1. Exposure. A hallmark treatment approach for all anxiety disorders is exposure. Specific to social anxiety disorder, the individual is encouraged to engage in social situations where they are likely to experience increased anxiety. Initially, the clinician will role-play various social situations with the patient so they 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 situations with random strangers at various locations such as fast-food restaurants, local stores, libraries, etc. The patient is encouraged to continue with these exposures outside of treatment to help reduce anxiety related to social situations.

13.5.6.2. Social skills training. This treatment is specific to social anxiety disorder as it focuses on the patient's skill deficits or inadequate social interactions that contribute to their negative social experiences and anxiety. During a 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 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 and improve their overall social interactions while reducing ongoing social anxiety.

13.5.6.3. Cognitive restructuring. While exposure and social skills training are suitable treatment options, research routinely supports the need to incorporate cognitive restructuring as

an additive component in treatment to provide substantial symptom reduction. Like cognitive restructuring previously discussed in the Mood Disorder module, 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).

13.5.7. Panic Disorder

13.5.7.1. 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).

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.

Self-monitoring. **Self-monitoring**, or the act 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. Then, the patient is encouraged to recognize 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 learns the fundamental processes with which they can manage their panic symptoms (Craske & Barlow, 2014).

Relaxation training. Similar to that in exposure-based treatment for phobias, prior to engaging in exposure training, the individual must learn relaxation techniques to apply during onset of panic attacks. Though breathing training was once included as the relaxation training technique of choice for panic disorder more recent research has failed to support this technique as effective in the use of panic disorder due to the high incidence of hyperventilation during panic attacks (Schmidt et al., 2000). Findings suggest that breathing retraining is more commonly misused as a safety behavior or means for avoiding physical symptoms as opposed to an effective physiological response to stress (Craske & Barlow, 2014).

Progressive muscle relaxation. To replace the breathing retraining, Craske & Barlow (2014) suggest **progressive muscle relaxation** (PMR). In PMR, the patient learns to tense and relax various large muscle groups throughout the body. The patient is encouraged to start at either the head or the feet, and gradually work their way 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, the individual exhausts those muscles, forcing them (and eventually) the entire body to engage in relaxation (McCallie, Blum, & Hood, 2006).

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. As noted previously, 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 beliefs to be questioned and challenged. This is where the detailed recordings produced by self-monitoring are helpful. By discussing what the patient has recorded for the relationship between physiological arousal and thoughts/behaviors, the clinician can help the patient restructure the maladaptive thought processes to more positive thought processes, which in return, helps to reduce fear and anxiety.

Exposure. As discussed in detail 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 to reduce and eliminate ongoing distress. **Interoceptive exposure** involves inducing panic-specific symptoms to the individual repeatedly for a prolonged 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 include spinning a patient repeatedly in a chair to induce dizziness and breathing in a paper bag to cause hyperventilation. These treatment approaches can be presented gradually; 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 ultimately leads to habituation across treatment and 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 are generally effective in eliminating symptoms again.

13.5.7.2. 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 and 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 the medication was discontinued post-treatment, the CBT+ medication groups fared worse than the CBT treatment-only groups, thus supporting the theory that immersion in interoceptive exposure is limited due to the use of medication. Therefore, it is suggested that medications be reserved for those who do not respond to CBT therapy alone (Kampman, Keijers, Hoogduin & Hendriks, 2002).

You should have learned the following in this section:

- Treatment options for separation anxiety disorder include exposure, relaxation training, and cognitive restructuring.
- Treatment options for selective mutism include exposure, relaxation training, and coping skills training.
- Treatment options for generalized anxiety disorder include benzodiazepines, rational-emotive therapy, CBT, and biofeedback.
- Treatment options for specific phobias include exposure treatments such as systematic desensitization, flooding, and modeling.
- Treatment options for agoraphobia include exposure and CBT techniques.
- Treatment options for social anxiety disorder include exposure treatment, social skills training, and cognitive restructuring.
- Treatment options for panic disorder include CBT, psychoeducation, self-monitoring, relaxation training, cognitive restructuring, exposure, and pharmacological interventions.

Section 13.5 Review Questions

1. Discuss the types of exposure treatments for individuals with anxiety disorders? Which are most effective? What have been some concerns with exposure treatment?
2. What is biofeedback? How is biofeedback used to treat anxiety related disorders?
3. What are the concerns with using pharmacological interventions in the treatment of anxiety disorders? Is there a time when it is helpful to use this treatment method?

Apply Your Knowledge

CASE VIGNETTE

Francesca, an 8-year-old girl, is terrified of going to the doctor. She is so terrified of going to the doctor that she had to be hospitalized for three days last month due to becoming severely dehydrated. Because she refused to go to the doctor, her condition of bronchitis worsened into pneumonia with severe dehydration. Francesca's mother has pleaded with Francesca to help her understand what she is so afraid of, but unfortunately Francesca cannot verbalize why this is. When her parents try to take her to the doctors' office, she screams, kicks, flops, runs, and ultimately will hide under her bed. Her behaviors are so severe that her parents worry Francesca will unintentionally injure herself when they attempt to get her in the car. The only reason they were able to get her to the hospital was because they had aid from paramedics. Francesca also cannot tolerate when she is separated from both of her parents. She has often reported to the school nurse feeling ill while at school, but as soon as she gets home, she feels fine. She frequently cries when she is dropped off at school and pleads with her parents to not have to go in. She has never had a sleepover at a friend's house either.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What anxiety disorder/disorders might Francesca be diagnosed with? Explain your reasoning and the symptoms you are noticing.
2. What other information would you want to know?
3. What treatment would you recommend?

Module Recap

This concludes our discussion of anxiety disorders. This discussion included separation anxiety disorder, selective mutism, generalized anxiety disorder, specific phobia, 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 14, we will discuss obsessive-compulsive disorders.

Part V. Mood and Anxiety-Related Disorders

Module 14:

Obsessive-Compulsive and Related Disorders

Module 14: Obsessive-Compulsive and Related Disorders

Module Overview

In Module 14, we will discuss matters related to obsessive-compulsive and related disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include obsessive compulsive disorder (OCD), body dysmorphic disorder (BDD), trichotillomania, and excoriation disorder. Be sure you refer to 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 therapies (Module 3).

Module Outline

- 14.1. Clinical Presentation
- 14.2. Prevalence and Comorbidity
- 14.3. Etiology
- 14.4. Assessment and Treatment

Module Learning Outcomes

- Describe how obsessive-compulsive disorders present.
- Describe prevalence and comorbidity of obsessive-compulsive disorders.
- Describe the etiology of obsessive-compulsive disorders.
- Describe how obsessive-compulsive disorders are assessed.
- Describe treatment options for obsessive-compulsive disorders.

14.1. Clinical Presentation

Section Learning Objectives

- Describe how obsessive-compulsive disorder presents.
- Describe how body dysmorphic disorder presents.
- Describe how trichotillomania and excoriation disorder present.

14.1.1. Obsessive-Compulsive Disorder

Obsessive-compulsive disorder, more commonly known as OCD, requires the presence of obsessions, compulsions, or both**. Obsessions** are defined as repetitive and persistent thoughts, urges, or images. These obsessions are intrusive, time-consuming (i.e., take more than an hour a day), and unwanted, often causing significant distress or impairment in an individual's daily functioning. Common obsessions are contamination (dirt on self or objects), errors of uncertainty regarding daily behaviors (locking the 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 compulsory behaviors to gain temporary relief from the distress or anxiety.

Compulsions are time-consuming, repetitive behaviors or mental acts that an individual performs in response to an obsession. Common examples of compulsions are checking (e.g., repeatedly checking if the stove is turned off even though the first four-times they checked it was), counting (e.g., flicking the lights off and on exactly five times), hand washing, symmetry, fears of harm to self or others, or repeating specific words (APA, 2022). 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 some 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 at restaurants, or they may bring their utensils from home. The frequency and severity of the obsessions and compulsions varies by patient, with some having mild to moderate symptoms and only spending 1-3 hours a day obsessing or engaging in compulsive behaviors, while other patients present with severe symptoms and have nearly constant intrusive thoughts or compulsions that can become incapacitating (APA, 2022).

14.1.2. Body Dysmorphic Disorder

Body dysmorphic disorder (BDD) is another obsessive disorder; however, the focus of the obsessions is with perceived defects or flaws in one's physical appearance. A key feature of these obsessions is that they are *not* observable or appear slight 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 body dysmorphic disorder. 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 body dysmorphic disorder, the most commonly reported areas are skin (acne, wrinkles, skin color), hair (particularly thinning or excessive body hair), and nose (size or shape; APA, 2022). Interestingly, the disorder can occur *by proxy* meaning the individual is not concerned with their own defects but those of another person, often a spouse or partner but at times, a parent, child, sibling, or stranger.

Due to the distressing nature of the obsessions regarding one's body, individuals with body dysmorphic disorder also engage in compulsive behaviors that take up a considerable amount of time in their day. For example, they may repeatedly compare their body to other people's bodies in the general public; frequently look at themselves in the mirror; engage in excessive grooming, which includes using make-up to modify their appearance. Some individuals with body dysmorphic disorder 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 body dysmorphic disorder, 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. Some individuals excessively tan, change their clothes repeatedly, or compulsively shop such as for beauty products. Camouflaging perceived defects is a common behavior and could involve applying makeup, adjusting a hat or one's clothes, or covering the forehead or eyes with one's hair, all to hide or cover the perceived defect or problem area (APA, 2022).

As the DSM-5-TR notes, body dysmorphic disorder has been associated with, "abnormalities in emotion recognition, attention, and executive function, as well as information-processing biases and inaccuracies in interpretation of information and social situations" (APA,

2022, pg. 273). These individuals tend to express a bias for negative and threatening interpretations of facial expressions and situations that would be classified as ambiguous, for instance.

14.1.2.1. 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 the appropriate amount of muscle definition (Ahmed, Cook, Genen & Schwartz, 2014). While the severity of BDD between individuals with and without muscle dysmorphia appears to be the same, some studies have found higher use of substance abuse (i.e., steroid use), poorer quality of life, and increased reports of suicide attempts in those with muscle dysmorphia (Pope, Pope, Menard, Fay Olivardia, & Philips, 2005). The DSM-5-TR instructs clinicians to specify if body dysmorphic disorder occurs with muscle dysmorphia.

14.1.2.2. Insight specifiers. Those diagnosed with body dysmorphic disorder vary in the degree of insight they have about the accuracy of their body dysmorphic disorder beliefs, ranging from good to absent/delusional. On average, insight is poor and at least one-third of those diagnosed with the disorder display absent/delusional insight. Mental health professionals would indicate the degree of insight regarding body dysmorphic disorder beliefs using *with good or fair insight*, *with poor insight*, or *with absent insight/delusional beliefs*. See page 272 of the DSM-5-TR for more information. Note that the insight specifier is used with OCD and hoarding disorder (not discussed in this module) as well.

14.1.3. Trichotillomania and Excoriation

14.1.3.1. Trichotillomania. **Trichotillomania (Hair-Pulling) disorder** is characterized by an individual recurrently pulling their hair out and results in hair loss. Despite attempts to cease the behavior, the individual continues with it. These behaviors cause distress and impairment and may also lead to other dermatological/medical conditions (APA, 2022).

Hair pulling may be accompanied by a range of behaviors or rituals such as searching for a particular kind of hair to pull such as those with a specific texture or color, pulling out hair in a specific way so that the root comes out intact, or visually examining or tactilely or orally manipulating the hair after it is pulled out such as pulling the strand between the teeth. Pain does not routinely accompany hair pulling.

It can be triggered by feelings of anxiety, boredom, and/or an increasing sense of tension, and may lead to gratification, pleasure, or a sense of relief once the hair is pulled out. It has been noted that for some, an "itch-like" or tingling sensation occurs and is alleviated once the hair has been pulled out. The act of hair pulling does not usually occur in the presence of other people with the exception of immediate family members. Some even have the urge to pull hair from other people or from pets, dolls, and other fibrous materials (APA, 2022).

Hair pulling can occur in infancy and will typically resolve during early development. Onset, though, most commonly coincides with, or follows, the onset of puberty. The usual course of trichotillomania is chronic with waxing and waning of symptoms being typical if untreated. The disorder manifests similarly across cultures and ethnic groups though there is not much data from non-Western regions.

14.1.3.2. Excoriation. **Excoriation (Skin-Picking) disorder** is similar to trichotillomania, except it involves skin-picking which results in skin lesions. Despite attempts to

cease the behavior, the individual continues with it. The skin picking causes impairment and may also lead to other medical consequences (APA, 2022).

Skin picking can be triggered by feelings of anxiety, boredom, and/or an increasing sense of tension, and may lead to gratification, pleasure, or a sense of relief once the skin or scab has been picked. It involves behaviors such as searching for a particular kind of scab to pull and examining, playing with, or swallowing the skin after it has been pulled. The act of skin picking usually does not occur in the presence of other people, except immediate family members, and some individuals report picking the skin of others. Pain is not typically associated with skin picking (APA, 2022).

Excoriation disorder usually begins during adolescence, commonly with or following the onset of puberty, and begins with a dermatological condition such as acne. The course is chronic with some waxing and waning if untreated. It appears clinical features are similar across cultures, though there is limited data currently.

You should have learned the following in this section:

- As part of OCD, obsessions are repetitive and persistent thoughts, urges, or images while compulsions are repetitive behaviors or mental acts that an individual performs in response to an obsession.
- Body dysmorphic disorder is characterized by obsessions over perceived defects or flaws in one's physical appearance.
- Muscle dysmorphia refers to the belief that one's body is too small or lacks the appropriate amount of muscle definition and is a type of body dysmorphic disorder common to men.
- Trichotillomania (Hair-Pulling) disorder is characterized by an individual recurrently pulling their hair our and results in hair loss.
- Excoriation (Skin Picking) disorder involves skin picking which results in skin lesions.

Section 14.1 Review Questions

1. Define obsessions and compulsions. Provide a list of examples of each thought/behavior.
2. What is body dysmorphic disorder? Give examples of characteristics that would *not* be consistent with a body dysmorphic disorder diagnosis.
3. What is the difference between trichotillomania disorder from excoriation disorder? How much pain do both involve for the afflicted?

14.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence and comorbidity of OCD.
- Describe the prevalence and comorbidity of body dysmorphic disorder.
- Describe the prevalence and comorbidity of trichotillomania and excoriation disorder.

14.2.1. OCD

14.2.1.1. Prevalence. The prevalence rate for OCD is approximately 1.2% both in the U.S. and worldwide (APA, 2022). Women are diagnosed with OCD more often than males; however, in childhood, boys are diagnosed more frequently than girls (APA, 2022). With respect to gender and symptoms, females are more likely to be diagnosed with cleaning related obsessions and compulsions. In contrast, males are more likely to display symptoms related to forbidden thoughts and symmetry (APA, 2022). The DSM-5-TR reports that the mean age of onset of OCD is 19.5 years with a quarter of cases starting by 14 years of age. Additionally, males have an earlier age of onset (5-15 yrs.) compared to females (20-24 yrs.; Rasmussen & Eisen, 1990).

14.2.1.2. Comorbidity. There is a high comorbidity 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. Additionally, due to the nature of OCD and its symptoms, nearly 41% of those with OCD will also be diagnosed with a depressive or bipolar disorder (APA, 2022).

There is a high comorbidity 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 ADHD in children. Due to this psychological disorder triad, it is believed there is a neurobiological mechanism at fault for the development and maintenance of the disorders.

It should be noted that there are several disorders—schizophrenia, bipolar disorder, eating disorders, body dysmorphic disorder, and Tourette's disorder – that OCD is much more common in. Therefore, clinicians who have a patient diagnosed with one of the disorders should also routinely assess patients for OCD (APA, 2022).

Finally, OCD has a mean rate of lifetime suicide attempts of 14.2%, a mean rate of lifetime suicidal ideation of 44.1%, and a mean rate of current suicidal ideation of 25.9%. Severity of OCD, the symptom dimension of unacceptable thoughts, a history of suicidality, and severity of comorbid depressive and anxiety symptoms are predictors of greater suicide risk (APA, 2022).

14.2.2. Body Dysmorphic Disorder

14.2.2.1. Prevalence. The point prevalence rate for body dysmorphic disorder among U.S. adults is 2.4% while outside the U.S., the point prevalence is 1.7% to 2.9%. Gender-based prevalence rates indicate that women are more likely to be diagnosed with body dysmorphic disorder than men, though muscle dysmorphia is diagnosed more frequently in men. Additionally, women are more likely to be preoccupied with weight, breasts, buttocks, legs, hips,

and excessive body or facial hair while men have preoccupations with their genitals, body build, and thinning hair (APA, 2022).

14.2.2.2. Comorbidity. Major depressive disorder is the most common comorbid psychological disorder with body dysmorphic disorder and typically occurs after the onset of body dysmorphic disorder. Additionally, there are some reports of social anxiety disorder, OCD, and substance-related disorders (likely related to muscle enhancement; APA, 2022). Those with body dysmorphic disorder are four times more likely to have experienced suicidal thoughts and 2.6 times more likely to have made suicide attempts compared to healthy control subjects and those diagnosed with eating disorders, OCD, or any anxiety disorder.

14.2.3. Trichotillomania and Excoriation Disorder

14.2.3.1. Trichotillomania. Trichotillomania occurs in 1% to 2% of adults and adolescents. Females are more often diagnosed with this disorder than males, in adulthood. However, in childhood, males and females are equally impacted. The most common comorbid disorders are MDD and excoriation disorder. Other repetitive body-focused symptoms such as nail-biting occur in most individuals with trichotillomania and may warrant an additional diagnosis of other specified obsessive-compulsive and related disorder (APA, 2022).

14.2.3.2. Excoriation disorder. In terms of excoriation disorder, an online survey of over 10,000 adults between the ages of 18-69 and gender-matched to the U.S. population found that 2.1% self-identified as having current excoriation disorder while 3.1% reported lifetime excoriation disorder. Community samples show that 75% or more of individuals diagnosed with the disorder are women. OCD, trichotillomania, and MDD are comorbid with excoriation disorder, with depression comorbidity being more common in women. Other repetitive body-

focused symptoms such as nail-biting occur in most individuals with excoriation disorder and may warrant an additional diagnosis of other specified obsessive-compulsive and related disorder (APA, 2022).

You should have learned the following in this section:

- The prevalence rate for OCD is about 1.2% while body dysmorphic disorder is 2.4%. Trichotillomania occurs in 1% to 2% of adults and adolescents and excoriation disorder has a prevalence rate of 2.1% (current, or 3.1% lifetime).
- In terms of gender, females are more likely to be diagnosed with the four disorders, though in terms of body dysmorphic disorder, males receive the muscle dysmorphia specifier more than females. Additionally, in childhood, males and females are equally impacted by trichotillomania.
- Gender differences are also present for symptom presentation in OCD and the area of the body focused on in body dysmorphic disorder.
- OCD is shown to have a high comorbidity with anxiety and depressive disorders as well as tic disorder and ADHD in children.
- Body dysmorphic disorder has a high comorbidity with major depressive disorder.
- Trichotillomania and excoriation disorder are both comorbid with each other and MDD, with the latter also being comorbid with OCD.

Section 14.2 Review Questions

1. What are the key gender differences related to OCD and body dysmorphic disorder?
2. How do the prevalence rate of the four disorders compare?
3. What are the most common comorbidities for OCD? Be specific.
4. This section discussed the OCD triad in children. What two other disorders complete this triad?
5. Which disorder is body dysmorphic disorder most comorbid with?
6. What disorders are trichotillomania and excoriation disorder comorbid with?

14.3. 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.

14.3.1. Biological

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

14.3.1.1. 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, the familial relationship with regards to obsessions were stronger than that of compulsions suggesting 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 hereditary influence; however, environmental factors appear to play a more significant role in the development of these disorders than that of OCD (Ahmed, et al., 2014; Lervolino et al., 2009). The DSM reports evidence for a genetic vulnerability to trichotillomania and excoriation disorder with the disorder being more common in individuals with OCD and their first-degree relatives than in the general population (APA, 2022).

14.3.1.2. Neurotransmitters. Neurotransmitters, particularly serotonin, have been identified as a contributing factor to obsessive and compulsive behaviors. This discovery was made accidentally, when individuals with depression and comorbid OCD were given antidepressant medications clomipramine and 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 a substantial improvement in their OCD symptoms (Bokor & Anderson, 2014). Antidepressant medications that do not affect serotonin levels are *not* effective in managing obsessive and compulsive symptoms, thus offering additional support for deficits of serotonin levels as an explanation of obsessive and compulsive behaviors (Sinopoli, Burton, Kronenberg, & Arnold, 2017; Bokor & Anderson, 2014). More recently, there has been some research implicating the involvement of additional neurotransmitters—glutamate, GABA, and dopamine—in the development and maintenance of

OCD, although future studies are still needed to draw definitive conclusions (Marinova, Chuang, & Fineberg, 2017).

14.3.1.3. Brain structures. Seeing as neurotransmitters have 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 to act (Beucke et al., 2013). It is believed that individuals with obsessive compulsive behaviors experience overactivity of the orbitofrontal cortex and a lack of filtering in the caudate nuclei, thus causing too many impulses to transfer 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).

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

We have shown that individuals with OCD experience cognitive biases and that these biases contribute to the obsessive and compulsive behaviors, but why do these cognitive biases occur so often? 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 concede to their thoughts once, or even forgo acknowledging 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 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).

14.3.3. Behavioral

The behavioral explanation of obsessive compulsive-related disorders focuses on compulsions rather than obsessions. Behaviorists believe that these compulsions begin with and are maintained through **operant conditioning**. How so? Well, an individual with OCD may experience negative thoughts or anxieties related to an unpleasant event (obsession; the event is a stimulus). These thoughts/anxieties cause significant distress to the individual, and therefore, they seek out some behavior (compulsion; the response) to alleviate these threats (i.e., escape behavior associated with negative reinforcement). This provides temporary relief to the individual, thus reinforcing the compulsive behaviors used to lessen the threat. Over time, the compulsive behaviors are reinforced due to the repeated exposure of the obsession and the temporary relief that comes with engaging in these compulsive behaviors (escape behavior).

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 patient's operant conditioning associated with the obsessions and compulsions by preventing the individual from engaging in the compulsive behavior until anxiety is reduced.

You should have learned the following in this section:

- Biological causes of obsessive-compulsive disorders include hereditary transmission, neurotransmitter deficits particularly in relation to serotonin, and abnormal functioning in brain structures.
- Cognitive causes of obsessive-compulsive disorders include distorted thinking such as overestimating the probability of harm, loss of control, or uncertainty in their life, and negative cognitive biases such as disconfirmation bias.
- Behavioral causes of obsessive-compulsive disorders include operant conditioning.

Section 14.3 Review Questions

1. What are the biological implications regarding the etiology of OCD and related disorders? What brain structures have been linked to these disorders?
2. Discuss identified cognitive biases that are related to the development and maintenance of OCD and related disorders?
3. The behavioral model discusses how respondent conditioning may explain the development and maintenance of these disorders. What type of reinforcement is at work and how?

14.4. Assessment and Treatment

Section Learning Objectives

- Provide an overview of assessment options.
- Describe treatment options for OCD.
- Describe treatment options for body dysmorphic disorder.
- Describe treatment options for trichotillomania and excoriation disorder.

14.4.1 Assessment

Overall, assessing for obsessive-compulsive and related disorders is largely based on interviewing and objective measures. Information is obtained about the various behaviors and thoughts to include their frequency, context, and severity. A thorough understanding of which obsessions and compulsions are present needs to be obtained. Interviews will often occur with caregivers, and perhaps teachers. Interviews should certainly include the child/adolescent as well, as many of these symptoms require a deeper understanding that can only come from the child/adolescent. The Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS) is a helpful objective measure to understand the presence of a wide variety of obsessions and compulsions as well as the severity of these symptoms.

14.4.2. Treatment of OCD

14.4.2.1. Exposure and Response Prevention (ERP). Treatment of OCD has come a long way in recent years. Among the most effective treatment options is exposure and response prevention (March, Frances, Kahn, & Carpenter, 1997). First developed by psychiatrist Victor

Meyer (1966), 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* (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 refrain 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). Combination treatments such as ERP with family counseling (utilizing CBT techniques) may increase this response rate even higher (Bolleau, 2011; Krebs & Heyman, 2015). Like most OCD related treatments, the largest barrier to treatment is getting

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

14.4.2.2. 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 and SSRIs. Reportedly, up to 60% of patients 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).

