Behavioral Disorders of Childhood

Discovering Psychology Series

Behavioral Disorders of Childhood

2nd edition

Kristy McRaney, Ph.D., Alexis Bridley, Ph.D., and Lee Daffin, Ph.D.

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WASHINGTON STATE UNIVERSITY

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PULLMAN, WA

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2nd edition

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

I

PART I. SETTING THE STAGE

Part I. Setting the Stage

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 missing important opportunities during the game – his peers are getting 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 took Ben to see a child psychologist.

Ben's story, though hypothetical, is true of children. Although many young children may 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 impairment for him in multiple domains of life such as home, work, school, and social circles.

In Module 1, we will explore what it means to display abnormal behavior, what mental disorders are and how society views it both today and throughout history. Then we will overview research methods used by psychologists in general and how they are adapted to study abnormal behavior/mental disorders. We will conclude with an overview of what mental health professionals do.

Module Outline

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

Module Learning Outcomes

- Explain what it means to display abnormal behavior.
- Clarify the manner in which mental health professionals classify mental disorders.
- Describe the effect of stigma on those afflicted with mental illness.
- Outline the history of mental illness.
- Describe research methods used to study abnormal behavior and mental illness.
- Identify types of mental health professionals, societies they may join, and journals they can publish their work in.

1.1. UNDERSTANDING ABNORMAL BEHAVIOR

Section Learning Objectives

- Describe the disease model and its impact on the field of psychology throughout history.
- Describe positive psychology.
- Defined abnormal behavior.
- Explain the concept of dysfunction as it relates to mental illness.
- Explain the concept of distress as it relates to mental illness.
- Explain the concept of deviance as it relates to mental illness.
- Explain the concept of dangerousness as it relates to mental illness.
- Define culture and social norms.
- Clarify the cost of mental illness on society.
- Define abnormal psychology, psychopathology, and mental disorders.

1.1.1. Understanding Abnormal Behavior

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

Psychology worked with the disease model for over 60 years, from about the late 1800s into the middle part of the 19th century. The focus was to cure 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 supported the dominant worldview still present at the time – mechanism – the idea that the world could be seen as a great machine and explained through the principles of physics and chemistry. In it, human beings were smaller machines in the larger machine of the universe.

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

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

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

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

(http://www.positivepsychologyinstitute.com.au/what_is_positive_psychology.html).

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

1.1.2. How do we determine what is abnormal behavior?

In the previous section we showed that what we might consider normal behavior is difficult to define. Equally difficult is understanding what abnormal behavior is which may be surprising to you. You will become intimately familiar throughout this book with The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5 for short). It states that though "no definition can capture all aspects of all disorders in the range contained in the DSM-5," certain aspects are required. These include:

- **Dysfunction** – includes "clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning" (pg. 20). Abnormal behavior, therefore, has the capacity to make our well-being difficult to obtain and can be assessed by looking at an individual's current performance and comparing it to what is expected in general or how the person has performed in the past. As such, a good employee who suddenly demonstrates poor performance may be experiencing an environmental demand leading to stress and ineffective coping mechanisms. Once the demand resolves itself, the person's performance should return to normal according to this principle.
- **Distress** – When the person experiences a disabling condition "in social, occupational, or other important activities" (pg. 20). Distress can take the form of psychological or physical pain, or both concurrently. Alone though, distress is not sufficient enough to describe behavior as abnormal. Why is that? The loss of a loved one would cause even the most "normally" functioning individual pain. An athlete who experiences a career ending injury would display distress as well. Suffering is part of life and cannot be avoided. And some people who display abnormal behavior are generally positive while doing so.
- **Deviance** – Closer examination of the word abnormal shows that it indicates a move away from what is normal, or the mean (i.e. what would be considered average and in this case in relation to behavior), and so is behavior that occurs infrequently (sort of an outlier in our data). Our **culture**, or the totality of socially transmitted behaviors, customs, values, technology, attitudes, beliefs, art,

and other products that are particular to a group, determines what is normal. A person is said to be deviant when he or she fails to follow the stated and unstated rules of society, called **social norms**. What is considered "normal" by society can change over time due to shifts in accepted values and expectations. For instance, homosexuality was considered taboo in the U.S. just a few decades ago but today, it is generally accepted. Likewise, PDAs, or public displays of affection, do not cause a second look by most people unlike the past when these outward expressions of love were restricted to the privacy of one's own house or bedroom. In the U.S., crying is generally seen as a weakness for males, However, if the behavior occurs in the context of a tragedy such as the Las Vegas mass shooting on October 1, 2017 in which 58 people were killed and about 500 were wounded while attending the Route 91 Harvest Festival, then it is appropriate and understandable. Finally, consider that statistically, deviant behavior is not necessarily negative. Genius is an example of behavior that is not the norm.

Though not part of the DSM conceptualization of what abnormal behavior is, many clinicians add **dangerousness** to this list, or when behavior represents a threat to the safety of the person or others. It is important to note that having a mental disorder does not mean you are also automatically dangerous. The depressed or anxious individual is often no more a threat than someone who is not depressed and as Hiday and Burns (2010) showed, dangerousness is more the exception than the rule. Still, mental health professionals have a duty to report to law enforcement when a mentally disordered individual expresses intent to harm another person or themselves. It is important to point out that people seen as dangerous are also not automatically mentally ill.

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

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

https://www.washingtonpost.com/news/wonk/wp/2012/12/17/seven-facts-about-americas-mental-health-care-system/?utm_term=.12de8bc56941

In conclusion, though there is no one behavior that we can use to classify people as abnormal, most clinical practitioners agree that any behavior that strays from what is considered the norm or is unexpected, and has the potential to harm others or the individual, is abnormal behavior. Armed with this understanding, let's discuss what mental disorders are.

1.1.3. Definition of abnormal psychology or psychopathology

Our discussion so far has concerned what is normal and abnormal behavior. We saw that the study of normal behavior falls under the providence of positive psychology. Similarly, the scientific study of abnormal behavior, with the intent to be able to reliably predict, explain, diagnose, identify the causes of, and treat maladaptive behavior is what we refer to as **abnormal psychology**. Abnormal behavior can become pathological in nature, leading to the scientific study of psychological disorders, or **psychopathology**. This begs the question of what the accepted definition of a psychological or mental disorder is. From our previous discussion we can fashion the following definition – **mental disorders** are characterized by psychological dysfunction which causes physical and/or psychological distress or impaired functioning and is not an expected behavior according to societal or cultural standards. **Child psychopathology** simply means abnormal psychology that is present during childhood.

1.2. CLASSIFYING MENTAL DISORDERS

Section Learning Objectives

- Define and exemplify classification.
- Define nomenclature.
- Define epidemiology.
- Define presenting problem and clinical description.
- Differentiate prevalence and incidence and any subtypes.
- Define comorbidity.

- Define etiology.
- Define course.
- Define prognosis.
- Define treatment.

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

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

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

Occurrence can be investigated in several ways. First, **prevalence** is the percentage of people in a population that has a mental disorder or can be viewed as the number of cases per some number of people. For instance, if 20 people out of 100 have bipolar disorder, then the prevalence rate is 20%. Prevalence can be measured in several ways:

- **Point prevalence** indicates the proportion of a population that has the characteristic at a specific point in time. In other words, it is the number of active cases.
- **Period prevalence** indicates the proportion of a population that has the characteristic at any point during a given period of time, typically the past year.
- **Lifetime prevalence** indicates the proportion of a population that has had the characteristic at any time during their lives.

According to the National Survey on Drug Use and Health (NSDUH), in 2015 there was an estimated 9.8 million U.S. adults, aged 18 years or older, with a *serious* mental illness or 4% of all U.S. adults, and 43.4 million adults, aged 18 years or older, with any mental illness or 17.9% of all U.S. adults.

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

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

Finally, **comorbidity** describes when two or more mental disorders are occurring at the same time and in the same person. The National Comorbidity Survey Replication (NCS-R) study conducted by the National Institute of Mental Health (NIMH) and published in the June 6, 2005 issue of the Archives of General Psychiatry, sought to discover trends in prevalence, impairment, and service use during the 1990s. It should be noted that the first study was conducted from 1980 to 1985 and surveyed 20,000 people from five different geographical regions in the U.S.A., followed by a second study 1990-1992 which was called the National Comorbidity Survey (NCS). The third study, the NCS-R, used a new nationally representative sample of the U.S. population, and found that 45% of those with one mental disorder met the diagnostic criteria for two or more disorders. The authors also found that the severity of mental illness, in regards to disability, is strongly related to comorbidity, and that substance use disorders often result from disorders such as anxiety and bipolar mood disorders. The implications of this are great as services to treat substance abuse and mental disorders are often separate, despite their appearing together.

The **etiology** is the cause of the disorder. There may be social, biological, or psychological explanations for the disorders beginning which need to be understood to identify the appropriate treatment. Likewise, the effectiveness of a treatment may give some hint at the cause of the mental disorder. More on this later.

The **course** of the disorder is its particular pattern. A disorder may be *acute* meaning that it lasts a short period of time, or *chronic,* meaning it lasts a long period of time. It can also be classified as *time-limited*, meaning that recovery will occur in a short period of time regardless of whether any treatment occurs.

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

Finally, we will discuss several treatment strategies in this book in relation to specific disorders, and in a general fashion in Module 3. **Treatment** is any procedure intended to modify abnormal behavior into

normal behavior. The person suffering from the mental disorder seeks the assistance of a trained professional to provide some degree of relief over a series of therapy sessions. The trained mental health professional may prescribe medication or utilize psychotherapy to bring about this change. Treatment may be sought from the primary care provider, in an outpatient fashion, or through inpatient care or hospitalization at a mental hospital or psychiatric unit of a general hospital.

1.3. THE STIGMA OF MENTAL ILLNESS

Section Learning Objectives

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

In the previous section we discussed the fact that care can be sought out in a variety of ways. The problem is that many people who need care never seek it out. Why is that? We already know that society dictates what is considered abnormal behavior through culture and social norms, and you can likely think of a few implications of that. But to fully understand society's role in why people do not seek care, we need to determine the psychological processes underlying this phenomena in the individual.

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

One way in which 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. It is during our initial interaction with someone that we collect information about him/her, assign the person to a category for which we have a schema, and then use that to affect how we interact with her or him. First impressions, called the *primacy effect*, are important because even if we obtain new information that should override an incorrect initial assessment, the initial impression is unlikely to change. We call this the *perseverance effect*, or *belief perseverance*.

Stereotypes are special types of schemas that are very simplistic, very strongly held, and not based on firsthand experience. They are **heuristics**, or mental shortcuts, that allow us to assess this collected information very quickly. One piece of information, such as skin color, can be used to assign the person to a schema for which we have a stereotype. This can affect how we think or feel about the person and behave toward them. Again, human beings have a tendency to imply things about 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 a group outside our immediate one as the same. This leads to a tendency to show favoritism to, and exclude or hold a negative view of members outside of, one's immediate group, called the *in-group/out-group bias*. The negative view or set of beliefs about a group of people is what we call *prejudice* and this can result in acting in a way that is negative against a group of people, called *discrimination*. It should be noted that a person can be prejudicial without being discriminatory since most people do not act on their attitudes toward others due to social norms against such behavior. Likewise, a person or institution can be discriminatory without being prejudicial such as if a company requires that a person have a certain education level or be able to lift 80 pounds as part of normal job responsibilities. Individuals without a degree or ability to lift will be removed from consideration for the job, but this discriminatory act does not mean that the company has negative views of people without degrees or the inability to lift heavy weight. You might even hold a negative view towards a certain group of people and not be aware of it. An attitude we are unaware of is called an *implicit attitude***,** which stands in contrast to *explicit attitudes,* which are the ones we publicly state.

We have spent quite a lot of space and time understanding background information on how people gather information about the world and people around them, process this information, use it to make snap judgements about others, form groups for which stereotypes may exist, and then potentially hold negative views of this group and behave negatively toward them as a result. Just one piece of

information can be used to set this series of mental events into motion. Outside of skin color, the label associated with having a mental disorder can be used. Stereotypes about people with a mental disorder can quickly and easily transform into prejudice when people in a society determine the schema to be correct and form negative emotions and evaluations of this group (Eagly & Chaiken, 1993). This in turn can lead to discriminatory practices such as an employer refusing to hire, a landlord refusing to rent an apartment, or a romantic relationship being avoided, all due to the person having a mental illness.

Overlapping with prejudice and discrimination in terms of how people with mental disorders are treated is **stigma**, or when negative stereotyping, labeling, rejection, and loss of status occur. Stigma takes on three forms as described below:

- *Public stigma* – when members of a society endorse negative stereotypes of people with a mental disorder and discriminate against them. They might avoid them all together resulting in social isolation. An example is when an employer intentionally does not hire a person because their mental illness is discovered.
- *Label avoidance* – In order to avoid being labeled as "crazy" or "nuts" people needing care may avoid seeking it all together or stop care once started. Due to these labels, funding for mental health services could be restricted and instead, physical health services funded.
- *Self-stigma* – When people with mental illnesses internalize the negative stereotypes and prejudice, and in turn, discriminate against themselves. They may experience shame, reduced self-esteem, hopelessness, low self-efficacy, and a reduction in coping mechanisms. An obvious consequence of these potential outcomes is the *why try* effect, or the person saying 'Why should I try and get that job. I am not 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 the person with a mental disorder. Karnieli-Miller et. al. (2013) found that families of the afflicted were often blamed, rejected, or devalued when others learned that their family member had a serious mental illness (SMI). Due to this, they felt hurt and betrayed and an important source of social support during the difficult time had disappeared, resulting in greater levels of stress. To cope, they decided to conceal their relative's illness and some parents struggled to decide whether it was their place to disclose versus the relative's place. Others fought with the issue of confronting the stigma through attempts at education or to just ignore it due to not having enough energy or desiring to maintain personal boundaries. There was also a need to understand responses of others and to attribute it to a lack of knowledge, experience, and/or media coverage. In some cases, the reappraisal allowed family members to feel compassion for others rather than feeling put down or blamed. The authors concluded that each family "develops its own coping strategies which vary according to its personal experiences, values, and extent of other commitments" and that "coping strategies families employ change over-time."

Other effects of stigma include experiencing work-related discrimination resulting in higher levels of self-stigma and stress (Rusch et al., 2014), higher rates of suicide especially when treatment is not

available (Rusch, Zlati, Black, and Thornicroft, 2014; Rihmer & Kiss, 2002), and a decreased likelihood of future help-seeking intention in a university sample (Lally et al., 2013). The results of the latter study also showed that personal contact with someone with a history of mental illness led to a decreased likelihood of seeking help. This is important because 48% of the 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 important to also point out that social distance, a result of stigma, has also been shown to increase throughout the life span suggesting that anti-stigma campaigns should focus on older people primarily (Schomerus, et al., 2015).

One potentially disturbing trend is that mental health professionals have been shown to hold negative attitudes toward the people that 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 is less stigmatized, their work places were in the county council as they were more likely to encounter patients who recover and return to normal life in society compared to municipalities where patients have long-term and recurrent mental illness, and they have or had one close friend with mental health issues.

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

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

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

Stigma has been shown to lead to health inequities (Hatzenbuehler, Phelan, & Link, 2013) prompting calls for stigma change. Targeting stigma leads to two different agendas. The *services agenda* attempts to remove stigma so the person can seek mental health services while the *rights agenda* tries to replace discrimination that "robs people of rightful opportunities with affirming attitudes and behavior" (Corrigan, 2016). The former is successful when there is evidence that people with mental illness are seeking services more or becoming better engaged while the latter is successful when there is an increase in the number of people with mental illnesses in the workforce and receiving reasonable accommodations. The federal government has tackled this issue with landmark legislation such as the Patient Protection and Affordable Care Act of 2010, Mental Health Parity and Addiction Equity Act of 2008, and the Americans with Disabilities Act of 1990 though protections are not uniform across all subgroups due to "1) explicit language about inclusion and exclusion criteria in the statute or implementation rule, 2) vague statutory language that yields variation in the interpretation about which groups qualify for protection, and 3) incentives created by the legislation that affect specific groups differently" (Cummings, Lucas, and Druss, 2013). More on this in Module 15.

1.4. THE HISTORY OF MENTAL ILLNESS

Section Learning Objectives

- Describe prehistoric and ancient beliefs about mental illness.
- Describe Greco-Roman thought on mental illness.
- Describe thoughts on mental illness during the Middle Ages.
- Describe thoughts on mental illness during the Renaissance.
- Describe thoughts on mental illness during the 18th and 19th centuries.
- Describe thoughts on mental illness during the 20th and 21st centuries.
- Describe the status of mental illness today.
- Outline the use of psychoactive drugs throughout time and their impact.
- Clarify the importance of managed health care for the treatment of mental illness.
- Define and clarify the importance of multicultural psychology.

- State the issue surrounding prescription rights for psychologists.
- Explain the importance of prevention science.

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

1.4.1. Prehistoric and Ancient Beliefs

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

1.4.2. Greco-Roman Thought

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

Also important was Greek philosopher, Plato (429-347 B.C.), who said that the mentally ill were not responsible for their own actions and should not be punished. It was the responsibility of the community and their families to care for them. Greek physician, Galen (A.D. 129-199) said mental disorders had either physical or mental causes and included fear, shock, alcoholism, head injuries, adolescence, and changes in menstruation.

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

1.4.3. The Middle Ages – 500 AD to 1500 AD

The progress made during the time of the Greeks and Romans was quickly reversed during the Middle Ages with the increase in power of the Church and the fall of the Roman Empire. Mental illness was yet again explained as possession by the Devil and methods such as exorcism, flogging, prayer, the touching of relics, chanting, visiting holy sites, and holy water were used to rid the person of his influence. In extreme cases, the afflicted were exposed to confinement, beatings, and even execution. Scientific and medical explanations, such as those proposed by Hippocrates, were discarded.

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

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

1.4.4. The Renaissance – 14th to 16th centuries

The most noteworthy development in the realm of philosophy during the Renaissance was the rise of **humanism**, or the worldview that emphasizes human welfare and the uniqueness of the individual. This helped continue the decline of supernatural views of mental illness. In the mid to late 1500s, Johann Weyer (1515-1588), a German physician, published his book, *On the Deceits of the Demons,* that rebutted the Church's witch-hunting handbook, the *Malleus Maleficarum*, and argued that many accused of being witches and subsequently imprisoned, tortured, and/or burned at the stake, were

mentally disturbed and not possessed by demons or the Devil himself. He believed that like the body, the mind was susceptible to illness. Not surprisingly, the book was met with vehement protest and even banned from the church. It should be noted that these types of acts occurred not only in Europe, but also in the United States. The most famous example was the Salem Witch Trials of 1692 in which more than 200 people were accused of practicing witchcraft resulting in 20 people being killed.

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

1.4.5. Reform Movement – 18th to 19th centuries

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

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

Reform in the United States started with the figure largely considered to be the father of American psychiatry, Benjamin Rush (1745-1813). Rush advocated for the humane treatment of the mentally ill, showing them respect, and even giving them small gifts from time to time. Despite this, his practice included treatments such as bloodletting and purgatives, the invention of the "tranquilizing chair," and

a reliance on astrology, showing that even he could not escape from the beliefs of the time.

Due to the rise of the moral treatment movement in both Europe and the United States, asylums became habitable 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 main proponent in the United States was Dorothea Dix (1802-1887), a New Englander who observed the deplorable conditions suffered by the mentally ill while teaching Sunday school to female prisoners. Over the next 40 years, from 1841 to 1881, she motivated people and state legislators to do something about this injustice and raised millions of dollars to build over 30 more appropriate mental hospitals and improve others. Her efforts even extended beyond the U.S. to Canada and Scotland.

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

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

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

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

1.4.6. 20th – 21st Centuries

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

1.4.6.1. Biological or Somatogenic Perspective. Recall that Greek physicians Hippocrates and Galen said that mental disorders were akin to physical disorders and had natural causes. Though the idea fell into oblivion for several centuries it re-emerged in the late 19th century for two reasons. First, German psychiatrist, Emil Kraepelin (1856-1926), discovered that symptoms occurred regularly in clusters which he called **syndromes**. These syndromes represented a unique mental disorder with its own cause, course, and prognosis. In 1883 he published his textbook, *Compendium der Psychiatrie* (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 (published in 2013).

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

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

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

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

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

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

The use of hypnosis to treat hysteria was also carried out by fellow Frenchman Pierre Janet (1859-1947), and student of Charcot, who believed that hysteria had psychological, not biological causes. Namely, these included unconscious forces, fixed ideas, and memory impairments. In Vienna, Josef Breuer (1842-1925) induced hypnosis and had patients speak freely about past events that upset them. Upon waking, he discovered that patients sometimes were free of their symptoms of hysteria. Success was even greater when patients not only recalled forgotten memories, but also relived them emotionally. He called this the **cathartic method** and our use of the word *catharsis* today indicates a purging or release, in this case, of pent up emotion.

By the end of the 19th century, it had become evident that mental disorders were caused by a

combination of biological and psychological factors and the investigation of how they develop began. Sigmund Freud's development of psychoanalysis followed on the heels of the work of Bruner, and others who came before him.

1.4.7. Current Views/Trends

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

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

Also of interest was that the female participants were more likely to have had anxiety and mood disorders while male participants 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 psychotropic medications which are not more readily available now. The article concludes, “Survey researchers also suggest that we need more outreach and voluntary screening, more education about mental illness for the public and physicians, and more effort to treat substance abuse and impulse control disorders.” We will explore several of these issues in the remainder of this section to include the use of psychiatric drugs and deinstitutionalization, managed health care, private psychotherapy, positive psychology and prevention science, Multicultural psychology, and prescription rights for psychologists.

For more on the Harvard article, please see:

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

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

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

Frank (2006) found that by 1996, psychotropic drugs were used in 77% of mental health cases and spending on these drugs to treat mental disorders grew from $2.8 billion in 1987 to about $18 billion in 2001 (Coffey et al., 2000; Mark et al., 2005), representing a greater than sixfold increase. The largest classes of psychotropic drugs are anti-psychotic and anti-depressant, followed closely by anti-anxiety. Frank, Conti, and Goldman (2005) point out, “The expansion of insurance coverage for prescription drugs, the introduction and diffusion of managed behavioral health care techniques, and the conduct of the pharmaceutical industry in promoting their products all have influenced how psychotropic drugs are used and how much is spent on them.” Is it possible then that we are overprescribing these medications? Davey (2014) provides ten reasons why this may be including (1) leading suffers to believe that recovery is out of their hands and instead in the hands of their doctors, (2) increased risk of relapse, (3) drug companies causing the “medicalization of perfectly normal emotional processes, such as bereavement” to ensure their own survival, (4) side effects, and (5) a failure to change the way the way the person thinks or the socioeconomic environments that may be the cause of the disorder. For more on this article, please see: https://www.psychologytoday.com/blog/why-we-worry/201401/overprescribing-drugs-treat-mental-health-problems. Smith (2012) echoed similar sentiments in an article on inappropriate prescribing and cites the approval of Prozac by the Food and Drug Administration (FDA) in 1987 as when the issue began and the overmedication/overdiagnosis of children with ADHD as a more recent example.

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

1.4.7.3. Managed health care. Managed health care is a term used to describe a type of health insurance in which the insurance company determines the cost of services, 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 the majority of their care. The PCP refers the subscriber to specialists or other health care providers as is necessary. This is the most restrictive option.
- *Preferred Provider Organizations (PPO)* – Usually pay more if the subscriber obtains care within the network but if care outside the network is sought, the PPO plan pays a smaller portion of the service.
- *Point of Service (POS)* – These plans provide the most flexibility and allow the subscriber to choose between an HMO or a PPO each time care is needed.

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

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

In August 2002, the American Psychological Association's (APA) Council of Representatives put forth six guidelines based on the understanding that "race and ethnicity can impact psychological practice and interventions at all levels" and that there is a need for respect and inclusiveness. They further state, "psychologists are in a position to provide leadership as agents of prosocial change, advocacy, and social justice, thereby promoting societal understanding, affirmation, and appreciation of

multiculturalism against the damaging effects of individual, institutional, and societal racism, prejudice, and all forms of oppression based on stereotyping and discrimination." The guidelines from the 2002 document are as follows:

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

Source: https://www.apa.org/about/policy/multicultural-guidelines

This type of sensitivity training is important because bias on the basis of ethnicity, race, and culture has been found in the diagnosis and treatment of autism (Harrison et al., 2017; Burkett, 2015), borderline personality disorder (Jani et al., 2016), and schizophrenia (Neighbors et al., 2003'; Minsky et al., 2003). Despite these findings, Schwartz and Blankenship (2014) 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 while women were offered an appointment time in their preferred time range more than men were, though average appointment offer rates were similar between genders (Kugelmass, 2016). These findings collectively show that though we are becoming more culturally sensitive, we have a lot more work to do.

1.4.7.5. Prescription rights for psychologists. To reduce inappropriate prescribing as described in 1.4.7.2, it has been proposed to allow appropriately trained psychologists the right to prescribe.