14.4.3. Treatment of Body Dysmorphic Disorder

Seeing as though there are strong similarities between OCD and body dysmorphic disorder, it should not come as a surprise that the only two effective treatments for body dysmorphic disorder are those that are effective in OCD. Exposure and response prevention has been successful in treating symptoms of body dysmorphic disorder, 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 body dysmorphic disorder. Similar to OCD, medications such as clomipramine and SSRIs are generally prescribed. While these are effective in reducing body dysmorphic disorder symptoms, once medication is discontinued, symptoms resume nearly

immediately suggesting this is not an effective long-term treatment option for those with body dysmorphic disorder.

Treatment of body dysmorphic disorder appears to be difficult, with one study finding that only 9% of participants 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 8 years (Bjornsson, Dyck, et al., 2011).

14.4.3.1. Plastic surgery and medical treatments. Many individuals with body dysmorphic disorder seek out plastic surgery to attempt to correct their deficits. Phillips and colleagues (2001) evaluated treatments of patients with body dysmorphic disorder 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 medical treatment.

14.4.4. Treatment of Trichotillomania and Excoriation Disorder

Cognitive-behavioral therapy (thoroughly described in our chapter on depression) and habit reversal training (HRT, thoroughly described in our chapter on motor disorders) have been utilized for these disorders. Additionally, Acceptance and Commitment Therapy (ACT) has been noted to have some promise for reducing symptoms. The individual learns to accept that negative thoughts and feelings are part of being human and to respond in new ways to these negative

thoughts and feelings more in line with their overall values and goals (e.g., not skin picking or hair pulling; Lochner, Ross, & Stein, 2017).

Pharmacological options include SSRIs and glutamatergic medications and have shown promising results in research. Other, less common drugs, such as opioid antagonists and anti-epileptic drugs have been theorized to be beneficial. However, studies of these drugs either show no benefit or are not in-depth random control trials (RCTs; Lochner, Ross, & Stein, 2017).

You should have learned the following in this section:

- Assessing for obsessive-compulsive and related disorders is largely based on interviewing and objective measures.
- Treatment options for OCD include exposure and response prevention, as well as SSRIs though the drug does not provide an added benefit in treatment.
- Treatment options for body dysmorphic disorder include exposure and response prevention and drugs clomipramine and SSRIs.
- Treatment options for trichotillomania and excoriation disorder include CBT and HRT as well as pharmacological options such as SSRIs, glutamatergic medications, opioid antagonists, and anti-epileptic drugs

Section 14.4 Review Questions

1. What methods and tools are used to assess for obsessive-compulsive and related disorders?
2. Discuss the various types of treatments for OCD. Which treatment option has the best outcome?
3. What are the different components of Exposure and Response Prevention? How do they work together to reduce OCD symptoms?
4. What are the most effective treatment approaches for body dysmorphic disorder?
5. What are the most effective treatment approaches for trichotillomania and excoriation disorder?

Apply Your Knowledge

CASE VIGNETTE

Frank, an 11-year-old boy, was brought in for services due to severe and persistent behaviors. Frank reportedly has intrusive and repetitive thoughts and fears of illness. He consistently scans his body for injury and illness. He experiences significant somatic symptoms when he notices anything slightly wrong such as a bruise. He is unable to attend his health class because the topics lead to him become overwhelmed with fear of illness in himself and scanning himself for any potential symptom of a condition they are covering. He is able to recognize that these thoughts are irrational, but he cannot seem to put a pause on them.

Frank also must scan his body every morning exactly three times. He scans his body from head to toe three times while standing in the mirror, inspecting for any obvious external injury and internally monitoring himself for any symptoms of illness. This routine takes him about 1 hour each morning and Frank cannot go about his day unless he engages in this routine. It often leads to him arriving late to school and events.

His grades are average, but he is often distracted by his thoughts in class, and Frank's school had to grant him a specific exception to his health class.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What symptoms of OCD does Frank have? Obsessions? Compulsions?
2. What other disorders would you consider for Frank and why? Would it be hard to differentiate between OCD and the other disorder you might consider?
3. Would you need more information to make a full diagnosis for Frank?
4. What would you recommend for his treatment?

Module Recap

As in all preceding modules, 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 our discussions of mood and anxiety-related disorders.

In our final block of disorders, we will cover trauma-related, eating, and substance-related disorders.

3rd edition as of December 2022

Part VI. Trauma, Eating, and Substance-related Disorders

Part VI. Trauma, Eating, and Substance-related Disorders

Module 15:
Trauma- and Stressor-Related Disorders

Module 15: Trauma- and Stressor-Related Disorders

Module Overview

In Module 15, we will discuss matters related to trauma- and stressor-related disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment. Our discussion will include PTSD, acute stress disorder, and adjustment disorder. Prior to discussing these clinical disorders, we will explain what stressors are, as well as identify common stressors that may lead to a trauma- or stressor-related disorder. We will also discuss adverse childhood events and Children's Advocacy Centers (CACs). Be sure you refer to Modules 1-3 for explanations of key terms (Module 1), an overview of models to explain psychopathology (Module 2), and descriptions of various therapies (Module 3).

Module Outline

Module Learning Outcomes

- Define and identify common stressors.
- Describe how trauma- and stressor-related disorders present.
- Describe the prevalence and comorbidity of trauma- and stressor-related disorders.
- Describe the etiology of trauma- and stressor-related disorders.
- Describe how trauma- and stressor-related disorders are assessed.
- Describe treatment options for trauma- and stressor-related disorders.

15.1. Stressors, ACEs, and CACs

Section Learning Objectives

- Define stressor and trauma.
- Identify and describe common stressors.
- Describe types of childhood trauma.
- Describe Adverse Childhood Events (ACE).
- Describe CACs.

15.1.1. Stressors and Types of Trauma

Before we dive into the clinical presentations of three of the trauma and stress-related disorders, let's discuss 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 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 was not 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 focused on posttraumatic stress disorder (PTSD) symptoms due to the large number of service members returning from deployments and reporting significant trauma symptoms.

Physical assault, and 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 likely underestimates 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).

Specific to children, two thirds of children report experiencing at least one traumatic event by the time they reach age 16 (SAMHSA, 2017, December). To give a bit more clarity on the prevalence of traumatic events, 1 in 5 students are bullied and 1 in 6 are cyberbullied. Also,

54% of children have been impacted by a natural disaster and, in 2015, for every 1,000 children, 9.2 experienced either abuse or neglect (SAMHSA, 2017, December).

Trauma in childhood can take many different forms. Childhood trauma may either be a trauma that is unrelated to a specific event such as death of a loved one, a natural disaster, and other adverse childhood events (see ACE discussion below) or it may be specific to maltreatment. Childhood maltreatment refers to neglect or abuse of a child (Table 15.1 provides an overview of the various types of childhood maltreatment). Childhood trauma may include physical abuse, sexual abuse, neglect, medical trauma, witnessing domestic violence, traumatic grief, bullying, community violence, terrorism/violence, refugee trauma, natural disasters, complex trauma, early childhood trauma or any other life-threatening stressor. While some of these forms of abuse might seem clearly defined (e.g., physical abuse, sexual abuse, witnessing domestic violence), others may need a bit more clarification. For example, early childhood trauma (trauma that occurs prior to age 6) and complex trauma (exposure to multiple traumatic events) are not terms that are frequently mentioned in general societal conversations about childhood trauma. Neglect is the most common form of childhood maltreatment followed by physical abuse.

Early childhood trauma is trauma that occurs in very young children. Typically, people have a belief that if trauma occurs before a child can remember it, then it does not impact them. However, this is a misconception. Children's brain structures may even be impacted such that their brain cortex may be reduced in size. It can also lead to significant disruptions in the attachment a child forms with their caregivers. Let's think about why that might be. As an infant, our only responsibility is to grow (physically, cognitively, and emotionally) and we rely on caregivers to provide stability, protection, and soothing. When caregivers provide nurture,

soothing, food, and stability to an infant, then the infant's body can focus on making important neural connections, learning from their caregivers how to regulate their distress, and use the nurture and nutrition provided to physically and cognitively grow. However, if an infant does not feel safe and is not provided constant protection and care, they do not have the luxury of only focusing on growing and learning. The infant now must shift their attention from growing to surviving. They also do not learn how to sooth themselves or appropriately recognize danger (we tend to perceive benign things as dangerous in efforts to stay safe). They may struggle to regulate their emotions and behaviors, appropriately react to their environment and surroundings, and develop close and meaningful attachments (NCTSN, n.d.c.).

Complex trauma occurs when a child experiences multiple traumatic events. Those traumatic events are also interpersonal, meaning these events are directed at them from another person (typically the caregiver) and are not natural disasters or a painful medical procedure. The traumatic events are severe, and they impact the child's development. The repeated events disrupt the child's ability to feel secure with safety and stability. As such, their development is impacted. Because these children are often in a stress-activated state, their bodies do not appropriately regulate physiological responses to stress. They tend to recognize non-threating situations as threatening and others perceive them as "overreacting." For example, a child that has been repeatedly abused by a caregiver may jump when their classmate slams their locker shut. Other children may perceive this child as overreacting and even point it out or make fun of him or her. However, for that child, his body is "stuck" in an overactive "flight or fight" state and perceived the small benign threat – a locker slamming - as a major threat. The constant stress the body is under can lead to physical difficulties and even a compromised immune system (NCTSN, n.d.c.).

Emotionally, children with a complex trauma history have a very difficult time recognizing, expressing, and regulating their emotions. They may also disassociate, often to cope with ongoing trauma. Their ability to attach to caregivers may also be compromised (NCTSN, n.d.c.).

Behaviorally, they may be "set off" easily and struggle to regulate their own behaviors and reactions. They may appear impulsive and unpredictable. They may attempt to exert control in their environment which may lead to behavioral disturbances as well. Moreover, children with this history may have trouble with problem-solving and acquiring new cognitive skills (NCTSN, n.d.c.).

Table 15.1. Types of Maltreatment, From Highest to Lowest Prevalence

Maltreatment Type	Definition	Prevalence
Neglect (Impaired Caregiving)	When a caregiver fails to properly care for a child. This includes providing appropriate shelter, food, supervision for safety, etc. This may also include a child being in environments that are unsafe (e.g., home with illegal substance, etc.)	74.8%
Physical Abuse	Legally defined in each state, but in general, "nonaccidental" event in which a caregiver leaves a mark (e.g., bruise, etc.) on a child, even if leaving a mark was unintentional. This typically occurs during punishment or when a caregiver acts in anger.	18.2%
Sexual Abuse	Again, this varies by state. Each statute defines sexual abuse. Broadly, it is defined as any interaction between a child and adult in which a child is used for sexual stimulation. This may include touching behaviors or non-touching behaviors such as exposing a child to pornography.	8.5%
Other	Parental substance abuse, threatened abuse, or any other maltreatment	6.9%
Emotional Abuse	Typically, language that is aggressive and harmful that leads to a change in a child's behavior and emotional well-being.	5.6%
Medical neglect	Failure to provide appropriate medical care to a child. For example, if a child is sick/injured and a caregiver fails to take the child to the doctor.	2.1%

Note. Table heavily informed by NCTSN (n.d.a, n.d.b., n.d.c., & n.d.d) and Child Welfare Information Gateway (2016a, 2016b) information.

15.1.2. Adverse Childhood Events (ACE)

Kaiser Permanente conducted a massive study in the years of 1995 to 1997 (Felitti, Anda, Nordenberg, Williamson, Spitz, Edwards, & Marks, 1998). The study focused on various adverse childhood experiences and how those experiences impacted children's overall development. They divided adverse childhood events into 2 main areas (i.e., abuse and household dysfunction) with seven separate categories: psychological abuse, physical abuse, sexual abuse, substance abuse in the home, mental illness of a household member, violent behavior by mother, and criminal behavior of a household member. The study was groundbreaking. The results of the

study found that over half of the participants had experienced at least one major childhood adverse event. Moreover, about a quarter of participants had experienced two separate types of adverse events. With increased adverse events, adult outcomes were increasingly negative such that more adverse events lead to higher likelihood of smoking, increased sexual partners and sexually transmitted disease, obesity, and other health concerns (Felitti, et al., 1998). This study made it clear that adverse events have long-lasting impacts into adulthood that impact overall health and quality of life. As such, the need to implement prevention efforts to reduce the frequency in which children experience adverse events, including maltreatment and neglect, was obvious.

15.1.3 Children's Advocacy Centers (CACs)

Children's Advocacy Centers (CACs) are designed to improve a child's experience with investigations following abuse. The first CAC was established in 1985. Before CACs, children would have to disclose their abuse to several different individuals (e.g., first the police, then a doctor, then a social worker, investigator, and counselor). With the implementation of a CAC, multidisciplinary teams (MDTs) were designed. MDTs are comprised of several professionals including but limited to law enforcement, medical professionals, mental health providers, child protective services, victim advocates, and legal prosecutors. The idea was that a child would complete one forensic interview with multiple team members viewing the interview (either live or videoed). A **forensic interview** is a recorded interview with the goal to allow a child to provide information about their experiences of abuse in a non-leading and supportive method (National Children's Advocacy Center, n.d.). The forensic interview is conducted by a trained individual and is videotaped so that it can be used in litigation and does not require the

child to testify or recount abuse in court. This reduced the number of times that a child had to disclose their abuse/trauma, on average from 8 different people to just 1 time. CACs appear to improve a child and caregivers' satisfaction with the investigation process (Jones, Cross, Walsh, Simone, 2007). The CAC team also works to connect the child and family with mental health and other needed or appropriate support services following their initial contact.

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 and diagnostic criteria for posttraumatic stress disorder, acute stress disorder, and adjustment disorder.

You should have learned the following in this section:

- A stressor is any event that increases physical or psychological demands on an individual.
- It does not have to be personally experienced but can be witnessed or occur to a close family member or friend to have the same effect.
- Only a small percentage of people experience significant maladjustment due to these events.
- The most studied triggers for trauma-related disorders include physical/sexual assault and combat.
- Adverse events have long-lasting impacts into adulthood that impact overall health and quality of life.
- Children's Advocacy Centers (CACs) are designed to improve a child's experience with investigations following abuse.
- A **forensic interview** is a recorded interview with the goal to allow a child to provide information about their experiences of abuse in a non-leading and supportive method

Section 15.1 Review Questions

1. Given an example of a stressor you have experienced in your own life.
2. Why are the triggers of physical/sexual assault and combat more likely to lead to a trauma-related disorder?
3. What forms does childhood trauma take?
4. What are CACs?
5. What is the principal tool used by CACs?

15.2. Clinical Presentation

Section Learning Objectives

- Describe how PTSD presents (take note of differences for children 6 and younger)
- Describe how acute stress disorder presents.
- Describe how adjustment disorder presents.

15.2.1. 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, sexual violence, or serious injury 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; directly experiencing a traumatic event; or being exposed to repeated events where one experiences an aversive event (e.g., victims of child abuse/neglect, ER physicians in trauma centers, etc.).

It is important to understand that while the presentation of these symptoms varies among individuals, to meet the criteria for a diagnosis of PTSD, individuals need to report symptoms among the four different categories of symptoms.

15.2.1.1. Category 1: Recurrent experiences. The first category involves *recurrent experiences* of the traumatic event, which can occur via dissociative reactions such as flashbacks; recurrent, involuntary, and intrusive distressing memories; or even recurrent distressing dreams (APA, 2022, pgs. 301-2). These recurrent experiences must be specific to the traumatic event or

the moments immediately following to meet the criteria for PTSD. Regardless of the method, the recurrent experiences can last several seconds or extend for several days. They are often initiated by physical sensations similar to those experienced during the traumatic events or 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. One or more of the intrusion symptoms must be present.

15.2.1.2. Category 2: Avoidance of stimuli. The second category involves *avoidance* of *stimuli* related to the traumatic event and either one or both of the following must be present. First, individuals with PTSD may be observed trying to avoid the distressing thoughts, memories, and/or feelings related to the memories of the traumatic event. Second, they may prevent these memories from occurring by avoiding physical stimuli such as locations, individuals, activities, or even specific situations that trigger the memory of the traumatic event.

15.2.1.3. Category 3: Negative alterations in cognition or mood. The third category experienced by individuals with PTSD is *negative alterations in cognition or mood* and at least two of the symptoms described below must be present. 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 lead 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 oneself or others is also reported by those with PTSD. Similar to those with depression, individuals with PTSD may report a reduced interest in participating in previously enjoyable activities, as well as the desire to engage with others socially. They also report not being able to experience positive emotions.

15.2.1.4. Category 4: Alterations in arousal and reactivity. The fourth and final category is *alterations in arousal and reactivity* and at least two of the symptoms described below must be present. Because of the negative mood and increased irritability, individuals with PTSD may be quick-tempered and act out aggressively, 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 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. They also experience significant sleep disturbances, with difficulty falling asleep, as well as staying asleep due to nightmares; engage in reckless or self-destructive behavior, and have problems concentrating.

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 symptoms have *not* been present for a month, the individual may meet criteria for acute stress disorder (see below).

15.2.1.5. Diagnosing PTSD in children under 6 years of age. Historically, diagnosing PTSD in children was difficult. The criteria required the presence of internal symptoms that children sometimes have difficulty reporting and describing. For example, assessing if a child has persistent negative beliefs about a traumatic event or about themselves, or has difficulty with remembering aspects of the event was difficult. As such, when the new DSM-5 was published, the taskforce created new criteria for younger children (aged 6 and under).

While some of the specific criteria are the same, such as experiencing a traumatic event, other components are different. To better understand the differences, see Table 15.2 below:

Table 15.2. Differences in PTSD Criteria for Children Aged 6 and Younger and All Other Individuals (Aged 7 and Up)

Criterion	Older than 6 Years Old	Younger than 6 Years Old
A – Exposure to actual or threatened death, serious injury, or sexual violence (at least one)	• Directly experiencing the event • Witnessing, in person • Learning that the event occurred to a close family member or friend • Experiencing repeated or extreme exposure to aversive details of the event	• Directly experiencing the event • Witnessing, in person • Learning that the event occurred to a parent or caregiver figure
B – Presence of at least one intrusion symptom	• Distressing memories • Distressing dreams • Dissociative reactions in which the individual feels or acts as if the traumatic event were recurring • Prolonged psychological distress at exposure to reminders • Physiological reactions to cues	All are the same
C – Avoidance of stimuli associated with the event (at least one)	• Avoidance of distressing memories, thoughts, or feelings • Avoidance of external reminders that cause distressing memories	At least one of the following associated with persistent avoidance or negative alterations • Avoidance of activities, places, or physical reminders • Avoidance of people, conversations, or interpersonal situations • Increased frequency of negative emotional states • Diminished interest in activities • Socially withdrawn behavior • Persistent reduction in expression of positive emotions
D – Negative alterations in cognitions and mood associated with the event (two or more)	• Not remembering an aspect of the event • Exaggerated negative beliefs • Distorted cognitions about the cause or consequences of the event • Negative emotional state • Diminished interest in activities • Detachment or estrangement from others • Inability to experience positive emotions	

Criterion	Older than 6 Years Old	Younger than 6 Years Old
E – Alterations in arousal and reactivity (two or more)	• Irritable behavior/angry outbursts • Reckless or self-destructive behavior • Hypervigilance • Exaggerated startle response • Concentration problems • Sleep disturbance	• Irritable behavior/angry outbursts • Hypervigilance • Exaggerated startle response • Concentration problems • Sleep disturbance

Source: DSM-5-TR pgs. 301-304 (APA, 2022)

15.2.2. 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 one month, the individual would then meet the criteria for PTSD. Additionally, if symptoms present immediately following the traumatic event but resolve by day 3, an individual would not meet the 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; note that in total, there are 14 symptoms across these five categories). 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 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 the criteria for acute stress disorder.

Making Sense of the Disorders

In relation to trauma- and stressor-related disorders, note the following:

Diagnosis PTSD ….. if symptoms have been experienced for at least one month

Diagnosis acute stress disorder … if symptoms have been experienced for 3 days to one month

15.2.3. Adjustment Disorder

Adjustment disorder is the least intense of the three disorders discussed so far in this module. An adjustment disorder occurs following an identifiable stressor that happened 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 and causes marked distress "…that is out of proportion to the severity or intensity of the stressor" (APA, 2022, pg. 319).

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 if the behaviors do not meet criteria for one of the aforementioned categories. Based on the individual's presenting

symptoms, the clinician will determine which category best classifies the patient's condition.

These modifiers are also important when choosing treatment options for patients.

You should have learned the following in this section:

- In terms of stress disorders, symptoms lasting over 3 days but not exceeding one month, would be classified as acute stress disorder while those lasting over a month are typical of PTSD.
- If symptoms begin after a traumatic event but resolve themselves within three days, the individual does not meet the criteria for a stress disorder.
- Symptoms of PTSD fall into four different categories for which an individual must have at least one symptom in each category to receive a diagnosis. These categories include recurrent experiences, avoidance of stimuli, negative alterations in cognition or mood, and alterations in arousal and reactivity.
- To receive a diagnosis of acute stress disorder an individual must experience nine symptoms across five different categories (intrusion symptoms, negative mood, dissociative symptoms, avoidance symptoms, and arousal symptoms).
- Adjustment disorder is the last intense of the three disorders and does not have a specific set of symptoms of which an individual has to have some number. Whatever symptoms the person presents with, they must cause significant impairment in areas of functioning such as social or occupational, and several modifiers are associated with the disorder.

Section 15.2 Review Questions

1. What is the difference in diagnostic criteria for PTSD, acute stress disorder, and adjustment disorder?
2. What are the four categories of symptoms for PTSD? How do these symptoms present in acute stress disorder and adjustment disorder?

15.3. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence and comorbidity of PTSD.
- Describe the prevalence and comorbidity of acute stress disorder.
- Describe the prevalence and comorbidity of adjustment disorders.

15.3.1. PTSD

15.3.1.1. Prevalence. The national lifetime prevalence rate for PTSD using DSM-IV criteria is 6.8% for U.S. adults and 5.0% to 8.1% for U.S. adolescents. There are currently no definitive, comprehensive population-based data using DSM-5 though studies are beginning to emerge (APA, 2022). 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 ethnically or politically motivated genocide (APA, 2022).

Concerning gender, PTSD is more prevalent among females (8% to 11%) than males (4.1% to 5.4%), likely due to their higher occurrence of exposure to traumatic experiences such as childhood sexual abuse, rape, domestic abuse, and other forms of interpersonal violence. Women also experience PTSD for a longer duration. (APA, 2022). 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. More specifically, prevalence rates of PTSD are highest for African Americans, followed by Latinx Americans and European Americans, and lowest for Asian Americans (Hinton & Lewis-Fernandez, 2011). According to the DSM-5-TR, there are higher rates of PTSD among Latinx, African-Americans, and American Indians compared to whites, and likely due to exposure to past adversity and racism and discrimination (APA, 2022).

15.3.1.2. Comorbidity. 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. Individuals with PTSD are more likely than those without PTSD to report clinically significant levels of depressive, bipolar, anxiety, or substance abuse-related symptoms (APA, 2022). There is also a strong relationship between PTSD and major neurocognitive disorders, which may be due to the overlapping symptoms between these disorders.

15.3.2. Acute Stress Disorder

15.3.2.1. Prevalence. The prevalence rate for acute stress disorder varies across the country and by traumatic event. Accurate prevalence rates for acute stress disorder are difficult to determine as patients must seek treatment within 30 days of the traumatic event. Despite that, 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).

As with PTSD, acute stress disorder is more common in females than males; however, unlike PTSD, there may be some neurobiological differences in the stress response, gender differences in the emotional and cognitive processing of trauma, and sociocultural factors that contribute to females developing acute stress disorder more often than males (APA, 2022). With that said, the increased exposure to traumatic events among females may also be a strong reason why women are more likely to develop acute stress disorder.

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

15.3.3. Adjustment Disorder

15.3.3.1. Prevalence. Adjustment disorders are relatively common as they describe individuals who are having difficulty adjusting to life after a significant stressor. In psychiatric hospitals in the U.S., Australia, Canada, and Israel, adjustment disorders accounted for roughly 50% of the admissions in the 1990s. It is estimated that anywhere from 5-20% of individuals in

outpatient mental health treatment facilities have an adjustment disorder as their principal diagnosis. Adjustment disorder has been found to be higher in women than men (APA, 2022).

15.3.3.2. Comorbidity. Unlike most of the disorders we have reviewed thus far, adjustment disorders have a high comorbidity rate with various other medical conditions (APA, 2022). Often following a critical or terminal medical diagnosis, an individual will meet the 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. For example, an individual with adjustment disorder with depressive mood must not meet the criteria for a major depressive episode; otherwise, the diagnosis of MDD should be made over adjustment disorder. As the DSM-5-TR says, "adjustment disorders are common accompaniments of medical illness and may be the major psychological response to a medical condition" (APA, 2022).

You should have learned the following in this section:

- Regarding PTSD, rates are highest among people who are likely to be exposed to high traumatic events, women, and minorities.
- As for acute stress disorder, prevalence rates are hard to determine since patients must seek medical treatment within 30 days, but females are more likely to develop the disorder.
- Adjustment disorders are relatively common since they occur in individuals having trouble adjusting to a significant stressor, though women tend to receive a diagnosis more than men.
- PTSD has a high comorbidity rate with psychological and neurocognitive disorders while this rate is hard to establish with acute stress disorder since it becomes PTSD after 30 days.
- Adjustment disorder has a high comorbidity rate with other medical conditions as people process news about their health and what the impact of a new medical diagnosis will be on their life.

Section 15.3 Review Questions

1. Compare and contrast the prevalence rates among the trauma and stress-related disorders.
2. What are the most common comorbidities among trauma and stress-related disorders?
3. Why is it hard to establish comorbidities for acute stress disorder?

15.4. 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.

15.4.1. 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 dysfunction within this axis is to blame for the development of trauma symptoms. Within the brain, the **amygdala** serves as the integrative system that inherently elicits the physiological response to a traumatic/stressful environmental situation. The amygdala sends this response to the HPA axis to prepare the body for "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 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 responsiveness 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).

15.4.2. Cognitive

Preexisting conditions of depression 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 and leading 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).

15.4.3. Social

While this may hold 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 being judged for their actions before the rape, report fewer trauma symptoms and faster psychological improvement (Street et al., 2011).

15.4.4. 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 associated with 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).

You should have learned the following in this section:

- In terms of causes for trauma- and stressor-related disorders, an over-involvement of the hypothalamic-pituitary-adrenal (HPA) axis has been cited as a biological cause, with rumination and negative coping styles or maladjusted thoughts emerging as cognitive causes.
- Culture may lead to different interpretations of traumatic events thus causing higher rates among Hispanic Americans.
- Social and family support have been found to be protective factors for individuals most likely to develop PTSD.

Section 15.4 Review Questions

1. Discuss the four etiological models of the trauma- and stressor-related disorders. Which model best explains the maintenance of trauma/stress symptoms? Which identifies protective factors for the individual?

15.5. Assessment

Section Learning Objectives

- Outline the assessment process when screening for trauma experiences and PTSD.

Overall, every child should be screened for trauma experiences despite the setting. If a clinician sees a child that is being assessed for ADHD, they should still screen for trauma. This is because we never know when a child may disclose a trauma, and trauma and PTSD reactions may explain some behaviors. For example, is the child impulsive and explosive due to a behavioral disorder such as ADHD *or* are they experiencing hyper-arousal and emotion regulation difficulties associated with a complex trauma history and/or PTSD? This question cannot be answered if the clinician does not screen for trauma.

When assessing for PTSD, two questions must be answered: (1) has a trauma occurred and (2) is the child exhibiting trauma reactions/symptoms? To answer the first part, *has a trauma occurred,* a trauma screening is utilized. Screening for trauma can be formal or informal. Utilizing a standard trauma screener can be helpful so that a provider does not fail to screen for a particular type of trauma. For example, the Childhood Trust Events Inventory screens for various types of traumas and takes only a few minutes to complete. The UCLA PTSD Reaction Index for DSM-5 also has a helpful trauma screener at the beginning of the measure. Trauma screening is often done directly with a child but can also be used with caregivers or other adults involved in the child's life (e.g., social worker, case worker).

To answer the second part, *is the child exhibiting trauma reactions/symptoms,* we must understand if there is a presence of avoidance behaviors, intrusive memories, hyperarousal,

irritability, behavioral regulation difficulties, interpersonal difficulties, or developmental problems. We can do this by interviewing the child, parent, or other adults. We can also use objective measures that assess these areas. For example, the Trauma Symptom Checklist for Young Children (TSCYC, caregiver report) and the Trauma Symptom Checklist for Children (TSCC, child report) can be used to assess for general, related symptoms. The UCLA PTSD Reaction Index for DSM-5 can be used to assess for the presence of specific criteria of PTSD to understand the likelihood the child meets full DSM-5 criteria of PTSD.

An alternative option that may also be helpful is to create a timeline with the adults involved in the child's life to understand when traumatic events occurred and when symptoms started. This helps mental health practitioners understand if the symptoms are related to a trauma or not. For example, if a child presents as irritable and moody prior to any trauma, the irritability may not necessarily be a trauma-reaction. Conversely, if a child was happy and did not display frequent irritability but following a trauma presented with significant irritability, this may be indicative of a trauma reaction. Thus, building a timeline with the caregiver can be helpful. Utilizing a timeline with a child, however, is not suggested as this may be too distressing for them.

You should have learned the following in this section:

- When assessing for PTSD, two questions must be answered: (1) has a trauma occurred and (2) is the child exhibiting trauma reactions/symptoms?
- To answer the first part, *has a trauma occurred,* a trauma screening is utilized.
- To answer the second part, *is the child exhibiting trauma reactions/symptoms,* we must understand if there is a presence of avoidance behaviors, intrusive memories, hyperarousal, irritability, behavioral regulation difficulties, interpersonal difficulties, or developmental problems.
- An alternative option that may also be helpful is to create a timeline with the adults involved in the child's life to understand when traumatic events occurred and when symptoms started.

Section 15.5 Review Questions

1. What tools are used to assess for PTSD in children?
2. How might a timeline be used and with whom?

15.6. 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 other psychological interventions.
- Describe the treatment approach of parent-child interventions.
- Describe the use of psychopharmacological treatment.

15.6.1. 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 follow the same general 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 posttraumatic event (Kinchin, 2007). Research across a variety of traumatic events (i.e., 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 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 for professionals employing psychological debriefing, thus ensuring that those who are providing treatment are properly trained to do so.

While this might be used in instances of natural disasters and mass traumas, this is not commonly used with children. As such, it is important to understand debriefing and the varying options, but it is not necessary to understand the intricacies of debriefing.

15.6.2. Exposure Therapy

While exposure therapy is predominately used in anxiety disorders, it has also shown great success in treating 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 mentally re-creates specific details of the traumatic event. The patient is then asked to repeatedly discuss the event in increasing detail, providing more information regarding their thoughts and feelings at each step of the event. During 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 fewer details of the event, and slowly gaining information over time. In vivo starts with images or 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 method, it is also the most distressing and places patients at risk for dropping out of treatment (Resick, Monson, & Rizvi, 2008).

These exposure techniques are often used in other treatments that also incorporate other components such as cognitive strategies. Particularly for children, the exposure typically occurs in developmentally appropriate ways. See below discussion of TF-CBT and ITCT/ITCT-A.

15.6.3. 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 replace 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 and 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 provides the patient with strong social support and a 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 to prepare for when they experience these triggers out in the real world, as well as any future challenges that may come their way.

TF-CBT is also beneficial for children that have experienced traumatic grief. The same principles are implemented; however, the intervention provides specifics about implementing the treatment in the context of traumatic grief as well.

Alternatives for Families – A Cognitive Behavioral Therapy (AF-CBT) is another adaptation of CBT for children that have experienced trauma. It is composed of three different general components: child-directed components, caregiver-directed components, and parent-child/family-system directed components. Child-directed components has a similar focus as TF-CBT (e.g., psychoeducation, emotional and cognitive skills, exposure). The caregiver-directed components include a focus on the caregiver's own psychoeducation, cognitive processing and skills, rapport building with the caregiver, as well as parenting strategies to improve child behaviors. Finally, the parent-child/family-system directed component focuses on communication, problem-solving, and safety/relapse planning. Because there is a strong focus on family, this is sometimes a preferred treatment when it has been observed that caregivers engage in coercive parenting, especially in the context of child physical abuse.

Cognitive Behavioral Intervention for Trauma in Schools (CBITS) is a school-based cognitive behavioral intervention. Although this intervention has some limitations, it is particularly helpful for children and families that do not have easy access to transportation to attend regular mental health therapy appointments in an outpatient setting.

15.6.4. Other Psychological Interventions

Integrative Treatment of Complex Trauma for Children (ITCT-C) and Integrative Treatment of Complex Trauma for Adolescents (ITCT-A) are modular treatments that incorporate several components of various treatment modalities. These treatments allow for more flexibility and tailored intervention plans which can be helpful when children/adolescents present with complex trauma. Because individuals with complex trauma may not have the typical "acute PTSD" symptom presentation, or they may have that presentation with other related symptoms, the flexibility of this treatment allows for many benefits. The provider uses an assessment to identify major problem areas and uses this to target their first goals in therapy. Problem areas may include concerns of safety, issues related to sexual/physical victimization, caretaker support issues, anxiety, depression, aggression, self-esteem, posttraumatic stress, attachment insecurity, identity issues, relationship problems, suicidality, substance use/abuse, grief, sexual behaviors, self- injury, binging/purging, other risky behaviors, legal issues, emotion regulation, flashbacks, and others. The areas of greatest concern are identified as the primary goals that need to be addressed and these goals then align with specific treatment modules.

Throughout therapy, new "problem area" assessments are completed. If the goals are the same, the treatment stays the same. If they are not, then the goals are realigned and new modules, if needed, are implemented. The modules include: cognitive skills, exposure, mindfulness, affect regulation training, trigger management, psychoeducation, relational building/support, safety planning, relational processing, identity issues, interventions, caregiver interventions, and substance abuse interventions. Although the specifics of these interventions vary from ITCT-C and ITCT-A, the concept is similar in both. However, the specific problem areas and modules vary slightly.

15.6.5. Parent-Child Interventions

Parent-Child Interaction Therapy (PCIT) was not originally designed for children that have experienced trauma but has proven useful since. The therapy focuses on increasing the positive interaction between a parent and child. The parent learns how to interact with and impose appropriate consequences on a child by watching the therapist interact with the child. Then, they practice what they have learned from the clinician. The therapy typically involves a one-way mirror and an "earpiece" often referred to as "bug in the ear." This allows the parent to first observe the therapist, but more importantly, it allows the therapist to observe the parent and then coach them through the earpiece on how to interact with the child. This gives the parent real-time coaching.

Child-Parent Psychotherapy (CPP) is used for younger children, as young as infants. The principal behind this treatment is to increase attachment between the child and caregiver. Caregiver needs are addressed in this therapy. For example, often, the caregiver themselves have experienced a trauma. Thus, assessing and addressing PTSD in the caregiver may occur. Also understanding the caregiver's thoughts and feelings about the child is an important component. With the child, understanding and addressing symptoms of trauma and other emotional or behavioral concerns are addressed. Within the therapy, the child-parent relationship remains the focus. Safety of the child and caregiver home environment is ensured, and limit-setting is established. Parents learn what to expect from their child (e.g., how children express emotion and regulate emotion), and increase the parent's ability to respond to those emotions. Children and parents learn how to express and receive love and support from each other as well.

15.6.6. 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, SSRIs 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 has been limited. Future studies exploring other medication options are needed to determine if there are alternative medication options for stress/trauma disorder patients.

Of course, with any of these medications, the age of the child and severity of the symptoms will be considered before determining if medicinal intervention is appropriate, safe, and necessary.

You should have learned the following in this section:

- Several treatment approaches are available to clinicians to alleviate the symptoms of trauma- and stressor-related disorders.
- The first approach, psychological debriefing, has individuals who have recently experienced a traumatic event discuss or process their thoughts related to the event and within 72 hours.
- Another approach is to expose the individual to a fear hierarchy and then have them use positive coping strategies such as relaxation techniques to reduce their anxiety or to toss the fear hierarchy out and have the person experience the most distressing memories or images at the beginning of treatment.
- The third approach is Cognitive Behavioral Therapy (CBT) and attempts to identify and challenge the negative cognitions surrounding the traumatic event and replace them with positive, more adaptive cognitions. AF-CBT and CBITS are used with children.
- Integrative Treatment of Complex Trauma for Children (ITCT-C) and Integrative Treatment of Complex Trauma for Adolescents (ITCT-A) are modular treatments that incorporate several components of various treatment modalities.
- Parent-child interventions include Parent-Child Interaction Therapy (PCIT) and Child-Parent Psychotherapy (CPP).
- Finally, when psychotherapy does not produce relief from symptoms, psychopharmacology interventions are an effective second line of treatment and may include SSRIs, TCAs, and MAOIs.

Section 15.6 Review Questions

1. Identify the different treatment options for trauma and stress-related disorders. Which treatment options are most effective? Which are least effective?
2. Which treatment approaches are used with children? Describe each.

Apply Your Knowledge

CASE VIGNETTE

Nina is a 14-year-old girl. When Nina was 18 months old, a neighbor called CPS due to concerns that Nina was often dirty, and the neighbor had witnessed aggressive speech and posturing toward Nina from her parents. However, there was not sufficient or concrete evidence of abuse, thus Nina remained in her parent's custody. During this CPS investigation, Nina's mother stated to CPS that she was incredibly stressed and also experienced intimate partner violence. She admitted that caring for Nina was difficult because Nina's was a cranky baby that did not respond to her attempts to soothe. CPS also learned that both parents had a history of substance abuse concerns; however, both parents reportedly were sober and drug/substance free. CPS developed a plan for the family which included parenting classes and a social worker that followed their case for 6 months. After 6-months, Nina's family appeared to be more stable, and thus, CPS contact ended. However, shortly after, Nina's parents began using drugs again, and their relationship was more volatile than ever.

When Nina was 13, she had developed a close relationship with the school counselor. One day, the counselor noticed a bruise on her upper arm. Nina trusted her counselor, and after the counselor asked what happened, Nina explained her father had hurt her and showed the counselor other significant bruises. She also told her counselor that her parents often confined her to her room and blamed her for family problems. Nina being the oldest of three also often tried to protect her younger siblings from being hurt. The counselor called CPS and this time, Nina and her siblings were removed from her parents' custody. Ultimately, Nina's parents' parental rights were terminated, and she was adopted by a family after living with 3 foster families. Her siblings were also adopted, but to different families.

CONTINUED BELOW

Apply Your Knowledge Continued

After being placed in a foster home, and eventually adopted, Nina became withdrawn and quiet – she spent significant periods alone in her room. Although she was often polite and respectful toward adults, she struggled to engage with peers. She wasn't interested in extracurricular activities or making friends. Her grades were often Fs, and she was frequently distracted at school. She complained of physical complaints often, with no founded medical conditions. Often, when her adoptive parents raised their voice slightly, she would tear up and run. If her parents dropped something, she would jump. It often seemed to her adoptive parents that Nina had significant difficulty relaxing and always appeared tense. And nearly every night, Nina woke up in a sweat and got little sleep. Her moods shifted from apathetic to hostile and angry quickly. She often displayed emotional outbursts that included verbal and physical aggression. She was recently diagnosed with oppositional defiant disorder (ODD) and ADHD. Nina didn't talk much about how she was feeling, and her adoptive parents were struggling to figure out how to help Nina.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. Do you think Nina is experiencing a trauma-related disorder? If so which one?
2. Do you think Nina has ADHD and ODD, or are her experiences better captured by a trauma reaction? Explain your thoughts.
3. What treatment options may be best for Nina?
4. How can her new family support Nina?
5. What protective factors are present for Nina? What risk factors are present?

Nina's vignette was heavily informed by Joshua's vignette published by NCTSN (NCTSN, 2008).

The National Child Traumatic Stress Network. (2008, March). *Child welfare trauma training toolkit: Supplemental handouts.* http://www.trauma-informed-california.org/wp- content/uploads/2012/02/child_welfare_trauma_training_toolkit_supplements.pdf

Module Recap

In Module 15, we discussed trauma- and stressor-related disorders to include PTSD, acute stress disorder, and adjustment disorder. We clarified what stressors and traumas were, forms of childhood maltreatment their impact on children. We also learned about CACs. Next, we discussed how trauma-related disorders present and what the diagnostic criteria are for each. In addition, we clarified the prevalence, comorbidity, and etiology of each disorder. Finally, we discussed the assessment process and potential treatment options for the trauma- and stressor-related disorders, with specific strategies identified for children.

Our discussion in Module 16 moves to eating disorders.

Part VI. Trauma, Eating, and Substance-related Disorders

Module 16:
Eating Disorders

Module 16: Eating Disorders

Module Overview

In Module 16, we will discuss matters related to eating disorders to include their clinical presentation, epidemiology, comorbidity, etiology, assessment, and treatment options. Our discussion will include anorexia nervosa, bulimia nervosa, and binge eating 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 therapies (Module 3). As a reminder, feeding disorders, also part of the chapter in the DSM-5-TR, were covered separately in Module 5.

Module Outline

- 16.1. Clinical Presentation
- 16.2. Prevalence and Comorbidity
- 16.3. Etiology
- 16.4. Assessment
- 16.5. Treatment

Module Learning Outcomes

- Describe how eating disorders present.
- Describe the prevalence and comorbidity of eating disorders.
- Describe the etiology of eating disorders.
- Describe how eating disorders are assessed, diagnosed, and treated.

16.1. Clinical Presentation

Section Learning Objectives

- Clarify the concept of mutually exclusive.
- Describe how anorexia nervosa presents.
- Describe how bulimia nervosa presents.
- Describe how binge-eating disorder (BED) presents.

Feeding and eating disorders are "…characterized by a persistent disturbance of eating or eating-related behavior that results in the altered consumption or absorption of food and that significantly impairs physical health or psychosocial functioning" (APA, 2022, pg. 371). They are very serious, yet relatively common mental health disorders, particularly in 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 has been identified as major contributors in both the development and maintenance of eating disorders.

Within the DSM 5-TR (APA, 2022), six disorders are classified under the Feeding and Eating Disorders chapter: pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa, and binge-eating disorder. In this book, we will cover the latter three whose diagnostic criteria are **mutually exclusive**, meaning that only one of these diagnoses can be assigned at any given time due to substantial differences in their clinical course, outcome, and treatment needs, despite a number of common psychological and behavioral features.

For more on eating disorders in general, please visit the National Eating Disorders Association website below:

https://www.nationaleatingdisorders.org/what-are-eating-disorders

16.1.1. Anorexia Nervosa

Anorexia nervosa involves the *restriction* of energy intake, 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.

Typical warning signs and symptoms 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;" eating a restricted range of foods; making excuses to avoid mealtimes; and not eating in public. Physical changes may include dizziness, difficulty concentrating, feeling cold, sleep problems, thinning hair/hair loss, and muscle weakness, to name a few. When the individual loses weight, they view this as an impressive achievement and a sign of extraordinary discipline, while weight gain is seen as an unacceptable failure of self-control (APA, 2022).

The onset of the disorder typically begins with mild dietary restrictions such as eliminating carbs or specific fatty foods. As weight loss is achieved, the dietary restrictions progress to more severe, e.g., under 500 calories/day. Symptoms present in adolescence or young adulthood and rarely before puberty or after age 40. The onset of the disorder typically is preceded by a stressful life event such as leaving home for college.

For more on anorexia nervosa, please visit the National Eating Disorders Association website below:

https://www.nationaleatingdisorders.org/learn/by-eating-disorder/anorexia

16.1.2. 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. Individuals with bulimia nervosa often report a sense of lack of

control over-eating during these binge-eating episodes. While not always the case, these binge-eating episodes are followed by a feeling of disgust with oneself, which leads to a **compensatory behavior** to rid the body of the excessive calories. These compensatory behaviors include vomiting, use of laxatives, fasting (or severe restriction), diuretics or other medications, or excessive exercise. This cycle of binge eating and compensatory behaviors occurs on average, at least once a week for three months (National Eating Disorder Association website; APA, 2022).

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 episode—if they do, they do *not* meet the criteria for anorexia nervosa.

Signs 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 the bathroom after eating a 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.

Making Sense of the Disorders

Though anorexia and bulimia share some common features, they differ as follows:

Diagnosis anorexia …... if severe calorie restriction occurs alone

Diagnosis bulimia … if severe calorie restriction occurs **AND** there is a binge-eating episode

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

Additionally, those with bulimia are often ashamed of their eating problems and attempt to hide the symptoms. The binge eating occurs in secrecy or as inconspicuously as possible. Common antecedents of binge eating include negative affect; interpersonal stressors; dietary restraint; boredom; and negative feelings linked to body weight, shape, and food.

For more on bulimia nervosa, please visit the National Eating Disorders Association website below:

https://www.nationaleatingdisorders.org/learn/by-eating-disorder/bulimia

16.1.3. 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. The binge-eating episodes are associated with at least three of the following: eating quicker than usual, eating until uncomfortably full, eating large amounts even if not hungry, eating alone, and feeling disgust with oneself or being depressed. Despite the feelings of shame and guilt post-binge, individuals with BED will not engage in vomiting, excessive exercise, 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. Individuals 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.

Making Sense of the Disorders

Though bulimia and BED are similar, they differ as follows:

Diagnosis BED …... if binge eating occurs alone

Diagnosis bulimia … if binge eating occurs **AND** there are compensatory behaviors to prevent weight gain

While much is still being researched about binge-eating disorder, current research indicates that the onset of BED is adolescence to early adulthood but can begin later in life. Those who seek treatment tend to be older than those with either bulimia or anorexia. Binge eating has been found to be common in adolescent and college-age samples and for all, is associated with social role adjustment issues, impaired health-related quality of life and life satisfaction, and increased medical morbidity and mortality (APA, 2022).

For more on binge eating disorder, please visit the National Eating Disorders Association website below:

https://www.nationaleatingdisorders.org/learn/by-eating-disorder/bed

You should have learned the following in this section:

- Anorexia nervosa involves the restriction of food, which leads to significantly low body weight relative to the individual's age, sex, and development, and an intense fear of gaining weight or becoming fat.
- Bulimia nervosa is characterized by a pattern of recurrent binge eating behaviors followed by compensatory behaviors.
- Binge-eating disorder is characterized by recurrent binge eating episodes along with a feeling of lack of control but no compensatory behavior to rid the body of the calories.

Section 16.1 Review Questions

1. What does mutually exclusive mean? What does it mean with respect to eating disorders?
2. What are the key differences in diagnostic criteria for anorexia, bulimia, and binge eating disorder?
3. Define compensatory behavior. What disorder is this found in?

16.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the prevalence of anorexia nervosa.
- Describe the comorbidity of anorexia nervosa.
- Describe the prevalence of bulimia nervosa.
- Describe the comorbidity of bulimia nervosa.
- Describe the prevalence of binge eating disorder.
- Describe the comorbidity of binge eating disorder.

16.2.1. Prevalence of Eating Disorders

16.2.1.1. Anorexia nervosa. According to the National Eating Disorder Alliance (NEDA) website, at any point in time more women (0.3-0.4%) than men (0.1%) will be diagnosed with anorexia. Anorexia nervosa is most prevalent in postindustrialized, high-income countries such as the United States, Australia, New Zealand, Japan, and many European countries. In the U.S., prevalence is lower among Latinx and non-Latinx Black Americans than non-Latinx Whites (APA, 2022).

16.2.1.2. Bulimia nervosa. According to the NEDA website, at any point in time, 1.0% of women and 0.1% of men will meet the diagnostic criteria for bulimia nervosa. A study by Stice and Bohon (2012) found that between 1.1% and 4.6% of females and 0.1% to 0.5% of males will develop bulimia and that subthreshold bulimia occurs in 2.0% to 5.4% of adolescent females. The DSM reports that the 12-month prevalence ranges from 0.14% to 0.3% with higher

rates in females and high-income countries. Rates are similar across ethnoracial groups across the U.S. (APA, 2022).

16.2.1.3. Binge eating disorder. Hudson et al. (2007) reports that BED is three times more common than anorexia and bulimia and is more common than breast cancer, HIV, and schizophrenia. It has also been found that between 0.2% and 3.5% of females and 0.9% and 2.0% of males will develop binge eating disorder with subthreshold binge eating disorder occurring in 1.6% of adolescent females (Stice & Bohon, 2012). The DSM reports a 12-month prevalence of 0.44% to 1.2% with rates 2-3 times higher in women, similar rates across ethnoracial groups in the United States and between most high-income industrialized countries (APA, 2022).

16.2.1.4. General comments. Not all individuals with an eating disorder end up being diagnosed or in treatment. For example, Austin et al., (2008) indicated that, when screening high-school students for an eating disorder, 25% of girls and 11% of boys reported disordered eating and weight controlling methods severe enough to necessitate clinical evaluation of the symptoms. However, very few of those students indicated that they had received treatment.

For more on statistics and research related to feeding and eating disorders, please visit the National Eating Disorders Association website below:

https://www.nationaleatingdisorders.org/statistics-research-eating-disorders

16.2.2. Comorbidity of Eating Disorders

16.2.2.1. Anorexia nervosa. Anorexia is rarely a single diagnosis. High rates of bipolar, depressive, and anxiety disorders are common among individuals with anorexia nervosa. Obsessive-compulsive disorder is more often seen in those with the restricting 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, with rates reported to be 18 times greater than in an age- and gender-matched comparison group. It is also estimated that between 9% and 25% of individuals with anorexia have attempted suicide (APA, 2022).