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

1.4.7.6. Prevention science. As a society, we used to wait for a mental or physical health issue to emerge and then we would scramble to treat it. More recently, medicine and science has taken a **prevention** stance, or identifying the factors that cause specific mental health issues and implementing interventions to stop them from happening, or at least minimize their deleterious effects. Our focus has shifted from individuals to the population. Mental health promotion programs have been instituted with success in schools (Shoshani & Steinmetz, 2014; Weare & Nind, 2011; Berkowitz & Beer, 2007), in the workplace (Czabała, Charzyńska, & Mroziak, B., 2011), with undergraduate and graduate students (Conley et al., 2017; Bettis et al., 2016), in relation to bullying (Bradshaw, 2015), and with the elderly (Forsman et al., 2011). Many researchers believe the time is ripe to move from knowledge to action and to expand public mental health initiatives (Wahlbeck, 2015). The growth of positive psychology in the late 1990s has further propelled this movement forward. For more on positive psychology, please see Section 1.1.1.

1.5. RESEARCH METHODS IN PSYCHOPATHOLOGY

Section Learning Objectives

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

1.5.1. The Scientific Method

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

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

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

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

Table 1.1: The Steps of the Scientific Method

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

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

1. *Observation* – In order to know about the world around us we have to be able to see it firsthand. When an individual is afflicted by a mental disorder, we can see it through the overt behavior they make. An individual with depression may withdraw from activities he/she enjoys, those with social anxiety disorder will avoid social situations, people with schizophrenia may express concern over being watched by the government, and individuals with dependent personality disorder may wait to

make any decision in life until trusted others tell them what to do. In these examples, and numerous others we can suggest, the behaviors that lead us to a diagnosis of a specific disorder can easily be observed by the clinician, the patient, and/or family and friends.

2. *Experimentation* – To be able to make *causal* or cause and effect statements, we must isolate variables. We have to manipulate one variable and see the effect of doing so on another variable. Let's say we want to know if a new treatment for bipolar disorder is as effective as existing treatments…or more importantly, better. We could design a study with three groups of bipolar patients. One group would receive no treatment and serve as a control group. A second group would receive an existing and proven treatment and would also be considered a control group. Finally, the third group would receive the new treatment and be the experimental group. What we are manipulating is what treatment the groups get – no treatment, the older treatment, and the newer treatment. The first two groups serve as controls since we already know what to expect from their results. There should be no change in bipolar disorder symptoms in the no treatment group, a general reduction in symptoms for the older treatment group, and the same or better performance for the newer treatment group. As long as patients in the newer treatment group don't perform worse than their older treatment counterparts, we can say the new drug is a success. You might wonder why we would get excited about the performance of the new drug being the same as the old drug. Does it really offer any added benefit? In terms of a reduction of symptoms, maybe not, but it could cost less money than the older drug and so that would be of value to patients.
3. *Measurement* – How do we know that the new drug has worked? Simply, we can measure the person's bipolar 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 a home, school, or forest. The researcher counts, measures, and rates behavior in a systematic way and at times uses multiple judges to ensure accuracy in how the behavior is being measured. The advantage of this method is that you see behavior as it occurs in its natural environment and it is not tainted by the experimenter. The disadvantage is that it could take a long time for the behavior to occur and if the researcher is detected it may influence the behavior of those being observed. **Laboratory observation** involves observing people or animals in a laboratory setting. The researcher might want to know more about parent-child interactions and so brings a mother and her child into the lab to engage in preplanned tasks such as playing with toys, eating a meal, or the mother leaving the room for a short period of time. The advantage of this method over naturalistic method is that the experimenter can use

sophisticated equipment and videotape the session to examine it at a later time. The problem is that since the subjects know the experimenter is watching them, their behavior could become artificial. Clinical observation is a commonly employed research method to study psychopathology and we will talk about it more throughout this course.

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

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

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

A special from of correlational research is the **epidemiological study** in which the prevalence and

incidence of a disorder in a specific population are measured (See Section 1.2 for definitions).

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

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

Finally, the study of mental illness does not always afford us a large sample of participants to study and so we have to focus on one individual This is called a **single-subject experimental design** and differs from a case study in the sheer number of strategies that can be used to reduce potential **confounding variables**, or variables not originally part of the research design but contribute to the results in a meaningful way. One type of single-subject experimental design is the **reversal** or **ABAB design**. Kuttler, Myles, and Carson (1998) used social stories to reduce tantrum behavior in two social environments in a 12-year old student diagnosed with autism, Fragile-X syndrome, and intermittent explosive disorder. Using an ABAB design, they found that precursors to tantrum behavior decreased when the social stories were available (B) and increased when the intervention was withdrawn (A). A more recent study (Balakrishnan & Alias, 2017) also established the utility of social stories as a social learning tool for children with autism spectrum disorder (ASD) using an ABAB design. Four students were included in the study and during the baseline phase (A) they were observed and data recorded on an observation form. During the treatment phase (B), they were read the social story and data recorded in the same manner. Upon completion of the first B, the student was returned to A which was followed one more time by B and the reading of the social story. Once the second treatment phase ended, the participation was monitored again to obtain a final outcome. All students showed improvement during the treatment phases in terms of the number of positive peer interactions they had and then dropped back down in terms of the number of such interactions. From this the researchers concluded that the social story led to the increase in positive peer interactions of children with ASD.

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

1.6. MENTAL HEALTH PROFESSIONALS, SOCIETIES, AND JOURNALS

Section Learning Objectives

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

1.6.1. Types of Professionals

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

Table 1.2: Types of Mental Health Professionals

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

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

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

1.6.2. Professional Societies and Journals

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

1.6.2.1. Professional Societies

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

- **Society of Clinical Child and Adolescent Psychology – Division 53 of the American Psychological Association**
    - Website – https://www.clinicalchildpsychology.org/
    - Mission Statement – "The purpose of Division 53: Society of Clinical Child and Adolescent Psychology is to encourage the development and advancement of clinical child and adolescent psychology through integration of its scientific and professional aspects. The division promotes scientific inquiry, training, professional practice, and public policy in clinical child and adolescent psychology as a means of improving the welfare and mental health of children, youth, and families. In the service of these goals, the division promotes the general objectives of the American Psychological Association."
    - Publication – Journal of *Clinical Child and Adolescent Psychology*

- ***American Academy of Clinical Psychology***
    - Website – https://www.aacpsy.org/
    - Mission Statement – "The American Academy of Clinical Psychology is an organization of Board Certified psychologists in the specialty of Clinical Psychology who have joined together to promote high quality services in Clinical Psychology, through encouraging high standards and ethical practice in the field. The Academy also provides member services, promotes the value and recognition of Board Certification in the specialty of Clinical Psychology, and encourages those qualified by training and experience to become candidates for Board Certification."
    - Publication – Bulletin of the American Academy of Clinical Psychology (newsletter)

- **The Society for a Science of Clinical Psychology (SSCP)**

- Website – http://www.sscpweb.org/
- Mission Statement – "**The Society for a Science of Clinical Psychology (SSCP)** was established in 1966. Its purpose is to affirm and continue to promote the integration of the scientist and the practitioner in training, research, and applied endeavors. Its members represent a diversity of interests and theoretical orientations across clinical psychology. The common bond of the membership is a commitment to empirical research and the ideal that scientific principles should play a role in training, practice, and establishing public policy for health and mental health concerns. SSCP has organizational affiliations with both the American Psychological Association (Section III of Division 12) and the Association for Psychological Science."
- *Other Information – Offers ten awards ranging from early career award, outstanding mentor award, outstanding student teacher award, and outstanding student clinician award.*

- ***American Society of Clinical Hypnosis***

  - Website – http://www.asch.net/
  - Mission Statement – "The American Society of Clinical Hypnosis is the largest U.S. organization for health and mental health care professionals using clinical hypnosis. Founded by Milton H. Erickson, MD in 1957, ASCH promotes greater acceptance of hypnosis as a clinical tool with broad applications. Today, ASCH offers professional hypnosis training workshops, certification, and networking opportunities that can enhance both professional and personal lives. ASCH is unique among organizations for professionals using hypnosis. Members must be licensed healthcare workers and, at a minimum, have obtained a master's degree."
  - Publication – American Journal of Clinical Hypnosis
  - *Other Information – Offers certification in clinical hypnosis*

1.6.2.2. Professional Journals

- **Clinical Psychology: Science and Practice**

  - Website – http://onlinelibrary.wiley.com/journal/10.1111/(ISSN)1468-2850
  - Published by – American Psychological Association, Division 12
  - Description – "*Clinical Psychology: Science and Practice* presents cutting-edge developments in the science and practice of clinical psychology by publishing scholarly topical reviews of research, theory, and application to diverse areas of the field, including assessment, intervention, service delivery, and professional issues."

- **Journal of *Clinical Child and Adolescent Psychology***
    - Website – https://www.clinicalchildpsychology.org/JCCAP
    - Published by – American Psychological Association, Division 53
    - Description – "It publishes original contributions on the following topics: (a) the development and evaluation of assessment and intervention techniques for use with clinical child and adolescent populations; (b) the development and maintenance of clinical child and adolescent problems; (c) cross-cultural and sociodemographic issues that have a clear bearing on clinical child and adolescent psychology in terms of theory, research, or practice; and (d) training and professional practice in clinical child and adolescent psychology, as well as child advocacy."

- **American Journal of Clinical Hypnosis**
    - Website – https://www.tandfonline.com/action/journalInformation?journalCode=ujhy20
    - Published by – American Society of Clinical Hypnosis
    - Description – "The *Journal* publishes original scientific articles and clinical case reports on hypnosis, as well as reviews of related books and abstracts of the current hypnosis literature."

Module Recap

In Module 1, we undertook a fairly lengthy discussion of what abnormal behavior is by first looking at what normal behavior is. What emerged was a general set of guidelines focused on mental illness as causing dysfunction, distress, deviance, and at times, being dangerous for the afflicted and others around him/her. From this we classified mental disorders in terms of their occurrence, cause, course, prognosis, and treatment. We acknowledged that mental illness is stigmatized in our society and provided a basis for why this occurs and what to do about it. This involved a discussion of the history of mental illness and current views and trends. Psychology is the scientific study of behavior and mental processes. The word *scientific* is key as psychology adheres to the strictest aspects of the scientific method and uses five main research designs in its investigation of mental disorders – observation, case study, surveys, correlational research, and experiments. These designs are used by various mental health professionals, societies and journals in providing 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.

2nd edition

Module 2 - Models of Abnormal Psychology

Module Overview

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

Module Outline

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

Module Learning Outcomes

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

2.1. UNI- VS. MULTI-DIMENSIONAL MODELS OF ABNORMALITY

Section Learning Objectives

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

2.1.1. Uni-Dimensional

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

2.1.2. Multi-Dimensional

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

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

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

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

2.2. THE BIOLOGICAL MODEL

Section Learning Objectives

- Describe how communication in the nervous system occurs.
- List the parts of the nervous system.
- Describe the structure of the neuron and all key parts.
- Outline how neural transmission occurs.
- Identify and define important neurotransmitters.
- List the major structures of the brain.
- Clarify how specific areas of the brain are involved in mental illness.
- Describe the role of genes in mental illness.
- Describe the role of hormonal imbalances in mental illness.
- Describe 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. While doing this, we will identify areas of concern for psychologists focused on the treatment of mental disorders.

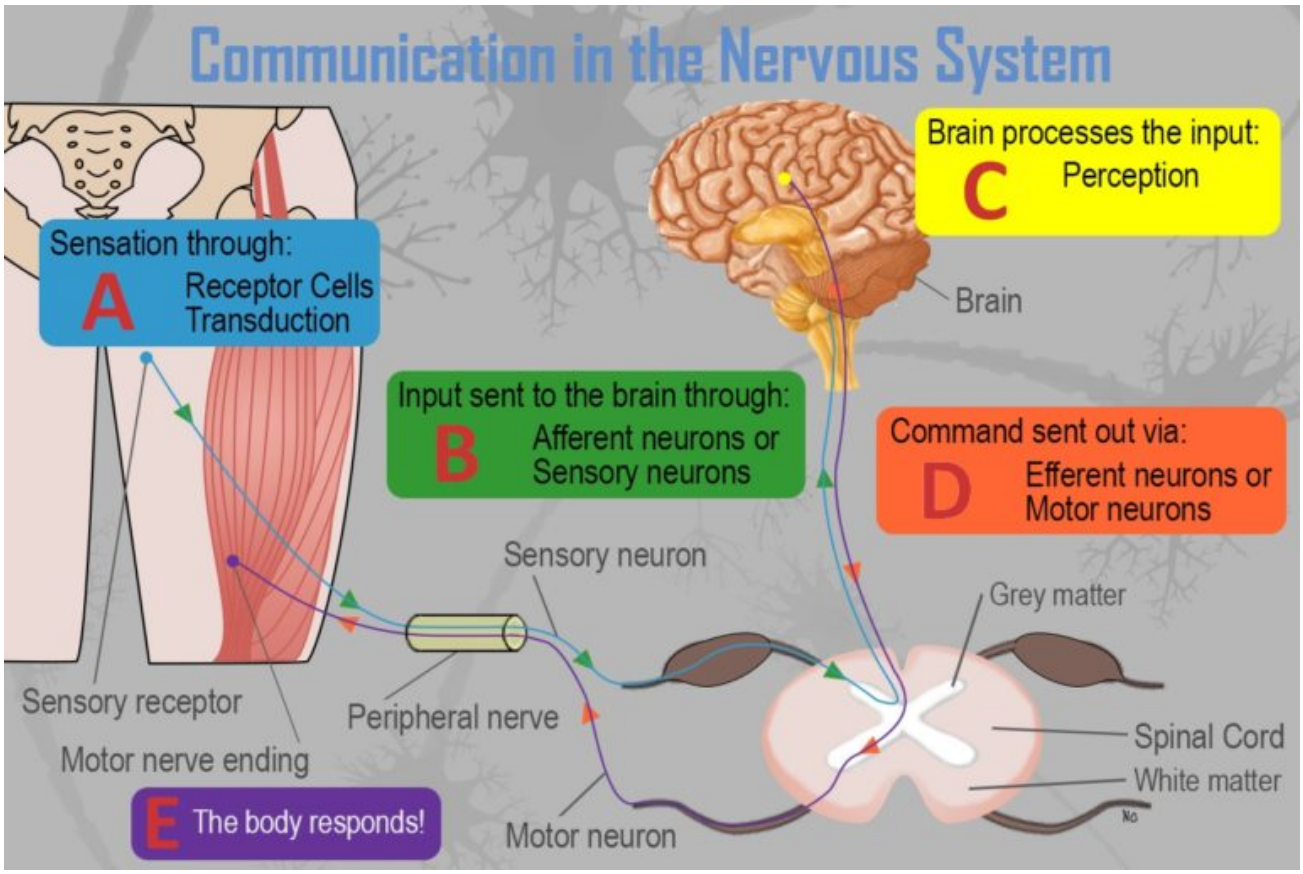
2.2.1. Brain Structure and Chemistry

2.2.1.1. Communication in the nervous system. To really understand brain structure and chemistry,

it is a good idea to understand how communication occurs within the nervous system. See Figure 2.1 below. Simply:

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

Figure 2.1. Communication in the Nervous System

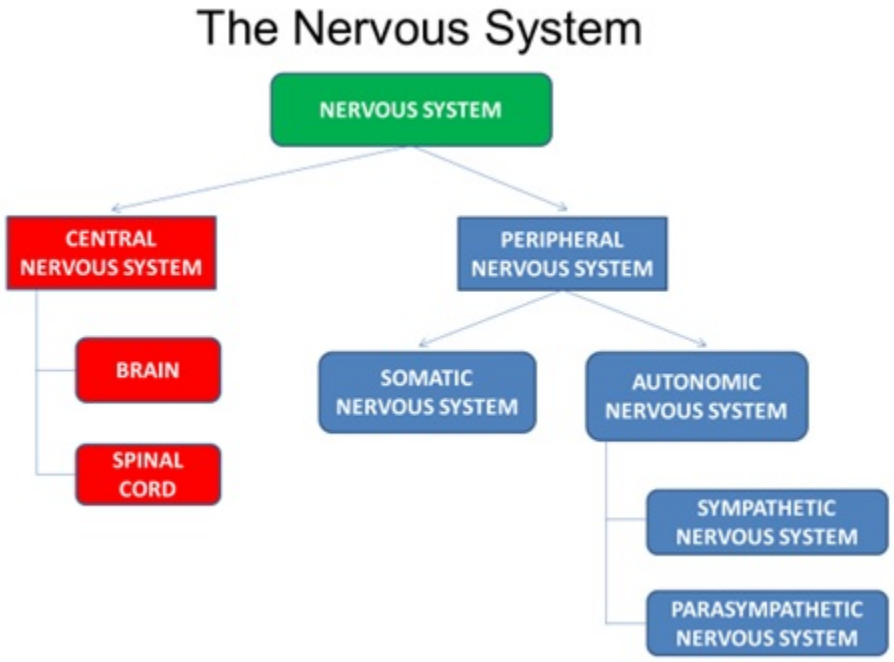


Please note that we will not cover this process in full but will focus on the parts relevant to our topic of psychopathology.

2.2.1.2. The nervous system. The nervous system consists of two main parts – the central and peripheral nervous systems. The **central nervous system (CNS)** is the control center for the nervous system which receives, processes, interprets, and stores incoming sensory information. It consists of the

brain and spinal cord. The **peripheral nervous system** consists of everything outside the brain and spinal cord. It handles the CNS's input and output and divides into the somatic and autonomic nervous systems. The **somatic nervous system** allows for voluntary movement by controlling the skeletal muscles and carries sensory information to the CNS. The **autonomic nervous system** regulates functioning of blood vessels, glands, and internal organs such as the bladder, stomach, and heart. It consists of sympathetic and parasympathetic nervous systems. The **sympathetic nervous system** is involved when a person is intensely aroused. It provides the strength to fight back or to flee (fight-or-flight instinct). Eventually the response brought about by the sympathetic nervous system must end. The **parasympathetic nervous system** calms the body.

Figure 2.2. The Structure of the Nervous System



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

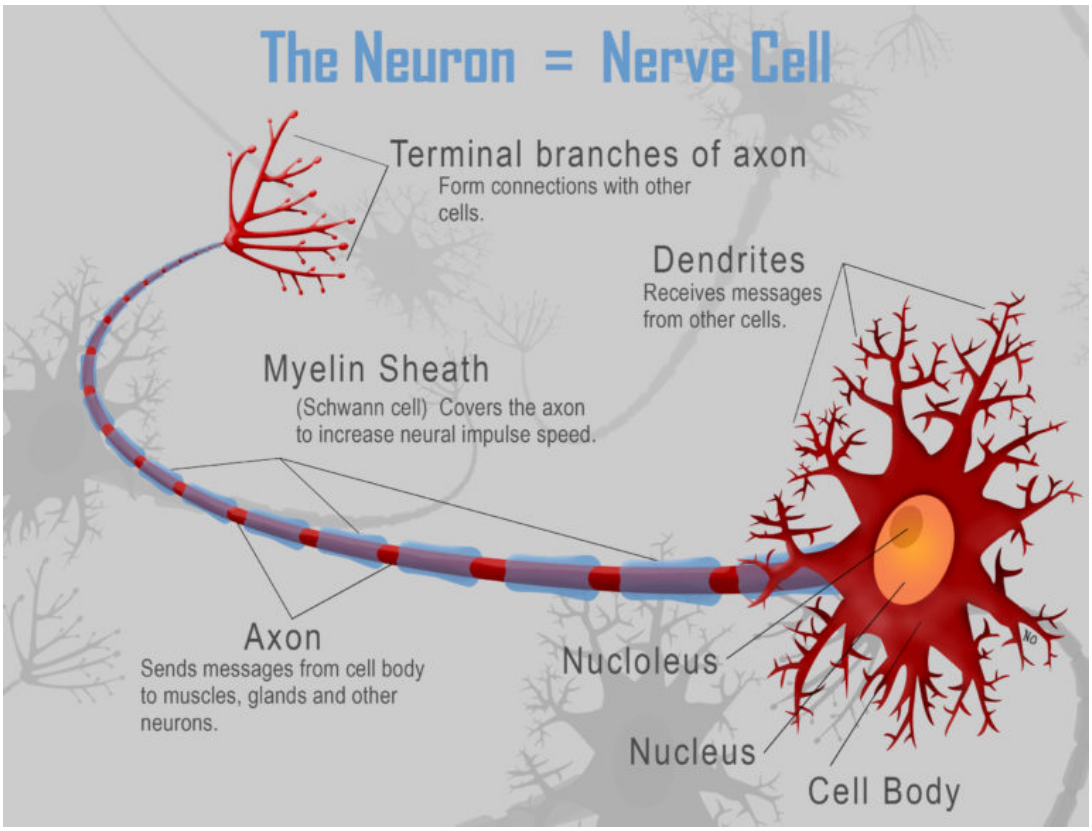
end of the axon where the electrical impulse becomes a chemical message and is passed to an adjacent neuron.

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

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

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

Figure 2.3. The Structure of the Neuron



2.2.1.4. Neural transmission. Transducers or receptor cells in the major organs of our five sensory systems – vision (the eyes), hearing (the ears), smell (the nose), touch (the skin), and taste (the tongue) – convert the physical energy that they detect or sense, and send it to the brain via the neural impulse. How so? See Figure 2.4 below. We will cover this process in three parts.

Part 1. The Axon and Neural Impulse

Figure 2.4. The Neural Impulse

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

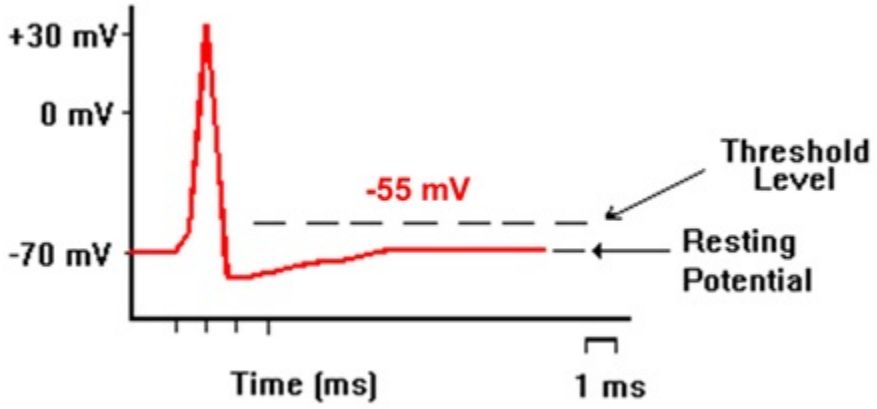
- Step 1 – Neurons waiting to fire are said to be in **resting potential** and **polarized**, or having a negative charge inside the neuron and a positive charge outside.
- Step 2 – If adequately stimulated, the neuron experiences an **action potential** and becomes **depolarized**. When this occurs, ion gated channels open allowing positively charged Sodium ions to enter. This shifts the polarity to positive on the inside and negative outside.
- Step 3 – Once the action potential passes from one segment of the axon to the next, the previous segment begins to **repolarize**. This occurs because the sodium (Na) channels close and potassium (K) channels open. K has a positive charge and so the neuron becomes negative again on the inside and positive on the outside.
- Step 4 – After the neuron fires it will not fire again no matter how much stimulation it receives. This is called the **absolute refractory period**. The neuron ABSOLUTELY will not fire, no matter what, during this period of time.
- Step 5 – After a short period of time, the neuron can fire again, but needs greater than normal levels of stimulation to do so. This is called the **relative refractory period**.
- Step 6 – Please note that the process is cyclical. We start at resting potential in Step 1 and notice

that Step 6 is the same as Step 1.

Part 2. The Action Potential

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

Figure 2.5. The Action Potential



- Recall that a neuron is normally at resting potential and polarized. The charge inside is -70mV at rest.
- If it receives sufficient stimulation meaning that the polarity inside the neuron rises from -70 mV to -55mV defined as the **threshold of excitation**, the neuron will **fire** or send an electrical impulse down the length of the axon (the action potential or depolarization). It should be noted that it either hits -55mV and fires or it does not. This is the **all-or-nothing principle**. The threshold must be reached.
- Once the electrical impulse has passed from one segment of the axon to the next, the neuron begins the process of resetting called repolarization.
- During repolarization the neuron will not fire no matter how much stimulation it receives. This is called absolute refractory period.
- The neuron next moves into relative refractory period meaning it can fire, but needs greater than normal levels of stimulation. Notice how the line has dropped below -70mV. Hence, to reach -55mV and fire, it will need more than the normal gain of +15mV (-70 to -55 mV).
- And then we return to resting potential, as you saw in Figure 2.4

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

exit and return the neuron to a polarized state.