16.2.2.2. Bulimia nervosa. The majority of individuals diagnosed with bulimia nervosa also present with at least one other mental disorder. Similar to anorexia nervosa, there is a high frequency of depressive symptoms (i.e., low self-esteem), as well as bipolar and depressive disorders. While some experience mood fluctuations because of their eating pattern (occurring at the same time or following the development of bulimia), some individuals will identify mood symptoms prior to the onset of bulimia nervosa (APA, 2022).

Anxiety, particularly social anxiety, is often present in those with bulimia nervosa. However, most mood and anxiety symptoms resolve once an effective treatment of bulimia is established. Substance use disorder, and in particular alcohol use disorder, is also prevalent in those with bulimia, with about a 30% prevalence among those with bulimia. The substance abuse begins as a compensatory behavior (e.g., stimulant use is used to control appetite and weight) and over time, as the eating disorder progresses, so does the substance abuse. There is also a percentage of individuals with bulimia nervosa who display personality features that meet the criteria for at least one personality disorder, most often borderline personality disorder. Finally,

about one-quarter to one-third of individuals with bulimia have had suicidal ideation and a comparable amount have attempted suicide.

16.2.2.3. Binge eating disorder. Research shows that BED shares similar comorbidities with anorexia nervosa and bulimia nervosa. Common comorbidities include major depressive disorder and alcohol use disorder. About 25% of those with BED have shown suicidal ideation (APA, 2022).

You should have learned the following in this section:

- BED is three times more common than anorexia and bulimia.
- All eating disorders are more common in women and high-income, industrialized countries.
- Only anorexia shows differences across ethnoracial groups in the United States.
- Anorexia has a high comorbidity with bipolar, depressive, and anxiety disorders. OCD and alcohol use disorder are also comorbid but depend on the type of anorexia (restricting or binge-eating/purging).
- Bulimia has a high comorbidity with bipolar disorder, depressive symptoms and disorders, social anxiety, and substance use disorder.
- BED is highly comorbid with MDD and alcohol use disorder.
- There is a high rate of suicidal ideation with all three disorders.

Section 16.2 Review Questions

1. Which eating disorder is most common?
2. What gender differences occur with regards to the eating disorders?
3. Are there any other noteworthy similarities or differences in the prevalence rates of the three disorders?
4. Discuss the comorbidity rates among the three main eating disorders.

16.3. 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 many contributing factors 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 their presentation. 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.

16.3.1. Biological

There is some evidence of a genetic predisposition for eating disorders, with relatives of those diagnosed with an eating disorder being up to six times more likely than other individuals to be diagnosed also. 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 anorexia in their lifetime. The concordance rate for fraternal twins (who share less genes) is 20%. While not

as strong for bulimia, identical twins still display a 23% concordance rate, compared to the 9% rate for fraternal twins.

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 have been caused *by* the disorder, as manipulation of eating patterns is known to trigger 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 that cause 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 calories or experience periods of overeating.

16.3.2. 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 preferred treatment for eating disorders.

16.3.3. Sociocultural

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 viewed as more desirable (Polivy & Herman, 2002). While eating disorders were once thought of as disorders of higher SES, recent research suggests that as our country becomes more homogenized, the more universal eating disorders become.

16.3.3.1. Media. One commonly discussed contributor to eating disorders is the media. The idealization of thin models and actresses sends the message to young women (and adolescents) that 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 emergence of social media (e.g., 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 people become dissatisfied with their body (Polivy & Herman, 2004).

16.3.3.2. 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 would appear to be less likely to develop anorexia; however, findings suggest this is not the case. 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).

16.3.3.3. Gender. Males account for only a small percentage of eating disorders. 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 men being "muscular" or "strong."

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

16.3.3.4. Family. Family influences are one of the strongest external contributors to maintaining eating disorders. Often family members are praised 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? The odds are likely 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, they are indirectly perpetuating the disorder.

While family involvement can help maintain the disorder, it can also contribute to the development of an eating disorder. 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 a feeding/eating disorder than mothers without eating disorders (Whelan & Cooper, 2000). Additional family characteristics that are common among patients receiving treatment for eating disorders are enmeshed, intrusive, critical, hostile, or overly concerned with parenting (Polivy & Herman, 2002). 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.

16.3.4. 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.

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

16.3.4.2. Self-Esteem. Self-esteem, or one's belief in their 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).

You should have learned the following in this section:

- Biological causes of eating disorders include a genetic predisposition and disruption in the neuroendocrine system.
- Cognitive causes of eating disorders include distorted thought patterns and an over-evaluation of body size.
- Sociocultural causes of eating disorders include the idealization of thin models and actresses by the media, SES, gender, and family involvement.
- The personality trait of perfectionism and low self-esteem are contributing factors to disorders related to eating, weight, and body shape.

Section 16.3 Review Questions

1. Define multidimensional disorders?
2. What evidence is there to suggest eating disorders are biologically driven?
3. According to the cognitive theory, eating disorders may be a variant of what other disorder?
4. Discuss the four sociocultural subgroups that explains development of eating disorders.
5. What are the two personality traits most commonly used to describe behaviors associated with eating disorders?

16.4. Assessment

Section Learning Objectives

- Outline assessment processes for eating disorders.

When assessing for eating disorders, a multidisciplinary team is necessary. This is because many of the symptoms of an eating disorder require an understanding of physical symptoms as well as psychological symptoms and so the team may include a medical doctor, dietician, nurse, psychologist, therapist, etc.

16.4.1. Physical Assessment

A full medical and physical exam should occur. The medical exam should rule out inflammatory bowel disease, hyperthyroidism, chronic infection, diabetes, and other serious medical conditions. A female's menstruation history should also be obtained to include information about their first menstrual period (if menarche has occurred) and if their menstrual period has become irregular or completely stopped (Lask & Bryant-Waugh, 2013).

The physical exam should be done alone unless the child requests otherwise. The provider will look for physical consequences of the eating disorder and rule out other serious disorders. Physical growth will be assessed, and weight/body measurements will be obtained such as body mass index. Additionally, laboratory tests will be utilized to assess various systems and nutritional information including protein levels, liver functioning, and hormone levels (Lask & Bryant-Waugh, 2013).

16.4.2. Psychological Assessment

A clinical interview is conducted not just with the individual, but also family members. During the interview, a timeline will be established which includes life events and the onset or progression of eating behaviors. Eating behaviors will be thoroughly assessed. Additionally, weight-controlling behaviors such as excessive exercise, induced vomiting, laxatives, etc.; their overall body image; and an assessment of mental health checking for suicidal ideation, will be assessed (Lask & Bryant-Waugh, 2013).

Although interviews can be conducted informally, structured interviews can also be used, and are preferred at times. The gold standard for assessing for eating disorders in children is the Child Eating Disorder Examination (ChEDE). Additionally, standardized objective measures to screen for levels of symptoms such as the Eating Disorder Examination Questionnaire (EDEQ) and Eating Disorder Inventory for Children (EDIC) are used. Helpful screening tools include Children's Eating Attitudes Test (ChEAT) or the Kids' Eating Disorder Survey (KEDS; Lask & Bryant-Waugh, 2013).

You should have learned the following in this section:

- Assessment of eating disorders involves a multidisciplinary team.
- A full medical and physical exam should occur.
- A clinical interview is conducted not just with the individual, but also family members.

Section 16.4 Review Questions

1. How do mental health professionals screen for eating disorders?
2. Why is a multidisciplinary team needed to assess for eating disorders? Who is part of this team?

16.5. 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.

16.5.1. Anorexia Nervosa

The immediate goal for the 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).

16.5.1.1. 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—and emotional behaviors—feelings related to the food. In addition to these behavioral strategies, it is also important to address the maladaptive thought patterns associated with their negative body image and desire to control their physical characteristics. Changing the *fear* related to gaining weight is essential in recovery.

16.5.1.2. Family based therapy (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 has been shown to elicit 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 fewer 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 take charge of weight restoration, (2) client's gradual control of overeating, and (3) addressing 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-aged students and above) is still undetermined. As with adolescents, the goal for a family-based treatment program should center around helping the patient separate their feelings and needs from that of their family.

16.5.1.3. Refeeding. Refeeding is a nutritional rehabilitation effort to prevent **refeeding syndrome** which can lead to serious complications (e.g., serious cardiovascular, neurological, and hematologic events) due to a significant and potentially fatal shift in fluid and electrolyte levels following the individual increasing nutritional intake/calories (Fuentebella & Kerner, 2009). Refeeding therapy is done to ensuring adequate nutrition and hydration yet minimize the risk of refeeding syndrome. In severe cases, nasogastric (nasal tube) and gastrostomy (tube inserted into stomach) tubes to appropriately achieve hydration and electrolyte balance may be required. More commonly, graded refeeding can occur. Oral refeeding, the preferred refeeding approach, when possible, occurs when a dietician helps outline appropriate nutritional

expectations. Then, a plan for gradually increasing food for the child/adolescent as they become more comfortable and less fearful of food, is established and implemented.

16.5.2. Bulimia Nervosa

Just as anorexia nervosa treatment initially focuses on weight gain, the first goal of bulimia nervosa treatment is to eliminate binge eating episodes and compensatory behaviors. The aim is to replace both negative behaviors with positive eating habits. One of the most effective ways to establish this is through Cognitive Behavioral Therapy (CBT).

16.5.2.1. 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 method for modifying behaviors 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 action, while simultaneously using relaxation strategies to reduce anxiety associated with not engaging in the negative behavior. Therefore, to prevent an individual from purging post-binge episodes, the individual would be encouraged to partake in an activity that directly competes with their ability to purge, e.g., 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 is 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 feelings, which in return, can help them manage their behaviors.

16.5.2.2. 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 typically covered in weekly sessions over 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 interpersonal issues. 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 main 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 can better understand their problems, and as a result, 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 throughout 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 extensive 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).

16.5.3. Binge Eating Disorder

Given the similar presentations of BED and bulimia nervosa, it should not be surprising that the most effective treatments for BED are 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 a separate eating disorder, treatment research specific to this disorder is expected to grow.

16.5.3.1. Antidepressant medications. Given the high comorbidity 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.

16.5.4. 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 a short history of the disorder. Conversely, unfavorable features are a 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%. Most 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. While treatment outcome for BED is still in its infancy, initial findings suggest that remission rates of BED are much higher than that for anorexia nervosa and bulimia nervosa.

You should have learned the following in this section:

- Treatment options for anorexia nervosa include CBT and FBT.
- Treatment options for bulimia nervosa include CBT, exposure and response prevention, and the three phases of interpersonal psychotherapy.
- Treatment options for BED include the taking of antidepressants to manage depressive symptoms, CBT, and interpersonal therapy.

Section 16.5 Review Questions

1. What is the initial (main) goal of treatment for anorexia?
2. What are the three phases of family-based treatment?
3. What is the goal for interpersonal psychotherapy? Discuss the three phases of IPT.
4. What is the overall treatment effectiveness of eating disorders?

Apply Your Knowledge

CASE VIGNETTE

Sally is a 16-year-old teenager. She has been a high performer in school, achieving a 4.0. She strives for perfection. She has a large group of friends and interacts well socially. She started dating her first boyfriend. Recently, however, he cheated on her. Sally is incredibly broken by this. She does not feel supported or validated by her friends and family's reaction. Around this time, Sally started to feel depressed and decreased appetite due to her depression. As a result, she lost weight and people noticed. They complimented her indicating how wonderful she looked.

Following this, Sally becomes motivated to lose more weight. She begins restricting the food she eats. This continues for several months. She loses so much weight that, although she is keeping her grades and social interactions up, her friends and family begin to worry. She becomes consumed with monitoring how much she is eating, the calories within those foods, etc. She has also begun to increase the amount of exercise she completes and occasionally intentionally purges her food by inducing vomiting after meals. Every night, she analyzes her body in the mirror and points out all of her flaws. No matter how thin she gets, she continues to see herself as needing to lose weight. Sally is now 5 foot 5 inches and weighs 105 pounds.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. Which eating disorder would you diagnose Sally with?
2. What other disorders would you consider?
3. Would you need more information to make a full diagnosis for Sally? If so, what information would you need?
4. What would you recommend for her treatment?

Module Recap

Module 16 covered eating disorders in terms of their clinical presentation, prevalence, comorbidity, etiology, and treatment options. The discussion included anorexia nervosa, bulimia nervosa, and binge eating disorder.

Our next and final chapter will cover substance use disorders.

Part VI. Trauma, Eating, and Substance-related Disorders

Module 17: Substance-Related and Addictive Disorders

Module 17: Substance-Related and Addictive Disorders

Module Overview

In Module 17, will cover matters related to substance-related and addictive disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, 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 to 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 therapies (Module 3).

Module Outline

- 17.1. Clinical Presentation
- 17.2. Prevalence and Comorbidity
- 17.3. Etiology
- 17.4. Assessment
- 17.5. Treatment

Module Learning Outcomes

- Describe how substance-related and addictive disorders present.
- Describe the prevalence and comorbidity of substance-related and addictive disorders.
- Describe the etiology of substance-related and addictive disorders.
- Describe how substance-related and addictive disorders are assessed, diagnosed, and treated.

17.1. Clinical Presentation

Section Learning Objectives

- Define substances and substance abuse.
- Describe properties of substance abuse.
- Describe how substance use disorder presents.
- Describe how substance intoxication presents.
- Describe how substance withdrawal presents.
- Define depressants and describe types.
- Define stimulants and describe types.
- Define hallucinogens/cannabis/combination and describe types.
- Describe the effects of using drugs in combination.

17.1.1. Defining Terms and Adding Context

Substance-related and addictive 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). It is worth noting that the DSM-5 shifted terminology from *drug addiction* to *substance use disorder*, "…to describe the wide range of the disorder, from a mild form to a severe state of chronically relapsing, compulsive pattern of drug taking." The DSM-5 acknowledges that many clinicians will use the term *drug addiction* to describe more severe presentations, but it is omitted from the DSM-5 due to "…its uncertain definition and its potentially negative connotation" (APA, 2022, pg. 543).

What are substances? **Substances** are any ingested materials that cause temporary cognitive, behavioral, or physiological symptoms within the individual. The DSM uses 10 classes of substances: alcohol, caffeine, cannabis, hallucinogens, inhalants, opioids, sedatives, stimulants, tobacco, and other (or unknown).

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 or must 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 known as **tolerance.** As tolerance builds, additional physical and psychological symptoms present, often causing significant disturbances in an individual's personal and professional life. Individuals with substance abuse often spend 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. 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 abuse of multiple substances, withdrawal should be closely monitored in a hospital setting to avoid severe consequences such as seizures, stroke, or even death.

According to the DSM-5-TR (APA, 2022), the substance-related disorders are divided into two groups: substance use disorders and substance-induced disorders which include substance intoxication and substance withdrawal. While there are some subtle differences in

symptoms, particularly psychological, physical, and behavioral symptoms, the general diagnostic criteria for substance use disorder, substance intoxication, and substance withdrawal remains the same across substances. These criteria are reviewed below, with more specific details of psychological, physical, and behavioral symptoms in the Section 11.1.5.: Types of Substances Abused.

17.1.2. Substance Use Disorder

The essential feature of **substance use disorder**, is a "…cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues using the substance despite significant substance-related problems" (APA, 2022, pg. 544) and can be diagnosed for all the substance classes except caffeine. Distress or impairment can be described as any of the following: inability to complete or lack of participation in work, school or home activities; increased time spent on activities obtaining, using, or recovering from substance use; impairment in social or interpersonal relationships; use of a substance in a potentially hazardous situation; psychological problems due to recurrent substance abuse; craving the substance; an increase in the amount of substance used over time (i.e., tolerance); difficulty reducing the amount of substance used despite a desire to reduce/stop using; and/or withdrawal symptoms (APA, 2022). While the number of these symptoms may vary among individuals, only two symptoms are required for a diagnosis of substance use disorder.

17.1.3. Substance Intoxication

For a diagnosis of **substance intoxication**, the individual must have recently ingested a substance. 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). This said, the most common changes involve disturbances of perception, wakefulness, attention, thinking, psychomotor behavior, interpersonal behavior, and judgment (APA, 2022).

17.1.4. Substance Withdrawal

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 psychological symptoms within a few hours after cessation/reduction. These symptoms cause significant distress or impairment in daily functioning (APA, 2022). As with substance intoxication, physiological and 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.

17.1.5. Types of Substances Abused

For our purposes, the most abused substances will be divided into three categories based on how they impact one's physiological state: depressants, stimulants, and hallucinogens/cannabis/combination.

17.1.5.1. Depressants. Depressants include alcohol, sedative-hypnotic drugs, and opioids are known to have an 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 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-aged students report engaging in binge drinking, with 14% binge drinking at least 5 days per month (SAMHSA, 2013). In addition to these high levels of alcohol consumption, students also engage in other behaviors such as skipping meals, which can impact the rate of alcohol intoxication and place them at risk for dehydration, blacking out, and developing alcohol-induced seizures (Piazza-Gardner & Barry, 2013).

The "active" 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 bloodstream, 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 readily in conversation, and in general, produce a confident and happy personality. However, when consumption is increased or excessive, the central nervous system is unable to metabolize

the ethyl alcohol adequately, and adverse 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 those high in fat and carbohydrates, slow the absorption rate of ethyl alcohol, thus reducing its effects. Regarding gender, women absorb and metabolize alcohol differently than men, 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 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, can impact the liver's ability to metabolize alcohol, thus affecting 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 appropriate dosage, they can have a sedative effect, thus making them a suitable drug for treating anxiety-related disorders. In the early 1900s, **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— Xanax, 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 (pre-flight or before surgery) or long-term use (generalized anxiety disorder). While they do not produce respiratory distress in large dosages like barbiturates, 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 1800s, **morphine** was isolated from opium by German chemist Friedrich Wilhelm Adam Serturner. Due to its analgesic effect, it was named after the Greek god of dreams, Morpheus (Brownstein, 1993). Its popularity grew during the American 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 an effort 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 more addictive than morphine. In 1917, Congress stated 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 had abused prescription narcotic pain relievers in the past year (SAMHSA, 2016). In an effort to reduce such abuse, the FDA developed programs to educate prescribers about the risks of misuse and abuse of opioid medications.

17.1.5.2. 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 commonly 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 potent natural stimulant known to date (Acosta et al., 2011). 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 effects of cocaine are due to an increase of *dopamine, norepinephrine*, and *serotonin* in various brain structures (Hart & Ksir, 2014; Haile, 2012).

One key feature of cocaine use is the rapid high of *cocaine intoxication,* followed by the quick depletion, 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 delusions (Haile, 2012). Conversely, as the drug leaves the system, the individual will experience adverse 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 in various ways. While cocaine was initially snorted via the nasal cavity, individuals found that if the drug was smoked or injected, its effects were more potent 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% pure. Due to its low melting point, freebased cocaine is easy to smoke via a glass pipe. Inhaled cocaine is absorbed into the bloodstream and brain within 10-15 seconds suggesting its effects are felt almost immediately (Addiction Centers of America).

Crack is a derivative 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, they 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 we are sure you 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 form of caffeine each 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 have been observed with the simultaneous popularity of energy drinks. Common energy drinks such as Monster and Red Bull have nearly double the amount of caffeine of tea and coke (Bigard, 2010). While adults commonly consume these drinks, 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 a rise in ER visits due to the intoxication effects (SAMHSA, 2013).

17.1.5.3. 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 the tops of plants (SAMHSA, 2014). Many external factors impact the potency of cannabis, such as the climate it was grown in, the method of preparation, 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 hunger, 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 Americans report regular use of marijuana, only 10% 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 reported 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 of developing 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.

17.1.5.4. Using drugs in combination. 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, 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, judgment 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.

You should have learned the following in this section:

- An individual is diagnosed with substance use disorder, substance intoxication, or substance withdrawal specific to the substance or substances being ingested though the symptoms remain generally the same across substances.
- Substance use disorder occurs when a person experiences significant impairment or distress for 12 months due to the use of a substance.
- Substance intoxication occurs when a person has recently ingested a substance leading to significant behavioral and/or psychological changes.
- Substance withdrawal occurs when there is a cessation or reduction of a substance that has been used for a long period of time.
- Depressants include alcohol, sedative-hypnotic drugs, and opioids.
- Stimulants include cocaine and amphetamines, but caffeine as well.
- Hallucinogens come from natural sources and produce powerful changes in sensory perception.
- Cannabis is also derived from a natural plant and produces psychoactive effects.
- Many drugs are taken by users in combination which can have dangerous results depending on the interactions between the substances.

Section 17.1 Review Questions

1. What is a substance?
2. What is the difference between substance intoxication and substance abuse?
3. What is the difference between tolerance and withdrawal?
4. Create a table listing the three types of substances abused, as well as the specific substances within each category.
5. What are the common factors that affect alcohol absorption?
6. What are the effects of sedative-hypnotic drugs?
7. What receptors are responsible for increasing activity in alcohol and benzodiazepines?
8. What is responsible for the addictive nature of opioids?
9. Which neurotransmitters are implicated in cocaine use?
10. What are the different ways cocaine can be ingested?
11. List the common types of amphetamines.

17.2. Prevalence and Comorbidity

Section Learning Objectives

- Describe the epidemiology of depressants.
- Describe the epidemiology of stimulants.
- Describe the epidemiology of hallucinogens.
- Describe the comorbidity of substance-related and addictive disorders.

17.2.1. Prevalence of Substance-Related and Addictive Disorders

It has been estimated that nearly 9% 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 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).

17.2.1.1. Depressants. Concerning depressant substances, men outnumber women in alcohol abuse 2 to 1 (Johnston et al., 2014). Ethnically, Native Americans have highest rate of alcoholism, followed by White, Hispanic, African, and Asian Americans. With regards to opioid use, roughly 1% of the population has this disorder, with 80% of those being addicted to pain-

reliever opioids such as oxycodone or morphine; the remaining 20% are heroin (SAMHSA, 2014).

17.2.1.2. Stimulants. Nearly 1.1% 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 growing concern is the abuse of stimulant medication among college students as 17% of college students reported abusing stimulant medications. Greek organization membership, academic performance, and other substance use were the most highly correlated variables related to stimulant medication abuse.

17.2.1.3. Hallucinogens. Up to 14% of the 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 (APA, 2022). 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.

17.2.2. General Comorbidity

Substance abuse, in general, has a high comorbidity 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 consensus among researchers is that there is a strong relationship between substance abuse and mood, anxiety, PTSD, and personality disorders (Grant et al., 2016).

You should have learned the following in this section:

- More men and Native Americans are addicted to depressants.
- Cocaine is more prevalent in suburban neighborhoods due to its cost and methamphetamine is used equally by men and women.
- Hallucinogens are used by up to 14% of the general population.
- Substance abuse has a high comorbidity within itself and with mental health disorders such as mood, anxiety, PTSD, and personality disorders.

Section 17.2 Review Questions

1. Identify the gender and ethnicity differences of substance abuse across the three substance categories.
2. Are these substances abused by other unique groups of people?
3. With what other conditions are substance-related and addictive disorders highly comorbid?

17.3. 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.

17.3.1. Biological

17.3.1.1. 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 abuse is among the most well studied substances, likely because it is the only legal substance (except cannabis in some states). Twin studies have indicated a range of 50-60% heritability risk for alcohol disorder (Kendler et al., 1997). 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) 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

substance abuse; additionally, the individual may also be at risk due to their familial environment where their parents 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).

17.3.1.2. 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. Most of the 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 decrease dopamine. Cannabis has been shown to reduce the production of endocannabinoids.

17.3.2. 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 expectancies are thought to increase drug-seeking behavior, while negative expectancies would decrease substance use (Oei &

Morawska, 2004). Several 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 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).

17.3.3. 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 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 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. Studies of substance use on animals routinely support 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). Secondly, negative reinforcement is involved in 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, an individual will consume more of the substance, thus again escaping the negative symptoms and enjoying the "highs" of the substance.

17.3.4. Sociocultural

Arguably, one of the strongest influences of substance abuse is the impact of one's friends and the immediate environment. Peer attitudes, perception of others' 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 possibility 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 a 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 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 are higher among poorer people (SAMHSA, 2014). Furthermore, additional stressors such as childhood abuse and 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).