Part 3. The Synapse

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

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

- **Dopamine** – controls voluntary movements and is associated with the reward mechanism in the brain
- **Serotonin** – controls pain, sleep cycle, and digestion; leads to a stable mood and 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** – responsible for blocking the signals of excitatory neurotransmitters responsible for anxiety and panic.
- **Glutamate** – associated with learning and memory

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

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

- **Medulla** – Regulates breathing, heart rate, and blood pressure.
- **Pons** – Acts as a bridge connecting the cerebellum and medulla and helps to transfer messages between different parts of the brain and spinal cord.

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

Of course, this is not an exhaustive list of structures found in the brain but gives you a pretty good idea of function and which structure is responsible for it. What is important to mental health professionals is that for some disorders, specific areas of the brain are involved. For instance, Parkinson's disease is a brain disorder which results in a gradual loss of muscle control and arises when cells in the **substantia nigra**, a long nucleus considered to be part of the basal ganglia, stop making dopamine. As these cells die, the brain fails to receive messages about when and how to move. In the case of depression, low levels of serotonin are responsible, at least partially. New evidence suggests "nerve cell connections, nerve cell growth, and the functioning of nerve circuits have a major impact on depression…and areas that play a significant role in depression are the amygdala, the thalamus, and the hippocampus" (Harvard Health, nd). Also, individuals with borderline personality disorder have been shown to have structural and functional changes in brain areas associated with impulse control and emotional regulation while imaging studies reveal differences in the frontal cortex and subcortical structures for those suffering from OCD.

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

2.2.2. Genes, Hormonal Imbalances, and Viral Infections

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

Recent research has discovered that autism, ADHD, bipolar disorder, major depression, and schizophrenia all share genetic roots. They, "were more likely to have suspect genetic variation at the same four chromosomal sites. These included risk versions of two genes that regulate the flow of calcium into cells" (Losik, 2016). For more on this development, please check out the article at: https://www.nih.gov/news-events/nih-research-matters/common-genetic-factors-found-5-mental-disorders 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 is one and the endocrine system is the second. 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 organize the nervous system and body tissues at certain stages of development and activate behaviors such as alertness or sleepiness, sexual behavior, concentration, aggressiveness, reaction to stress, a desire for companionship. The **pituitary gland** is the "master gland" which regulates other endocrine glands. It influences blood pressure, thirst, contractions of the uterus during childbirth, milk production, sexual behavior and interest, body growth, the amount of water in the body's cells, and other functions as well. The **pineal gland** helps regulate the sleep-wake cycle while the **thyroid gland** regulates the body's rate of metabolism and how energetic people are.

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

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

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

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

2.2.3. Treatments

2.2.3.1. Psychopharmacology and psychotropic drugs. One option to treat severe mental illness is psychotropic medications. These medications fall under five major categories.

The *antidepressants* are used to treat depression, but also anxiety, insomnia, or pain. The most common types of antidepressants are SSRIs or selective serotonin reuptake inhibitors and include Citalopram, Paroxetine, and Fluoxetine (Prozac). Possible side effects include weight gain, sleepiness, nausea and vomiting, panic attacks, or thoughts about suicide or dying.

Anti-anxiety medications help with the symptoms of anxiety and include the benzodiazepines such as

Clonazepam, Alprazolam, and Lorazepam. “Anti -anxiety medications such as benzodiazepines are effective in relieving anxiety and take effect more quickly than the antidepressant medications (or buspirone) often prescribed for anxiety. However, people can build up a tolerance to benzodiazepines if they are taken over a long period of time and may need higher and higher doses to get the same effect.” Side effects include drowsiness, dizziness, nausea, difficulty urinating, and irregular heartbeat, to name a few.

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

Antipsychotics are used to treat psychosis or, “conditions that affect the mind, and in which there has been some loss of contact with reality, often including delusions (false, fixed beliefs) or hallucinations (hearing or seeing things that are not really there).” They can be used to treat eating disorders, severe depression, PTSD, OCD, ADHD, and Generalized Anxiety Disorder. Common antipsychotics include Chlorpromazine, Perphenazine, Quetiapine, and Lurasidone. Side effects include nausea, vomiting, blurred vision, weight gain, restlessness, tremors, and rigidity.

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

For more information on psychotropic medications, please visit:

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

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

2.2.3.2. Electroconvulsive therapy. According to Mental Health America, “Electroconvulsive therapy (ECT) is a procedure in which a brief application of electric stimulus is used to produce a generalized seizure.” Patients are placed on a padded bed and administered a muscle relaxant to avoid injury during the seizures. Annually, approximately 100,000 are treated using ECT for conditions including severe depression, acute mania, suicidality, and some forms of schizophrenia. The procedure is still the most controversial available to mental health professionals due to “its effectiveness vs. the side effects, the objectivity of ECT experts, and the recent increase in ECT as a quick and easy solution, instead of long-term psychotherapy or hospitalization” (http://www.mentalhealthamerica.net/ect). Its popularity has declined since the 1960s and 1970s.

2.2.3.3. Psychosurgery. Another option to treat mental disorders is to perform brain surgeries. In the past, we have conducted trephining and lobotomies, neither of which are used today. Today's techniques are much more sophisticated and have been used to treat schizophrenia, depression, and some personality and anxiety disorders, though critics cite obvious ethical issues with conducting such surgeries as well as scientific issues.

> For more on psychosurgery, check out this article from Psychology Today:
>
> https://www.psychologytoday.com/articles/199203/psychosurgery

2.2.4. Evaluation of the Model

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

2.3. PSYCHOLOGICAL PERSPECTIVES

Section Learning Objectives

- Describe psychodynamic theory.
- Outline the structure of personality and how it develops over time.
- Describe ways to deal with anxiety.
- Clarify what psychodynamic techniques are used.
- Evaluate the usefulness of psychodynamic theory.
- Describe learning.
- Outline respondent conditioning and the work of Pavlov and Watson.
- Outline operant conditioning and the work of Thorndike and Skinner.
- Outline observational learning/social-learning theory and the work of Bandura.
- Evaluate the usefulness of the behavioral model.

- Define the cognitive model.
- Exemplify the effect of schemas on creating abnormal behavior.
- Exemplify the effect of attributions on creating abnormal behavior.
- Exemplify the effect of maladaptive cognitions on creating abnormal behavior.
- List and describe cognitive therapies.
- Evaluate the usefulness of the cognitive model.
- Describe the humanistic perspective.
- Describe the existential perspective.
- Evaluate the usefulness of the humanistic and existential perspectives.

2.3.1. Psychodynamic Theory

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

cathartic method.

For more on Anna O., please see:

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

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

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

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

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

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

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

- **Repression** – When unacceptable ideas, wishes, desires, or memories are blocked from consciousness such as forgetting a horrific car accident that you caused. Eventually it must be dealt with or else the repressed memory can cause problems later in life.
- **Reaction formation** – When an impulse is repressed and then expressed by its opposite. For example, we are angry with our boss but cannot lash out at him, and so are super friendly instead. Another example is having lustful thoughts of a coworker than you cannot express because you are married, and so you are extremely hateful to this person.
- **Displacement** – When we satisfy an impulse with a different object because focusing on the primary object may get us in trouble. A classic example is taking out your frustrations with your boss on your wife and/or kids when you get home. If we lash out at our boss we could be fired. The substitute target is less dangerous than the primary target.

- **Projection** – When we attribute threatening desires or unacceptable motives to others. An example is when we do not have the skills necessary to complete a task but we blame the other members of our group for being incompetent and unreliable.
- **Sublimation** – When we find a socially acceptable way to express a desire. If we are stressed out or upset, we may go to the gym and box or lift weights. A person who desires to cut things may become a surgeon.
- **Denial** – Sometimes life is so hard all we can do is deny how bad it is. An example is denying a diagnosis of lung cancer given by your doctor.
- **Identification** – This is when we find someone who has found a socially acceptable way to satisfy their unconscious wishes and desires and we model that behavior.
- **Regression** – When we move from a mature behavior to one that is infantile in nature. If your significant other is nagging you, you might regress and place your hands over your ears and say, "La la la la la la la…"
- **Rationalization** – When we offer well thought out reasons for why we did what we did but in reality, these are not the real reasons. Students sometimes rationalize not doing well in a class by stating that they really are not interested in the subject or saying the instructor writes impossible to pass tests.
- **Intellectualization** – When we avoid emotion by focusing on intellectual aspects of a situation such as ignoring the sadness we are feeling after the death of our mother by focusing on planning the funeral.

For more on defense mechanisms, please visit:

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

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

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

Finally, Freud used **dream analysis** to understand a person's inner most wishes. The content of dreams include the person's actual retelling of the dream, called **manifest content**, and the hidden or symbolic

meaning, called **latent content**. In terms of the latter, some symbols are linked to the person specifically while others are common to all people.

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

2.3.2. The Behavioral Model

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

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

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

(discussed as social learning theory).

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

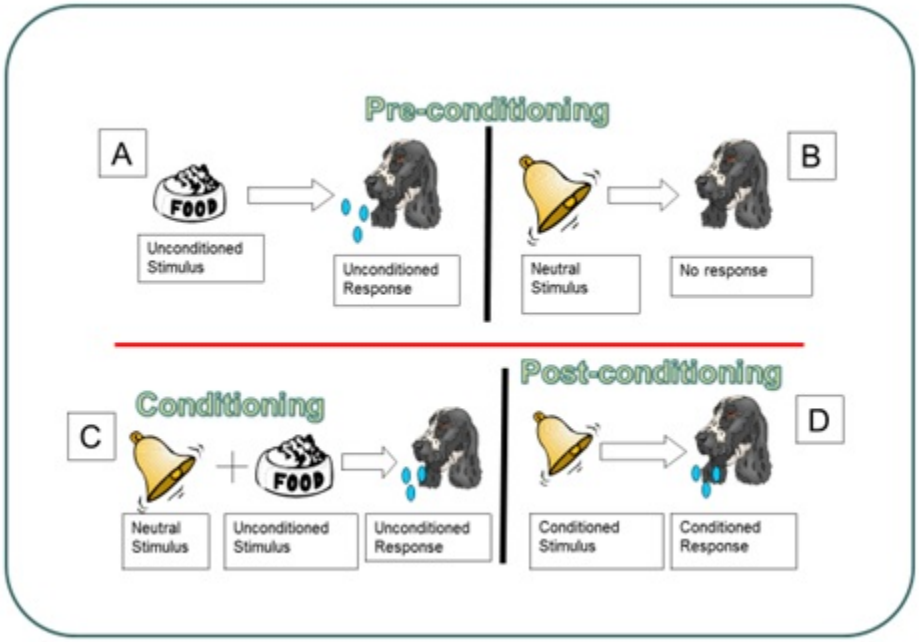
The important thing to understand is that not all behaviors occur due to reinforcement and punishment as operant conditioning says. In the case of respondent conditioning, stimuli exert complete and automatic control over some behaviors. We see this in the case of reflexes. When a doctor strikes your knee with that little hammer, it extends out automatically. You do not have to do anything but watch. Babies will root for a food source if the mother's breast is placed near their mouth. If a nipple is placed in their mouth, they will also automatically suck, as per the sucking reflex. Humans have several of these reflexes, though not as many as other animals due to our more complicated nervous system.

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

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

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

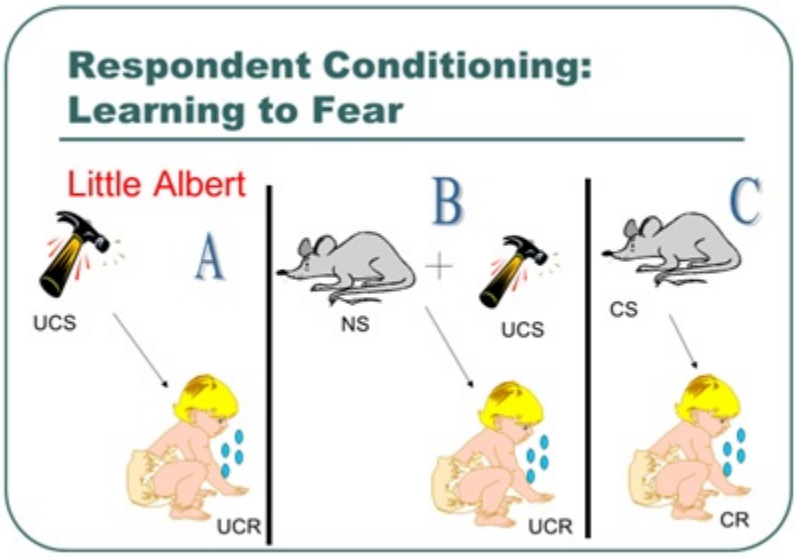
Figure 2.6. Pavlov's Classic Experiment



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

One of the most famous studies in psychology was conducted by Watson and Rayner (1920). Essentially, they wanted to explore "the possibility of conditioning various types of emotional response(s)." The researchers ran a 9-month-old child, known as Little Albert, through a series of trials in which he was exposed to a white rat to which no response was made outside of curiosity (NS–NR not shown). In Panel A of Figure 2.7, we have the naturally occurring response to the stimulus of a loud sound. On later trials, the rat was presented (NS) and followed closely by a loud sound (UCS; Panel B). After several conditioning trials, the child responded with fear to the mere presence of the white rat (Panel C).

Figure 2.7. Learning to Fear



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

Figure 2.8. Unlearning Fears

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

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

Finally, several properties of respondent conditioning should be mentioned:

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

2.3.2.3. Operant conditioning. Influential on the development of Skinner's operant conditioning, Thorndike proposed the **law of effect** (Thorndike, 1905) or the idea that if our behavior produces a favorable consequence, in the future when the same stimulus is present, we will be more likely to make the response again, expecting the same favorable consequence. Likewise, if our action leads to dissatisfaction, then we will not repeat the same behavior in the future. He developed the law of effect thanks to his work with the Puzzle Box. Cats were food deprived the night before the experimental procedure was to occur. The next morning, they were placed in the puzzle box and a small amount of food was placed outside the box close enough to be smelled, but the cat could not reach the food. To get out, a series of switches, buttons, levers, etc. had to be manipulated and once done, the cat could escape the box and eat some of the food. But just some. The cat was then promptly placed back in the box to figure out how to get out again, the food being its reward for doing so. With each subsequent escape and re-insertion into the box, the cat became faster until he/she knew exactly what had to be done to escape. This is called **trial and error learning,** or making a response repeatedly if it leads to success. Thorndike also said that stimulus and responses were connected by the organism and this lead to learning. This approach to learning was called **connectionism**.

Operant conditioning is a type of associate learning which focuses on consequences that follow a response or behavior that we make (anything we do, say, or think/feel) and whether it makes a behavior more or less likely to occur. This should sound much like what you just read about in terms of

Thorndike's work. Skinner talked about **contingencies** or when one thing occurs due to another. Think of it as an If-Then statement. If I do X then Y will happen. For operant conditioning, this means that if I make a behavior, then a specific consequence will follow. The events (response and consequence) are linked in time.

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

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

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

Figure 2.9. Contingencies in Operant Conditioning

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

Let's go through each:

- **Positive Punishment (PP)** – If something bad or aversive is given or added, then the behavior is less likely to occur in the future. If you talk back to your mother and she slaps your mouth, this is a

PP. Your response of talking back led to the consequence of the aversive slap being delivered or given to your face. Ouch!!!

- **Positive Reinforcement (PR)** – If something good is given or added, then the behavior is more likely to occur in the future. If you study hard and earn, or are given, an A on your exam, you will be more likely to study hard in the future. Similarly, your parents may give you money for your stellar performance. Cha Ching!!!
- **Negative Reinforcement (NR)** – This is a tough one for students to comprehend because the terms don't seem to go together and are counterintuitive. But it is really simple and you experience NR all the time. This is when something bad or aversive is taken away or subtracted due to your actions, making it that you will be more likely to make the same behavior in the future when the same stimuli presents itself. For instance, what do you do if you have a headache? You likely answered take Tylenol. If you do this and the headache goes away, you will take Tylenol in the future when you have a headache. NR can either result in current escape behavior or future avoidance behavior. What does this mean? *Escape* occurs when we are presently experiencing an aversive event and want it to end. We make a behavior and if the aversive event, like the headache, goes away, we will repeat the taking of Tylenol in the future. This future action is an *avoidance* event. We might start to feel a headache coming on and run to take Tylenol right away. By doing so we have removed the possibility of the aversive event occurring and this behavior demonstrates that learning has occurred.
- **Negative Punishment (NP)** – This is when something good is taken away or subtracted making a behavior less likely in the future. If you are late to class and your professor deducts 5 points from your final grade (the points are something good and the loss is negative), you will hopefully be on time in all subsequent classes.

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

Secondary or conditioned reinforcers and punishers are not inherently reinforcing or punishing, but must be learned. An example was the attention I received for saying I was Chicken Little. Over time I learned that attention was good. Other examples of secondary reinforcers include praise, a smile, getting money for working or earning good grades, stickers on a board, points, getting to go out dancing, and getting out of an exam if you are doing well in a class. Examples of secondary punishers

include a ticket for speeding, losing television or video game privileges, being ridiculed, or a fee for paying your rent or credit card bill late. Really, the sky is the limit with reinforcers in particular.

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

Figure 2.10. Key Components of Reinforcement Schedules

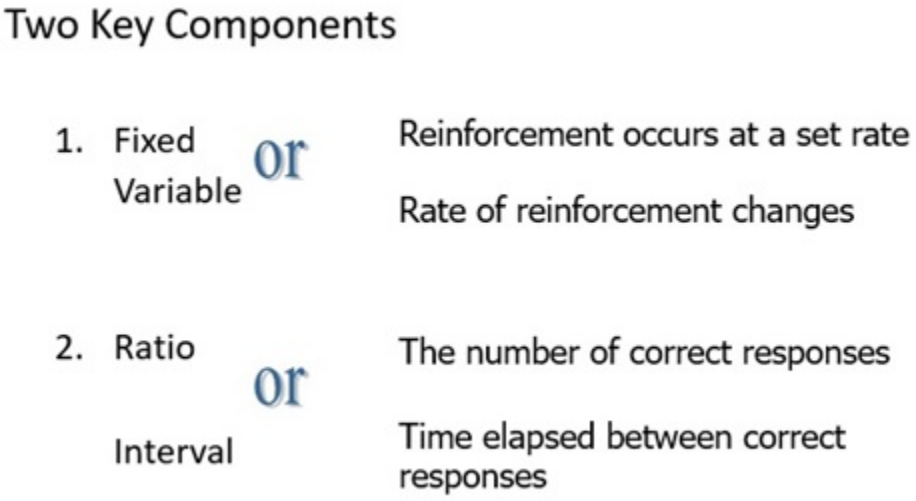


Figure 2.10 shows that that are two main components that make up a reinforcement schedule – when you will reinforce and what is being reinforced. In the case of when, it will be either fixed or at a set rate, or variable and at a rate that changes. In terms of what is being reinforced, we will either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

- **Fixed Ratio schedule (FR)** – With this schedule, we reinforce some set number of responses. For instance, every twenty problems (fixed) a student gets correct (ratio), the teacher gives him an extra credit point. A specific behavior is being reinforced – getting problems correct. Note that if we reinforce each occurrence of the behavior, the definition of continuous reinforcement, we could also describe this as a FR1 schedule. The number indicates how many responses have to be made and in this case, it is one.
- **Variable Ratioschedule (VR)** – We might decide to reinforce some varying number of responses such as if the teacher gives him an extra credit point after finishing between 40 and 50 correct problems. This is useful after the student is obviously learning the material and does not need regular reinforcement. Also, since the schedule changes, the student will keep responding in the

absence of reinforcement.

- **Fixed Interval schedule (FI)** – With a FI schedule, you will reinforce after some set amount of time. Let's say a company wanted to hire someone to sell their product. To attract someone, they could offer to pay them $10 an hour 40 hours a week and give this money every two weeks. Crazy idea but it could work. J Saying the person will be paid *every* indicates fixed, and *two weeks* is time or interval. So, FI.
- **Variable Interval schedule (VI)** – Finally, you could reinforce someone at some changing amount of time. Maybe they receive payment on Friday one week, then three weeks later on Monday, then two days later on Wednesday, then eight days later on Thursday. Etc. This could work, right? Not for a job but maybe we could say we are reinforced on a VI schedule if we are.

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

Do you suppose continuous? Or partial?

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

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

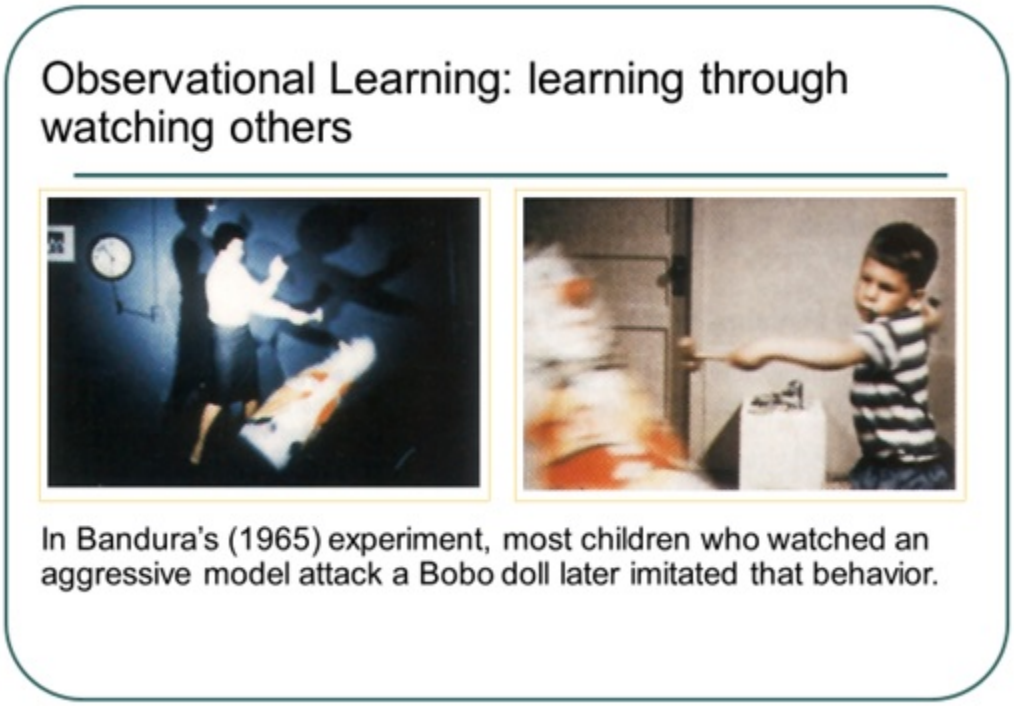
Second, you might have wondered if a person or animal will try to make the response again in the future even though it stopped being reinforced in the past. The answer is yes and one of two outcomes

is possible. First, the response is made and nothing happens. In this case extinction continues. Second, the response is made and a reinforcer is delivered. The response re-emerges. Consider a rat that has been trained to push a lever to receive a food pellet. If we stop delivering the food pellets, in time, the rat will stop pushing the lever. The rat will push the lever again sometime in the future and if food is delivered, the behavior spontaneously recovers. Hence why this phenomenon is called **spontaneous recovery.**

2.3.2.4. Observational learning. There are times when we learn by simply watching others. This is called **observational learning** and is contrasted with **enactive learning**, which is learning by doing. There is no firsthand experience by the learner in observational learning unlike enactive. As you can learn desirable behaviors such as watching how your father bags groceries at the grocery store (I did this and still bag the same way today) you can learn undesirable ones too. If your parents resort to alcohol consumption to deal with the stressors life presents, then you too might do the same. What is critical is what happens to the model in all of these cases. If my father seems genuinely happy and pleased with himself after bagging groceries his way, then I will be more likely to adopt this behavior. If my mother or father consumes alcohol to feel better when things are tough, and it works, then I might do the same. On the other hand, if we see a sibling constantly getting in trouble with the law then we may not model this behavior due to the negative consequences.

Albert Bandura conducted the pivotal research on observational learning and you likely already know all about it. Check out Figure 2.11 to see if you do. In Bandura's experiment, children were first brought into a room to watch a video of an adult playing nicely or aggressively with a Bobo doll. This was a model. Next, the children are placed in a room with a lot of toys in it. In the room is a highly prized toy but they are told they cannot play with it. All other toys are fine and a Bobo doll is in the room. Children who watched the aggressive model behaved aggressively with the Bobo doll while those who saw the nice model, played nice. Both groups were frustrated when deprived of the coveted toy.

Figure 2.11. Bandura's Classic Experiment



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

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

2.3.2.5. Evaluating the behavioral model. Within the context of abnormal behavior or psychopathology, the behavioral perspective is useful because it says that maladaptive behavior occurs when learning goes awry. The good thing is that what is learned can be unlearned or relearned through **behavior modification, which** is the process of changing behavior. To begin, an applied behavior analyst will identify a target behavior, or behavior to be changed, define it, work with the client to develop goals, conduct a functional assessment to understand what the undesirable behavior is, what causes it, and what maintains it. Armed with this knowledge, a plan is developed and consists of

numerous strategies to act on one or all of these elements – antecedent, behavior, and/or consequence. The strategies arise from all three learning models. In terms of operant conditioning, strategies include antecedent manipulations, prompts, punishment procedures, differential reinforcement, habit reversal, shaping, and programming. Flooding and desensitization are typical respondent conditioning procedures used with phobias and modeling arises from social learning theory/observational learning. Watson and Skinner defined behavior as what we do or say, but later, behaviorists added what we think or feel. In terms of the latter, cognitive behavior modification procedures arose after the 1960s and with the rise of cognitive psychology. This led to a cognitive-behavioral perspective which combines concepts from the behavioral and cognitive models, the latter discussed in the next section.

Critics of the behavioral perspective point out that it oversimplifies behavior and often ignores inner determinants of behavior. Behaviorism has also been accused of being mechanistic and seeing people as machines. This criticism would be true of behaviorism's first two stages, though sociobehaviorism steered away from this proposition and even fought against any mechanistic leanings of behaviorists.

The greatest strength or appeal of the behavioral model is that its tenets are easily tested in the laboratory unlike those of the psychodynamic model. Also, a large number of treatment techniques have been developed and proven to be effective over the years. For example, desensitization (Wolpe, 1997) teaches clients to respond calmly to fear producing stimuli. It begins with the individual learning a relaxation technique such as diaphragmatic breathing. Next, a fear hierarchy, or list of feared objects and situations, is constructed in which the individual moves from least to most feared. Finally, the individual either imagines (systematic) or experiences in real life (in-vivo) each object or scenario from the hierarchy and uses the relaxation technique while doing so. This represents individual pairings of feared object or situation and relaxation and so if there are 10 objects/situations in the list, the client will experience ten such pairings and eventually be able to face each without fear. Outside of phobias, desensitization has been shown to be effective in the treatment of Obsessive Compulsive Disorder symptoms (Hakimian and Souza, 2016) and limitedly with the treatment of depression when co-morbid with OCD (Masoumeh and Lancy, 2016).

2.3.3. The Cognitive Model

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

around them. How so?

2.3.3.2. Schemas and cognitive errors. First, consider the topic of **social cognition** or the process of collecting and assessing information about others. So, what do we do with this information? Once *collected* or sensed (**sensation** is the cognitive process of detecting the physical energy given off or emitted by physical objects), the information is sent to the brain. This occurs through the neural impulse. Once in the brain, it is processed and interpreted. This is where *assessing information about others* comes in and involves the cognitive process of **perception,** or adding meaning to raw sensory data. We take the information just detected and use it to assign people to **categories**, or groups. For each category, we have a **schema**, or a set of beliefs and expectations about a group of people, presumed to apply to all members of the group, and based on experience.

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

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

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

specifically, cognitive distortions/maladaptive cognitions can take the following forms:

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

For more on cognitive distortions, check out this website:

http://www.goodtherapy.org/blog/20-cognitive-distortions-and-how-they-affect-your-life-0407154

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

Cognitive restructuring, also called rational restructuring, replaces maladaptive cognitions with more adaptive ones. To do this, the client must be aware of the distressing thoughts, when they occur, and their effect on them. Next, help the client stop thinking these thoughts and replace them with more rational ones. It's a simple strategy, but an important one. Psychology Today published a great article on January 21, 2013 which described 4 ways to change one's thinking through cognitive restructuring. Briefly, these strategies included:

1. Notice when you are having a maladaptive cognition such as making "negative predictions." They suggest you figure out what is the worst thing that could happen and what other outcomes are possible.
2. Track the accuracy of the thought as if you believe focusing on a problem generates a solution. Write down each time you ruminate and then the result. You can generate a percentage of times you ruminated to the number of successful problem-solving strategies you generated.
3. Behaviorally, for example, test your thought by figuring out if you really do not have time to go to

the gym. Record what you do each day and then look at open times of the day. Add them up and see if you make some minor, or major, adjustments to your schedule to allow yourself an hour to get in valuable exercise.

4. Examine the evidence both for and against your thought. If you do not believe you do anything right, list evidence of when you did not do something right and then evidence of when you did. Then write a few balanced statements such as the one the article suggests, "I've made some mistakes that I feel embarrassed about but a lot of the time, I make good choices."

The article also suggested a few non-cognitive restructuring techniques to include mindfulness meditation and self-compassion. For more on this topic, visit:

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

A second major strategy is to use what is called **cognitive coping skills training**. This strategy teaches social skills, communication, and assertiveness through direct instruction, role playing, and modeling. For social skills, identify appropriate social behavior such as making eye contact, saying no to a request, or starting up a conversation with a stranger and determine whether the client is inhibited from making this behavior due to anxiety. For communication, determine if the problem is with speaking, listening, or both and then develop a plan for use in various interpersonal situations. Finally, assertiveness training aids the client in protecting their rights and obtaining what they want from others. Those who are not assertive are often either overly passive and never get what they want or are overly aggressive and only get what they want. Treatment starts with determining situations in which assertiveness is lacking and coming up with a hierarchy of assertiveness opportunities. Least difficult situations are handled first, followed by more difficult situations, all while rehearsing and mastering all the situations present in the hierarchy. For more on these techniques, visit http://cogbtherapy.com/cognitive-behavioral-therapy-exercises/.

Finally, **acceptance techniques** can be used to reduce a client's worry and anxiety. Life involves a degree of uncertainty and at times we need to just accept this. Techniques might include weighing the pros of fighting uncertainty against the cons of doing so. The cons should outweigh the pros and help you to end the struggle and accept what is unknown. Chances are you are already accepting the unknown in some areas of life and identifying these can help you to see why it is helpful in these areas, and how you can also think like this in more difficult areas. Finally, does uncertainty unnecessarily lead to a negative end? We may think so but review of the evidence for and against this statement will show that it does not and reduce how threatening it seems.

2.3.3.6. Evaluating the cognitive model. The cognitive model made up for an obvious deficit in the behavioral model – overlooking the role cognitive processes play in our thoughts, feelings, and behaviors. Right before his death, Skinner (1990) reminded psychologists that the only thing we can truly know and study was the observable. Cognitive processes cannot be empirically and reliably measured and so should be ignored. Is there merit to this view? **Social desirability** states that sometimes participants do not tell us the truth about what they are thinking, feeling or doing (or have

done) because they do not want us to think less of them or to judge them harshly if they are outside the social norm. In other words, they present themselves in a favorable light. If this is true, how can we really know anything about controversial matters? The person's true intentions or thoughts and feelings are not readily available to us, or are covert, and so do not make for good empirical data. Still, cognitive-behavioral therapies have proven their efficacy for the treatment of OCD (McKay et al., 2015); perinatal depression (Sockol, 2015); insomnia (de Bruin et al., 2015), bulimia nervosa (Poulsen et al., 2014), hypochondriasis (Olatunji et al., 2014), and social anxiety disorder (Leichsenring et al., 2014) to name a few. Other examples will be discussed throughout this book.

2.3.4. The Humanistic and Existential Perspectives

2.3.4.1. The humanistic perspective. The humanistic perspective, or third force psychology (psychoanalysis and behaviorism being the other two forces), emerged in the 1960s and 1970s as an alternative viewpoint to the largely deterministic view of personality espoused by psychoanalysis and the view of humans as machines advocated by behaviorism. Key features of the perspective include a belief in human perfectibility, personal fulfillment, valuing self-disclosure, placing feelings over intellect, an emphasis on the present, and hedonism. Its key figures were Abraham Maslow who proposed the hierarchy of needs and Carl Rogers who we will focus on here.

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

2.3.4.2. The existential perspective. This approach stresses the need for people to continually re-create themselves and be self-aware, acknowledges that anxiety is a normal part of life, focuses on free will and self-determination, emphasizes that each person has a unique identity known only through relationships and the search for meaning, and finally, that we develop to our maximum potential. Abnormal behavior arises when we avoid making choices, do not take responsibility, and fail to actualize our full potential. Existential therapy is used to treat substance abuse, "excessive anxiety,

apathy, alienation, nihilism, avoidance, shame, addiction, despair, depression, guilt, anger, rage, resentment, embitterment, purposelessness, psychosis, and violence. It also focuses on life-enhancing experiences like relationships, love, caring, commitment, courage, creativity, power, will, presence, spirituality, individuation, self-actualization, authenticity, acceptance, transcendence, and awe" (For more information, please visit: https://www.psychologytoday.com/therapy-types/existential-therapy).

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

2.4. THE SOCIOCULTURAL MODEL

Section Learning Objectives

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

Outside of biological and psychological factors on mental illness, race, ethnicity, gender, religious orientation, socioeconomic status, sexual orientation, etc. also play a role, and this is the basis of the **sociocultural model**. How so? We will explore a few of these factors in this section.

2.4.1. Socioeconomic Factors

Low socioeconomic status has been linked to higher rates of mental and physical illness (Ng, Muntaner, Chung, & Eaton, 2014) due to persistent concern over unemployment or under-employment, low wages,

lack of health insurance, no savings, and the inability to put food on the table, which then leads to feeling hopeless, helpless, and dependent on others. This situation places considerable stress on an individual and can lead to higher rates of anxiety disorders and depression. Borderline personality disorder has also been found to be higher in people in low income brackets (Tomko et al., 2012) and group differences for personality disorders have been found between African and European Americans (Ryder, Sunohara, and Kirmayer, 2015).

2.4.2. Gender Factors

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

- Rates of eating disorders are higher among women than, men, though both genders are affected. In the case of men, *muscle dysphoria* is of concern and is characterized by extreme concern over being more muscular.
- OCD has an earlier age of onset in girls than boys, with most people being diagnosed by age 19.
- Females are at greater risk for developing an anxiety disorder than men.
- ADHD is more common in males than females, though females are more likely to have inattention issues.
- Boys are more likely to be diagnosed with Autism Spectrum Disorder.
- Depression occurs with greater frequency in women than men.
- Women are more likely to develop PTSD compared to men.
- Rates of SAD (Seasonal Affective Disorder) are four times greater in women than men. Interestingly, younger adults are more likely to develop SAD than older adults.

Consider this...

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

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

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

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

2.4.3. Environmental Factors

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

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

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

2.4.4. Multicultural Factors

Racial, ethnic, and cultural factors are also relevant to understanding the development and course of mental illness. Multicultural psychologists assert that both normal behavior and abnormal behavior need to be understood in relation to the individual's unique culture and the group's value system. Racial and ethnic minorities must contend with prejudice, discrimination, racism, economic hardships, etc. as part of their daily life and this can lead to disordered behavior (Lo & Cheng, 2014; Jones, Cross, & DeFour, 2007; Satcher, 2001), though some research suggests that ethnic identity can buffer against these stressors and protect mental health (Mossakowski, 2003). To address this unique factor, **culture-sensitive therapies** have been developed and include increasing the therapist's awareness of cultural values, hardships, stressors, and/or prejudices faced by their client; the identification of suppressed anger and pain; and raising the client's self-worth (Prochaska & Norcross, 2013). These therapies have proven efficacy for the treatment of depression (Kalibatseva & Leong, 2014) and schizophrenia (Naeem et al., 2015).

2.4.5. Evaluation of the Model

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

Module Recap

In Module 2, we first distinguished uni- and multi-dimensional models of abnormality and made a case that the latter was better to subscribe to. We then discussed biological, psychological, and sociocultural models of abnormality. In terms of the biological model, neurotransmitters, brain structures, hormones, genes, and viral infections were discussed as potential causes of mental illness and three treatment options were given. In terms of psychological perspectives, Freud's psychodynamic theory; learning which includes the work of Watson, Skinner, and Bandura and Rotter; the cognitive model; and the humanistic and existential perspectives were discussed. Finally, the sociocultural model indicated the role of socioeconomic, gender, environmental, and multicultural factors on abnormal behavior.

2nd edition

Module 3 - Classification, Assessment, Diagnosis

Module Overview

Module 3 covers the issues of clinical assessment, diagnosis, and treatment. We will define assessment and then describe key issues such as reliability, validity, standardization, and specific methods that are used. In terms of clinical diagnosis, we will discuss the two main classification systems used around the world – the DSM-5 and ICD-10. Finally, we discuss reasons why people may seek treatment and what to expect when doing so. As this is the last module in Part 1, please make sure you are preparing for your first exam.

Module Outline

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

Module Learning Outcomes

- Describe clinical assessment and methods used in it.
- Clarify how mental health professionals 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?

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

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

3.1.2. Key Concepts in Assessment

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

In addition to reliability, we want to make sure the test measures what it says it measures. This is called **validity**. Let's say a new test is developed to measure symptoms of depression. It is compared against an existing, and proven test, such as the Beck Depression Inventory (BDI). If the new test measures depression, then the scores on it should be highly comparable to the ones obtained by the BDI. This is called *concurrent* or *descriptive validity*. We might even ask if an assessment tool looks valid. If we answer yes, then it has *face* validity, though it should be noted that this is not based on any statistical or evidence-based method of assessing validity. An example would be a personality test that asks about how people behave in certain situations. It therefore seems to measure personality or we have an overall feeling that it measures what we expect it to measure.

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

Finally, we want to make sure that the experience one patient has when taking a test or being assessed is the same as another patient taking the test the same day or on a different day, and with either the same tester or another tester. This is accomplished with the use of clearly laid out rules, norms, and/or

procedures, and is called **standardization**. Equally important is that mental health professionals interpret the results of the testing in the same way or otherwise it will be unclear what the meaning of a specific score is.

3.1.3. Methods of Assessment

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

3.1.3.1. Observation. In Section 1.5.2.1 we talked about two types of observation – naturalistic, or observing the person or animal in their environment, and laboratory, or observing the organism in a more controlled or artificial setting where the experimenter can use sophisticated equipment and videotape the session to examine it at a later time. One-way mirrors can also be used. A limitation of this method is that the process of recording a behavior causes the behavior to change, called **reactivity.** Have you ever noticed someone staring at you while you sat and ate your lunch? If you have, what did you do? Did you change your behavior? Did you become self-conscious? Likely yes and this is an example of reactivity. Another issue is that the behavior that is made in one situation may not be made in other situations, such as your significant other only acting out at the football game and not at home. This is a form of validity is called **cross-sectional validity.** We also need our 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 are able to follow up on specific issues that catch their attention. A **mental status examination** is used to organize the information collected during the interview and systematically evaluates the patient through a series of questions assessing appearance and behavior to include grooming and body posture, thought processes and content to include disorganized speech or thought and false beliefs, mood and affect such that whether the person feels hopeless or elated, intellectual functioning to include speech and memory, and awareness of surroundings to include where the person is and what the day and time are. The exam covers areas not normally part of the interview and allows the mental health professional to determine which areas need to be examined further. The limitation of the interview is that it lacks reliability, especially in the case of the unstructured interview.

3.1.3.3. Psychological tests and inventories. Psychological tests are used to assess the client's personality, social skills, cognitive abilities, emotions, behavioral responses, or interests and can be administered either individually or to groups in paper or oral fashion. **Projective tests c**onsist of simple ambiguous stimuli that can elicit an unlimited number of responses. They include the Rorschach test or

inkblot test and the **Thematic Apperception Test** which asks the individual to write a complete story about each of 20 cards shown to them and give details about what led up to the scene depicted, what the characters are thinking, what they are doing, and what the outcome will be. From the response, the clinician gains perspective on the patient's worries, needs, emotions, conflicts and how the individual connects with one of the people on the card. Another projective test is the *sentence completion test which* asks individuals to finish an incomplete sentence. Examples include 'My mother' …. or 'I hope' …..

Personality inventories ask clients to state whether each item in a long list of statements applies to them, and could ask about feelings, behaviors, or beliefs. Examples include the MMPI or Minnesota Multiphasic Personality Inventory and the NEO-PI-R which is a concise measure of the five major domains of personality – Neuroticism, Extroversion, Openness, Agreeableness, and Conscientiousness. Six facets define each of the five domains and the measure assess emotional, interpersonal, experimental, attitudinal, and motivational styles (Costa & McCrae, 1992). These inventories have the advantage of being easy to administer by either a professional or the individual taking it, are standardized, objectively scored, and are completed either on the computer or through paper and pencil. That said, personality cannot be directly assessed and so you do not ever completely know the individual.

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

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

3.1.3.6. Behavioral assessment. Within the realm of behavior modification and applied behavior analysis, we talk about what is called **behavioral assessment** which simply is the measurement of a target behavior. The **target behavior** is whatever behavior we want to change and it can be in excess or needing to be reduced, or in a deficit state and needing to be increased. During behavioral assessment we learn about the ABCs of behavior in which **Antecedents** are the environmental events or stimuli that trigger a behavior; **Behaviors** are what the person does, says, thinks/feels; and **Consequences** are the outcome of a behavior that either encourages it to be made again in the future

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

3.1.3.7. Intelligence tests. Intelligence testing is used to determine the patient's level of cognitive functioning and consists of a series of tasks asking the patient to use both verbal and nonverbal skills. An example is the *Stanford-Binet Intelligence test* which is used to assess fluid reasoning, knowledge, quantitative reasoning, visual-spatial processing and working memory. Intelligence tests have been criticized for not predicting future behaviors such as achievement and reflecting social or cultural factors/biases and not actual intelligence. Also, can we really assess intelligence through one dimension, or are there multiple dimensions?

3.2. DIAGNOSING AND CLASSIFYING ABNORMAL BEHAVIOR

Section Learning Objectives

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

3.2.1. Clinical Diagnosis and Classification Systems

To begin any type of treatment, the client/patient must be clearly diagnosed with a mental disorder. **Clinical diagnosis** is the process of using assessment data to determine if the pattern of symptoms the person presents with is consistent with the diagnostic criteria for a specific mental disorder set forth in an established classification system such as the DSM-5 or ICD-10 (both will be described shortly). Any diagnosis should have clinical utility meaning it aids the mental health professional determine

prognosis, the treatment plan, and possible outcomes of treatment (APA, 2013). Receiving a diagnosis does not necessarily mean the person requires treatment. This decision is made based upon how severe the symptoms are, level of distress caused by the symptoms, symptom salience such as expressing suicidal ideation, risks and benefits of treatment, disability, and other factors (APA, 2013). Likewise, a patient may not meet full criteria for a diagnosis but require treatment nonetheless.

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

The most widely used classification system in the United States is the *Diagnostic and Statistical Manual of Mental Disorders* currently in its 5th edition and produced by the American Psychiatric Association (APA, 2013). Alternatively, the World Health Organization (WHO) produces the *International Statistical Classification of Diseases and Related Health Problems (ICD)* currently in its 11th edition (although we are currently using the 10th edition and the 11th edition will not go into effect until 2022). We will begin by discussing the DSM and then move to the ICD.

3.2.2. The DSM Classification System

3.2.2.1. A brief history of the DSM. The DSM 5 was published in 2013 and took the place of the DSM IV-TR (TR means Text Revision; published in 2000) but the history of the DSM goes back to 1844 when the American Psychiatric Association published a predecessor of the DSM which was a "statistical classification of institutionalized mental patients" and "…was designed to improve communication about the types of patients cared for in these hospitals" (APA, 2013, p. 6). The DSM evolved through four major editions after World War II into a diagnostic classification system to be used psychiatrists and physicians, but also other mental health professionals. The Herculean task of revising the DSM began in 1999 when the APA embarked upon an evaluation of the strengths and weaknesses of the DSM in coordination with the World Health Organization (WHO) Division of Mental Health, the World Psychiatric Association, and the National Institute of Mental Health (NIMH). This resulted in the

publication of a monograph in 2002 called, *A Research Agenda for DSM-V*. From 2003 to 2008, the APA, WHO, NIMH, the National Institute on Drug Abuse (NIDA), and the National Institute on Alcoholism and Alcohol Abuse (NIAAA) convened 13 international DSM-5 research planning conferences, "to review the world literature in specific diagnostic areas to prepare for revisions in developing both DSM-5 and the International Classification of Disease, 11th Revision (ICD-11)" (APA, 2013).

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

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

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

- Diagnostic Criteria and Descriptors – Diagnostic criteria are the guidelines for making a diagnosis. When the full criteria are met, mental health professionals can add severity and course specifiers to indicate the patient's current presentation. If the full criteria are not met, designators such as "other specified" or "unspecified" can be used. If applicable, an indication of severity (mild, moderate, severe, or extreme), descriptive features, and course (type of remission – partial or full – or recurrent) can be provided with the diagnosis. The final diagnosis is based on the clinical interview, text descriptions, criteria, and clinical judgment.
- Subtypes and Specifiers – *Subtypes* denote "mutually exclusive and jointly exhaustive phenomenological subgroupings within a diagnosis" (APA, 2013). For example, non-rapid eye movement sleep arousal disorders can have either a sleep walking or sleep terror type. Enuresis is nocturnal only, diurnal only, or both. *Specifiers* are not mutually exclusive or jointly exhaustive and so more than one specifier can be given. For instance, binge eating disorder has remission and severity specifiers. Somatic symptom disorder has a specifier for severity, if with predominant pain, and/or if persistent. Again, the fundamental distinction between subtypes and specifiers is that there can be only one subtype but multiple specifiers.
- Principle Diagnosis – A *principal diagnosis* is used when more than one diagnosis is given for an

individual. It is the reason for the admission in an inpatient setting, or the reason for a visit resulting in ambulatory care medical services in outpatient settings. The principal diagnosis is generally the main focus of treatment.

- Provisional Diagnosis – If not enough information is available for a mental health professional to make a definitive diagnosis, but there is a strong presumption that the full criteria will be met with additional information or time, then the *provisional* specifier can be used.

3.2.2.3. DSM-5 disorder categories. The DSM-5 includes the following categories of disorders:

Table 3.1. DSM-5 Classification System of Mental Disorders

Disorder Category	Short Description	Module
Neurodevelopmental disorders	A group of conditions that arise in the developmental period and include intellectual disability, communication disorders, autism spectrum disorder, motor disorders, and ADHD	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, cyclothymic disorder	12
Depressive	Characterized by sad, empty, or irritable mood, as well as somatic and cognitive changes that affect functioning; includes major depressive and persistent depressive disorders	12
Anxiety	Characterized by excessive fear and anxiety and related behavioral disturbances; Includes phobias, separation anxiety, panic attack, generalized anxiety disorder	13
Obsessive-Compulsive	Characterized by obsessions and compulsions and includes OCD, hoarding, and body dysmorphic disorders	14
Trauma- and Stressor-Related	Characterized by exposure to a traumatic or stressful event; PTSD, acute stress disorder, and adjustment disorders	4 & 15
Dissociative	Characterized by a disruption or disturbance in memory, identity, emotion, perception, or behavior; dissociative identity disorder, dissociative amnesia, and depersonalization/derealization disorder	Not covered
Somatic Symptom	Characterized by prominent somatic symptoms to include 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	5 & 16
Elimination	Characterized by the inappropriate elimination of urine or feces; usually first diagnosed in childhood or adolescence	6
Sleep-Wake	Characterized by sleep-wake complaints about the quality, timing, and amount of sleep; includes insomnia, sleep terrors, narcolepsy, and sleep apnea	Not covered
Sexual Dysfunctions	Characterized by sexual difficulties and include premature ejaculation, female orgasmic disorder, and erectile disorder	Not covered
Gender Dysphoria	Characterized by distress associated with the incongruity between one’s experienced or expressed gender and the gender assigned at birth	Not covered
Disruptive, Impulse-Control, Conduct	Characterized by problems in self-control of emotions and behavior and involve the violation of the rights of others and cause the individual to be in violation of societal norms; Includes oppositional defiant disorder, antisocial personality disorder, kleptomania, etc.	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 and the NCD has not been present since birth or early in life	Not covered
Personality	Characterized by a pattern of stable traits which are inflexible, pervasive, and leads to distress or impairment	Not covered

Paraphilic	Characterized by recurrent and intense sexual fantasies that can cause harm to the individual or others; includes exhibitionism, voyeurism, and sexual sadism	Not covered

3.2.3. The ICD-10

In 1893, the International Statistical Institute adopted the International List of Causes of Death which was the first international classification edition. The World Health Organization was entrusted with the development of the ICD in 1948 and published the 6th version (ICD-6). The ICD-10 was endorsed in May 1990 by the 43rd World Health Assembly. The ICD-11 has been created and will go into effect in 2022. The WHO states:

ICD is the foundation for the identification of health trends and statistics globally, and the international standard for reporting diseases and health conditions. It is the diagnostic classification standard for all clinical and research purposes. ICD defines the universe of diseases, disorders, injuries and other related health conditions, listed in a comprehensive, hierarchical fashion that allows for:
– easy storage, retrieval and analysis of health information for evidenced-based decision-making;
– sharing and comparing health information between hospitals, regions, settings and countries; and
– data comparisons in the same location across different time periods.

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

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

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

3.2.4. Harmonization of DSM-5 and ICD-10

According to the DSM-5, there is an effort to harmonize the two classification systems so that there can be more accurate collection of national health statistics and design of clinical trials, increased ability to replicate scientific findings globally, and to rectify the issue of DSM-V and ICD-10 diagnoses not agreeing (APA, 2013).