You should have learned the following in this section:

- Biological causes of substance-related and addictive disorders include the brain reward system and a genetic predisposition, though if the individual is not exposed to the substance they will not develop the substance abuse.
- Cognitive causes of substance-related and addictive disorders include the expectancy effect, and research provides stronger support for positive expectancy over negative expectancy.
- Behavioral causes of substance-related and addictive disorders include positive and negative reinforcement.
- Sociocultural causes of substance-related and addictive disorders include friends and the immediate environment.

Section 17.3 Review Questions

1. Discuss the *brain reward system.* What neurobiological regions are implicated within this system?
2. Define the *expectancy effect.* How does this explain the development and maintenance of substance abuse?
3. Discuss operant conditioning in the context of substance abuse. What are the reinforcers?
4. How does the sociocultural model explain substance abuse?

17.4. Assessment

Section Learning Objectives

- Outline the assessment process for substance-use disorders.

Overall, assessment is based on verbal report from caregivers, teachers, and the individual themselves. It is very important to be clear to the adolescent about who will have access to the information. Moreover, it may be helpful to discuss with parents the importance of transparency in the interview, and thus, the need for assurance that punishment for behaviors will not occur. Essentially, the adolescent can report on their behaviors without fear of repercussions. Nevertheless, full reports on use can be difficult to obtain. At times, using objective screeners and measures may also help. There are a host of them, some that target general substance use behaviors such as the CRAFFT, others that target specific drug use such as the Cannabis Use Disorders Identification Test (CUDIT). Additionally, screening for other risky behaviors, delinquent peer groups, and other serious mental health conditions should occur. Family history of substance use disorders should be obtained due to its strong etiological relevance and may inform prognosis.

You should have learned the following in this section:

- Assessment is based on verbal report from caregivers, teachers, and the individual themselves.
- CRAFFT and CUDIT are typical assessment tools that are used.

Section 17.4 Review Questions

1. How do you assess for substance-induced disorders?

17.5. 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 affected 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).

17.5.1. Biological

17.5.1.1. Detoxification. Detoxification refers to the medical supervision of withdrawal from a specified drug. While most detoxification programs are inpatient for increased monitoring, some programs allow for outpatient detoxification, particularly if the addiction is not as severe. There are two main theories of detoxification—gradually decreasing the amount of the substance until the individual is off the drug completely, or eliminating 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.

17.5.1.2. 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 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 to heroin with an 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 adverse 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, others claim that the combination of methadone with education and psychotherapy can successfully help individuals off both illicit drugs and methadone medications (Jhanjee, 2014).

17.5.1.3. Antagonist drugs. Unlike agonist drugs, **antagonist drugs** block or change the effects of the addictive drug. 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. In theory, this treatment appears promising, but it is extremely dangerous as it can send the individual into immediate, severe withdrawal symptoms (Alter, 2014). This type of treatment requires appropriate medical supervision to ensure the safety of the patient.

17.5.2. Behavioral

17.5.2.1. Aversion therapy. Based on respondent 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. By pairing this aversive stimulus to the abused substance, the individual will begin to independently pair the substance with an aversive thought, thus reducing their craving/desire for the substance. Some view the use of agonist and antagonist drugs as a form of aversion therapy as these medications utilize the same treatment strategy as traditional aversion therapy.

17.5.2.2. Contingency management. Contingency management is a treatment approach that emphasizes *operant conditioning*—increasing sobriety and adherence to treatment programs 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 individuals to gain incentives specific to their

interests, 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 with the treatment program (Mignon, 2014). Despite its success, dissemination of this type of treatment has been rare. To rectify this, the federal government has provided financial resources through SAMHSA for the development, implementation, and evaluation of contingency management as a treatment to reduce alcohol and drug use (Mignon, 2014).

17.5.3. Cognitive-Behavioral

17.5.3.1. 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 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).

17.5.4. Sociocultural

17.5.4.1. 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 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. To achieve this, the participants are encouraged to “take one day at a time.” In using the 12 steps, participants are emboldened to admit that they have a disease, that they are powerless over this disease, and that their disease is more powerful than any person. Therefore, participants turn their addiction over to God and ask for help to right their wrongs and remove their negative character defects and shortcomings. The final steps include identifying and making 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 over 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.

17.5.4.2. 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 community while also attending regular 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. Several also incorporate 12-step programs into treatment, as many patients transition from a residential treatment center to a 12-step program post discharge. As one would expect, the residential treatment goal is 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 useful 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.

17.5.4.3. 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.

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.

You should have learned the following in this section:

- Biological treatment options for substance-related and addictive disorders include detoxification programs, agonist drugs, and antagonist drugs.
- Behavioral treatment options for substance-related and addictive disorders include aversion therapy and contingency management.
- Cognitive-behavioral treatment options for substance-related and addictive disorders include relapse prevention training.
- Sociocultural treatment options for substance-related and addictive disorders include Alcoholics Anonymous, residential treatment centers, and community reinforcement.

Section 17.5 Review Questions

1. Discuss the differences between agonist and antagonist drugs. Give examples of both.
2. What are the two behavioral treatments discussed in this module? Discuss their effectiveness.
3. What are the main components of the 12-step programs? How effective are they in substance abuse treatment?

Apply Your Knowledge

CASE VIGNETTE

Antonio, a 14-year-old boy, was recently suspended because he was caught on school premises with marijuana. Antonio lives in an area in which marijuana is legalized; however, he is not of permitted age to be in possession and there is a strict school policy stating that marijuana is not allowed on school premises. Antonio's mother occasionally uses marijuana, legally, in a social setting; however, his father dislikes this and is strongly against the use of marijuana. Antonio explained that he uses marijuana only occasionally with friends. However, through school-mandated counseling, his parents learned that Antonio has also tried other substances including Adderall and other simulant-based pills. Antonio said he only did this once or twice when studying for an exam, especially because he has a desire to keep his grades up to get a scholarship to his dream school. His parents have always communicated the importance of grades, but recently, keeping his grades up had been a struggle, and he tried stimulants in hopes it would help. With this new knowledge, his parents are more concerned. They report that Antonio appears to have a good group of friends, but that Antonio has been a bit more isolated recently. Antonio swears he will not use any more substances, but his parents aren't sure what to do now.

QUESTIONS TO TEST YOUR KNOWLEDGE

1. Do you think Antonio has or is at risk for a substance use disorder? If so, what disorders are you concerned about?
2. Do you think Antonio has any non-substance related disorders?
3. Does Antonio need treatment? What might his parents do next?
4. Does Antonio have protective factors? If so, what are they?
5. Does Antonio have risk factors? If so, (1) what are they and (2) what can be done to mitigate that risk?
6. Are there societal factors at play here?

Module Recap

And that concludes the final chapter of this book. 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, comorbidity, and etiology of the disorders. We then also discussed biological, behavioral, cognitive-behavioral, and sociocultural treatment approaches.

We hope you enjoyed the book and learned a lot.

Glossary

A

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

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

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

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

Acquired capability for suicidality - The idea that, over time, an individual who has been exposed to pain or life-threatening danger are desensitized, to a degree, to death or bodily harm

Action potential – When the neuron depolarizes and fires

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

Adaptive skills - Skills that help us navigate our daily lives successfully

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

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

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

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

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

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

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

Antecedents - The environmental events or stimuli that trigger a behavior

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

Attention-Deficit/Hyperactivity Disorder (ADHD) - A disorder in which individuals have difficulty with executive functioning – an individual's decision-making ability, which involves working memory, inhibition of inappropriate or unhelpful responses, and ability to focus in on relevant information while dismissing unimportant or irrelevant information

Attribution theory - The idea that people are motivated to explain their own and other people's behavior by attributing causes of that behavior to personal reasons or *dispositional factors* that

are in the person themselves or linked to some trait they have; or *situational factors* that are linked to something outside the person

Autism spectrum disorder - A neurodevelopmental concern related to social and adaptive functioning characterized by two major areas – deficits in social communication and interaction and significant concern related to restricted and receptive behaviors and/or interests

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

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

Avoidant/Restrictive Food Intake Disorder (ARFID) - Characterized by simply a low interest in eating/feeding which ultimately leads to a deficit in obtaining appropriate calories and nutrients

Axon - Sends signals/information to neighboring neurons

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

B

Behavior modification - The process of changing behavior

Behavioral assessment - The measurement of a target behavior

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

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

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

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

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

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

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

C

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

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

Child psychopathology - Abnormal psychology that is present during childhood.

Classical conditioning - When two events that occur close together become strongly associated with one another, despite their lack of causal relationship

Classification - The way in which we organize or categorize things

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

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

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

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

Clinical diagnosis - The process of using assessment data to determine if the pattern of symptoms the person presents with is consistent with the diagnostic criteria for a specific mental disorder set forth in an established classification system such as the DSM-5 or ICD-10

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

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

Cognition or intellectual functioning - Our ability to problem solve, understand and analyze complex material, absorb information from our environment, and reason

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

Cognitive triad – When a person interprets negative thoughts about their experiences, themselves, and their futures

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

Complex trauma - Occurs when a child experiences multiple traumatic events

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

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

Conduct Disorder - A more severe behavioral disorder in which an individual displays a disregard not only for rules and authority, but also the rights and conditions of humans and/or animals

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

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

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

Contingencies - When one thing occurs due to another

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

Cortisol - A hormone released as a stress response

Counterconditioning - The reversal of previous learning

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

Course – The particular pattern a disorder displays

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

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

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

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

Cyclothymic disorder – A mood disorder characterized by hypomanic symptoms and *mild* depressive symptoms (i.e. do not fully meet criteria for a depressive episode)

D

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

Deinstitutionalization - The release of patients from mental health facilities

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

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

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

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

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

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

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

Differential reinforcement - When we attempt to get rid of undesirable or problem behaviors by using the positive reinforcement (providing a reward of some sort) of desirable behaviors

Disinhibited Social Engagement Disorder (DSED) – Children with DSED tend to be overly social and interact with complete strangers. They experience impaired caregiving, which means that the caregiver does not sufficiently care for the child on a consistent basis.

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

Disruptive Mood Dysregulation Disorder (DMDD) - a depressive disorder in which a child presents as persistently irritable– they are likely often described as an irritable/unhappy child. The child displays extreme outbursts over minor stressors in their environment

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

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

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

Dyscalculia – Disorder in math

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

Dyslexia – Disorder in reading

E

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

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

Enactive learning - Learning by doing

Encopresis - Incontinence with bowel movements (i.e., the inability to remain absent of bowel accidents)

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

Enuresis - Urinary incontinence or the inability to remain absent of urinary accidents

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

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

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

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

Etiology - The cause of the disorder

Excoriation - Characterized by an individual recurrently skin picking

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

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

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

Exposure treatments – When an individual is *exposed* to their feared stimuli

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

F

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

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

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

Forensic interview - A recorded interview with the goal to allow a child to provide information about their experiences of abuse in a non-leading and supportive method

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

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

Functional behavioral assessment - When we closely scrutinize the antecedents and consequences to behaviors to see what affects the occurrence or nonoccurrence of a desired or problem behavior

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

G

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

Gaps - Holes in the literature of a given area

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

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

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

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

Glutamate – Neurotransmitter associated with learning and memory

H

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

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

Hypertension - -Chronically elevated blood pressure

Hypomanic episode - Persistently elevated, expansive, or irritable mood; May present as persistent increased activity or energy; Symptoms last at least 4 consecutive days and present

most of the day, nearly every day; Includes at least three of the following: inflated self-esteem or grandiosity, decreased need for sleep, more talkative or pressured speech, flight of ideas, distractibility, increase in goal-directed activity or psychomotor agitation, or excessive involvement in activities that have a high potential for painful consequences

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

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

Hypothesis – A specific, testable prediction

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

Hyperactive/impulsive symptoms - Symptoms of ADHD related to excessive energy and movement as well as impulsivity

I

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

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

Inattentive symptoms - Children with these symptoms tend to lose things frequently, have a hard time following directions because they get distracted, are disorganized, and make a lot of careless mistakes on classwork; Part of ADHD

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

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

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

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

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

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

Ions - Charged particles found both inside and outside the neuron

J

K

L

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

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

Latent content - The hidden or symbolic meaning of a dream

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

Learned helplessness – When a person or animal learns that they cannot avoid a painful stimulus on one day and then the next, when given the chance to escape the stimulus, choose not to still believing they cannot escape

Learning - Any relatively permanent change in behavior due to experience

Learning disorder - Characterized by the inability or difficulty processing academic or functional information in our environment

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

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

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

Locus coeruleus - The brain structure that serves as an "on-off" switch for norepinephrine neurotransmitters

M

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

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

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

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

Mathematics learning disorder - This may be related to simple calculation abilities such as math facts or more complex problem-solving and reasoning abilities

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

Melatonin - A hormone released when it is dark outside to assist with the transition to sleep

Mental disorders - Characterized by psychological dysfunction which causes physical and/or psychological distress or impaired functioning and is not an expected behavior according to societal or cultural standards

Mental health epidemiology - Refers to the occurrence of mental disorders in a population

Mental hygiene movement - An idea arising in the late 18th century to the early 19th century with the fall of the moral treatment movement, it focused on the physical well-being of patients

Mental status examination - Used to organize the information collected during the clinical interview and systematically evaluates the patient through a series of questions assessing appearance and behavior to include grooming and body posture, thought processes and content to include disorganized speech or thought and false beliefs, mood and affect such that whether the

person feels hopeless or elated, intellectual functioning to include speech and memory, and awareness of surroundings to include where the person is and what the day and time are

Model - A representation or imitation of an object

Modeling - Techniques used to change behavior by having subjects observe a model in a situation that usually causes them some anxiety

Mood lability - Rapid shifts in mood

Moral treatment movement – An idea arising in Europe in the late 18th century and then in the United States in the early 19th century, it stressed affording the mentally ill respect, moral guidance, and humane treatment, all while considering their individual, social, and occupational needs

Myelin sheath - The white, fatty covering which: 1) provides insulation so that signals from adjacent neurons do not affect one another and, 2) increases the speed at which signals are transmitted

Multicultural psychology – The area of psychology which attempts to understand how the various groups, whether defined by race, culture, or gender, differ from one another

Multidimensional disorders – States that there are many contributing factors that lead to the development of an eating disorder

Multi-dimensional model – An explanation for mental illness that integrates multiple causes of psychopathology and affirms that each cause comes to affect other causes over time

Muscle Dysmorphia - The belief that one's body is too small, or lacks appropriate amount of muscle definition

Mutually exclusive - Meaning that only one diagnosis can be assigned at any given time

N

Naturalistic observation - A research method in which the scientist studies human or animal behavior in its natural environment which could include the home, school, or a forest

Negative Punishment (NP) – This is when something good is taken away or subtracted making a behavior less likely in the future

Negative Reinforcement (NR) – This is when something bad or aversive is taken away or subtracted due to your actions, making it that you will be more likely to make the same behavior in the future when the same stimuli presents itself

Nerves - A group of axons bundled together like wires in an electrical cable

Neurological tests - Used to diagnose cognitive impairments caused by brain damage due to tumors, infections, or head injury; or changes in brain activity

Neuron - The fundamental unit of the nervous system

Neurotransmitter – When the actual code passes from one neuron to another in a chemical form

Nomenclature – A naming system

Noncontingent reinforcers – When reinforcement (i.e. food or liquid) is provided for a period of time, and does not require a particular behavior from the individual for the individual to receive the reinforce

Nonsuicidal self-injury (NSSI) - Also frequently referred to as self-harm, are self-injurious actions that an individual engages in without the intent to end one's life

Norepinephrine – Neurotransmitter which increases the heart rate and blood pressure and regulates mood

Nucleus - The control center of the body

O

Observation – Observing others either naturalistically or in a controlled environment

Observational learning - When we learn by observing the world around us

Obsessions - Repetitive and persistent thoughts, urges, or images

Obsessive compulsive disorder - More commonly known as OCD, the disorder requires the presence of both obsessions and compulsions

Operant conditioning - A type of associate learning which focuses on consequences that follow a response or behavior that we make (anything we do, say, or think/feel) and whether it makes a behavior more or less likely to occur

Oppositional Defiant Disorder (ODD) - Characterized by a child that is defiant and vindictive at times

Oral Stage – Beginning at birth and lasting to 24 months, the libido is focused on the mouth and sexual tension is relieved by sucking and swallowing at first, and then later by chewing and biting as baby teeth come in

P

Panic disorder - When an individual experiences recurrent panic attacks consisting of physical and cognitive symptoms

Parasympathetic nervous system – The part of the autonomic nervous system that calms the body after sympathetic nervous system arousal

Parietal lobe – The part of the cerebrum that contains the somatosensory cortex and receives information about pressure, pain, touch, and temperature from sense receptors in the skin, muscles, joints, internal organs, and taste buds

Perceived burdensomeness - The idea that an individual cannot meaningfully contribute to one's own life, other's life, or society (e.g., physical impairment, unemployment)

Peripheral nervous system - Consists of everything outside the brain and spinal cord; It handles the CNS's input and output and divides into the somatic and autonomic nervous systems

Period prevalence - Indicates the proportion of a population that has the characteristic at any point during a given period of time, typically the past year

Persistent Depressive Disorder – A mood disorder characterized by poor appetite or overeating, insomnia or hypersomnia, low self-esteem, low energy, and feelings of hopelessness lasting most of the day, for more days than not, for at least 2 years

Persistent (chronic) motor or vocal tic disorder - When either one or more motor tics or one or more vocal tic is present

Personality inventories - Ask clients to state whether each item in a long list of statements applies to them, and could ask about feelings, behaviors, or beliefs

Phallic Stage – Occurring from about age 3 to 5-6 years, the libido is focused on the genitals and children develop an attachment to the parent of the opposite sex and are jealous of the same sex parent

Pica - The act of eating items that are not food, on a regular or recurring basis

Pineal gland - Helps regulate the sleep-wake cycle

Pituitary gland - The "master gland" which regulates other endocrine glands; It influences blood pressure, thirst, contractions of the uterus during childbirth, milk production, sexual

behavior and interest, body growth, the amount of water in the body's cells, and other functions as well

Placebo - Or a sugar pill made to look exactly like the pill given to the experimental group

Plasticity - The ability for our brain to modify its neural connections, our brains have

Point prevalence - Indicates the proportion of a population that has the characteristic at a specific point in time

Polarized – When the neuron has a negative charge inside and a positive charge outside

Pons – The part of the brain that acts as a bridge connecting the cerebellum and medulla and helps to transfer messages between different parts of the brain and spinal cord

Posttraumatic stress disorder - More commonly known as PTSD, is identified by the development of physiological, psychological, and emotional symptoms following exposure to a traumatic even

Positive psychology – The position in psychology that holds a more positive conception of human potential and nature

Positive Punishment (PP) – If something bad or aversive is given or added, then the behavior is less likely to occur in the future

Positive Reinforcement (PR) – If something good is given or added, then the behavior is more likely to occur in the future

Preconscious – According to Freud, the level of personality that includes all of our sensations, thoughts, memories, and feelings

Presenting problem – The issue the person displays

Prevalence - The percentage of people in a population that has a mental disorder or can be viewed as the number of cases per some number of people

Prevention – When we identify the factors that cause specific mental health issues and implement interventions to stop them from happening, or at least minimize their deleterious effects

Prognosis - The anticipated course the mental disorder will take

Projection – When we attribute threatening desires or unacceptable motives to others

Projective tests – A psychological test which consists of simple ambiguous stimuli that can elicit an unlimited number of responses

Psychoanalysis - Psychoanalytic therapy used to understand the personality of a therapist's patient and to expose repressed material

Psychological debriefing - A type of crisis intervention that requires individuals who have recently experienced a traumatic event to discuss or process their thoughts and feelings related to the traumatic event, typically within 72 hours of the event

Psychological model – includes learning, personality, stress, cognition, self-efficacy, and early life experiences and how they affect mental illness

Psychological or psychogenic perspective - States that emotional or psychological factors are the cause of mental disorders and represented a challenge to the biological perspective

Psychological tests - Used to assess the client's personality, social skills, cognitive abilities, emotions, behavioral responses, or interests and can be administered either individually or to groups in paper or oral fashion

Psychopathology - The scientific study of psychological disorders

Public stigma – When members of a society endorse negative stereotypes of people with a mental disorder and discriminate against them

Punishment – Due to the consequence, a behavior/response is less likely to occur in the future

R

Random assignment – When participants have an equal chance of being placed in the control or experimental group

Rape - Forced sexual intercourse or other sexual act committed without an individual's consent

Rationalization – When we offer well thought out reasons for why we did what we did but in reality these are not the real reason

Reaction formation – When an impulse is repressed and then expressed by its opposite

Reactive Attachment Disorder (RAD) – A child with RAD presents as detached from others and like DSED, often experience impaired caregiving.

Reactivity – When the observed changes behavior due to realizing they are being observed

Reading learning disorder - This essentially relates to an individual having difficulty in reading, may that be in comprehending material, reading fluently and quickly, or reading words accurately

Receptor sites – Locations where neurotransmitters bind to

Regression – When we move from a mature behavior to one that is infantile in nature

Reinforcement – Due to the consequence, a behavior/response is more likely to occur in the future

Reinforcement schedule - The rule for determining when and how often we will reinforce a desired behavior

Relapse prevention training - Identifying potentially high-risk situations for relapse and then learning behavioral skills and cognitive interventions to prevent the occurrence of a relapse

Relative refractory period - After a short period of time, the neuron can fire again, but needs greater than normal levels of stimulation to do so

Reliable – When our assessment is consistent

Replication - Repeating a study to confirm its results

Repolarization – When the Na channels close and Potassium channels open; K has a positive charge and so the neuron becomes negative again on the inside and positive on the outside, or polarizes

Repression – When unacceptable ideas, wishes, desires, or memories are blocked from consciousness

Research design - Our plan of action of how we will go about testing the hypothesis

Resistance – According to psychoanalytic theory, is the point during free association that the patient cannot or will not proceed any further

Respondent conditioning (also called classical or Pavlovian conditioning) - Occurs when we link a previously neutral stimulus with a stimulus that is unlearned or inborn

Respondent Discrimination – When the CR is elicited by a single CS or a narrow range of CSs

Respondent Extinction – When the CS is no longer paired with the UCS

Respondent Generalization – When a number of similar CSs or a broad range of CSs elicit the same CR

Resting potential – When the neuron is waiting to fire

Reticular formation – The part of the brain responsible for alertness and attention

Reuptake - The process of the presynaptic neuron taking up excess neurotransmitters in the synaptic space for future use

Reversal or **ABAB design** – A study in which the control is followed by the treatment, and then a return to control and second administration of the treatment condition; builds replication in to the design

Rumination Disorder - The frequent act of regurgitating food with no medical explanation (e.g. gastro concerns, reflux) and in the absence of a body-image/weight-related reason (e.g., anorexia/bulimia)

S

Schema - A set of beliefs and expectations about a group of people, presumed to apply to all members of the group, and based on experience

Self-stigma – When people with mental illnesses internalize the negative stereotypes and prejudice, and in turn, discriminate against themselves

Scientific method - A systematic method for gathering knowledge about the world around us

Sedative-Hypnotic drugs - More commonly known as anxiolytic drugs**,** these drugs have a calming and relaxing effect on individuals

Selective Mutism – A disorder is characterized by an absence of speech in particular social situations in which a person is expected to speak

Self-monitoring – When the person does their own measuring and recording of the ABCs

Self-serving bias - When we attribute our success to our own efforts (dispositional) and our failures to outside causes (situational)

Sensitization - When our reactions are increased due to a strong stimulus

Separation Anxiety Disorder - A disorder that is characterized by excessive fear of separating from a caregiver. A child may worry about the caregiver becoming seriously ill, dying, or being permanently separated from them

Serotonin – Neurotransmitter which controls pain, sleep cycle, and digestion; leads to a stable mood and so low levels leads to depression

Single-subject experimental design – When we have to focus on one individual in a study

Social anxiety disorder - Occurs when an individual experiences anxiety related to social or performance situations, where there is the possibility that they will be evaluated negatively

Social cognition - The process of collecting and assessing information about others

Social desirability - When a participant answers questions dishonestly so that he/she is seen in a more favorable light

Social norms - The stated and unstated rules of society

Social Pragmatic Communication Disorder - Characterized by overall difficulty with understanding how social communication should occur (e.g., to and fro), flexible understand of places and contexts of conversation (e.g., we talk about personal things to friends and not to acquaintances, we talk quietly in library and loudly at a football game), and subtle social cues

Sociocultural Model – includes factors such as one's gender, religious orientation, race, ethnicity, and culture that affect mental illness

Soma - The cell body

Somatic nervous system - Allows for voluntary movement by controlling the skeletal muscles and carries sensory information to the CNS

Specific phobia - Observed when an individual experiences anxiety related to a specific object or subject

Spontaneous recovery – When the CS elicits the CR after extinction has occurred

Standardization – When we use clearly laid out rules, norms, and/or procedures in the process of assessing clients

Statistical significance - An indication of how confident we are that our results are due to our manipulation or design and not chance

Stereotypic Movement Disorder (SMD) - A disorder in which an individual engages in repetitive movements and those movements have no clear functional purpose

Stereotypy - Stereotyped movement

Stigma - When negative stereotyping, labeling, rejection, and loss of status occur

Stimulus generalization - The tendency for the conditioned stimulus to evoke similar responses to other conditions

Stressors - Any event- either witnessed firsthand, experienced personally or experienced by a close family member- that increases physical or psychological demands on an individual

Sublimation – When we find a socially acceptable way to express a desire

Substance abuse - Occurs when an individual consumes the substance for an extended period of time, or has to ingest large amounts of the substance to get the same effect a substance provided previously

Substance Intoxication – A substance use disorder characterized by recent ingestion of substance, significant behavioral or psychological changes immediately following the ingestion of substance, physical and physiological symptoms develop after ingestion of substance, and changes in behavior not attributable to a medical condition or other psychological disorder

Substance Use Disorder – A substance use disorder diagnosed when the individual presents with at least two criteria to include: substance is consumed in larger amounts over time, desire or inability to reduce quantity of substance use, cravings for substance use, use of the substance in potentially hazardous situations, tolerance of substance use, and withdrawal, to name a few (11 total criteria)

Substance Withdrawal - A substance use disorder characterized by cessation or reduction in substance that has been previously used for a long or heavy period of time, physiological and/or

psychological symptoms within a few hours after cessation/reduction, physiological and/or psychological symptoms cause significant distress or impairment in functioning, and symptoms not attributable to a medical condition or other psychological disorder

Substances - Any ingested materials that cause temporary cognitive, behavioral, and/or physiological symptoms within the individual

Superego - According to Freud, the part of personality which represents society's expectations, moral standards, rules, and represents our conscience

Sympathetic nervous system - Involved when a person is intensely aroused; It provides the strength to fight back or to flee (fight-or-flight instinct)

Synapse - The point where the code passes from one neuron to another; Consists of three parts – the *axon* of the sending neuron; the *space* in between called the synaptic space, gap, or cleft; and the *dendrite* of the receiving neuron

Syndrome - Symptoms occurred regularly in clusters

Systematic desensitization - An exposure technique that utilizes relaxation strategies to help calm the individual as they are presented with the fearful object

T

Target behavior - Whatever behavior we want to change and it can be in excess or needing to be reduced, or in a deficit state and needing to be increased

Thalamus – The major sensory relay center for all senses but smell

Thanatos - Our death instinct which is either directed inward as in the case of suicide and masochism or outward via hatred and aggression

Thematic Apperception Test – A projective test which asks the individual to write a complete story about each of 20 cards shown to them and give details about what led up to the scene depicted, what the characters are thinking, what they are doing, and what the outcome will be

Theory – A systematic explanation of a phenomenon

Threshold of excitation - -55mV or the amount of depolarization that must occur for a neuron to fire; It rises from -70mV to -55mV

Thwarted belongingness - Not feeling connected to others or feeling isolated

Thyroid gland – The endocrine gland which regulates the body's rate of metabolism and so how energetic people are.