3.3. TREATMENT OF MENTAL DISORDERS – AN OVERVIEW

Section Learning Objectives

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

3.3.1. Seeking Treatment

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

For more on this article, please visit:

https://www.psychologytoday.com/blog/where-science-meets-the-steps/201303/5-signs-its-time-seek-therapy

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

3.3.1.3. So what exactly is psychotherapy? APA states (article quoted below can be found at: https://www.apa.org/helpcenter/understanding-psychotherapy) that in **psychotherapy**, "psychologists apply scientifically validated procedures to help people develop healthier, more effective habits." Several different approaches can be utilized to include behavior, cognitive and cognitive-behavior, humanistic-experiential, psychodynamic, couples and family, and biological therapies/treatments. We will discuss each of these in Section 3.3.2.

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

So how do you find a psychotherapist? Several strategies may prove fruitful. You could ask family and friends, your primary care physician (PCP), look online, consult an area community mental health center, your local university's psychology department, state psychological association, or use APA's Psychologist Locator Service (https://locator.apa.org/?_ga=2.160567293.1305482682.1516057794-1001575750.1501611950). Once you find a list of psychologists or other practitioners, choose the right one for you by determining if you plan on attending alone or with family, what you wish to get out of your time with a psychotherapist, how much your insurance company pays for and if you have to pay out of pocket how much you can afford, if the provider is networked with your employee assistance program (a common benefit employers offer), 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, your therapist and you will work to develop a full history which could take several visits. From this, a treatment plan will be developed. “This collaborative goal-setting is important, because both of you need to be invested in achieving your goals. Your psychologist may write down the goals and read them back to you, so you’re both clear about what you’ll be working on. Some psychologists even create a treatment contract that lays out the purpose of treatment, its expected duration and goals, with both the individual’s and psychologist’s responsibilities outlined.”

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

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

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

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 discussed clinical assessment, diagnosis, and treatment. In terms of assessment, we covered key concepts such as reliability, validity, and standardization; and discussed methods of assessment such as observation, the clinical interview, psychological tests, personality inventories, neurological tests, the physical examination, behavioral assessment, and intelligence tests. In terms of diagnosis, we discussed the classification systems of the DSM-5 and ICD-10. For treatment, we discussed reasons why someone may seek treatment, self-treatment, psychotherapy, the client-centered relationship, and how well psychotherapy works. We discussed some of the specific therapies in Module 2 but will cover others throughout this book and in terms of the disorders they are used to treat.

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II

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

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 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 presents.
- 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 is assessed, diagnosed, and treated.

4.1. CLINICAL PRESENTATION

Section Learning Objectives

- Describe the presentation and associated features of Disinhibited Social Engagement Disorder (DSED).
- Describe the presentation and associated features of Reactive Attachment Disorder (RAD).
- Describe attachment and the impact attachment disruption has on the development of DSED and RAD.

Although we are covering these disorders under the "early childhood disorders" these disorders are technically considered trauma-related disorders. The reason we are covering these under the early childhood disorder section is because these disorders are related to clinical presentations that emerge due to traumatic experiences in early childhood. We do not see these disorders spontaneously present in late childhood or adulthood. The reason for this is because the trauma that impacts these disorders occurs in infancy to early childhood.

4.1.1. Clinical Presentation of Disinhibited Social Engagement Disorder (DSED)

This disorder can be thought of in two components: (1) how the child presents and (2) the history of the child.

Let's start with "how the child presents." These children, opposite to children with RAD (described below), tend to be overly social and interact with complete strangers. This child may walk up to someone in a store that 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.

Now for "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 try to socialize the child and interact with them regularly. Perhaps the child is not cared for appropriately regarding basic needs of food, hygiene, and shelter. Perhaps the child is in a setting in which there are not enough

caregivers to care for the child consistently (e.g., an orphanage in which there are only a few caretakers with several infants), or they have not had one consistent caregiver (e.g., moved from one foster care setting to another regularly). These situations interrupt the attachment and security of a developing child.

This disorder is present, as I mentioned, early in life and, as such, a child must evidence these symptoms prior to age 5 to be diagnosed with this disorder.

4.1.2. Clinical Presentation of Reactive Attachment Disorder (RAD)

This disorder can also be thought of in two components: (1) how the child presents and (2) the history of the child.

Let's start with "how the child presents." Children with this disorder typically present as detached from others. They do not seek comfort from caregivers, don't respond to physical touch or praise in a typically way, and generally, although they can't effectively self-regulate, reject or ignore others attempts to help them regulate their emotions (e.g., comfort or soothing). These children typically have low levels of expressed emotions, particularly positive emotion. They may be irritable and experience significant negative emotionality at times.

Let's move into "the history of the child." The "history" is the same as described for DSED.

This disorder is present, as I mentioned, early in life and, as such, a child must evidence these symptoms prior to age 5 to be diagnosed with this disorder.

As you may realize now, both disorders require the same history; however, the presentation of the child resulting from that history may vary, thus, impacting which disorder they may be diagnosed with (RAD versus DSED).

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 and we can begin to recognize attachment 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, concerns of attachment are warranted, potentially. A child can form attachments with more than one caregiver; however, we have a "threshold" or "maximum" number of individuals we 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 attachment appropriately (Zeanah, et al., 2016). However, if they have tried to attach to multiple individuals, perhaps their ability to fully attach may be somewhat compromised.

Mary Salter Ainsworth created the Strange Situation Procedure in 1969 to assess attachment. The procedure involves having the child experience times of separating and reuniting with the child's mother (originally with their mother, although this system would now be done with the primary caregiver, may that be a mother or not). Strangers are also introduced in this procedure. During all of the various trials and interactions, the infant's reaction is monitored and recorded (Krapp, 2005).

The Strange Situation Procedure involves having the mother, baby, and observer first enter a room, then after a short time (30 seconds), the observer leaves the room. This allows the mother and baby to explore the room together. Next, a stranger enters the room quietly, interacts with the mother, and approaches the infant as the mother leaves. The mother will be absent and the stranger will stay with the infant for three minutes. After three minutes, the mother comes back, engages with the child, and the stranger leaves. Next, the mother says goodbye to the child and leaves the room (again, for 3 minutes). At this point the baby is alone in the room but is being observed for behaviors (and safety) through a one-way mirror. The baby is alone for three minutes before the stranger (but not the mother) enters the room. After another three minutes, the mother returns to the room and the stranger leaves. During each of these variations, the infant's behaviors are being monitored and recorded. Observers will note things such as if the child moves close to their mother, if they cling to their mother, if they ignore either their mother or the stranger, if they avoid or reject contact from an adult, if they look around for their mother, or if they vocalize or interact across the room with their mother 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 considered to be a risk factor for later psychopathology and negative trajectories whereas secure attachment is considered to be a protective factor. During the Strange Situation Procedure, a child that is securely attached is likely to explore a room while the mother is present, feeling confident that their mother 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 and a child that is difficult to sooth. Disorganized attachment typically involves patterns of interactions that are not fully described above or are significantly inconsistent. Generally speaking attachment is considered either secure, avoidant, resistant, or disorganized (Zeanah, et al., 2016).

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

The prevalence of DSED is largely unknown (APA, 2013) and considered to be extremely rare. However, rates are noted to be up to 20% in high populations risk (e.g., foster care and institutionalized settings; APA, 2013).

DSED must be differentiated from ADHD. This is because children with DSED may present as socially impulsive and mimic impulsivity related to ADHD. However, children with DSED only, while they may impulsively interact with strangers, don't display overall levels of impulsivity and hyperactivity (APA, 2013).

Additionally, the medical condition of Williams syndrome, which is caused by a partial chromosomal deletion, may mimic DSED symptoms (Zeanah, et al., 2016). These children struggle with socially discriminating 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, DSED 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.

Cognitive and language delays as well as ADHD may be commonly comorbid with DSED (APA, 2013).

4.2.2 Reactive Attachment Disorder

The prevalence of RAD is largely unknown (APA, 2013) and considered to be extremely rare. However, higher rates are noted in particular populations. For example, in severely neglected children,

prevalence is noted to be approximately 10% (APA, 2013) and in young, institutionalized children, up to 40% (Zeanah, r, Boris, and AACP CQI, 2016).

Due to the significant deficit in emotional-social reciprocity, Autism must be differentiated from RAD. The child's history (neglect versus no neglect) and the presence or absence of restricted or repetitive behaviors/interests help differentiate the two disorders (APA, 2013). For example, if a child has no history of severe neglect or abuse, the diagnosis of Autism is more likely than RAD. If the child does not have restricted/repetitive behaviors/interests, the child is not likely to be diagnosed with Autism, and as such, deficits in social/emotional reciprocity is more likely to be explained by RAD (particularly if there is a significant neglect/abuse history).

Intellectual disabilities may be highly comorbid with RAD (APA, 2103). Clinician's must be careful to tease apart cognitive deficits from attachment, as attachment is unrelated to intellectual functioning. As such, differentiating between the two disorders is important to arrive a correct diagnosis, and also account for any comorbidity with ID and RAD.

4.3. ETIOLOGY

Section Learning Objectives

- Describe enviromental causes of Disinhibited Social Engagement Disorder and Reactive Attachment Disorder

4.3.1. Environmental

4.3.1.1. General. For both RAD and DSED, 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 in attachment results in behavioral patterns that are troublesome and problematic not only in the short term, but long term, particularly if no interventions are implemented.

4.3.1.2. DSED specific information. The quality of caregiving has been noted to moderate the progression of DSED (APA, 2013). Essentially, severely impaired caregiving may lead to more DSED symptoms but improved caregiving may lead to less DSED symptoms. Moreover, some genetic vulnerabilities (we won't get into the specifics, here, but the genetic vulnerabilities involve 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 DSED that a child in the same setting without the genetic vulnerability.

4.4. ASSESSMENT AND TREATMENT

Section Learning Objectives

- Describe assessment tools commonly used
- Describe treatment options for Disinhibited Social Engagement Disorder and Reactive Attachment Disorder

4.4.1. Assessment

In general, assessment is going to include a thorough interview with a caregiver. This caregiver may be the biological parent of the child; however, it may be a foster parent, social worker, or other relative. An understanding of not only the child's life history, such as trauma, access to care and nurturing, etc., but also developmental progress and social interacts, 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 key.

Observations may be largely informal. For example, a psychologist may note several things as they happen to occur. For example, they may note 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 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 commonly known and well validated observation procedure to assess attachment is the Strange Situate Procedure, this is most often done in a research setting, and is less commonly used in clinical settings. However, Zeanah, et al. (2016) reviewed parameters that may be helpful. Below is a table, copied directly from their article that outlines a brief and informal procedure that can be used. This

procedure is used to obtain qualitative information and does not result in an objective score, necessarily.

Table 4.1 Variation of Attachment Observation

Episode	Duration (minutes)	Action	Observation
1	5	Clinician observes parent-child "free play."	Note especially familiarity, comfort, and warmth in child as he/she interacts with attachment figure.
2	3	Clinician talks with, then approaches, then attempts to engage child in play.	Most young children exhibit some reticence, especially initially, about engaging with an unfamiliar adult.
3	3	Clinician picks up child and shows him/her a picture on the wall or looks out window with child.	This increases the stress for the child. Again, note the child's comfort and familiarity with this stranger.
4	3	Caregiver picks up child and shows him/her a picture on the wall or looks out window with child.	In contrast to stranger pick-up, child should feel obviously more comfortable during this activity.
4a (optional)	1	Child is placed between caregiver and stranger and remote control (e.g., scary/exciting) toy is introduced.	Child should seek comfort preferentially from parent. If interested rather than frightened, child should share positive affect with parent.
5	3	Clinician leaves the room.	This separation should not elicit much of a reaction in the child, as the clinician is a stranger.
6	1	Clinician returns.	Similarly, the child should not be much affected by the stranger's return.
7	3	Caregiver leaves the room.	Child should definitely take notice of caregiver's departure, although not necessarily exhibit obvious distress. If the child is distressed, the clinician should be of little comfort to the child.
8	1	Caregiver returns.	Child's reunion behavior with caregiver should be congruent with separation behavior. That is, distressed children should seek comfort, and nondistressed children should re-engage positively with caregiver, by introducing him or her to the toy or activity or talking with him or her about what occurred during the separation.

Note: The general rationale for the procedure is to compare the child's behavior with the putative attachment figure to the child's behavior with the stranger, especially with regard to degree of comfort, showing warmth and affection, reliance for help, cooperation, and seeking comfort when afraid or distressed.

Note. Table is directly derived from Zeanah, et. al., , 2016, p. 998)

Ultimately, the child should prefer the caregiver and separation from the caregiver should be mildly upsetting. The child should seek support, comfort, and protection from the caregiver (Zeanah, et al., CQI, 2016).

As a final piece of assessment, the actual safety and appropriateness of the child's current care and living situation and caregiver relationship should also be assessed according to (Zeanah, et al., 2016).

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, before getting too far into the discussion of treatment, it should be noted that little research directly investigates the impact of therapies on these disorders.

A primary 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 to attach, the primary goal of the intervention, the caregiver has to be emotionally ready to do so (Zeanah, et al., 2016). What I mean by this is, 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 have the caregiver talk about their relationship with the child so that the therapist can begin to 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 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 (CPP – discussed later in the semester) or Attachment and Biobehavioral Catch Up videotape review and clinician shape 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).

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 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, her 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 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 RAD and DSED. We discussed the various behaviors and symptoms of DSED, and RAD and how they relate to the various presentations. We also learned about attachment styles and how they related to RAD and DSED. We then discussed the prevalence of these disorders, frequently comorbid disorders, and the etiology of RAD and DSED. We ended on a discussion of how RAD and DSED is assessed and treated. In our next module we will discuss another category of disorders that appear in infancy and early childhood – feeding disorders.

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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, and Avoidant/Restrictive Food Intake disorders. 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

- 5.1. Clinical Presentation and DSM-5 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 Disorders present.
- Describe the prevalence of these feeding-related disorders.
- Describe the etiology of these disorders.
- Describe how these disorders are assessed, diagnosed, and treated.

5.1. CLINICAL PRESENTATION

Section Learning Objectives

- 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.

Before we jump into this module, please keep in mind that *feeding* and *eating* disorders used to be considered two different areas of concern in the previous edition of the DSM. Theoretically, 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) An eating disorder is related more to eating habits, rather than feeding concerns, whether it be under or over eating, and generally is considered to occur more frequently in typically developing (meaning no developmental or cognitive delays) individuals from childhood/adolescent to adulthood. Although on the surface, these two things seem notably different, conceptually, the DSM-5 has combined both feeding and eating disorders into one category (APA, 2013). However, given that they have some differences, for the purposes of this class, to help facilitate easy learning, we will be splitting the disorders up into two chapters. We are going to discuss Pica, Rumination Disorder, and Avoidant/Restrictive Food Intake Disorder now (feeding-related disorders), and later in the semester, we will discuss Anorexia, Bulimia, and Binge Eating Disorders (eating-related disorders).

5.1.1. Pica

In simple terms, Pica is the act of eating items that are not food, on a regular or recurring basis. For example, eating grass, chalk, dirt, paper, etc. on a recurring basis would be characteristic of Pica. 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 acts of eating nonfood items are 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, 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, one would not be diagnosed with Pica. Also, if the behavior is culturally-expected, then Pica would not be diagnosed. For example, ingesting a particular grass for medicinal or spiritual purposes would not lead to a diagnosis of Pica APA, 2013).

5.1.2. Rumination Disorder

This is 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). An individual may just regurgitate the food and dispose of it by spitting, or they may also re-chew or swallow the food after regurgitation APA, 2013).

5.1.3. Avoidant/Restrictive Food Intake Disorder (ARFID)

This disorder is new to the DSM 5. In the previous edition of the DSM, criteria that was more limited and stringent outlined Feeding Disorder of Infancy. However, the literature and field began to recognize the importance to expand the criteria and conceptualization of the phenomenon (Norris & Katzzman, 2015). As such, ARFID was created. Because it is new to the DSM-5, like the other new disorders, we are limited in our knowledge about the disorder. ARFID is characterized by simply a low interest in eating/feeding which ultimately leads to a deficit in obtaining appropriate calories and nutrients. An individual may begin to lose a large amount of weight, or evidence a failure to gain weight for children. They may also require alternative feeding methods such as tubal feeding or supplementation of nutrients with oral supplements. It should be noted that avoidance or restriction of food, in this disorder, has no relation to a desire to lose weight, become thin, or issue with body image. This is completely unrelated to the concerns we may see, for example, in anorexia (APA, 2013).

5.2. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe the prevalence and course of Pica, Rumination Disorder, and Avoidant/Restrictive Food Intake Disorders.
- Describe comorbid disorders of Pica, Rumination Disorder, and Avoidant/Restrictive Food Intake Disorders

5.2.1. Pica

Specific prevalence rates of Pica are somewhat unknown (APA, 2013; Murray, Thomas, Hinz, Munsch, & Hilbert, 2018). Pica tends to present more severely in individuals with an intellectual deficit (APA, 2013). 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 (Murray et al., 2018).

Autism and Intellectual Disability are the two most common comorbid diagnoses. Obsessive-compulsive disorders related to hair pulling or skin picking or Obsessive-Compulsive Disorder (OCD) may be comorbid (APA, 2013).

5.2.2. Rumination

Similar to Pica, prevalence rates of Rumination Disorder are unknown (APA, 2013; Murray et al., 2018). However, it tends to occur more frequently in individuals with an intellectual disability (Olden, 2001). Information on comorbidity specific to Rumination Disorder is limited. Ensuring that regurgitation is not occurring due to a specific medical or gastro-related condition (e.g., Sandifer syndrome, gastroparesis) is imperative (APA, 2013). Medical doctors will often need to screen for these conditions before Rumination Disorder is diagnosed.

5.2.3 Avoidant/Restrictive Food Intake Disorder

Again, prevalence rates are largely unknown. However, it does seem that this disorder is more common in children with Autism. It is likely this disorder occurs more commonly in Autism due to sensory concerns and rigidity that leads to children having very specific preferences on foods. For example, children may actively refuse to eat many foods due to texture aversions. Keeping in mind that ARFID is comorbid with Autism, it may also lead to a higher prevalence of ARFID for males (as Autism is more common in males). However, when considering the disorder in populations without Autism, the prevalence appears to be equally common in males and females (APA, 2013). Again, ruling out medical conditions, is necessary.

Some structural atypicality may also make feeding hard and lead to ARFID (APA, 213). As mentioned, Autism is a common comorbid disorder. Other neurodevelopmental disorders may be highly comorbid such as ADHD and Intellectual Disability. Anxiety is also often comorbid with this disorder, as well as OCD (APA, 2013).

5.3. ETIOLOGY

Section Learning Objectives

- Describe various biological, behavioral, and sociocultural causes of feeding-related 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 may also be a risk factor for the development of Pica (APA, 2013).

5.3.2. Rumination

Information related to Rumination Disorder etiology is limited. However, similar to Pica, neglectful caregiving, difficult parent-child relationship, and early life stress may be important risk factors to consider (APA, 2018).

5.3.3 Avoidant/Restrictive Food Intake Disorder

Various factors may contribute to the development of ARFID. Parent-child interactions may be particularly important to consider (APA, 2013). 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 and neglect/abuse may foster the above interactions as well.

Children with ARFID 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).

5.4. ASSESSMENT AND TREATMENT

Section Learning Objectives

- Describe assessment methods
- Examine treatment options

5.4.1. General Assessment of Feeding-related Concerns

Often times, assessment begins with parent/caregiver reports during interviews. A psychologist will likely do 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. Moreover, 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 screens 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 a parent-child feeding session.

5.4.2. Therapy for Feeding-related Concerns

Similar to assessment, treatment frequently, but not always, occurs in a feeding clinic. Because these disorders have higher frequencies of occurrences in the developmentally delayed, although treatment may occur in a feeding clinic, it is also very likely to occur in other settings. Moreover, because ARFID is highly comorbid with Autism due to sensory concerns, feeding therapy may also be incorporated into a child's ABA goals (this will be discussed in the chapter on Autism), 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, supplementation of iron may be attempted to increase the individual's iron levels, and thus, reduce Pica behaviors. Moreover, 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). If you have taken Self-Control, you learned all about this concept. As a brief overview, **differential reinforcement** is when we attempt to get rid of undesirable or problem behaviors (in this case, Pica) by using positive reinforcement (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 the discarding of 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. 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 to behaviors to see what affects the occurrence or nonoccurrence of a desired or problem behavior. Through functional behavioral assessments, research appears to indicate that rumination is automatically reinforced by a 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 desired by the individual, this is considered *reinforcement*. Because the reinforcement (the 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 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 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 to specifically have parents learn how to implement these behavioral strategies around meals shows promising improvement for the child (Najdowski, Wallace, Reagon, Penrod, Higbee, & Tarbox, 2010; Sharp, Burrell, & Jaquess, 2014). Essentially, 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. Again, 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 (described in the Chapter on Autism).

It should be noted that we outlined that child-parent interactions may also strongly impact the development and maintenance of ARFID. As such, carefully exploring the feeding patterns during meal time between the parent and child is imperative. If irritability or frustration are perpetuating 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.

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

Module Recap

In Module 5, we discussed feeding-related disorders including Pica, Rumination Disorder, and Avoidant/Restrictive Food Intake Disorder. We discussed how these disorders present themselves. 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 chapter, we will continue to discuss disorders of infancy and early childhood by reviewing elimination disorders.

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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 disorders. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 6.1. Clinical Presentation and DSM-5 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 is assessed, diagnosed, and treated.

6.1. CLINICAL PRESENTATION

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, essentially, urinary incontinence (i.e., the inability to remain absent of urinary accidents). It is developmentally appropriate to not be able to remain dry during the day and/or night for a period of time in young children. However, it is expected that children will learn to remain dry during the day (first) and eventually remain dry and accident free at nighttime. If this is not achieved by the age of five, it is considered to be of clinical concern and likely indicative of enuresis. Occasional accidents are okay and expected to a certain degree, but when accidents occur regularly, then it becomes clinically concerning. Because daytime and nighttime dryness may be indicative of different concerns, psychologists can specify by using the terms 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) when describing enuresis (APA, 2013).

6.1.2. Encopresis

Encopresis is, essentially, incontinence with bowel movements (i.e., the inability to remain absent of bowel accidents). Similar to urinary incontinence, it is developmentally appropriate to not be toilet trained for a period of time. However, it is expected that children will learn to control their bowel movements and use the toilet when defecating, eventually. If this is not achieved by the age of four, it is considered to be of clinical concern and likely indicative of encopresis. Occasional accidents are okay and expected to a certain degree, although, the allowance for accidents is less liberal than with urinary accidents. Essentially, we expect a child’s control of bowel movements to be better and without accident more than we do for urinary training. Difficulties with bowel movements may be related or unrelated to issues of constipation and/or overflow (meaning fecal seepage). As such, psychologists will specify if encopresis is with or without constipation and overflow (APA, 2013).

6.2. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe the prevalence and course of Encopresis and Enuresis.
- Describe comorbid disorders of Encopresis and Enuresis.

6.2.1. Enuresis

Enuresis is slightly more common than encopresis. It is actually fairly common in 5-year-olds with a base rate of anywhere between 5 to 10% (APA, 2013). As children get older, the more infrequent this occurs. For example, in 10-year-olds, the prevalence drops to somewhere between 3 and 5% with prevalence dropping to 1% in 15-year-olds Nocturnal enuresis occur more in males whereas diurnal enuresis is more frequent in females (APA, 2013).

Specific mental health conditions are not commonly comorbid with enuresis. Urinary tract infections occur in a higher frequency in children with enuresis than in children without enuresis. Children with significant delays in development are also more likely to experience enuresis than children without delays.

6.2.2. Encopresis

Encopresis is fairly infrequent, occurring in only approximately 1% of 5-year-olds (APA, 2013). This condition is slightly more common in males than in females (APA, 2013). Similar to encopresis, urinary tract infections are common.

6.3. ETIOLOGY

Section Learning Objectives

- Describe 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 accounted for (APA, 2013). Additionally, some medication can make it difficult to remain continent (APA, 2013). As such, medical examinations to rule these things out is important before diagnosing enuresis. There also appears to be a heritability factor in enuresis with children being anywhere from 3.6 to 10.1 times higher risk for developing enuresis if their parents experienced enuresis themselves in childhood (von Gontard et al., 2011; APA, 2013).

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, again, a medical examination and/or consideration for particular medical conditions must be considered. This is because encopresis is not diagnosed if incontinence is explained by a medical condition rather than a psychological.

6.3.2. Environmental/Psychological

Ineffective toilet training procedures, or toilet-training procedures that occur later than necessary, may contribute to enuresis (APA, 2013) 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 as such avoid doing so. This perpetuates concerns with encopresis. Thus, they may avoid defecating for so long until they physically can no longer do so, resulting in accidents.

6.4. ASSESSMENT AND TREATMENT

Section Learning Objectives

- Examine 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 these elimination concerns. Also, utilizing a voiding diary may help parents recognize when and how frequently accidents

are occurring. 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 which has a few examples. The alarm will either vibrate or sound (or both) when it becomes wet. This awakens the child to prompt them to go to the restroom. 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 months (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. Moreover, 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. Of importance to note – 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).