Tics - Can be either *motor movements* (motor) or *vocalizations* (vocal)

Tolerance - The need to continually increase the amount of ingested substance

Tourette's Disorder - When *both* motor and vocal tics are present

Transference – In psychoanalytic theory, this technique involves patients transfering to the therapist attitudes he/she held during childhood

Trauma - When a stressor is significant enough that they pose a threat, whether real or imagined, to the individual or loved one

Trauma-focused cognitive-behavioral therapy (TF-CBT) - An adaptation of CBT, that utilizes both CBT techniques, as well as trauma sensitive principles to address the trauma related symptoms

Treatment - Any procedure intended to modify abnormal behavior into normal behavior

Trephination - In which a stone instrument known as a *trephine* was used to remove part of the skull, creating an opening

Trial and error learning - Making a response repeatedly if it leads to success

Trichotillomania - Characterized by an individual recurrently pulling their hair our and results in hair loss

U

Unconscious – According to Freud, the level of personality not available to us

Uni-dimensional model – A single factor explanation for mental illness

V

Validity – When the test measures what it says it measures

Variable Interval schedule (VI) – Reinforcing at some changing amount of time

Variable Ratio schedule (VR) – Reinforcing some varying number of responses

W

Written expression learning disorder - This may refer to simply the ability to accurately spell words or punctuate and use correct grammar, or it may also include one's ability or create written work that is well-organized and comprehendible

X

Y

Z

References

Aarnoudse-Moens, C. S. H., Weisglas-Kuperus, N., van Goudoever, J. B., & Oosterlaan, J. (2009). Meta-analysis of neurobehavioral outcomes in very preterm and/or very low birth weight children. *Pediatrics*, *124*(2), 717-728.

Acosta, M., Haller, D., & Schnoll, S. (2011). Cocaine and stimulants. In RJ. Frances, AH Mack, & SI. Miller (Eds.), *Clinical textbook of addictive disorders* (3rd ed. pp. 183-218). New York, NY: Guilford Press.

Addiction Centers of America – Differences between crack & Cocaine. Available at https://americanaddictioncenters.org/cocaine-treatment/differences-with-crack/ Accessed on November 20, 2017.

Advokat, C., Comaty, J., Julien, R. (2014). *Julien's primer of drug reaction.* New York: Worth Publishers.

Agras, W., Fitzsimmons-Craft, E., & Wilfley, D. (2017). Evolution of cognitive-behavioral therapy for eating disorders. *Behaviour Research and Therapy, 88,* 26-36.

Agras, W., Lock, J., Brandt, H., Bryson, S., Dodge, E., Halmi, K.,…Woodside, B. (2014). Comparison of 2 family therapies for adolescent anorexia nervosa: a randomized parallel trial. *Journal of American Medical Association Psychiatry, 71*(11), 1279-1286.

Ahmed, I., Cook, T., Genen, L., & Schwartz, R. (2014). Body dysmorphic disorder. Retrieved from http://emedicine.medscape.com/article/291182overview0aw2aab6b2b3.

Alcoholics Anonymous 2014 Membership Survey. (2014). AA World Services.

Alter, C. (2014. March 10). Holder urges use of drug to combat heroin overdoses. *Time.*

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders*. Text revision (4th ed.). Washington, DC: American Psychiatric Publishing.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th Ed.). Washington, DC: American Psychiatric Publishing.

American Psychiatric Association. (2013b). *Disruptive Mood Dysregulation Disorder*. Retrieved from https://www.psychiatry.org/File%20Library/Psychiatrists/Practice/DSM/APA_DSM-5-Disruptive-Mood-Disregulation-Disorder.pdf

American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders.* (5th ed. Text Revision). Arlington, VA: American Psychiatric Publishing.

Anderson, S. R., Taras, M., & Cannon, B. O. (1996). Teaching new skills to young children with autism. In C. Maurice (Ed.), *Behavioral intervention for young children with autism.* Austin, TX: Pro-Ed.

Astburry, J. (2010). The social causes of women's depression: A question of rights violated? In D.C., Jack & A. Ali (Eds.), *Silencing the self across cultures: Depression and gender in the social world* (pp. 19-45). New York: Oxford University Press.

Austin, S. B., Ziyadeh, N. J., Forman, S. F., Prokop, L. A., Keliher, A., & Jacobs, D. (2008). Screening high school students for eating disorders: Results of a national initiative. *Preventing Chronic Disease*.

Ayd, F. J. (1956). A clinical evaluation of frenquel. *The Journal of nervous and mental disease*, *124*(5), 507-509.

Balakrishnan, S., & Alias, A. (2017). Usage of Social Stories in Encouraging Social Interaction of Children with Autism Spectrum Disorder. *Journal of ICSAR*, *1*(2), 91-97.

Barahona-Corrêa, J. B., & Filipe, C. N. (2016). A concise history of Asperger syndrome: The short reign of a troublesome diagnosis. *Frontiers in Psychology*, *6*, 2024.

Barkley, R. A. (2015). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment.* (4th ed). Charleston, SC: Guilford Press

Beck, A. (1967). *Depression: Causes and treatment.* Philadelphia: University of Pennsylvania Press.

Beck, A. T. (Ed.). (1979). *Cognitive therapy of depression*. Guilford press.

Beck, A. (1991). Cognitive therapy: A 30-year retrospective, *American Psychologist, 46*(4), 368-375.

Beck, A. (2002). Cognitive models of depression. In R. L. Leahy & E.T. Dowd (Eds). *Clinical advance sin cognitive psychotherapy: Theory and Application* (pp. 29-61). New York: Springer.

Belendiuk, K. & Riggs, P. (2014). Treatment of adolescent substance use disorders. *Current Treatment Options in Psychiatry, 1,* 175-188.

Bender, E. (2004). Data show wide variation in addiction treatment costs. *Psychiatric News, 39,* 11.

Berkowitz, M. W., & Bier, M. C. (2007). What works in character education. *Journal of Research in Character Education*, *5*(1), 29.

Bernardy, N. & Friedman, M. (2015). Psychopharmacological strategies in the management of posttraumatic stress disorder (PTSD): What have we learned? *Current Psychiatry Report, 17,* 20-30.

Bettis, A. H., Coiro, M. J., England, J., Murphy, L. K., Zelkowitz, R. L., Dejardins, L., ... &

Burklow, K. A., Phelps, A. N., Schultz, J. R., McConnell, K., & Rudolph, C. (1998). Classifying complex pediatric feeding disorders. *Journal of Pediatric Gastroenterology and Nutrition, 27*, 143-147.

Compas, B. E. (2017). Comparison of two approaches to prevention of mental health problems in college students: enhancing coping and executive function skills. *Journal of American College Health*, 1-10.

Beucke, J., Sepulcre, J., Talukdar, T., Linnman, C., Zchenderliein, K., Endrass, T., . . . Kathmann, N. (2013). Abnormally high degree connectivity of the orbitofrontal cortex in obsessive-compulsive disorder. *American Journal of Psychiatry, 70,* 619-629.

Bienvenu, O., Davydow, D., & Kendler, K. (2011). Psychiatric "diseases" vs. behavioral disorders and degree of genetic influence. *Psychological Medicine, 41,* 33-40.

Bigard, A. (2010). Risks of energy drinks in youth. *Archives of Pediatrics, 17,* 1625-1631.

Bisaga, A., Sullivan, M., Glass, A., Mishlen, K., Pavlicova, M. Haney, M., … Nunes, E. (2015). The effects of dronabinol during detoxification and the initiation of treatment with extended release naltrexone. *Drug and Alcohol Dependence, 154,* 38-45.

Bjornsson, A., Dyck, I., Moitra, E., Stout, R., Weisberg, R., Keller, M., & Phillips, K. (2011). The clinical course of body dysmporhic disorder in the Harvard/Brown Anxiety Research Project (HARP). *Journal of Nervous and Mental Disease, 199,* 55-57.

Black, M., Basile, K., Breiding, M., Smith, S., Walters, M., Merrick, M., . . . Stevens, M. (2011). *The National Intimate Partner and Sexual Violence Survey: 2010 summary report.* Retrieved from the Centers for Disease Control and Prevention, National Center for Injury Prevention and Control: http://www.cdc.gov/ViolencePrevention/pdf/NISVS_Report2010-a.pdf

Bokor, G. & Anderson, P. (2014). Obsessive compulsive disorder. *Journal of Pharmacy Practice, 27,* 116-130.

Borkovec, T. D., & Ruscio, A. M. (2001). Psychotherapy for generalized anxiety disorder. *The Journal of Clinical Psychiatry, 62*, 37-42.

Bourin, M., Malinge, M., & Guitton, B. (1995). Provocative agents in panic disorder. *Therapie, 50*(4), 301-306.

Bradshaw, C. P. (2015). Translating research to practice in bullying prevention. *American Psychologist*, *70*(4), 322.

Brambrink, D. (2004). A comparative study for the treatment of anxiety in women using electromyographic biofeedback and progressive muscle relaxation and coping with stress: A manual for women. *Dissertation Abstracts International: Section B: The Science and Engineering, 65,* 3146.

Brewin, C., Andrews, B., Rose, S., & Kirk, M. (1999). Acute stress disorder and posttraumatic stress disorder in victims of violent crime. *American Journal of Psychiatry, 156,* 360-366.

Brown, S. (1985). Reinforcement expectancies and alcoholism treatment outcome after a one-year follow-up. *Journal of Studies on Alcohol, 46,* 304-308,

Brownstein, M. (1993). A brief history of opiates, opioid peptides, and opioid receptors. *Proceedings of the National Academy of Sciences, 90,* 5391-5393.

Bryant, R. (2010). The complexity of complex PTSD. *American Journal of Psychiatry, 167,* 879-881.

Bryant, R., Friedman, M., Spiegel, D., Ursano, R., & Strain, J. (2010). A review of acute stress disorder in DSM-5. *Depression and Anxiety, 28,* 802-817.

Bryant, R. & Harvey, A. (1998). The relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *American Journal of Psychiatry, 155,* 625-629.

Burkett, K., Morris, E., Manning-Courtney, P., Anthony, J., & Shambley-Ebron, D. (2015). African American families on autism diagnosis and treatment: The influence of culture. *Journal of autism and developmental disorders*, *45*(10), 3244-3254.

Cahill, S., Rothbaum, B., Resick, P., & Follette, V. (2009). Cognitive-behavioral therapy for adults. In Foa, E., Keane, T., Friedman, M., & Cohen, J. (Eds). *Effective Treatments of PTSD*, (pp. 139-222). New York, NY: Guilford Press.

California Evidence-Based Clearinghouse (2018, September). *Attachment and biobehavioral (ABC) catch-up.* Retrieved from http://www.cebc4cw.org/program/attachment-and-biobehavioral-catch-up/

Call, C., Walsh, T., & Attia, E. (2013). From DSM-IV to DSM-5: changes to eating disorder diagnoses. *Current Opinions on Psychiatry, 26*(6), 532-536.

Call, N. A., Simmons, C. A., Mevers, J. E. L., & Alvarez, J. P. (2015). Clinical outcomes of behavioral treatments for pica in children with developmental disabilities. *Journal of Autism and Developmental Disorders*, *45*(7), 2105-2114

Campbell, K. & Peebles, R. (2014). Eating disorders in children and adolescents: state of the artreview. *Pediatrics, 134*(3), 582-592.

Carey, G. & Gottesman, I., Twin and family studies of anxiety, phobic, and obsessive compulsive disorders. In D. F. Klein & J. Rabkin (Eds.), *Anxiety: New research and changing concepts* (pp. 117-136). New York: Raven Press.

Casey, E. History of Drug Use and Drug Users in the United States. Retrieved from www.druglibrary.org/schaffer/history/casey1.htm Accessed on November 4, 2017.

Center for Disease Control and Prevention Web-Based Injury Statistics Query and Reporting System. (2013). Leading causes of death reports. Retrieved from

http://www.cdc.gov/injury/wisqars/fatal_injury_reports.htm Accessed on November 4, 2017.

Centers for Disease Control and Prevention. (2018, April). *Tourette syndrome: Risk factors and causes.* Retrieved from https://www.cdc.gov/ncbddd/tourette/riskfactors.html

Centers for Disease Control and Prevention. (2018, May). *Autism: Basics.* Retrieved from https://www.cdc.gov/ncbddd/autism/facts.html

Centers for Disease Control and Prevention. (2018a, September). *Attention-deficit/hyperactivity disorder syndrome: Data & statistics.* Retrieved from https://www.cdc.gov/ncbddd/adhd/data.html

Centers for Disease Control and Prevention. (2018b, September). *Attention-Deficit/Hyperactivity Disorder syndrome: Treatment.* Retrieved from https://www.cdc.gov/ncbddd/adhd/treatment.html

Centers for Disease Control and Prevention. (2018, October). *Tourette syndrome: Data & statistics.* Retrieved from https://www.cdc.gov/ncbddd/tourette/data.html

Centers for Disease Control and Prevention. (2018, November). *Autism: Data & statistics.* Retrieved from https://www.cdc.gov/ncbddd/autism/data.html

Chen, E., Weissman, J., Zeffiro, T., Yiu, A., Eneva, K., Arlt, J., & Swantek, M. (2016). Family based therapy for young adults with anorexia nervosa restores weight. *International Journal of Eating Disorders,* 49(7), 701-707.

Chen, L., Zhang, G., Hu, M., & Liang, X. (2013). Eye movement desensitization and reprocessing versus cognitive-behavioral therapy for adult posttraumatic stress disorder: Systematic review and meta-analysis. *Journal of Nervous & Mental Disease, 203,* 443-451.

Chen, S., Zhou, R., Cui, H., & Chen, X. (2013). Deficits in cue detection underlie event-based prospective memory impairment in major depression: An eye tracking study. *Psychiatry Research, 209*(3), 453-458.

Child Welfare Information Gateway. (2012). *Trauma-Focused Cognitive Behavioral Therapy for Children Affected by Sexual Abuse or Trauma.* Washington, DC: US Department of Health and Human Services, Children's Bureau.

Child Welfare Information Gateway. (2016). *Definitions of child abuse and neglect.* Washington, DC: U.S. Department of Health and Human Services, Children's Bureau.

Child Welfare Information Gateway. (2018). *Child Maltreatment 2016: Summary of key findings.* Washington, DC: U.S. Department of Health and Human Services, Children's Bureau.

Chung, T., Sealy, L., Abraham, M., Ruglovsky, C., Schall, J., & Maisto, S. (2014). Personal network characteristics of youth substance use treatment: Motivation for and perceived difficulty of positive network change. *Substance Abuse, 36*(3), 380-388.

Cisler, J., Adams, T., Brady, R., Bridges, A., Lohr, J., & Olatunji, B. (2011). Unique affective and cognitive processes in contamination apparaisals: Implications for contamination fear. *Journal of Anxiety Disorders, 25,* 28-35.

Coffey, R. M., Mark, T., King, E., Harwood, H., McKusick, D., Genuardi, J., ... & Buck, J. A. (2000). National Estimates of Expenditures for Mental Health and Substance Abuse Treatment, 1997. SAMHSA pub no SMA-00-3499. Rockville, Md. *Center for Substance Abuse Treatment and Center for Mental Health Services, Substance Abuse and Mental Health Services Administration.*

Conley, C. S., Shapiro, J. B., Kirsch, A. C., & Durlak, J. A. (2017). A meta-analysis of indicated

mental health prevention programs for at-risk higher education students.

Constantino, J. N., & Gruber, C. P. (2012). *Social responsiveness scale (SRS)*. Torrance, CA: Western Psychological Services.

Conwell, Y., Duberstein, P. R., & Caine, E. D. (2002). Risk factors for suicide in later life. *Biological psychiatry*, *52*(3), 193-204.

Colvert E, Tick B, McEwen F, et al. Heritability of Autism Spectrum Disorder in a UK Population-Based Twin Sample. *JAMA Psychiatry.* 2015;72(5):415–423.

Cooper, P. J., Whelan, E., Woolgar, M., Morrell, J., & Murray, L. (2004). Association between childhood feeding problems and maternal eating disorder: role of the family environment. *The British Journal of Psychiatry*, *184*(3), 210-215.

Cortina, L. & Kubiak, S. (2006). Gender and posttraumatic stress: Sexual violence as an explanation for women's increased risk. *Journal of Abnormal Psychology, 115,* 753-759.

Craighhead, E., & Dunlop, B. (2014). Combination psychotherapy and antidepressant medication treatment for depression: For whom, when, and how. *Annual Review of Psychology, 65,* 267-300.

Craske, M. & Barlow, D. (2014). Panic disorder and agoraphobia. In D. Barlow (Ed), *Clinical handbook of psychological disorders: A step-by-step treatment manual.* (pp. 1-61). New York, NY: Guilford Press.

Culbert, K., Burt, S., McGue, M., Iacono, W., & Klump, K. (2009). Puberty and the genetic diathesis of disordered eating attitudes and behaviors. *Journal of Abnormal Psychology, 118*(4), 788-796.

Culbert, K., Racine, E., & Klump, K. (2015). Research review: What we have learned about the causes of eating disorders- a synthesis of sociocultural, psychological, and biological research. *Journal of Child Psychology and Psychiatry, 56*, 1141-1164.

Czabała, C., Charzyńska, K., & Mroziak, B. (2011). Psychosocial interventions in workplace mental health promotion: an overview. *Health promotion international*, *26*(suppl_1), i70-i84.

Davey, G.C. (2014). Psychopathology: Research, assessment, and treatment in clinical psychology (2nd ed.). Great Britain. British Psychological Society and Wiley

Diclemente, C., Garay, M., & Gemmell, L. (2008). Motivational enhancement. In HD Kleber & M. Galanter (Eds). *The American Psychiatric Publishing textbook of substance abuse treatment (*4th ed., pp. 361-371). Arlington, VA: American Psychiatric Publishing.

Ding, Y., Naganawa, M., Gallezot, J., Nabulsi, N., Lin, S., Ropchan, J., Weinzimmer, D., . . . Laurelle, M. (2014). Clinical doses of atomozetime significantly occupy both norepinephrine and serotonin transports: Implications on treatment of depression and ADHD. *Neuroimage, 86,* 164-171.

Dopp, A. R., Borduin, C. M., Wagner, D. V., & Sawyer, A. M. (2014). The economic impact of multisystemic therapy through midlife: A cost–benefit analysis with serious juvenile offenders and their siblings. *Journal of consulting and clinical psychology*, *82*(4), 694.

Donoghue, K., Doody, G., Murray, R., Jones, P., Morgan, C., Dazzan, P., … Maccabe, J. (2014). Cannabis use, gender and gage of onset of schizophrenia: Data from the /ESOP study. *Psychiatry Research, 215,* 528-532.

Dougherty, D., Rauch, S., & Jenike, M. (2002). Pharmacological treatments for obsessive compulsive disorder. In P.E. Nathan & J.M. Gordon (Eds.), *A guide to treatments that work* (2nd ed., pp. 387-410). New York: Oxford University Press.

Dudley, M. Z., Salmon, D. A., Halsey, N. A., Orenstein, W. A., Limaye, R. J., O'Leary, S. T., & Omer, S. B. (2018). Do Vaccines Cause Autism?. In *The Clinician's Vaccine Safety Resource Guide* (pp. 197-204). Springer, Cham.

Dunn, W. (2014). Sensory Profile 2 Manual: Strengths Based Approach to Assessment and Planning. *Bloomington, MN: Pearson*.

Edvardsen, J., Torgersen, S., Roysamb, E., Lygren, S., Skre, I., Onstad, S., & Oien, P. (2008). Heritability of bipolar spectrum disorders. Unity or heterogeneity. *Journal of Affective Disorders, 106*(3)*,* 229-240.

Einfeld, S., & Emerson, E. (2008). Intellectual disability. *Rutter's child and adolescent psychiatry*, *5*.

Eldevik, S., Hastings, R. P., Hughes, J. C., Jahr, E., Eikeseth, S., & Cross, S. (2009). Meta-analysis of early intensive behavioral intervention for children with autism. *Journal of Clinical Child & Adolescent Psychology*, *38*(3), 439-450.

El nemer, F. M., Alian, D. M., Eldin, M. S., Khalil, H. E. M. (2014). Prevalence of pica among children attending pediatrics clinic at El-Menoufiya University Hospital. *American Journal of BioScience*, *2*(4), 147-152.

Endrass, T., Kloft, L., Kaufmann, C., & Kaufmann, N. (2011). Approach and avoidance learning in obsessive-compulsive disorder. *Depression & Anxiety, 28,* 166-172.

Fairburn, C., Cooper, Z, & Shafran, R. (2003). Cognitive behavior therapy for eating disorders: A "transdiagnostic" theory. *Behavior Research and Therapy, 41*(5), 509-528.

Fairburn, C., Jones, R., Peveler, R., Hope, R., & O'Connor, M. (1993). Psychotherapy and bulimia nervosa: The longer term effects of interpersonal psychotherapy, behavior therapy, and cognitive behaviour therapy. *Archives of General Psychiatry, 50,* 419-428.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American journal of preventive medicine*, *14*(4), 245-258.

Ferster, C. (1973). A functional analysis of depression. *American Psychologist, 28*(10), 857-870.

Figley, C. (1978). Symptoms of delayed combat stress among a college sample of Vietnam veterans. *Military Medicine, 143,* 107-110.

Filip, M., Frankowska, M., Sadakierska-Chudy, A., Suder, A., Szumiec, L., Mierzejewski, P., …, Cryan, J. (2015). GABAB receptors as a therapeutic strategy in substance use disorders: Focus on positive allosteric modulators. *Neuropharmacology, 88,* 36-47.