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 elimination disorders. We then discussed the prevalence of elimination disorders and examined 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 chapter starts our discussion of developmental delays and motor disorders. We will start by reviewing intellectual disabilities and learning disorders.

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III

PART III. DEVELOPMENTAL AND MOTOR-RELATED DISORDERS

Part III. Developmental and Motor-related Disorders

Module 7 - Intellectual Disability Intellectual Developmental Disorder (IDIDD) & Learning Disorders

Module Overview

In Module 7, we will discuss matters related to intellectual disability intellectual developmental and learning disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include intellectual disability and learning disorders. 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 ID and Learning Disorders (LDs) present.
- Describe the prevalence of ID and LD.
- Describe the etiology of ID and LD.
- Describe how ID and LDs are assessed, diagnosed, and treated.

7.1. CLINICAL PRESENTATION

Section Learning Objectives

- Describe the presentation and associated features of ID.
- Describe the presentation and associated features of LDs.
- Clarify the differences and similarities between ID and LD.

7.1.1. 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. Due to significant stigma and social misuse of the term, when the DSM 5 was published, the term changed from Mental Retardation to Intellectual Disability (also described as an Intellectual Developmental Disorder). While the terms Intellectual Disability and Intellectual Developmental Disorder are considered interchangeable, we will use the term Intellectual Disability (ID) for the purposes of this class. When considering this disorder there are two primary areas of major deficits – cognitive functioning and adaptive functioning (APA, 2013).

7.1.1.1. Cognitive functioning. Cognition or intellectual functioning refers, in a general sense, to our ability to problem solve, understand and analyze complex material, absorb information from our environment, and reason. An individual with ID has a significant deficit in this area. Cognitive functioning is most often measured by the use of an intelligence test (more on this later in this chapter). Generally speaking, an IQ score under 70 – 75 indicates a severe deficit in cognitive functioning, although there is some flexibility within this particular criteria component.

7.1.1.2. Adaptive functioning. Adaptive skills are essentially skills that help us navigate our daily lives successfully. Our ability to understand safety signs in our environment, make appointments, interact with others, complete hygiene routines, etc. are examples of adaptive functioning. Essentially, these are the skills that one would ultimately need to live independently. Individuals with ID typically have adaptive skills that are far below what would be expected given their chronological age. This is typically measured by a standardized scale (more on this later, as well).

When both cognitive and adaptive functioning is delayed, a concern of ID is high. ID is also categorized into different severities, based on the level of delays related to adaptive functioning. Essentially, the more support someone needs, the more severe the ID diagnosis. Severity ranges from Mild (least severe), Moderate, Severe, and Profound (most severe; APA, 2013).

ID is present in the early neurodevelopmental period. As such, it is most frequently diagnosed in children. ID is not something one would “acquire” in adulthood. If an individual experiences cognitive and adaptive function decline in later years, this is not considered ID (which is a neurodevelopmental disorder), but is considered to be more likely a neurocognitive disorder that may be due to a number of things (e.g., traumatic brain injury, dementia). As such, although an individual can go undiagnosed until adulthood, and then as an adult be diagnosed with ID, there must be significant and undoubtable

evidence of cognitive delay and adaptive functioning delay in the early developmental time period. Otherwise, an adult would not be diagnosed with ID.

7.1.2. Specific Learning Disabilities

A learning disorder is characterized by the inability or difficulty processing academic or functional information in our environment (APA, 2013). Essentially, despite an ability to cognitively achieve similar to peers, an individual is delayed in learning in a particular area. More specifically, academic tasks are challenging within one or more areas, which results in significant academic impairment (APA, 2013). Historically, we diagnosed LDs when there was a significant discrepancy between an individual's cognitive/intellectual ability (as measured by an intelligence test) and their academic achievement (as measured by a standardized achievement test) as this was required by the DSM-IV-TR criteria. This method is referred to as the *discrepancy model.* While many still do this, and there is nothing in the DSM 5 that disallows this practice, the DSM 5 criteria was rewritten to allow for more flexibility in this. Ultimately, a discrepancy between one's IQ and academic achievement is no longer required; however, there must be specific data that indicates an individual is preforming significantly below what would be expected given their age.

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

When considering LDs, there are three specific areas that are considered: reading, mathematics, and written expression. For example, a professional would diagnose an individual a *specific learning disorder with impairment in reading*. An individual may have a diagnosis of only one LD, or multiple LDs.

Reading – This essentially relates to an individual having difficulty in reading, may that be in comprehending material, reading fluently and quickly, or reading words accurately.

Mathematics – This may be related to simple calculation abilities such as math facts or more complex problem-solving and reasoning abilities.

Written expression – 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.

7.1.2.1. Matters of dyslexia and dyscalculia. Technically, dyslexia and dyscalculia are not actual diagnoses in the DSM 5, rather they are alternative terms used to describe learning disorders in

reading (dyslexia) and math (dyscalculia). Dyslexia is the presence of significant deficit related to fluent word recognition, decoding, and spelling (APA, 2013). Dyscalculia is the presence of significant deficits related to "problems processing numerical information, learning arithmetic facts, and performing accurate or fluent calculations" (APA, 2013). Although these two terms are used very frequently in school systems and by other professionals such as Speech/Language Pathologists, as mentioned, they are considered alternative terms in the DSM 5, not diagnoses, and as such psychologists cannot actually diagnose these terms. Instead, they diagnose a s*pecific learning disorder with impairment in reading* (for dyslexia) and a *specific learning disorder with impairment in mathematics (*for dyscalculia). They can provide explanation and rationale that the deficits of the individual are characteristic of the pattern of deficits seen in individuals with dyslexia or dyscalculia. This is an excellent example of how professionals sometimes will discuss the same phenomenon but use different terminology.

7.1.3. Differences and Similarities between ID and LD

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

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

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

7.1.3.2. Learning disorders. If we *can* achieve an average level of skill (meaning our IQ is average), but we *are not* achieving an average level of academic achievement in an area, that leads us to be puzzled, right? If we *can* do something, but we *are not*, that doesn't make sense. But what if we *cannot* perform averagely (meaning our IQ is not average, but substantially below average)? Would we expect the individual to perform averagely on academic tasks? For example, if someone's IQ is 65 (*cannot* function typically on cognitive task*)* would we expect them to have an academic achievement score of 100 (remember, this is their "*is/are* or *is/are not* doing/performing)? That's a 30-point jump from their '*can do*' to their '*are doing*'. We wouldn't necessarily expect this, right? We would expect that if someone's IQ is 70 then they would have an academic score of around a 70. Even though the achievement score is low, this isn't necessarily indicative of an LD; rather, it is reflective of low

achievement due to low cognitive abilities resulting from ID. However, if that person's IQ was 100 (*can do*) and they scored a 70 (*is* not performing) on an academic achievement task, we would be concerned about an LD because what they *are* doing is not matching and measuring up to what they, theoretically, *can* do. Now keep in mind, I'm describing this in a very watered-down way, but this is the easiest way to differentiate between the two disorders if you are getting them confused.

7.1.3.3. LDs in the cognitively delayed and in the cognitively gifted. Individual's with extreme cognitive functioning abilities often get overlooked. For example, children that are gifted, but have a reading disorder, often go undiagnosed. Think about it, their weaknesses, although areas of deficit for themselves, actually look like average abilities to others around them. You might be asking yourself what I mean by this. Well, let me illustrate an example.

A 2nd grader with a high cognitive ability gets 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 has a great ability to 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 near as easily 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 I told you 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. The old DSM would have made it easy to diagnose this child with an LD in reading. The new DSM makes it a bit tougher. However, one would be inclined, if this reading deficit (compared to her own abilities) caused impairment (internal distress, preventing her from advancing in math and writing because her reading abilities were lagging behind the other abilities), then one would have a strong case to diagnose her with an LD in reading. However, it is easy to see how this child would be missed and go undiagnosed for years, right?

Now let's reverse the scenario. Let's take a 2nd grade girl who has a diagnosis of an ID. 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 an LD 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).

Essentially, when individual have an IQ that falls to the extreme (low or high), their weaknesses are often missed. As such, providers and educators have to be careful and mindful to not overlook potential

LDs in these individuals.

7.2. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe the prevalence and course of ID and LDs.
- Describe comorbid disorders of ID and LDs.

7.2.1. Intellectual Disability

ID occurs in approximately 1% of the population (APA, 2013) and is more common in males than females, although gender variations are inconsistent in some of the literature (APA, 2013; Einfeld & Emerson, 2008). It is hypothesized that the reason there is a higher occurrence of ID in males is due to the general genetic vulnerability, often linked to X chromosome issues that males experience (Harris, 2006).

ID is often comorbid with other medical and physical conditions (see etiology discussion) as well as other neurodevelopmental conditions including autism and ADHD. Moreover, depression, bipolar, and anxiety are often comorbid with ID. Lastly, stereotypic movement disorder is frequently comorbid with ID (APA, 2013)

7.2.2. Learning Disorders

LDs occur in approximately 5 to 15% of the population and are more common in males than females (APA, 2013).

ADHD is frequently comorbid with LDs. Additionally, autism and communication disorders are frequently comorbid with LDs (APA, 2013).

7.3. ETIOLOGY

Section Learning Objectives

- Describe biological basis/causes of ID and LDs.
- Describe environmental causes of ID and LDs

7.3.1. Biological

7.3.1.1. Intellectual disability. Biological factors heavily influence the development of ID. For example, 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 ID (Harris, 2006). Moreover, brain and central nervous system malformations such as spina bifida are risk factors and correlated with ID (Harris, 2006).

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

7.3.2. Environmental

7.3.2.1. Intellectual disability. Malnutrition of a mother during pregnancy, or medical conditions preventing nutrition absorption of the fetus, is a risk factor. Moreover, maternal illness or disease, such as diabetes or varicella (chickenpox), during pregnancy, increases risk of ID. Fetal exposure to alcohol, drugs, toxins, etc. also impacts potential development of ID. Events during labor and delivery or soon following 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 ID. Other events such as severe social deprivation, abuse, or exposure to high levels of lead or mercury lead to a higher risk of ID.

7.3.2.2. Learning disorders. 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 an LD. Early educational experiences may also heavily impact an individual's neural development and neural connections; thus, impacting LD risk (Goldstein, et al., 2011). For example, if an individual does not have proper exposure to educational material early on, this may negatively impact the neural connections established, and thus, lead to a higher risk for developing a LD.

7.4. ASSESSMENT

Section Learning Objectives

- Describe assessment tools commonly used.

7.4.1. Observations and Interviews

Although observation may be used for assessment of these disorders, it is the least clinically utilized in these disorders. A school observation may be conducted to ensure other disorders do not need to be investigated (for example, ADHD) that may explain some of the voiced concerns from parents and educators. However, from a diagnostic standpoint, the information that will be most helpful will be objective data (with some supplementation of information from interviews). Interviews are utilized to find out 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 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 skills quicker/earlier than him (or has even passed him up 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. Moreover, we want to get information from multiple individuals and we want to be able to recognize 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 begin to get very subjective. Common objective measures of adaptive functioning include the Adaptive Behavior Assessment System, Third Edition (ABAS-3) and the Vineland Adaptive Behavior Scales, Third 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 get 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 deficit is present, we want to utilize a full IQ battery, not an abbreviated version. Common IQ tests that are used to assess individual's IQ are the Stanford-Binet Intelligence Scales, Fifth Edition (SB-5) Wechsler Intelligence Scale for children, Fifth Edition (WISC-V), Woodcock Johnson Tests of Cognitive Abilities, Fourth Edition (WJ-IV), and the Kaufman Assessment Battery for Children, Second Edition, Normative Update (KABC-II NU). Remember, this score gives us the "*can*".

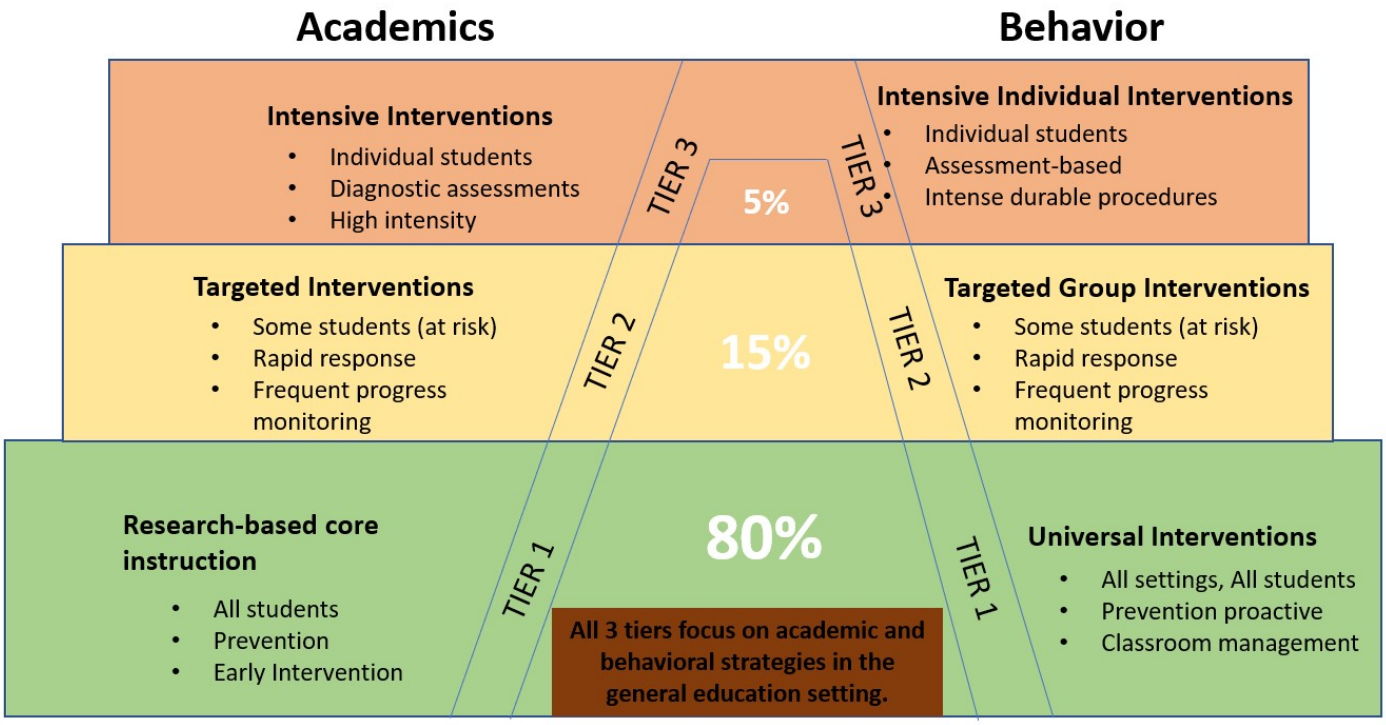
7.4.2.3. Academic achievement tests. Achievement tests assess academic achievement in an individual. We use these, *instead of only* looking at one's grades, because it allows for a more consistent comparison. For example, if we just relied on grades, we would not be able to control for Teacher A's grading or curriculum being more strict or difficult than Teacher B's grading. 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, Third Edition (WIAT-III), Woodcock Johnson Tests of Achievement, Fourth Edition (WJ-IV), and Kaufman Test of Educational Achievement, Third Edition (KTEA-III). Remember this score gives us the *"is/are".*

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

Responses 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. In a basic sense, all children receive basic academic instruction (oftentimes the general instruction all students receive is often referred to as Tier 1). If children begin to fall behind in academics in any particular area, they are identified and placed into a Tier 1 intervention group (or Tier 2, if all students are being considered as the Tier 1 group (see above). 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 that 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

7.5. TREATMENT

Section Learning Objectives

- Describe treatment options for ID and LDs.

7.5.1. Intellectual Disability

7.5.1.1. Community supports and programs. For individuals with ID, 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 ID. They often engage in advocacy efforts and offer training for the community and professionals. Moreover, they offer employment services for individuals with ID or other developmental delays. Additionally, local chapters will often host social gatherings and events for individuals and their families (The Arc, 2018). Typically, there 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 ID 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 and have constant supervision and medical care access as well as transportation. Another option, often referred to supported independent living, is a situation in which fewer, perhaps 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, and a nurse and other medical staff are not readily available. Moreover, individuals with ID are often capable of successful employment and these opportunities are provided in group and independent living home arrangements. Individuals with ID, 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 an Intellectual Disability 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) which was 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 what a child is entitled to in the educational setting so that they are able to access educational material to the fullest degree. Children with ID 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. Additionally, 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 kids that all have a severe disability, often times 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 go beyond academic specific areas. For example, social skills may be a focus of intervention.

Individuals with severe deficits related to ID will eventually have to have a determination of diploma track or not. 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 ID 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 that have a developmental delay, including ID. Students receive a "Certification of Completion" within a specific vocational area when they complete the program. They live on campus and are able to access the full college experience (MSU, n.d.).

7.5.1.3. Psychotherapy. Therapy is often underutilized in individuals with ID, despite beneficial impacts that research has shown when both behavioral and cognitive-behavioral therapies are utilized (Harris, 2006). Therapy often focuses on the emotional and behavioral impacts of ID, normalizing the individual's experiences, and treating comorbid depression, anxiety, or other mental health conditions (Harris, 2006). Another area of strong focus may be increasing adaptive functioning skills. For example, helping the individual learn and regularly implement daily hygiene, chores, etc. and learning to safely and successfully navigate within their home and community may be a focus of therapy.

7.5.1.4. Medication. Medications to manage emotional or behavioral concerns that are occurring comorbid with an individual's ID diagnosis may be beneficial. For example, if an individual has ID and depression, an antidepressant may be beneficial to utilize to help resolve some symptoms of depression. However, medications are not utilized to "treat" ID.

7.5.2. Learning Disorders

7.5.2.1. Education. Individuals with an LD receive an Individualized Education Plan (IEP) as well. Focus is placed on increasing instructional aids for the child. The child will often be pulled out 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 a study guide is not regularly given in a particular class. A child may also be allowed to have tests read to them, especially on nonreading-related tests, such as history, when a reading impairment is noted. 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 tests items and have a teacher write their answers. Moreover, the child may get opportunities to correct errors on test for additional credit, etc. These are just examples of accommodations and are not an exhaustive list. The specific accommodations and supports that are

implemented should be specific to the child, their particular deficits, and their current needs.

Tutoring, may that be in school or privately, is often useful as well. This simply 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 ID, medicine is not utilized to 'treat' an LD. However, given that ADHD is highly comorbid with LDs, ADHD-related medications may be utilized and beneficial, when this comorbidity is present for a child. Moreover, as chronic underachievement in an academic area may lead to some anxiety and depressive states for some children, medicinal intervention (or psychotherapy) may also be utilized and beneficial.

Apply Your Knowledge

CASE VIGNETTE

Review these two cases:

- https://harrywodehouse.weebly.com/uploads/3/8/4/0/3840641/case_study_-_sld.pdf
- https://ohioemploymentfirst.org/up_doc/Case_Study_Intellectual_Disability_accessible.pdf

QUESTIONS TO TEST YOUR KNOWLEDGE

1. What ways were Mary and Kesha's experiences similar?
2. What was the most notable take always from either Mary 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 Disability (ID) and Learning Disorders (LDs). We discussed the various symptoms of ID and LD and how they relate to the various presentations. We carefully examined the similarities and differences between ID and LD as well. We then discussed the prevalence of these disorders, frequently comorbid disorders, and the etiology of ID and LDs. We ended on a discussion of how ID and LDs are assessed and treated. Next week, we will learn about Autism

Spectrum Disorder.

2nd edition

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 and social communication disorders. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 8.1. Clinical Presentation and DSM-5 Criteria
- 8.2. Prevalence and Comorbidity
- 8.3. Etiology
- 8.4. Assessment
- 8.5. Treatment

Module Learning Outcomes

- Describe how Autism Spectrum Disorder (ASD) presents.
- Describe the prevalence of ASD.
- Describe the etiology of ASD.
- Describe how ASD is assessed, diagnosed, and treated.

8.1. CLINICAL PRESENTATION – AUTISM SPECTRUM DISORDER

Section Learning Objectives

- Provide history of Autism Spectrum Disorder and clarify misconceptions.
- Describe the common symptoms and associated features of Autism Spectrum Disorder.
- Describe a related disorder, Social (Pragmatic) Communication Disorder

8.1.1. History of Autism Spectrum Disorder

Autism spectrum disorder (ASD) is a disorder new to the DSM-5. Prior to the DSM 5, autism spectrum disorder was split into two different disorders – Asperger's syndrome and autistic disorder in which a presence of developmental delays and language delays had to be noted. However, for Asperger's syndrome, a presence of developmental delays and language delays were not noted. In addition, a presence of a cognitive delay could not be noted whereas cognitive delay was permitted when diagnosing autistic disorder. Moreover, restricted behaviors in autistic disorder typically presented as motor movements whereas in Asperger's syndrome, it most often presented as restricted and circumscribed interests (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). As such, with the publication of the new DSM-5, 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. Essentially, if the individual had any of the previous, old diagnoses, they were eligible to automatically be assigned with the new ASD diagnosis (APA, 2013).

8.1.2. Clinical Presentation of Autism Spectrum Disorder

At the core of autism spectrum disorder, the deficit is a neurodevelopmental concern related to social and adaptive functioning. The disorder is characterized by two major areas – deficits in social communication and interaction and significant concern related to restricted and receptive behaviors and/or interests (APA, 2013).

Social communication and interaction concerns may include things like poor eye contact, dominating a conversation/or lacking ability to maintain conversation due to no or limited **reciprocity** (i.e., "to and fro" conversation for 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.

Restricted and repetitive behaviors may include stereotyped movement, frequently called

stereotypy, such as hand flapping, spinning, or any receptive movement that does not have an obvious function. This can also include **restricted or repetitive play**. For example, lining up toys, fixating on a part of a toy or button, etc. Individuals may have excessive and **restricted interests** as well – for example, 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 they will engage in a conversation over. **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 many really 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 nowhere near 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 really want to 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. Individuals must exhibit both social communication concerns *and* restricted/repetitive behaviors/interests to truly be classified as having ASD. Moreover, these symptoms have to be present very early in development (APA, 2013).

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. There is a saying that a Dr. Stephen Shore originally said which is, "If you've met one person with autism, you've met one person with autism." What is meant by this is that, ASD is a very heterogenous disorder. One child with ASD may have symptoms that present very differently than another child with ASD. Because of this, it is important to recognize traps of *myths* about ASD and incorrect assumptions and information.

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 outline if there is any evidence of language impairments or intellectual impairments. Moreover, ASD is also diagnosed in light of severity. Clinicians diagnose ASD in the context of levels of severity in each main category with a Level 1 of severity being the least severe and a Level 3 of severity being the most severe.

8.1.3. Social (Pragmatic) Communication Disorder

Social pragmatic communication disorder is similar to autism spectrum disorder in that social communication, may that be verbal or nonverbal, is impacted. For the purposes of this class, we will not spend excessive time on this disorder. However, it is being mentioned because of the importance of differentiating this disorder from ASD. This disorder is characterized by overall difficulty with

understanding how social communication should occur (e.g., to and fro), flexible understanding 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. 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 get older, 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, given the individuals early history of the behaviors. This disorder is new to the DSM-5 and we are still early in our understanding of it. This disorder is considered to be more of a communication disorder rather than a pervasive developmental disorder or delay. We will not discuss this disorder further, but please be aware of the slight differences between ASD and Social (Pragmatic) Communication Disorder discussed in this section (APA, 2013).

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

One in 68 (59 according to some statistics; CDC, 2018, November) children are currently diagnosed with ASD (Smith & Iadarola, 2015). Prevalence rates have increased throughout the years. Previous prevalence rates indicate that in 2012, 1 in 88 children were diagnosed with an ASD. Even lower, in 2007 only 1 in 150 children were diagnosed with an ASD (CDC, 2018, November). Autism is more common, in fact, four times more common, in boys than it is in girls (CDC, 2018, May).

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.

Intellectual impairment and language disorders are commonly comorbid with ASD. Moreover, difficulties, sleep problems, avoidant/restrictive food intake disorder (often due to sensory concerns), epilepsy, and constipation are commonly comorbid with autism.

8.3. ETIOLOGY

Section Learning Objectives

- Describe biological basis/causes of ASD
- Describe sociocultural 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.

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. Moreover, 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 genetic predispositions are likely. However, as much as researchers try, they cannot pinpoint a specific genetic marker that accounts for autism or predispositions of autism.

Spontaneous gene mutations may also be related to autism. Moreover, genetic conditions such as Fragile X and tuberous sclerosis may lead to higher susceptibility to developing autism (APA, 2013).

Children that have a sibling with ASD have higher risk for later being diagnosed with ASD (CDC, 2018, May).

Parental Factors. Older parental age and complicated birth of the child are associated with higher risk for developing ASD. However, no singular parental factor could predict autism in the Gardener, Spielgeman, & Buka's 2009 study.

8.3.1.3. Vaccines. For a long period of time, there was a large misconception that vaccines, particularly the MMR vaccine, were causing autism Spectrum Disorder. Since that time, we have learned that is **not** true or accurate. To understand this misconception, we must first understand the background of such. The idea that vaccines caused autism began around in the late 90s/early 2000s when a team of researchers, headed by Andrew Wakefield, MD 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 the bias of the funding of the study was also not revealed. Moreover, there were parts of the paper that were criticized as being incorrect and, in whole, an ethical concern (e.g., cherry-picking data, misleading statements about the data). As such, a retraction to the study was later made 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; Taylor, Swerdfeger, & Eslick, 2014; Uno, Uchiyama, Aleksic, & Ozaki, 2015) Despite continued evidence disproving the link between the two, the general public continues to hold on to the concern of vaccines causing autism (Sheikh, Swetlik, & Wilson, 2018). So, 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.**

8.4. ASSESSMENT

Section Learning Objectives

- Describe assessment tools commonly used
- Examine differential diagnostic difficulties

When assessing for ASD, psychologists often rely heavily on observations. They also rely on parent-

report, particularly of early development as well. 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 getting 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 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. The observation is actually a bit more formal than observations of other disorders which we will discuss later. 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 severe issue in screening and diagnosing children with ASD. We have gotten better at diagnosing, 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 at 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 goes to 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. Module 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 pull for certain interactions and behaviors and allows the clinician to assess those abilities. For example, does the child point, does the child 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 derives one final score. This score will classify how likely a child is to meet diagnostic criteria. In this author’s clinical opinion, ASD should not be diagnosed without completing the ADOS-2 or a related, standardized observation protocol for ASD.

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 a 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 piece (with the ADOS-2 being the primary piece) 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, it is not as frequently utilized as the ADOS-2. However, it is an excellent tool.

8.4.3. Objective Measures

Other standardized measures are often utilized. A common form that is utilized in a comprehensive assessment is the Autism Spectrum Rating Scales (Goldstein & Naglieri, 2010; ASRS). This form includes several items that address various areas of 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 up to age 18 years old. The Social Responsiveness Scale (Constantino & Gruber, 2012; SRS) is also a common measure used in an assessment. It is similar to the ASRS but focuses a bit more on the social aspects and social impacts of symptoms and behaviors. In addition, at times, the Sensory Profile (Dunn, 2014; SR-2) may be included to understand in more detail various sensory experiences a child has. Commonly used to screen, but should never be used to exclusively diagnose a child, is the Gilliam Autism Rating Scale: 3rd Edition (Gilliam, 2014; GARS). This is a very helpful tool to understand the likelihood of a presence of autism in a child. The Modified Checklist for Autism in Toddlers, Revised with Follow-Up (Robins, Fein, & Barton, 2009; M-CHAT R/F) is an excellent screening tool as well. These are most helpful when used in a primary care office. They 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 to assess for the presence of autism.

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 and hearing 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.

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 has been used more loosely, but typically speaking, this intervention includes significant behavioral intervention (i.e., applied behavioral intervention), often times 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 (Eldevik, Hastings, Hughes, et al.; Makrygianni & Reed, 2010; Reichow, 2012). Early intervention is considered to be potentially most beneficial because the younger our brains are, the more **plasticity,** the ability for our brain to modify its neural connections, our brains have. As such, we are able to grow and change our brain connections and structures more easily than we can when we are older. As such, 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. In fact, many children receive a large bulk of their ABA services in the outpatient clinic. This is because receiving early intervention services with in-home ABA is very 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 this 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 (meaning insurance will not cover the service) for in-home or in-school services (Note: In-school services are often offered by a school, but at a much lower number of hours in which a particular 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 ABA. 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 of a behavior (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 tantrums every time his mother leaves a room, it may be that he is tantruming because he has been denied her attention or because he know he will be expected to do work when she leaves and he wants to escape an undesired task. The behavioral therapist's job is to begin 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 you work for 10 minutes, you can have a 3-minute iPad break (if function is to escape undesired task). Or, if you work for 10 minutes, you can play with mom for 3 minutes (if function is to gain mother's attention). This is a basic example to illustrate ABA. A component that sets ABA apart is that nearly everything is tracked in ABA. 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 to take a task and break it down to the smallest task possible. For example, when we discuss putting on a pair of pants, 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 then next step (may that be forward or backward) is achieved until the entire task

(e.g., putting on pants) is accomplished independently. to increase task success. *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 being recorded (Anderson et al., 1996).

The only people qualified to fully implement ABA are BCBAs (or BCaBA) or RBTs. A BCBA is a Board-Certified Behavioral Analyst that typically has at least a Master's degree or higher. A BCaBA 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 as well as creating treatment plans, analyzing data of clients, and supervising 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 a week. While we will keep our conversation about speech therapy brief, important to note is that many children that do not develop language (or are still struggling to develop functional language) will end up using either the PECS system or an augmented communication device (a fancy way of saying a tablet with particular programming that helps facilitate communication). While there are differences in the two, essentially, for the purpose of this class, I want you 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. Again, this is a very watered-down explanation. Essentially, know that when children do not develop language, they may be fitted with an alternative way to communicate that heavily relies on pictures to communicate.

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. Which 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, desire friendships but find it difficult to develop those friendships due to social skill deficits, or feel frustrated by their behaviors and symptoms. As such, 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 siblings. 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. As such, anti-anxiety medications may be beneficial. Moreover, stimulants to help reduce hyperactivity, either due to ASD or comorbid ADHD may also be helpful. Moreover, antidepressants may be helpful in decreasing repetitive behaviors and may

also help taper irritability and tantruming. Anti-psychotic medications may help decrease irritability, hyperactivity, stereotyped behavior, and aggression (NICHD, 2017, January).

APPLY YOUR KNOWLEDGE

CASE VIGNETTE

Howard is 7 years old and presents at his GP with his mother as she is concerned about his challenging behaviour 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 behaviour 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. 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 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 ASD. 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 chapter, we will discuss motor disorders such as Tourette's and Stereotypic Movement Disorder.

2nd edition

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 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 (SMD) is a disorder in which an individual engages in repetitive movements and those movements have no clear functional purpose. For example, flapping hands repetitively. This disorder may present with self-injurious behavior at times (e.g., head banging, pinching skin severely). This disorder is not a disorder that emerges in adulthood. In fact, the onset for this disorder occurs in very young children (e.g., onset by age 3 typically; APA, 2013). Although some of these behaviors may be reminiscent of behaviors seen in children with Autism, children with SMD do not display deficits in social communication and interaction and do not have circumscribed interests, etc. Essentially, the only symptoms related to autism that are displayed in SMD is stereotypy-related behavior. As such, these children are not diagnosed with Autism, rather, they are diagnosed with stereotypic movement disorder.

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. Although I'll break down the three different disorders, for the purposes of this class, I'd like you to understand the basic symptoms of this classification of disorders. As such, we will keep the conversation general and the specifics brief.

9.1.2.1. General symptoms. These disorders present before adulthood (i.e., 18 years old). Typically speaking, the time in which they generally 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. **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, 2013).

9.1.2.2. Tourette's disorder. Tourette's disorder is when *both* motor and vocal tics are present. Generally speaking, more than one motor tic must be present and at least one vocal tic must occur (APA, 2013) to be classified as Tourette's disorder. The tics do not have to occur together and do not have to be complex tics. Of course, complex tics could be present, they just are not necessarily required.

9.1.2.3. Persistent (chronic) motor or vocal tic disorder. This is when *either* one or more motor tics *or* one or more vocal tic is present. However, vocal and motor tics are *not* both present (APA, 2013). These again can be simple or complex and only the presence of one tic is required. If an individual used to meet the descriptors for Tourette's, despite the presentation of their tics now, they would be diagnosed with Tourette's and not persistent (chronic) motor or vocal tic disorder. For example, if an individual used to have several simple motor tics and one vocal tic; however, they no longer have a vocal tic, they would still be diagnosed with Tourette's rather than persistent (chronic) motor or vocal tic disorder.

9.1.2.4. Provisional tic disorder. For a diagnosis of Tourette's or persistent (chronic) motor or vocal tic disorder, tics have to be present for at least a year. As such, if a person has *any combination* of tics, but the tics have not been present for a year, then they would not be diagnosed with either Tourette's or persistent (chronic) motor or vocal tic disorder; rather, they would be diagnosed with provisional tic disorder (assuming the onset is before 18 years old).

9.2. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe the prevalence of Stereotypic Movement Disorders and Tic Disorders.
- Describe common disorders that are comorbid with Stereotypic Movement Disorders 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 an intellectual disability, the prevalence rate is higher and occurs in about 4 to 16% of individuals with these cognitive deficits.

SMD 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, presence of other neurodevelopmental disorders such as intellectual disability may be comorbid with SMD as well.

Below are common disorders that SMD must be differentiated from.

9.2.1.1. 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, as mentioned earlier, children with stereotypic movement disorder do not evidence social communication and reciprocity deficits. Moreover, they do not tend to have circumscribed interests, difficulty with transitions, delayed speech, etc. Essentially, the only symptoms related to Autism that are displayed is the stereotypy related to behavior (APA, 2013).

9.2.1.2. 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 than tics related to tic disorders do. Finally, tics typically are quick, brief, and fleeting whereas stereotypic movements tend to be more prolonged and repetitive (APA, 2013).

9.2.1.3. Obsessive-Compulsive Disorders (OCD). Professionals can distinguish between obsessive-compulsive disorders and stereotypic movement disorders by determining if there are any obsessions present. Essentially, does the individual feel compelled or driven to complete a behavior? If the answer is yes, it is more likely an OCD-related behavior. If the answer is no, it not likely OCD. 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. Moreover, again, stereotypic movement disorder has an earlier onset than OCD-related disorders (APA, 2013).

9.2.2. Tic Disorders

Tic disorders are relatively rare. The CDC estimates that approximately .3% of children are diagnosed with Tourette's, specifically (CDC, 2018, October)). of the general population. Tic disorders are more common in boys (with boys being three to five times more likely to be diagnosed, CDC, 2018, October) and there are lower rates in African American and Hispanic groups (APA, 2013).

Comorbid disorders are common, with 86% of individuals with Tourette's also having another psychological disorder (CDC, 2018, October). ADHD and obsessive-compulsive disorders are commonly comorbid with tic disorders (APA, 2013) with about two-thirds of individuals with Tourette's also having ADHD and one-third having OCD (CDC, 2018, October). Careful assessment to differentiate if tics or obsession/compulsions are present is important. However, often times, both are present, and if that occurs, both disorders are diagnosed.

Below are common disorders that must be stereotypic movement disorder must be differentiated from.

9.2.2.1. Obsessive-compulsive disorders (OCD). This can be a very hard disorder to differentiate. However, typically speaking, tics are experiences as an "urge" in which a person may feel a pressure or tension just prior to a tic (think about how you feel just before you sneeze!) where as an individual that is engage in a compulsion (related to OCD) may have a do something because they feel driven to (e.g., belief something bad will happen if they don't, etc.) or do something until "it feels just right" (APA, 2013). As such, tics are not goal directed whereas compulsions related to OCD disorders are goal directed.

9.2.2.2. Stereotypic movement disorder. Distinguishing between stereotypic movement disorder and tic disorders is done by examining the same factors mentioned above when discussing stereotypic movement disorders (see 9.2.1. discussion).

9.2.2.3. Other disorders to differentiate. Motor stereotypies such as chorea or athetosis related to medical conditions, dyskinesia and myoclonus (sudden, nonrhythmic, unidirectional movement) must be differentiated (APA, 2013) and requires a medical assessment. You don't need to understand the specifics about these medical phenomena, only that there are some medical conditions that may mimic tics, and thus, full medical histories and exams are often necessary.

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 SMD. Some medical condition and genetic syndromes such as Lesch-Nyhan syndrome or Rett syndrome (APA, 2013). However, overall, little is known about the genetic and biological vulnerabilities that lead to SMD (Zinner & Mink, 2010)

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

will pass the genetically susceptibility on to their child (CDC, 2018, April). 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, 2013). Thus, similar to what was discussed with rumination disorder, stereotypic movements may be automatically reinforced due the internal stimulation it provides (Ricketts, 2013).

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 (APA, 2013). 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. Moreover, research has indicated that 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).

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

Overall, assessing for a motor disorders is largely based on observation and interviewing. Very specific information about the various behaviors occurring, the frequency, the context, and the severity is obtained. Through understanding of the presence of voluntary versus involuntary movements is important to obtain. Some forms may be given to rule out other disorders, specifically measures to screen/assess for Autism and OCD-related concerns may be utilized. 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 SMD. The most commonly research and used intervention is behavioral therapy. Specifically, differential reinforcement or habit reversal therapy (described in CBITS intervention below) with some modification has shown some 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 (selective serotonin reuptake inhibitor), clomipramine (tricyclic antidepressant), and risperidone (atypical neuroleptic) have been noted to positively impact repetitive behaviors in children with Autism, and thus, are used with children diagnosed with SMD at times. However, there is not systematic research and literature that these medications are empirically efficacious and beneficial in reducing stereotypy in children with SMD (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 well-established and efficacious treatment for tics. The core of treatment utilizes habit reversal training which includes increasing awareness and then introducing an incompatible response to a behavior. CBIT treatment includes 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" 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. Common medications that may be used 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 start with 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 of medicinal intervention, 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).

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 disorders 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 discussions on developmental delays and motor disorders. Next week, we will start our discussion on behavior-related disorders, starting with attention-deficit/hyperactivity disorder.

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IV

PART IV. BEHAVIOR-RELATED DISORDERS

Part IV. Behavior-related Disorders

Module 10 - Attention-Deficit Hyperactivity Disorder

Module Overview

In Module 10, we will discuss matters related to attention deficit/hyperactivity disorder to include its clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 10.1. Clinical Presentation
- 10.2. 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 – ADHD

Section Learning Objectives

- Provide history of ADHD and clarify misconceptions.
- Describe the common symptoms and associated features of ADHD.

10.1.1. Brief Overview of the Recent History of ADHD

In previous editions of the DSM, ADHD was known as either Attention Deficit Disorder (ADD) or Attention-Deficit/Hyperactivity Disorder (ADHD). As years have gone by, the field has come to understand that both ADD and ADHD are, in fact, the same phenomenon that simply presented a little differently. What the field has come to realize is this – one presentation, ADD, led to more inattentive and distractibility symptoms whereas the other presentation, ADHD, resulted in more impulsive and hyperactive symptoms. However, while the symptoms seem different, psychologists realized they were related to a similar, etiological and psychological phenomenon. Because of this, the field decided to combine both disorders into one disorder, ADHD, with specifications on the type of ADHD – ADHD Predominantly Inattentive type, ADHD Predominately Hyperactive/Impulsive type, and ADHD Combined type). Since that time, we have come to understand ADHD even further, and we have revised the disorder again.

Now, with the DSM-5 (APA, 2013), we still consider all presentations to fall under one disorder, ADHD. However, instead of *types*, which give the impression of a stable, unchanging, diagnosis, we now use "*presentations*." This change occurred because we began to realize 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. With presentations, it allows for a better understanding that symptom presentation can be fluid and change. Thus, we now have *one* disorder, ADHD, with three *presentation* (predominantly inattentive presentation, predominantly hyperactive/impulsive presentation, and combined presentation). That being said, ADD is not actually a disorder now. Rather, someone who in years past might have been diagnosed with ADD, would now be diagnosed *ADHD, predominantly inattentive presentation*. What the person is experiencing is the same, but how we diagnose it and communicate those symptoms has changed over the years.

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 ability to focus in on relevant information while dismissing unimportant or irrelevant information (Barkley, 2015). Essentially, an individual's ability to regulate their cognitive, 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. Children with ADHD often have a low frustration tolerance (e.g., become frustrated more easily by minor things) as well. This disorder is considered a neurodevelopmental disorder meaning that it presents, initially, within an early childhood period. As such, although the disorder *can* be diagnosed in adulthood, there *must be evidence* of symptoms during childhood years (e.g., before the age of 12; APA, 2013). 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 categorized into hyperactive/impulsive symptoms and inattentive symptoms. An individual *must* exhibit several symptoms under one singular area which results in *predominantly inattentive presentation* (if this is where the six or more symptoms are) or *predominantly hyperactive/impulsive presentation* (if this is where the six or more symptoms are). If several symptoms are present in both areas, evidence for *combined presentation* is likely (APA, 2013).

Hyperactive/impulsive symptoms. These symptoms are related to excessive energy and movement as well as impulsivity. Individuals with these symptoms are often described as high energy, are described as "talkative" and fidgety. These children may have a hard time waiting their turn for things, standing still, remaining in their seats, or staying in line and remaining quiet. They tend to be described as loud and disruptive at times as well.

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. These children may forget to turn in homework, fail to bring home all of their assignments, struggle to complete their work to its entirety, and get distracted by minor occurrences in the environment.

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 is considered to occur across cultures and is noted in approximately 5% of children (APA, 2013). Although the DSM-5 (APA, 2013) indicates a prevalence rate of 5%, some studies report prevalence rates as high as 9.4% (CDC, 2018a, September). ADHD is more often diagnosed in males than in females. Boys are more likely to exhibit ADHD predominantly hyperactive/impulsive presentation or combined presentation whereas girls may be more likely to exhibit predominantly inattentive presentation (APA, 2013). 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.

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, APA 2013). Conduct Disorder is also highly comorbid with ADHD (i.e., about 25% of youth with ADHD combined presentation).

Learning disorders are also commonly comorbid with ADHD (APA, 2013). However, differentiating if a learning disorder is present, in addition to ADHD, requires through evaluation (see differential discussion below).

Mood and anxiety disorders 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, 2013).

10.2.3. Differential Diagnosis

10.2.3.1. ODD. Because inattention and impulsivity can lead to noncompliance with rules, psychologists have 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 and begin to clean their room and 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, they 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 actually be experiencing anxiety instead. 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).

10.3. ETIOLOGY

Section Learning Objectives

- Describe biological basis/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 are of the brain has also been linked to ADHD symptoms. Some research indicates that other areas involved may include the anterior cingulate, basal ganglia, cerebellum, and corpus callosum as well. Moreover, smaller anterior right frontal regions, caudate nucleus and globus pallidus have also been associated with ADHD. Moreover, 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

Low birth weight is one of the strongest and most consistently noted environmental risk factors for ADHD (APA, 2013). Moreover, premature delivery is also associated with ADHD. Prenatal exposure to toxins, specifically smoking, but also alcohol and other drugs, is associated with higher rates of ADHD. Moreover, environmental toxins such as heavy exposure to lead or pesticides is linked to ADHD symptoms (APA, 2013; 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 week evidence for psychosocial factors impacting the development of ADHD (Barkley, 2015).

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 lot 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 them [the psychologist] 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 the child themselves is 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.

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. That 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 times, 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. In a teacher interview, the psychologist often focuses on similar topics and questions as the parent interview. However, 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 is able to 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.

10.5. TREATMENT

Section Learning Objectives

- Describe treatment options for ADHD
- Examine efficacy of 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 not 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 no work as quickly as stimulants, typically; however, they may have longer lasting effects (CDC, 2018b, September). However, given that 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 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 reacts 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. 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.

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.

APPLY YOUR KNOWLEDGE

CASE VIGNETTE

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 sperate 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 chapter, we will learn about oppositional defiant disorder (ODD) and conduct disorder (CD), two behavioral disorders.

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Module 11 - Oppositional and Conduct Disorder

Module Overview

In Module 11, we will discuss matters related to oppositional defiant and conduct disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

- 11.1. Clinical Presentation
- 11.2. Prevalence and Comorbidity
- 11.3. Etiology
- 11.4. Assessment
- 11.5. Treatment

Module Learning Outcomes

- Describe how ODD and CD presents.
- Describe the prevalence of ODD and CD.
- Describe the etiology of ODD and CD.
- Describe how ODD and CD is assessed, diagnosed, and treated.

11.1. CLINICAL PRESENTATION

Section Learning Objectives

- Describe the presentation and associated features of ODD.
- Describe the presentation and associated features of CD.

11.1.1. Oppositional Defiant Disorder

Oppositional defiant disorder (ODD) is characterized by a child that is defiant and vindictive at times. Children with ODD may actively defy commands and requests made by authority figures. They may break rules, be easily angered, annoy others intentionally, and struggle to take responsibility for their own actions. These children also frequently present as irritable. The symptoms of ODD may be noticed across multiple settings (e.g., home and school), but do not necessarily have to be noted in more than just one setting.

11.1.2. Conduct Disorder

Conduct disorder (CD) 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, and running away. There are certain specifiers with Conduct disorder that help outline the level of risk and prognosis for the individual. For example, symptoms may begin in *childhood* (prior to age 10; APA, 2013) or *adolescence* (after the age of 10; APA, 2013). Adolescent onset is associated with a more positive prognosis than childhood onset.

Conduct disorder is often associated with limited prosocial emotions. Because of this, psychologists are able to specify if an individual lacks prosocial emotions, when diagnosing conduct disorder. Prosocial emotions are things such as showing guilt or remorse, empathy, and appropriate affect (display of emotions). If these things are absent, it is important to note. As such if an individual displays a *lack of remorse or guilt,* is *callous* or *lacks empathy,* is *unconcerned about performance,* or has *shallow* or *deficient affect* (APA, 2013) this must be noted. This is because limited prosocial emotions are associated with a riskier and more negative trajectory.

11.2. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe the prevalence and course of ODD and CD.
- Describe comorbid disorders of ODD and CD.
- Describe disorders with similar presentations that must be differentiated from ODD and CD.

11.2.1. Oppositional Defiant Disorder

Prevalence rates for ODD vary. Recent estimates indicate that there is an average prevalence rate of 3.3% (with a range between 1% – 11%; APA, 2013). ODD is slightly more common in males (APA, 2013). ODD typically emerges in early childhood and is very rarely first noticed in adolescence (APA, 2013); however, symptoms may increase as a child develops. ODD may also be a risk factor for later development of CD (see etiological section below).

11.2.2. Conduct Disorder

Rates of CD also vary. According to the DSM-5, a prevalence anywhere between 2% to 9% has been noted (APA, 2013). CD occurs more often in males than females. Specifically, individuals with childhood-onset of symptoms are more likely to be male than female (APA, 2013). There is a slightly more equal rate of diagnosed cases among males and females with adolescent-onset of symptoms (APA, 2013). Symptoms commonly occur prior to age 16 (APA, 2013). As mentioned, an early onset is associated with great risk and may be more predictive of further criminal, antisocial, or substance-related behaviors (APA, 2013).

11.3. ETIOLOGY

Section Learning Objectives

- Describe biological basis/causes of disruptive disorders.
- Describe sociocultural causes of disruptive disorders

11.3.1. Biological

11.3.1.1. Oppositional defiant disorder and conduct disorder. Individuals with ODD and CD

appear to have a slower resting heart rates and lower reaction to fear. Research has also revealed functional and structural differences in individuals with ODD and CD indicating that there are differences in areas in the brain, including the frontotemporal-limbic connections and the prefrontal cortex/amygdala, involved with the processing and regulating affect (APA, 2013).

Regarding CD, having a caregiver or close relative that has been diagnosed with CD leads to a higher risk of a child developing CD. Children of parents with severe alcohol use, depression, bipolar, or schizophrenia, ADHD, or CD are at higher risk as well (APA, 2013). Family history may be particularly predictive of a child developing *childhood onset* (considered to have the worse prognosis; APA, 2013). Risk for CD and prognosis is furthered when also diagnosed with ADHD (APA, 2013).

11.3.2. Sociocultural

11.3.2.1. Oppositional defiant disorder. The most consistent finding is that harsh and inconsistent parenting (APA, 2013).

11.3.2.2. Conduct disorder. Rejection from parents, significant neglect, minimal parental supervision, and abuse are strong risk factors. Moreover, harsh and inconsistent parenting reflective of coercive interactions, as described in ODD, are highly linked to the development of these behaviors: rejection and neglect. Moreover, if the child's parents have a criminal history, the child is at greater risk for developing CD. Additionally, if the child is rejected by peers, socializes with a particularly delinquent peer group, or lives in a violent neighborhood, the child is at higher risk for CD (APA, 2013).

11.3.2.3. Theories of parenting and sociocultural impacts. Although there are various developmental pathway theories on how ODD and CD develop, such as Multiple Pathways (Loeber & Stouthamer-Loeber, 1998), Gerald Patterson's Coercive Family Process Model (1982) is one of the most commonly referenced and utilized theories. His Coercive Family Process 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 (discussed more below) of coercive parent-child interactions. Most of the parent training protocols to treat behavioral problems are based on Patterson's model.