Foa, E., Hembree, E., Cahill, S., Rauch, S., Riggs, D., Feeny, N., & Yadin, E. (2005). Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: Outcome at academic and community clinics. *Journal of Consulting and Clinical Psychology, 73,* 953-964.

Foa, E., Liebowitz, M., Kozak, M., Davies, S., Campeas, R., Franklin, M., . . . (2005). Treatment of obsessive compulsive disorder by exposure and ritual prevention, clomipramine, and their combination: A randomized, placebo-controlled trial. *American Journal of Psychiatry, 162,* 151-161.

Foa, E., & McNally, R. (1996). Mechanisms of change in exposure therapy. In R. Rapee (Ed.), *Current controversies in the anxiety disorders* (pp. 329-343). New York, NY: Guilford Press.

Forbes, D., Creamer, M., Bisson, J., Cohen, J., Crow, B., Foa, E., . . . Ursano, R. (2010). A guide to guidelines for the treatment of PTSD and related conditions. *Journal of Trauma and Stress, 23,* 537-552.

Forsman, A. K., Nordmyr, J., & Wahlbeck, K. (2011). Psychosocial interventions for the promotion of mental health and the prevention of depression among older adults. *Health promotion international*, *26*(suppl_1), i85-i107.

Frank, R. G., Conti, R. M., & Goldman, H. H. (2005). Mental health policy and psychotropic drugs. *The Milbank Quarterly*, *83*(2), 271-298.

Fulgoni, V., Keast, D., & Lieberman, H. (2015). Trends in intake and sources of caffeine in diets of the US adults: 2001-2010. *American Journal of Clinical Nutrition, 101,* 1081-1087.

Galanter, M. (2014). Alcoholics Anonymous and twelve-step recovery: A model based on social and cognitive neuroscience. *American Journal of Addictions, 23,* 300-307.

Galen, L. & Rogers, W. (2004). Religiosity, alcohol expectancies, drinking motives and their interaction in the prediction of drinking among college students. *Journal of Studies on Alcohol, 65,* 469-476.

Gallagher, M., Payne, L., White, K., Shear, K., Woods, S., Gorman, J., & Barlow, D. (2013). Mechanisms of change in cognitive behavioral therapy for panic disorder: The unique effects of self-efficacy and anxiety sensitivity. *Behaviour Research and Therapy, 51,* 767-777.

Gallagher, R., Abikoff, H. B., & Spira, E. G. (2014). *Organizational skills training for children with ADHD: An empirically supported treatment*. Guilford Publications.

Garcia-Toro, M., Rubio, J., Gili, M., Roca, M., Jin, C., Liu, S., . . . Blanco, C. (2013). Persistence of chronic major depression: A national prospective study. *Journal of Affective Disorders, 151,* 306-312.

Gardener, H., Spiegelman, D., & Buka, S. L. (2009). Prenatal risk factors for autism: Comprehensive meta-analysis. *British Journal of Psychiatry, 195*(1), 7–14.

Ghaemi, S., Hsu, D., Soldani, F., Goodwin, F. (2003). Antidepressants in bipolar disorder: the case for caution. *Bipolar Disorders, 5,* 421-433.

Ghanizadeh, A., & Haghighat, R. (2012). Nortriptyline for treating enuresis in ADHD—a randomized double-blind controlled clinical trial. *Pediatric Nephrology*, *27*(11), 2091-2097.

Gijsman, H., Geddess, J., Rnedell, J., Nolen, W., Goodwin, G. (2004). Antidepressants for bipolar depression: a systematic review of randomized, controlled trials. *American Journal of Psychiatry, 161,* 1537-1547.

Gilliam, J. C. (2014). GARS-3: Gilliam Autism Rating Scale–Third Edition.

Goldstein, S., & Naglieri, J. A. (2010). *Autism Spectrum Rating Scales: ASRS*. North Tonawanda, NY: Multi-Health Systems.

Goldstein, S., Naglieri, J. A., & DeVries, M. (Eds.). (2011). *Learning and attention disorders in adolescence and adulthood: Assessment and treatment*. John Wiley & Sons.

Gonzalez, H., Tarraf, W., Whitfield, K., & Vega, W. (2010). The epidemiology of major depression and ethnicity in the United States. *Journal of Psychiatric Research, 44,* 1043-1051.

Gorman, J., Kent, J., Sullivan, G., & Coplan, J. (2000). Neuroanatomical hypothesis of panic disorder, revised. *American Journal of Psychiatry, 157,* 493-505.

Gotlib, I. & Hammen, C. (2009). Handbook of Depression, 2nd ed. New York, NY: Guilford Press.

Grant, B., Goldstein, R., Saha, T., Chou, P., Jung, J., Zhang, H., …Hasin, D. (2015). Epidemiology of DSM-5 alcohol use disorder: Results from the national epidemiologic survey on alcohol and related conditions III. *Journal of American Medical Association Psychiatry, 72*(8), 757-766.

Grant, B., Saha, T., Ruan, J., Goldstein, R., Chou, P., …Hasin, D. (2016). Epidemiology of DSM-5 Drug Use Disorder results from the national epidemiologic survey on alcohol and related conditions-III. *JAMA Psychiatry, 73,* 39-47.

Graziottin, A. & Serafini, A. (2009). Depression and the menopause: Why antidepressants are not enough? *Menopause International, 15,* 76-81.

Greenberg, D. & Laporte, D. (1996). Racial differences in body type preferences of men for women. *International Journal of Eating Disorders, 19,* 275-78.

Gruber, S., Sagar, A., Dahlgren, M., Racine, M., & Lukas, S. (2012). Age of onset of marijuana use and executive function. *Psycholoyg of Addictive Behaviors, 26,* 496-506.

Guerdjikova, A., Mori, N., Casuto, L., & McElroy, S. (2017). Binge eating disorder. *Psychiatric Clinics of North America, 40*, 255-266.

Gutman, D., Gorman, J., & Hirsch, J. (2004). Neuroanatomy of panic disorder: Implications of functional imaging in fear conditioning. In J. Gorman (Ed.), *Fear and anxiety: The benefits of translational research*. (pp. 119-134). Washington, DC: American Psychiatric Publishing.

Haile, C. (2012). History use, and basic pharmacology of stimulants. In TR Kosten, TF Newton, R De L Garza, & C Haile (Eds.), *Cocaine and methamphetamine dependence: Advances*

in treatment (pp. 13-84). Arlington, VA: American Psychiatric Publishing.

Hankin, B. (2009). Development of sex differences in depressive and co-occurring anxious symptoms during adolescence: Descriptive trajectories and potential explanations in a multiwave prospective study. *Journal of Clinical Child and Adolescent Psychology, 38,* 460-472.

Hanrahan, F., Field, A., Jones, F., & Davy, G. (2013). A meta-analysis of cognitive therapy for worry in generalized anxiety disorder. *Clinical Psychology Review, 33,* 120-132.

Happe, F. (2011). Why fold Asperger syndrome into autism spectrum disorder in the DSM-5. *Simons Foundation Autism Research Initiative News and Opinion*.

Harris, J. C. (2006). *Intellectual disability: Understanding its development, causes, classification, evaluation, and treatment*. Oxford University Press.

Harrison, A. J., Long, K. A., Tommet, D. C., & Jones, R. N. (2017). Examining the Role of Race, Ethnicity, and Gender on Social and Behavioral Ratings Within the Autism Diagnostic Observation Schedule. *Journal of Autism and Developmental Disorders*, 1-13.

Hart, C. & Ksir, C. (2014). *Drugs, society, and human behavior* (15th ed.). East Windsor, NJ: McGraw-Hill Higher Education.

Hartz, S., Pato, C., Medeiros, H., Cavazos-Rehg, P., Sobell, J., …Pato, M. (2014). Comorbidity of severe psychotic disorders with measures of substance use. *JAMA Psychiatry, 71,* 248-254.

Hartzler, B., Lash, S., & Roll, J. (2012). Contingency management in substance abuse treatment: A structured review of the evidence for its transportability. *Drug and Alcohol Dependence, 122,* 1-10.

Harvey, A & Bryant, R. (1998). The relationship between acute stress disorder and posttraumatic stress disorder: A prospective evaluation of motor vehicle accident survivors. *Journal of Consulting and Clinical Psychology, 66,* 507-512.

Heimberg, R., & Becker, R. (2002). *Cognitive-behavioral group therapy for social phobia: Basic mechanisms and clinical strategies.* New York, NY: Guilford Press.

Hewitt, P., Flett, G., & Ediger, E. (1995). Perfectionism traits and perfectionistic self-presentation in eating disorder attitudes, characteristics, and symptoms. *International Journal of Eating Disorders, 18,* 317-326.

Hinton, D. & Lewis-Fernandez, R. (2011). The cross-cultural validity of posttraumatic stress disorder: Implications for DSM-5. *Depression and Anxiety, 28,* 783-801.

Hirai AH, Kogan MD, Kandasamy V, Reuland C, Bethell C. Prevalence and Variation of Developmental Screening and Surveillance in Early Childhood. *JAMA Pediatr.* 2018;172(9):857–866

Hlastala, S. A., Kotler, J. S., McClellan, J. M., & McCauley, E. A. (2010). Interpersonal and social rhythm therapy for adolescents with bipolar disorder: treatment development and results from an open trial. *Depression and Anxiety*, *27*(5), 457-64.

Hofer, H., Frigerio, S., Frischknecht, E., Gassmann, D., Gutbrod, K., & Muri, R. (2013). Diagnosis and treatment of an obsessive-compulsive disorder following traumatic brain injury: A single case and review of the literature. *Neruocase, 19,* 390-400.

Horowitz, S. (2008). Shedding light on seasonal affective disorder. *Alternative and Complementary Therapies*, *14*(6), 282-287.

Hurd, N. M., Varner, F. A., Caldwell, C. H., & Zimmerman, M. A. (2014). Does perceived racial discrimination predict changes in psychological distress and substance use over time? An

examination among Black emerging adults. *Developmental Psychology*, *50*(7), 1910-1918.

Individuals with Disabilities Education Act (n.d.) *About IDEA.* Retrieved from https://sites.ed.gov/idea/about-idea/

Jacob, M., Larson, M., & Storch, E. (2014). Insight in adults with obsessive-compulsive disorder. *Comprehensive Psychiatry, 55,* 896-903.

Jacobson, N., Martell, C., & Dimidjian, S. (2001). Behavioral activation treatment for depression: Returning to contextual roots. *Clinical Psychology Science and Practice, 8*(3), 255-270.

Jang, K., Livesley, W., & Vernon, P. (1995). Alcohol and drug problems: a multivariate behavioral genetic analysis of co-morbidity. *Addiction, 90,* 1213-1221.

Jani, S., Johnson, R. S., Banu, S., & Shah, A. (2016). Cross-cultural bias in the diagnosis of borderline personality disorder. *Bulletin of the Menninger Clinic*, *80*(2), 146-165.

Jansen, R., Penninx, B., Madar, V., Xia, K., Milaneschi, Y., Hottenga, J., . . . Sullivan, P. (2016). Gene expression in major depressive disorder. *Molecular Psychiatry, 21,* 339-347.

Jayawant, S. & Balkrishnana, R. (2005). The controversy surrounding OxyContin abuse: Issues and solutions. *Therapeutics and Clinical Risk Management, 1,* 77-82.

Jhanjee, S. (2014). Evidence based psychosocial interventions in substance use. *Indian Journal of Psychological Medicine, 36,* 112-118.

Jobes, D. A., Rudd, M. D., Overholser, J. C., & Joiner Jr, T. E. (2008). Ethical and competent care of suicidal patients: Contemporary challenges, new developments, and considerations for clinical practice. *Professional Psychology: Research and Practice*, *39*(4), 405.

Johnston, L., O'Malley, P., Miech, R., Bachman, J., & Schulenberg, J. (2014). *Monitoring the future national results on drug use, 1975-2013: Overview, key findings on adolescent drug use.* Ann Arbor, MI: Institute for Social Research, University of Michigan.

Joiner Jr, T. E., Van Orden, K. A., Witte, T. K., Selby, E. A., Ribeiro, J. D., Lewis, R., & Rudd, M. D. (2009). Main predictions of the interpersonal–psychological theory of suicidal behavior: Empirical tests in two samples of young adults. *Journal of Abnormal Psychology*, *118*(3), 634.

Jones, B., Corbin, W., & Fromme, K. (2001). A review of expectancy theory and alcohol consumption. *Addiction, 96,* 57-72.

Jones, H. L., Cross Jr, W. E., & DeFour, D. C. (2007). Race-related stress, racial identity attitudes, and mental health among Black women. *Journal of Black Psychology*, *33*(2), 208-231.

Jones, L. M., Cross, T. P., Walsh, W. A., & Simone, M. (2007). Do Children's Advocacy Centers improve families' experiences of child sexual abuse investigations?. *Child Abuse & Neglect*, *31*(10), 1069-1085.

Kaczkurkin, A., Asnaai, A., Hall-Clark, B., Peterson, A., Yarvis, J., Foa, E., & the STRONG STAR Consortium. (2016). Ethnic and racial differences in clinically relevant symptoms in active duty military personnel with posttraumatic stress disorder. *Journal of Anxiety Disorders, 43,* 90-98.

Kalibatseva, Z., & Leong, F. T. (2014). A critical review of culturally sensitive treatments for depression: recommendations for intervention and research. *Psychological Services*, *11*(4), 433-450.

Kampman, C., Keijers, G., Hoogduin, C., & Hendriks, G. (2002). A randomized, double-blind, placebo-controlled study of the effects of adjunctive paroxetine in panic disorder patients uncessfully treated with cognitive-behavioral therapy alone. *Journal of Clinical Psychiatry, 63*(9), 772-777.

Kaufmann, W. E., Capone, G. T., Carter, J. C., & Lieberman, D. N. (2008). Genetic intellectual disability: neurobiological and clinical aspects. *Capute & Accardo's neurodevelopmental disabilities in infancy and childhood*, *1*, 155-73.

Kendler, K. S., & Karkowski-Shuman, L. (1997). Stressful life events and genetic liability to major depression: genetic control of exposure to the environment?. *Psychological medicine*, *27*(3), 539-547.

Kessler, R., Berguland, P., Demler, O., Jin, R., Koretz, D., Merikangas, K., . . . Wang, P. (2003). Results from the national comorbidity survey replication (NCS-R). *Journal of American Medical Association, 289*(23), 3095-3105.

Kinchin, D. (2007). *A guide to psychological debriefing: Managing emotional decompression and post-traumatic stress disorder.* London, England: Jessica Kingsley Publishers.

Klerman, G., Weissman, M., Rounsaville, B., & Chevron, E. (1984). *Interpersonal psychotherapy of depression.* London: Jason Aronson Inc. Parkinson's disease in the United States. *Movement Disorders, 28,* 311-318.

Krapp, K. M. (Ed.). (2005). *Psychologists and their theories for students* (Vol. 2). Farmington Hills, MI Thomson Gale.

Krishan, K. (2007). Revisiting monamine oxidase inibitors. *Journal of Clinical Psychiatry, 68*, 35-41.

Kugelmass, H. (2016). "Sorry, I'm Not Accepting New Patients" An Audit Study of Access to

Mental Health Care. *Journal of health and social behavior*, *57*(2), 168-183.

Kuhn, R. (1958). The treatment of depressive states with G-22355 (imipramine hydrochloride). *American Journal of Psychiatry, 115,* 459-464.

Kuttler, S., Myles, B. S., & Carlson, J. K. (1998). The use of social stories to reduce precursors to tantrum behavior in a student with autism. *Focus on Autism and Other Developmental Disabilities*, *13*(3), 176-182.

Lai, C., Zauszniewski, J., Tang, T., Hou, S., Su, S., & Lai, P. (2014). Personal beliefs, learned resourcefulness, and adaptive functioning in depressed adults. *Journal of Psychiatric and Mental Health Nursing, 21*(3), 280-287.

Lask, B., & Bryant-Waugh, Rachel. (2013). Eating disorders in childhood and adolescence (4th ed.). Hove, East Sussex ; New York, NY: Routledge/Taylor & Francis Group.

Le Grange, D., Lock, J., Accurso, E., Agras, W., Darcy, A., Forsberg, S., & Bryson, S. (2014). Relapse from remission at two- to four- year follow-up in two treatments for adolescent anorexia nervosa. *Journal of the American Academy of Child & Adolescent Psychiatry, 53*(11), 1162-1167.

Lervolino, A., Perroud, N., Fullana, M., Guipponi, M., Cherkas, L., Collier, D., & Mataix-Cols, D. (2009). Prevalence and heritability of compulsive hoarding: A twin study. *American Journal of Psychiatry, 166,* 1156-1161.

Leventhal, A. & Schmitz, J. (2006). The role of drug use outcome expectancies in substance abuse risk: An interactional-transformational model. *Addictive Behaviors, 31,* 2038-2062.

Levinson, D. & Nichols,W. (2014). *Major Depression and genetics.* Stanford, CA: Stanford, School of Medicine.

Lewinsohn, P., Antonuccio, D., Steinmetz, J. & Teri, L. (1984). *The coping with depression*

course. Eugene, OR: Castalia.

Lewinsohn, P., Clarke, G., Hops, H., & Andrews, J. (1990). Cognitive-behavioral treatment for depressed adolescents. *Behavior Therapist, 21,* 385-401.

Lewinsohn, P. (1974). A behavioral approach to depression. In R. M. Freidman & M. M. Katz (Eds.), *The psychology of depression: Contemporary theory and research* (pp. 157-185). New York: Wiley.

Lewinsohn, P., Biglan, A., & Zeiss, A. (1976). Behavioral treatment of depression. In P. O. Davidson (Ed), *The behavioral management of anxiety, depression and pain* (pp. 91-146). New York: Brunner/Mazel.

Lewis, M. & Petry, N. (2005). Contingency management treatments that reinforce completion of goal-related activities: Participation in family activities and its association with outcomes. *Drug and Alcohol Dependence, 79,* 267-271.

Lo, C. C., & Cheng, T. C. (2014). Race, unemployment rate, and chronic mental illness: a 15-year trend analysis. *Social psychiatry and psychiatric epidemiology*, *49*(7), 1119-1128.

Lochner, C., Roos, A., & Stein, D. J. (2017). Excoriation (skin-picking) disorder: a systematic review of treatment options. *Neuropsychiatric Disease and Treatment*, *13*, 1867-1872. doi:10.2147/NDT.S121138

Lord, C., Rutter, M., DiLavore, P., Risi, S., Gotham, K., & Bishop, S. (2012). Autism Diagnostic Observation Schedule Second Edition (ADOS-2) Manual (Part 1): Modules 1–4. *Torrance, CA: Western Psychological Services*.

Luiselli, J. K. (2015). Behavioral treatment of rumination: Research and clinical applications. *Journal of Applied Behavior Analysis*, *48*(3), 707-711.

Magugen, S., Luxton,D., Skopp, N., & Madden. E. (2012). Gender differences in traumatic experiences and mental health in active duty soldiers redeployed from Iraq and Afghanistan. *Journal of Psychiatric Research, 46,* 311-316.

Makrygianni, M. K., & Reed, P. (2010). A meta-analytic review of the effectiveness of behavioural early intervention programs for children with autistic spectrum disorders. *Research in Autism Spectrum Disorders*, *4*(4), 577-593.

March, J., Frances, A., Kahn, D., & Carpenter, D. (1997). The Expert Consensus Guideline series: Treatment of obsessive-compulsive disorder. *Journal of Clinical Psychiatry, 58,* 1-72.

Marinova, Z., Chuang, D., & Fineberg, N. (2017). Glutamate-modulating drugs as apotential therapeutic strategy in obsessive-compulsive disorder. *Current Neuropharmacology, 15,* 977-995.

Mark, T. L., Coffey, R. M., Vandivort-Warren, R., & Harwood, H. J. (2005). US spending for mental health and substance abuse treatment, 1991-2001. *Health Affairs*, *24*, W5.

Marlatt, G. & Donovan, D. (2005). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors.* New York, NY: Guilford.

Marsh, R., Horga, G., Parashar, N., Wang, Z., Peterson, B., & Simpson, H. (2014). Altered activation in fronto-striatal circuits during sequential processing of conflict in unmedicated adults with obsessive-compulsive disorder. *Biological Psychiatry, 75,* 615-622.

McCabe, R., Wilsnack, S., West, B., & Boyd, C. (2010). Victimization and substance use disorders in a national sample of heterosexual and sexual minority women and men. *Addiction, 105,* 2130-2140.

McCallie, M., Blum, C., & Hood, C. (2006). Progressive muscle relaxation. *Journal of Human Behavior in the Social Environment, 3,* 51-66.

McGillivray, J. A., & Evert, H. T. (2014). Group Cognitive Behavioural Therapy Program Shows Potential in Reducing Symptoms of Depression and Stress among Young People with ASD. Journal of Autism and Developmental Disorders, 44(8), 2041-2051.

McGorry, P., Nelson, B., Goldstone, S., & Yung, A. (2010). Clinical staging: a heuristic and practical strategy for new research and better health and social outcomes for psychotic and related mood disorders. *Canadian Journal of Psychiatry, 55,* 486-496.

McGuffin, P., Katz, R., Watkins, S., & Rutherford, J. (1996). A hospital-based twin register of the heritability of DSM-IV unipolar depression. *Archives of General Psychiatry, 53,* 129-136.

McLean, C. & Anderson, E. (2009). Brave men and timid women? A review of the gender differences in fear and anxiety. *Clinical Psychology Review, 29,* 496-505.

McNally, R. (2004). Psychological debriefing does not prevent posttraumatic stress disorder. *Psychiatric Times,* p.71.

Melrose, S. (2015). Seasonal affective disorder: an overview of assessment and treatment approaches. *Depression Research and Treatment*, *2015*.

Merikangas, K., Stolar, M., Stevens, D., Goulet, J., Preisig, M., Fenton, B., … Rounsaville, B. (1998). Familial Transmission of Substance Use Disorders. *Archives of General Psychiatry, 55*, 973-979.

Meyers, R. & Squires, D. (2001). *The community reinforcement approach: A guideline developed for the Behavioral Health Recovery Management project.* Albuquerque, NM: University of New Mexico Center on Alcoholism, Substance Abuse, and Addiction.

Michelson, D. J., Shevell, M. I., Sherr, E. H., Moeschler, J. B., Gropman, A. L., & Ashwal, S. (2011). Evidence Report: Genetic and metabolic testing on children with global developmental delay Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology*, *77*(17), 1629-1635.

Mignon, S. (2014). *Substance abuse treatment: Options, challenges, and effectiveness.* New York, NY: Springer Publishing.

Minsky, S., Vega, W., Miskimen, T., Gara, M., & Escobar, J. (2003). Diagnostic patterns in latino, african american, and european american psychiatric patients. *Archives of General Psychiatry*, *60*(6), 637-644.

Mississippi State University (n.d.). *ACCESS.* Retrieved from https://access.msstate.edu

Miklowitz, D. J., Simoneau, T. L., George, E. L., Richards, J. A., Kalbag, A., Sachs-Ericsson, N., & Suddath, R. (2000). Family-focused treatment of bipolar disorder: 1-year effects of a psychoeducational program in conjunction with pharmacotherapy. *Biological psychiatry*, *48*(6), 582-592.

Miklowitz, D. J. (2010). *Bipolar disorder: A family-focused treatment approach*. Guilford Press.

Moscovitch, D., Rowa, K., Paulitzki, J., Ierullo, M., Chiang, B., Antony, M., & McCabe, R. (2013). Self-portrayal concerns and their relation to safety behaviors and negative affect in social anxiety disorder. *Behavior Research and Therapy, 51,* 476-486.

Mossakowski, K. N. (2003). Coping with perceived discrimination: Does ethnic identity protect mental health?. *Journal of Health and Social Behavior*, 318-331.

Munafo, M., Brown, S, & Hariri, A. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry, 63*(9), 852-857.

Murphy, R., Straebler, S., Basden, S., Cooper, Z., & Fairbur, C. (2012). Interpersonal psychotherapy for eating disorders. *Clinical Psychology and Psychotherapy, 19,* 150-158.

Murray, H. B., Thomas, J. J., Hinz, A., Munsch, S., & Hilbert, A. (2018). Prevalence in primary school youth of pica and rumination behavior: The understudied feeding disorders. *International Journal of Eating Disorders*.

Naeem, F., Saeed, S., Irfan, M., Kiran, T., Mehmood, N., Gul, M., ... & Farooq, S. (2015). Brief culturally adapted CBT for psychosis (CaCBTp): a randomized controlled trial from a low income country. *Schizophrenia Research*, *164*(1), 143-148.

Najdowski, A. C., Wallace, M. D., Reagon, K., Penrod, B., Higbee, T. S., & Tarbox, J. (2010). Utilizing a home-based parent training approach in the treatment of food selectivity. *Behavioral Interventions: Theory & Practice in Residential & Community-Based Clinical Programs*, *25*(2), 89-107.

National Children's Advocacy Center (n.d.). *Forensic Interview Services*. Retrieved from https://www.nationalcac.org/forensic-interview-services

National Child Traumatic Stress Network (n.d.a). About Child Trauma. Retrieved from https://www.nctsn.org/what-is-child-trauma/about-child-trauma

National Child Traumatic Stress Network (n.d.b). Populations at Risk. Retrieved from https://www.nctsn.org/what-is-child-trauma/populations-at-risk

National Child Traumatic Stress Network (n.d.c). Trauma Types. Retrieved from https://www.nctsn.org/what-is-child-trauma/trauma-types

National Child Traumatic Stress Network (n.d.d). Treatments and Practices. Retrieved from https://www.nctsn.org/treatments-and-practices

National Institute of Health (NIH). (n.d.) *Suicide.* Retrieved from https://www.nimh.nih.gov/health/statistics/suicide.shtml#part_154969

National Institute of Health (NIH). (2017, November). *Post-traumatic Stress Disorder..* Retrieved from https://www.nimh.nih.gov/health/statistics/post-traumatic-stress-disorder-ptsd.shtml

National Institute of Child Health and Human Development (NICHD). (2017, January). *Medication Treatment for Autism.* Retrieved from https://www.nichd.nih.gov/health/topics/autism/conditioninfo/treatments/medication-treatment

Neighbors, H. W., Trierweiler, S. J., Ford, B. C., & Muroff, J. R. (2003). Racial differences in DSM diagnosis using a semi-structured instrument: The importance of clinical judgment in the diagnosis of African Americans. *Journal of Health and Social Behavior*, 237-256.

Nestadt, G., Samuels, J., Riddle, M., Bienvenu, J., Liang, K., LaBuda, M., . . . Hoehn-Saric, R. (2000). A family study of obsessive-compulsive disorder, *Archives of General Psychiatry, 57,* 358-363.

Newman, B. S., Dannenfelser, P. L., & Pendleton, D. (2005). Child abuse investigations: Reasons for using child advocacy centers and suggestions for improvement. *Child and Adolescent Social Work Journal*, *22*(2), 165-181.

Nezlek, J., Hampton, C. & Shean, G. (2000). Clinical depression and day-to-day social interaction in a community sample. *Journal of Abnormal Psychology, 109,* 11-19.

NIAAA (National Institute on Alcohol Abuse and Alcoholism). (1997). *Alcohol Alert: Alcohol Metabolism.* No. 35, PH 371. Bethesda, MD: The Institute.

Nillni, Y., Rohan, K., & Zvolensky, M. (2012). The role of menstrual cycle phase and anxiety sensitivity in catastrophic misinterpretation of physical symptoms during a CO2 challenge. *Archives of Women's Mental Health,* 1-10.

NIMH (National Institute of Mental Health; 2013). *Generalized anxiety disorder.* NIH Publications No. TR 10-4677. U.S. Department of Health and Human Services, National Institutes of Health.

Nivoli, A., Colom, F., Murru, A., Pacchiarotti, I., Castro-Loli, P., Gonzalez-Pinto, A., …Vieta, E. (2011). New treatment guidelines for acute bipolar depression: A systematic review. *Journal of Affective Disorders, 129*(1-3), 14-26.

Ng, E., Muntaner, C., Chung, H., & Eaton, W. W. (2014). Socioeconomic status and mental illness. *The Wiley Blackwell Encyclopedia of Health, Illness, Behavior, and Society*.

Nolen-Hoeksema, S. (2001). Gender differences in depression. *Current Directions in Psychological Research, 10,* 173-176.

Nolen-Hoeskema, S. (2012). Emotion regulation and psychopathology: The role of gender. *Annual Review of Clinical Psychology, 8,* 161-187.

Nolen-Hoeksema, S., Girgus, J., & Seligman, M.(1992). Predictors and consequences of childhood depressive symptoms: A 5-year longitudinal study. *Journal of Abnormal Psychology, 101*(3), 405-422.

Norris, M. L., & Katzman, D. K. (2015). Change is never easy, but It is possible: reflections on avoidant/restrictive food intake disorder Two years after its introduction in the DSM-5. *Journal of Adolescent Health*, *57*(1), 8-9.

NSDUH (National Survey on Drug Use and Health). (2013). *Results from the 2012 National Survey on Drug Use and Health: Mental health findings,* NSDUH Series H-47, HHS

Publication NO. (SMA) 13-4805. Rockville, MD: Substance Abuse and Mental Health Services Administration.

O'connor, S. S., Coggins, R., Gagnon, L., Rosenfeld, R. M., Shin, J. J., & Walsh, S. A. (2016). Plain language summary: otitis media with effusion. *Otolaryngology–Head and Neck Surgery*, *154*(2), 215-225.

Oei, T. & Morawska, A. (2004). A cognitive model of binge drinking: The influence of alcohol expectancies and drinking refusal self-efficacy. *Addictive Behaviors, 29,* 159-179.

Olden, K. W. (2001). Rumination. *Current Treatment Options in Gastroenterology*, *4*(4), 351-358.

Oliver, C., Petty, J., Ruddick, L., & Bacarese-Hamilton, M. (2012). The association between repetitive, self-injurious and aggressive behavior in children with severe intellectual disability. *Journal of autism and developmental disorders*, *42*(6), 910-919.

Owens, M., Herbert, J., Jones, P., Shakian, A., Wilkinson, P., Dunn, V., . . . Stuart, G. (2014). Elevated morning cortisol is a stratified population-level biomarker for major depression in boys only with high depressive symptoms. *Proceedings of the National Academy of Sciences of the United States of America, 111*(9), 3638-3643.

Patterson, G. R. (1982). *Coercive family process* (Vol. 3). Castalia Publishing Company.

Perilla, J. L., Norris, F. H., & Lavizzo, E. A. (2002). Ethnicity, culture, and disaster response: Identifying and explaining ethnic differences in PTSD six months after Hurricane Andrew. *Journal of social and clinical psychology*, *21*(1), 20-45.

Phillips, K., Pagano, M., Menard, W., & Stout, R. (2006). A 12-month follow-up study of thecourse of body dysmorphic disorder. *American Journal of Psychiatry, 163,* 907-912.

Piazza-Gardner, A. & Barry, G. (2013). The impact of alcohol on Alzheimer's disease: a systematic review. *Journal of Aging and Mental Health, 17,* 133-146.

Piper, B. J., Gray, H. M., & Birkett, M. A. (2012). Maternal smoking cessation and reduced academic and behavioral problems in offspring. *Drug and Alcohol Dependence*, *121*(1-2), 62-67.

Polivy, J. & Herman, C. (2002). Causes of eating disorders. *Annual Review of Psychology, 53,* 187-213.

Polivy, J. & Herman, C. (2004). Sociocultural idealization of thin female body shapes: anintroduction to the special issue on body image and eating disorders. *Journal of Social & Clinical Psychology, 23,* 1-6.

Pope, C., Pope, H., Menard, W., Fay, C., Olivardia, R., & Philips, K. (2005). Clinical features of muscle dysmorphia among males with body dysmorphic disorder. *Body Image, 2,* 395-400.

Possel, P. & Black, S. (2014). Testing three different sequential mediational interpretations of Beck's cognitive model of the development of depression. *Journal of Clinical Psychology, 70*(1), 72-94.

Prochaska, J., & Norcross, J. (2013). *Systems of psychotherapy: A transtheoretical analysis*. Nelson Education.

Raad, R., & Makari, G. (2010). Samuel Tuke's description of the retreat. *The American Journal of Psychiatry, 167(8)*, 898.

Rao, T. S., & Andrade, C. (2011). The MMR vaccine and autism: Sensation, refutation, retraction, and fraud. *Indian journal of psychiatry*, *53*(2), 95.

Rao U. (2014). DSM-5: disruptive mood dysregulation disorder. *Asian journal of psychiatry*, *11*, 119-23.

Rasmussen, S. & Eisen, J. (1990). Epidemiology of obsessive compulsive disorder. *The Journal of Clinical Psychiatry, 51,* 10-13.

Reichow, B. (2012). Overview of meta-analyses on early intensive behavioral intervention for young children with autism spectrum disorders. *Journal of autism and developmental disorders*, *42*(4), 512-520.

Reiner, W. G., & Kratochvil, C. J. (2008). Pharmacotherapy in the management of voiding and storage disorders, including enuresis and encopresis. *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(5), 491-498.

Reinhold, J., & Rickels, K. (2015). Pharmacological treatment for generalized anxiety disorder in adults: an update. *Expert Opinion in Pharmacotherapy, 16,* 1669-1681.

Resick, P., Monson, C., & Rizvi, S. (2008). Posttraumatic stress disorder. In. Barlow, D. (Ed), *Clinical Handbook of Psychological Disorders* (pp. 65-122). New York, NY: Guilford Press.

Ricketts, E. J., Bauer, C. C., Van der Fluit, F., Capriotti, M. R., Espil, F. M., Snorrason, I., ... & Woods, D. W. (2013). Behavior therapy for stereotypic movement disorder in typically developing children: a clinical case series. *Cognitive and Behavioral Practice*, *20*(4), 544-555.

Robins, D. L., Fein, D., & Barton, M. (2009). Modified checklist for autism in toddlers, revised, with follow-up (M-CHAT-R/F) TM. *Google Scholar*.

Roberts, J., Hunter, L., Gravel, J., Rosenfeld, R., Berman, S., Haggard, M., ... & Wallace, I. (2004). Otitis media, hearing loss, and language learning: controversies and current research. *Journal of Developmental & Behavioral Pediatrics*, *25*(2), 110-122.

Rodebaugh, T., Holaway, R., & Heimberg, R. (2004). The treatment of social anxiety disorder. *Clinical Psychology Review, 24,* 883-908.

Roy-Byrne, P., Arguelles, L., Vitek, M., Goldberg, J., Keane, T., Ture, W., & Pitman, R. (2004). Persistence and change of PTSD symptomatology: A longitudinal co-twin control analysis of the Vietnam Era Twin Registry, *Social Psychiatry and Psychiatric Epidemiology, 39,* 681-685.

Rudd, M. D., Berman, A. L., Joiner Jr, T. E., Nock, M. K., Silverman, M. M., Mandrusiak, M., ... & Witte, T. (2006). Warning signs for suicide: Theory, research, and clinical applications. *Suicide and Life-Threatening Behavior*, *36*(3), 255-262.

Rüsch, N., Zlati, A., Black, G., & Thornicroft, G. (2014). Does the stigma of mental illness contribute to suicidality?.

Rusyniak, D. (2011). Neurological manifestations of chronic methamphetamine abuse. *Neurologic Clinics, 29,* 641-655.

Rutter, M., Le Couteur, A., & Lord, C. (2003). Autism diagnostic interview-revised. *Los Angeles, CA: Western Psychological Services*, *29*, 30.

Salkovskis, P., Thorpe, S., Wahl, K., Wroe, A., & Forrester, E. (2003). Neutralizing increases discomfort associated with obsessional thoughts: An experimental study with obsessional patients. *Journal of Abnormal Psychology, 112,* 709-715.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2013). *National survey on drug use and health, 2011 and 2012.* Washington, DC: Department of Health and Human Services.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2014). *Results from the 2013 National Survey on Drug Use and Health: Summary of national findings*, NSDUH Series H-48, HSS Publication No. (SMA) 14-4863. Rockville, MD: SAMHSA.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2015). *National Survey on Drug Use and Health (NSDUH)—Alcohol Use in Lifetime, Past Year, and Past Month among Persons Aged 12 or Older, by Demographic Characteristics: Percentages, 2014 and 2015*. Washington, DC: Department of Health and Human Services.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2015, September). *Alcohol, tobacco, and prescription drug use by teens declines; Level of youth with major depressive episodes remains high.* Retrieved from https://www.samhsa.gov/newsroom/press-announcements/201509100930-0

SAMHSA (Substance Abuse and Mental Health Services Administration). (2016). *Prescription Drug Use and Misuse in the United States: Results from the 2015 National Survey on Drug Use and Health*. Washington, DC: Department of Health and Human Services.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2017, December). Understanding Child Trauma. Retrieved from https://www.samhsa.gov/child-trauma/understanding-child-trauma

SAMHSA (Substance Abuse and Mental Health Services Administration). (2018). Interpersonal and Social Rhythm Therapy. Washington, DC: Department of Health and Human Services.

Satcher, D. (2001). *Mental health: Culture, race, and ethnicity—A supplement to mental health: A report of the surgeon general*. US Department of Health and Human Services.

Schmidt, U., Kalgwasser, S., & Wotjak, C. (2013). Biomarkers in posttraumatic stress disorder: Overview and implications for future research. *Disease Markers, 35,* 43-54.

Schwartz, R., Brooner, R., Montoya, I., Currens, M., & Hayes, M. (2010). A 12-year follow-up of a methadone medical maintenance program. *The American Journal on Addictions, 8,* 293-299.

Schwartz, R. C., & Blankenship, D. M. (2014). Racial disparities in psychotic disorder diagnosis: A review of empirical literature. *World Journal of Psychiatry*, *4*(4), 133.

Seligman, M. (1975). *Helplessness*. San Francisco: Freeman.

Shalev, R. S., Manor, O., Kerem, B., Ayali, M., Badichi, N., Friedlander, Y., & Gross-Tsur, V. (2001). Developmental dyscalculia is a familial learning disability. *Journal of Learning Disabilities*, *34*(1), 59-65.

Sharp, W. G., Burrell, T. L., & Jaquess, D. L. (2014). The Autism MEAL Plan: A parent-training curriculum to manage eating aversions and low intake among children with autism. *Autism*, *18*(6), 712-722.

Sheikh, S., Swetlik, C., & Wilson, R. (2018). 'Just Google It'–The Effect of Media Events on Patterns of Public Interest in the 'Vaccines Cause Autism' Fallacy from 2004–2017 (P2. 182).

Shepard, J. A., Poler Jr, J. E., & Grabman, J. H. (2017). Evidence-based psychosocial treatments for pediatric elimination disorders. *Journal of Clinical Child & Adolescent Psychology*, *46*(6), 767-797.

Sherin, J. & Nemeroff, C. (2011). Post-traumatic stress disorder: The neurobiological impact of psychological trauma. *Dialogues in Clinical Neuroscience, 13,* 263-278.

Shulman, K., Herrmann, N., & Walker, S. (2013). Current place of monamine oxidase inhibitors in the treatment of depression. *CNS Drugs, 27*, 789-797.

Shoshani, A., & Steinmetz, S. (2014). Positive psychology at school: A school-based intervention to promote adolescents' mental health and well-being. *Journal of Happiness Studies*, *15*(6), 1289-1311.

Sieswerda, S., & Arntz, A. (2007). Successful psychotherapy reduces hypervigilance in borderline personality disorder. *Behavioral and Cognitive Psychotherapy, 35,* 387-402.

Sizoo, & Kuiper. (2017). Cognitive behavioural therapy and mindfulness-based stress reduction may be equally effective in reducing anxiety and depression in adults with autism spectrum disorders. *Research in Developmental Disabilities, 64,* 47-55.

Slocum, S. K., Mehrkam, L. R., Peters, K. P., & Vollmer, T. R. (2017). Using differential reinforcement of a discard response to treat pica. *Behavioral Interventions*, *32*(3), 234-241.

Smith, B.L. (2012). Inappropriate prescribing. *Monitor on Psychology, 43(6),* 36.

Smith, T., & Iadarola, S. (2015). Evidence base update for autism spectrum disorder. *Journal of Clinical Child & Adolescent Psychology*, *44*(6), 897-922.

Society of Clinical Psychology. (2016) *Family-focused therapy (FFT) for bipolar disorder.* Retrieved from https://www.div12.org/treatment/family-focused-therapy-fft-for-bipolar-disorder/

Soreff, S., & McInnes, L., (2014). Bipolar affective disorder. Retrieved from http://emedicine.medscape.com/article/286342-overview.

Stahl, S. & Wise, D. (2008). The potential role of a corticotrophin-releasing factor receptor-1 antagonist in psychiatric disorders. *CNS Spectrums, 13,* 467-478.

Stice, E., Marti, C., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-Year prospective community study of young women. *Journal of Abnormal Psychology, 122,* 445-457.

Street, A., Bell, M. & Ready, C. (2011). Sexual assault. In D.M. Benedek, & G.H. Wynn (Eds.) *Clinical manual for management of PTSD* (pp. 325-348) Arlington, VA: American Psychiatric Publishing.

Strother, E., Lemberg, R., Stanford, S., & Turberville, D. (2012). Eating disorders in men: underdiagnosed, undertreated, and misunderstood. *Eating Disorders, 20*(5), 346-355.

Sturmey, P., & Williams, D. E. (2016). Behavioral Treatment: Treatment Programs and Outcome Data. In *Pica in Individuals with Developmental Disabilities* (pp. 103-113). Springer, Cham.

Sue, D., Sue, D., Sue, D., & Sue, S. (2017). *Essentials of understanding abnormal behavior*, 3rd Ed. Boston: MA, Cengage Learning.

Tan, J. X., & Fajardo, M. L. R. (2017). Efficacy of multisystemic therapy in youths aged 10–17 with severe antisocial behaviour and emotional disorders: systematic review. *London journal of primary care*, *9*(6), 95-103.

Taylor, L. E., Swerdfeger, A. L., & Eslick, G. D. (2014). Vaccines are not associated with autism: an evidence-based meta-analysis of case-control and cohort studies. *Vaccine*, *32*(29), 3623-3629.

Terry-McElrath, Y., O'Malley, P., & Johnston, L. (2014). Energy drinks, soft drinks, and substance use among United States secondary school students. *Journal of Addiction Medicine, 8,* 6-13.

Tomko, R. L., Trull, T. J., Wood, P. K., & Sher, K. J. (2014). Characteristics of borderline personality disorder in a community sample: comorbidity, treatment utilization, and general functioning. *Journal of Personality Disorders*, *28*(5), 734-750.

Tsuang, M., Lyons, M., Eisen, S., Goldberg, J., True, W., Nang, L., … Eaves, L. (1996). Genetic influences on abuse of illicit drugs. *American Journal of Medical Genetics, 5,* 473-477.

The Arc. (2018) *What we do.* Retrieved from https://www.thearc.org/what-we-do

The National Child Traumatic Stress Network. (2008, March). *Child welfare trauma training toolkit: Supplemental handouts.* http://www.trauma-informed-california.org/wp-content/uploads/2012/02/child_welfare_trauma_training_toolkit_supplements.pdf

Tuckey, M. & Scott, J. (2014). Group critical incident stress debriefing with emergency services personnel: A randomized controlled trial. *Anxiety, Stress, and Coping, 27,* 38-54.

Turner, J. C. (1987). *Rediscovering the social group: A self-categorization theory*. Oxford, England: Basil Blackwell.

Unger, J., Schwartz, S., Huh, J., Soto, D., & Baezconde-Garbanati, L. (2014). Acculturation and perceived discrimination: Predictors of substance use trajectories from adolescence to emerging adulthood among Hispanics. *Addictive Behaviors, 39*(9), 1293-1296.

Uno, Y., Uchiyama, T., Kurosawa, M., Aleksic, B., & Ozaki, N. (2015). Early exposure to the combined measles–mumps–rubella vaccine and thimerosal-containing vaccines and risk of autism spectrum disorder. *Vaccine*, *33*(21), 2511-2516.

van Grootheest, D., Cath, D., Beekman, A., & Boomsma, D. (2005). Twin studies on obsessive-compulsive disorder: A review. *Twin Research and Human Genetics, 8,* 450-458.

Van Orden, K. A., Witte, T. K., Gordon, K. H., Bender, T. W., & Joiner Jr, T. E. (2008). Suicidal desire and the capability for suicide: Tests of the interpersonal-psychological theory of suicidal behavior among adults. *Journal of Consulting and Clinical Psychology*, *76*(1), 72.

Veale, D., Boocock, A., Goumay, K., Dryden, W., Shah, F., Wilson, R., Walburn, J. (1996). Body dysmorphic disorder. A survey of fifty cases. *The British of Journal of Psychiatry, 169,* 196-201.

Veiga-Martinez, C., Perez-Alvares, M., Garcia-Montes, G. (2008). Acceptance and commitment therapy applied to treatment of auditory hallucinations. *Clinical Case Studies, 7,* 118-135.

Volkmar, F. R., & Wiesner, L. A. (2017). *Essential Clinical Guide to Understanding and Treating Autism*. John Wiley & Sons.

von Gontard, A., Heron, J., & Joinson, C. (2011). Family history of nocturnal enuresis and urinary incontinence: results from a large epidemiological study. *The Journal of Urology*, *185*(6), 2303-2307.

Wagner, K., Ritt-Olson, A., Chou, C., Pokhrel, P., Duan, L., Baezconde-Garbanati, L., …Unger, J. (2012). Associations between parental family structure, family functioning, and substance use among Hispanic/Latino adolescents. *Psychology of Addictive Behaviors, 24,* 98-108.

Wahlbeck, K. (2015). Public mental health: the time is ripe for translation of evidence into practice. *World Psychiatry*, *14*(1), 36-42.

Watson, J. B. (1913). Psychology as the behaviorist views it. *Psychological Review*, *20*(2), 158.

Watson, J. & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology, 3,* 1-4.

Weare, K., & Nind, M. (2011). Mental health promotion and problem prevention in schools: what does the evidence say? *Health promotion international*, *26*, i29-i69.

Weaver, M. & Schnoll, S. (2008). Hallucinogens and club drugs. In H.D. Kleber & M Galanter (Eds), *The American Psychiatric Publishing textbook of substance abuse treatment* (4th ed. pp. 191-200). Arlington, VA: American Psychiatric Publishing.

Weissman, M. M., & Olfson, M. (1995). Depression in women: Implications for health care research. *Science*, *269*, 799-801.

Werle, M. A., Murphy, T. B., & Budd, K. S. (1993). Treating chronic food refusal in young children: Home-based parent training. *Journal of Applied Behavior Analysis*, *26*(4), 421-433.

Whelan, E. & Cooper, P. (2000). The association between childhood feeding problems andmaternal eating disorder: a community study. *Psychological Medicine, 30,* 69-77.

Whiffen, V. & Demidenko, N. (2006). Mood disturbance across the life span. In J. Worell & C. D. Goodheart (Eds.), *Handbook of girls' and women's psychological health* (pp. 51-59). New York: Oxford University Press.

Wise, R., & Koob, G. (2013). The development and maintenance of drug addiction. *Neuropsychopharmacology, 39,* 254-262.

Woods, D. W., Piacentini, J., Chang, S., Deckersbach, T., Ginsburg, G., Peterson, A., ... & Wilhelm, S. (2008). *Managing Tourette syndrome: A behavioral intervention for children and adults therapist guide*. Oxford University Press.

Wu, L., Ringwalt., C., Weiss, R., & Blazer, D. (2009). Hallucinogen-related disorders in a

national sample of adolescents: the influence of ecstasy/MDMA use. *Drug and Alcohol Dependence, 104*, 156-166.

Yadin, E. & Foa, E. (2009). How to reduce distress and repetitive behaviors in patients with OCD. *Current Psychiatry, 8,* 19-23.

Yes, Bayer Promoted Heroin – Here are the ads that prove it. Business Insider Website. http://www.businessinsider.com/yes-bayer-promoted-heroin-for-children-here-are-the-ads-that-prove-it-2011-11?op=1. Published on November 17, 2011. Accessed on November 4, 2017.

Zafra, J. (2016). The use of structural family therapy with a latino family: A case study. *Journal of Systemic Therapies*, *35*(4), 11-21.

Zeanah, C. H., Chesher, T., Boris, N. W., and the American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI). (2016). Practice parameter for the assessment and treatment of children and adolescents with reactive attachment disorder and disinhibited social engagement disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, *55*(11), 990-1003.

Zerbe, K. (2008). *Integrated treatment of eating disorders: Beyond the body betrayed.* New York, NY: W.W. Norton & Co.

Zeschel, E., Bingmann, T., Bechdolf, A., Kruger-Oezguerdal, S., Correll, C., Leopold, K., . . . Juckel, G. (2015). Temperament and prodromal symptoms prior to first manic/hypomanic episodes: Results from a pilot study. *Journal of Affective Disorders, 173,* 339-344.

Zinner, S. H., & Mink, J. W. (2010). Movement disorders I: Tics and stereotypies. *Pediatrics in Review*, *31*(6), 223.

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