Ultimately, Patterson (1982) theorized that various family factors impacts a parent's traits. Those parental traits then disrupt family dynamics which ultimately leads to child antisocial behavior. The interactions that occur 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

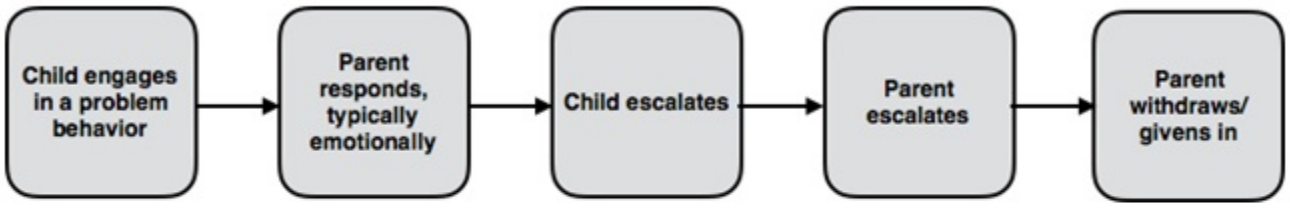
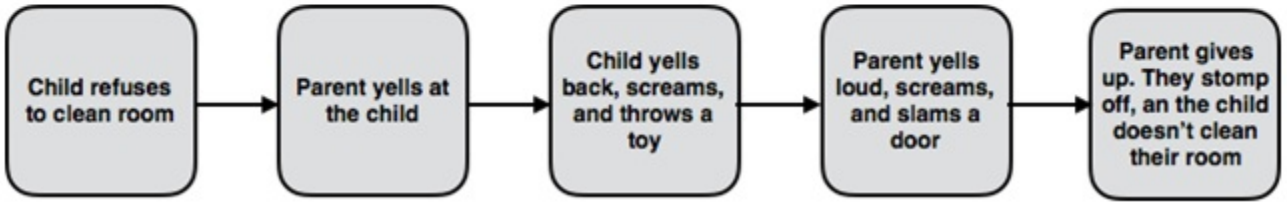


Figure 11.2 Specific Example of the Coercive Cycle



11.4. ASSESSMENT

Section Learning Objectives

- Describe assessment tools commonly used

11.4.1. Assessing for ODD and CD

When assessing for ODD or CD, the process is very similar to that outlined in the previous chapter of 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 and deny that they in fact 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 critical. Although, formal and inconspicuous observation of a child in the classroom is valuable, the behaviors we are looking for in ODD or CD are more disruptive and noticeable. As such, we tend to rely on parent and teacher reports more heavily. Nonetheless, a psychologist may get a good sample of observation 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 did not require the psychologist to observe the child in the waiting room or school to gain this information. Moreover, some symptoms, although severe, may occur infrequently. For example, fire setting, although severe, is not likely to occur during an observation period. Again, this is why, although useful when able to be obtained, observations are less critical for diagnosing ODD and/or CD than they are for a disorder such as ADHD.

11.4.3. Interview

An assessment for disruptive behaviors of ODD and CD should always include some version of an interview. This 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 started to occur will be a critical area of focus, especially when there is concern of CD. The need for a good understanding of age in when symptoms were first noticed is important due to the impact this has on the child's prognosis and trajectory. An important step in the interview process is to also understand and assess family and parenting practices. This is because family and parenting factors are closely related to etiology of these disorders as well as treatment implications.

Child interviews will typically be attempted. Sometimes with particularly defiant children, interviews are difficult. However, attempts should be made. Additionally, during the interview, a psychologist will also note displayed affect. A psychologist will attempt to understand the individual's presence of irritability, empathy, prosocial emotions, etc. This information is gained not just 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

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 to report on their own symptoms anywhere between the ages of 6-11, depending on the specific

questionnaire being used. Assessments will generally utilize similar assessments noted in the ADHD chapter. This is because ADHD, ODD, and CD, have high comorbidities with each other. Because of this, scales that were designed to assess ADHD also include subscales that measure ODD and CD 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, 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.

11.5 TREATMENT

Section Learning Objectives

- Describe treatment options for ODD and CD.
- Examine efficacy of varying treatment options.

11.5.1. Psychotherapy

11.5.1.1. Oppositional defiant disorder. 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 that they are more likely to be successful. This may mean breaking large chores down into more manageable pieces, etc. It also requires a parent to specifically outline the goal behavior (e.g., put your shoes in your closet) they want to see. They then have 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, state the expectation clearly, and remain firm with the directive.

PMT also focuses on giving more attention and praise to positive behaviors while ignoring negative minor misbehaviors. This is so that we can 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). This part is sometimes difficult for parents because they are often rewarding or praising 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. But, because the behavior is currently absent, we have to do something to increase that behavior. What we do is give it positive attention. 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, I often use this very analogy to help parents understand this.

PTM 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 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.

11.5.1.2. Conduct disorder. *Multisystemic therapy (MST***)** is an intensive treatment option that has demonstrated efficacious results for the treatment of CD, especially in cases of more extreme conduct problems. MST is a therapy that takes place in the child/adolescent's home as well as school and overall community. The therapist works with the child, their family, and other community members. Therapists can be accessed more readily than in other treatment modalities, meet with the child/family multiple times a week and follow a family for several months. This allows for 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 CD, it is costly and difficult to obtain in some areas of the country (due to resources). However, MST, although costly, may be less costly than typical services in the short term due to the reduction in crime and incarceration from MST treatment (Tan & Fajarado, 2017). Other studies (e.g., Dopp, Borduin, Wagner, & Sawyer, 2014) have also shown similar benefits found in Tan and Fajarado's (2017) study.

11.5.2. Psychopharmacology

Generally speaking, medications are not used to address symptoms of behavioral disorders. The primary treatment is psychotherapy. However, if a child has a behavioral disorder and comorbid impulsivity or mood concerns (see relevant chapters for more information), medications may be used to address those concerns as they may exacerbate ODD and CD symptoms.

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? What protective factors are present for William?
2. Does he meet criteria for ODD? CD? Why or why not?

Module Recap

In this module, we learned about ODD and CD. We discussed the various behaviors and symptoms of ODD, and CD and how they relate to the various presentations. We then discussed the prevalence of these disruptive, behavioral disorders, frequently comorbid disorders, and the etiology of ODD and CD. In our discussion of etiology, we also learned about the coercive cycle. We ended on a discussion of how ODD and CD is assessed and treated. This concludes our discussions on behavior-related disorders. Next week we will discuss mood-related disorders which includes depressive and bipolar-related disorders.

V

PART V. MOOD AND ANXIETY-RELATED DISORDERS

Part V. Mood and Anxiety-related 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, 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 and DSM-5 Criteria
- 12.2. Prevalence and Comorbidity
- 12.3. Etiology
- 12.4. Assessment
- 12.5. Treatment

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

- Describe the presentation and associated features of depressive and bipolar disorders
- Describe suicidality and self-harm.

Within mood disorders, in general, there are two distinct groups- individuals with depressive disorders and individuals with bipolar disorders. The key difference between the two groups is that those in the depressive disorder category *only* experience symptoms of depression, while those in the bipolar disorder category have periods of mania/hypomania that alternate with periods of depression.

12.1.1. Depressive Disorders

The two most common types of depressive disorders are **Major Depressive Disorder** and **Persistent Depressive Disorder**. Persistent Depressive Disorder, previously known as Dysthymia, is thought to be a more chronic, less severe depression. Like previously mentioned, in order to be diagnosed with either major depressive disorder or persistent depressive disorder, the individual must *never* have had a manic or hypomanic episode. In addition to Major Depressive Disorder and Persistent Depressive Disorder, **Disruptive Mood Dysregulation Disorder (DMDD)** is a newly created diagnosis that is classified as a depressive disorder. It will be discussed in this chapter as a sperate disorder because, although classified as a depressive disorder, holds its own unique characteristics and relevant content.

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

12.1.1.1. 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. However, in children, instead of depressed mood, children may display irritability. This is important to remember as many professionals, parents, and teachers do not recognize that irritability can be a very notable and common symptom of childhood depression, rather than sad or depressed mood.

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

12.1.1.3. 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. They often feel worthless, which creatives a negative feedback loop to their overall depressed mood. Individuals with depressive disorder also report difficulty concentrating on tasks, as they are easily distracted from outside stimuli. This is supported by research that has found individuals with depression perform worse than those without depression on tasks of memory, attention, and reasoning (Chen et al., 2013). Finally, thoughts of suicide and self-harm do occasionally occur in those with depressive disorders. This is covered in more detail in the epidemiology section.

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

Additional physical symptoms such as change in weight or eating behaviors are also observed. Some individuals who are experiencing depression report a lack of appetite, often forcing themselves to eat something during the day. On the contrary, others eat excessively, often seeking "comfort foods" such as those high in carbohydrates. Due to these changes in eating behaviors, there may be associated changes to weight.

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

According to the DSM-5 (APA, 2013), major depressive disorder and persistent depressive disorder are diagnosed according to the listed criteria. Although symptoms for both are nearly identical, the time frame of symptoms are significantly different, with symptoms presenting for a 2-week period for major depressive disorder and symptoms present for majority of 1-year (for children) for persistent depressive disorder. Cchildren do not necessarily have to exhibit symptoms to the same degree or duration to meet clinical criteria of diagnosis.

12.1.2. Disruptive Mood Dysregulation Disorder

Disruptive Mood Dysregulation Disorder (DMDD) is a new diagnosis and is classified as a depressive disorder. It emerged as a diagnosis with the publication of the DSM 5. There is some controversy in the

field about this diagnosis, but the rationale for its emergence, in many respects, was to reduce the frequency in which bipolar disorder was misdiagnosed in children. What researchers began to find is, children diagnosed with bipolar disorder (discussed in 12.1.3) that displayed persistent irritability, rather than episodic irritability, were at risk for future depression or anxiety, but they actually were not at risk for life-long bipolar-related symptoms (APA, 2013b). Ultimately, what this means is that children with persistent irritability, rather than episodic irritability associated with manic presentations of bipolar, were being diagnosed with bipolar, but were not likely to meet criteria for bipolar in the future. Rather, these children were more likely to be depressed or anxious. As such, these children were misdiagnosed with bipolar. In an effort to reduce this, DMDD emerged.

Essentially, DMDD is 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. These outbursts are often disproportional to the event (for example, tantruming for 30 minutes because the child did not get to pick the family game). The tantrums may seem to be consistent with what a younger child would display rather than what is typical for the child's actual age. For example, a 14-year-old adolescent stomping and crying over a minor obstacle in their day. These symptoms also present prior to age 10 (APA, 2013).

12.1.3. Bipolar Disorders

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

So, what defines a **manic episode**? The key feature of a manic episode is a specific period of time in which an individual experiences abnormally persistent expansive or irritable mood for nearly all day, every day, for at least one week (APA, 2013). Additionally, the individual will display increased activity or energy during this same time. With regards to mood, an individual in a manic episode will appear excessively happy, often engaging haphazardly in sexual or personal interactions. They also display rapid shifts in mood, also known as **mood lability**, ranging from happy, neutral, to irritable.

Inflated self-esteem, or grandiosity is also present during a manic episode. Occasionally these inflated self-esteem levels can appear delusional. Individuals may believe they are friends with a celebrity, do not need to abide laws, or even at times think they are God.

Despite their increased activity level, individuals experiencing a manic episode also require a decreased

need for sleep, sleeping as little as a few hours a night and still feel rested. In fact, decreased need for sleep may be an indicator that a manic episode is to begin imminently.

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

Sometimes symptoms of a manic episode present more mildly in a person, and this is an example of hypomania. Hypomanic states can often lead to productivity, initially, and thus may go unnoticed for a bit.

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

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

12.1.4 Suicide and Self-Harm

12.1.4.1. Suicide. Suicidal ideation is the act of thinking, considering or planning suicide. Suicide is death that is caused by an intention, self-directed injurious behavior with the purpose to ended 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 event “suicide attempt” from our day to day, and empirical, language. The reason for this is because it has negative connotation. For example, where else to we use the word committed? I’ll give you a moment to think on this. That’s right, “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” judgement. Negative connotations with both of these scenarios is

unnecessary and can be particularly painful for family members that have had a loved one die by suicide or for other individual that have themselves engaged in suicidal actions. As such, for the purpose of this class (and I hope for your any conversations you have moving forward), you will use terms such as ***died by suicide*** and perhaps even *suicidal actions,* rather than *committed suicide, suicide attempt, failed suicide attempt,* etc.. Also, please know that asking someone about suicide does **NOT** make someone suicidal.

While there are many theories about 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**, 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** is 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, and 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).

12.1.4.2. Nonsuicidal Self-Injury (NSSI). 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. For example, an individual with NSSI may have no suicidal ideation but regularly engage in NSSI. Examples of NSSI may include cutting, burning, or picking one's skin. Essentially, any action that injures one's self, 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. However, 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, self-harm should be seriously addressed and treated.

12.2. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe the prevalence and comorbidity of depressive disorders.
- Describe the prevalence and comorbidity of bipolar disorders.
- Describe the prevalence and comorbidity of depressive disorders and DMDD
- Describe the prevalence of suicidality.

12.2.1. Depressive Disorders

According to the DSM-5 (APA, 2013) the prevalence rate for major depression is approximately 7% within the United States. The prevalence rate for persistent depressive disorder is much lower, with a 0.5% rate among adults in the United states. The CDC indicates that 7.6% of individuals aged 12 and older experience depression. The CDC provided further information indicating that 2.1% of children between the ages of 3 and 17 have depression (CDC, 2018, March). Depression is approximately 1.5 to 3 times higher in females than males. The estimated lifetime prevalence for major depressive disorder in females is 21.3% compared to 12.7% in men (Nolen-Hoeksema, 2001).

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

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

12.2.2. Disruptive Mood Dysregulation Disorder

Disruptive Mood Dysregulation Disorder (DMDD) is a newer diagnosis and very little is known about its prevalence. Like depression, comorbidity is high for DMDD. The specific diagnoses that are comorbid with DMDD vary and include other mood (although one cannot be diagnosed with DMDD and bipolar disorder), anxiety, and other behavioral disorders.

12.2.3. Bipolar Disorders

Compared to depression, the epidemiological studies on the rates of bipolar Ddisorder suggest a significantly lower prevalence rate for both bipolar I and bipolar II. Within the two disorders, there is a very minimal difference in the prevalence rates with yearly rates reported as 0.6% and 0.8% in the United States for bipolar I and bipolar II, respectively (APA, 2013). In youth specifically, the prevalence rate is estimated to be approximately 1.8% to 2.6% (Goldstein & Bimaher, 2012; NIMH, 2017). As for gender differences, there are no apparent differences in the frequency of males and females diagnosed with bipolar I; however, bipolar II appears to be more common in females, with approximately 80-90% of individuals with rapid-cycling episodes being females (Bauer & Pfenning, 2005). Females are also more likely to experience rapid cycling between manic/hypomanic episodes and depressive episodes.

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

12.2.4. Suicidality

Suicidality in depressive disorders, particularly bipolar disorder, is much higher than the general public. In depressive disorders, males and those with a past history of suicide attempts/threats are most at risk for attempting suicide. Individuals with bipolar disorder are approximately 15 times greater than the general population to attempt suicide. Prevalence rates of suicide attempts in bipolar patients is estimated to be 33%. Furthermore, bipolar disorder may account for one-quarter of all completed suicides (APA, 2013). Males are more likely to die by suicide whereas females have higher numbers of suicidal actions. The reason for this is likely that males tend to choose more lethal means of suicide (for example, firearms), ultimately leading to death by suicide following suicidal actions whereas females typically engage in suicidal actions with less lethal means (for example, medication) with the opportunity for resuscitation following suicidal actions.

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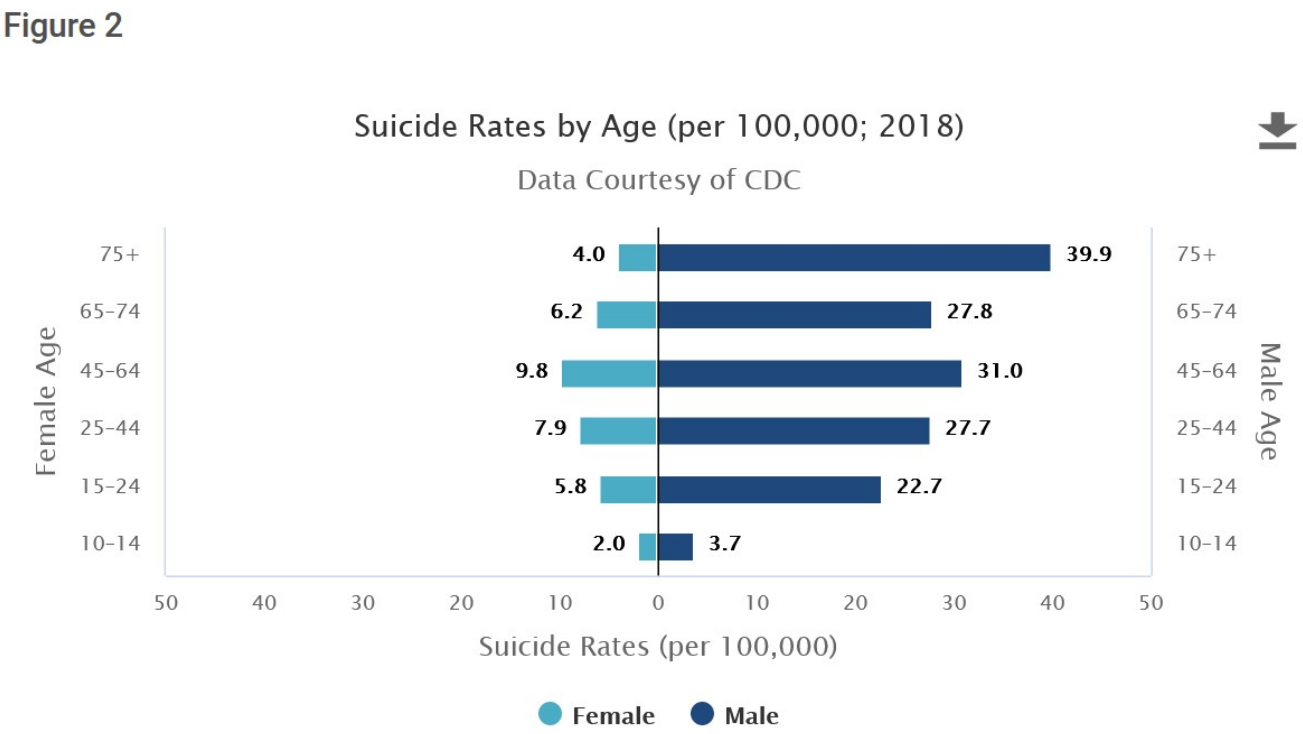
Suicide is the second leading cause of death in the age group of 10 to 34 years of age (NIH, n.d.). Below are charts, directly from NIH (n.d.) that depict rates of suicide for each age group, broken down by gender (Figure 12.1).

Figure 12.1 Data graph of Suicide Rates by Age



Note. Graph directly pulled from: https://www.nimh.nih.gov/health/statistics/suicide.shtml#part_154969

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 develop 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, in efforts to determine if there is some genetic component, whether it be a direct link or a predisposition. If there is a genetic predisposition to developing depressive disorders, one would expect a higher rate of depression within families than that of the general population. Researchers support this with regards to depressive disorders as there is nearly a 30 percent increase in relatives diagnosed with depression, compared to 10 percent of the general population (Levinson & Nichols, 2014). Similarly, there is also an elevated prevalence among first-degree relatives for both bipolar I and bipolar II disorders as well.

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

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

Bipolar disorders share a similar genetic predisposition to developing the disorder. Twin studies within bipolar disorder yield concordance rates for identical twins at as high as 72%, and 5-15% for fraternal twins, siblings, and other close relatives. Both of these percentages are significantly higher than that of the general population, suggesting a strong genetic component of bipolar disorder (Edvardsen et al., 2008).

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 was actually discovered accidently in the 1950's when MAOI's were given to tuberculosis patients, and miraculously, their depressive moods also improved. Soon thereafter, medical providers found that medications used to treat high blood pressure by causing a reduction in norepinephrine, also caused depression in their patients (Ayd, 1956).

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

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

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

There are a number of structural abnormalities in individuals with bipolar disorder; however, what or why these structures are abnormal is yet to be determined. Researchers continue to focus on areas of basal ganglia and cerebellum, which appear to be much smaller in individuals with bipolar disorder as opposed to the general public. Additionally, there appears to be a decrease in brain activity in regions

associated with regulating emotions, as well as increase in brain activity among structures related to emotional responsiveness (Houenou et al., 2011). Additional research is still needed to determine exactly how each of these brain structures may be implicated in the development of bipolar disorder.

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 the dogs regardless of their behavior. The following day, the dogs were placed in a similar apparatus; however, this time the dogs were not restrained and there was a small barrier placed between the "shock" floor and the "safe" floor. What Seligman observed was that despite the opportunity to escape the shock, the dogs flurried for a bit, and then ultimately laid down and whimpered while being shocked.

Based on this study, Seligman concluded that the animals essentially learned that they were unable to avoid the shock the day prior, and therefore, learned that they were helpless in avoiding the shocks. When they were placed in a similar environment but had the opportunity to escape the shock, their learned helplessness carried over and they continued to believe they were unable to escape the shock.

This study has been linked to humans through the research in **attributional style** (Nolen-Hoeksema, Girgus & Seligman, 1992). There are two types of attributional style- positive and negative. A negative attributional style focuses on the *internal, stable* and *global* influence of daily lives, whereas a positive attributional style focuses on the *external, unstable,* and *specific* influence of the environment. Research has found that individuals with a negative attributional style are more likely to experience depression. This is likely due to their negative interpretation of daily events. For example, if something bad were to happen to them, they conclude that it is *their* fault (internal), bad things *always* happen to them (stable), and bad things happen *all* day to them. Unfortunately, this maladaptive thinking style often takes over an individual's daily view, thus making them more vulnerable to depression.

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

Maladaptive attitudes, or negative attitudes about 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. Individual's with depressive

symptoms often develop these maladaptive attitudes regarding everything in their life, indirectly isolating themselves from others. The **cognitive triad** also plays into the maladaptive attitudes in that the individual interprets these negative thoughts about their *experiences, themselves*, and their *futures.*

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

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

12.3.3. Behavioral

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

12.3.4. Sociocultural

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

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. Stress can lead to increased rates of depression (Nezlek et al., 2000). Moreover, adverse life events, which may increase stress, increases risk for depression as well (APA, 2013).

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 hallmark in most societies, other symptoms may be more specific to one's nationality. More specifically, individuals from non-Western countries (China and other Asian countries) often focus on the physical symptoms of depression- tiredness, weakness, sleep issues, and less of an emphasis on the cognitive symptoms. Individuals from Latino and Mediterranean cultures often experience problems with "nerves" and headaches as primary symptoms of depression (American Psychiatric Association, 2013).

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

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

The first theory- *artifact theory*- suggests that the difference between genders is due to clinician or diagnostic systems being more sensitive to diagnosing females with depression than males. While females are often thought to be more "emotional," easily expressing their feelings and more willing to discuss their symptoms with clinicians and physicians, males often withhold their symptoms or will present with more traditionally "masculine" symptoms of anger or aggression. While this theory is often a possible explanation for the gender differences in the rate of depression, research has failed to support this theory suggesting that males and females 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 females more than males (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 females' ability to integrate and process emotional information, research fails to support this theory as the reason for higher rates of depression in females (Whiffen & Demidenko, 2006).

The third theory- *life stress theory*- suggests that females are more likely to experience chronic stressors than males, thus accounting for their higher rate of depression (Astbury, 2010). Females are at an increased risk for facing poverty, lower employment opportunities, discrimination, and poorer quality of housing than males, 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 females. For example, males are often encouraged to develop personal autonomy, seek out activities that interest them, and display achievement-oriented goals, females are encouraged to empathize and care for others, often fostering an interdependent functioning, which may cause females to value the opinion of others more highly than their male counterparts do.

The final theory- *rumination theory*– suggests that females 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 there are many theories trying to explain the gender discrepancy in depressive episodes, no one single theory has produced enough evidence to fully explain why females experience depression more than males. Due to the lack of evidence, gender differences in depression remains one of the most researched topics within depression, while simultaneously being the least understood phenomena in the clinical psychology world.

12.4. ASSESSMENT

Section Learning Objectives

- Describe assessment tools commonly used

12.4.1. Assessment

In general, assessment is going to include a thorough interview with a caregiver and the child/adolescent. The Diagnostic Interview Schedule for Children (DISC) may be utilized. It 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, a majority of diagnostic decision making will be reliant on interview reports and objective measures.

Objective measures are also 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.

12.5 TREATMENT

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 treatments include antidepressant medications, Cognitive-Behavioral Therapy (CBT; Beck et al., 1979), Behavioral Activation (BA; Jacobson et al., 2001), and Interpersonal Therapy (IPT; Klerman et al., 1984). Although CBT is the most widely known and used treatment for Major Depressive Disorder, there is minimal evidence to support one treatment modality over the other; treatment is generally dictated by therapist competence, availability, and patient preference (Craighhead & Dunlop, 2014).

12.5.1.1. Psychopharmacology – Antidepressant medications. Antidepressants are often the most common first line attempts 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). SSRI's are among the most common medications used to treat depression due to their relatively benign side effects. Additionally, the required dose to reach therapeutic levels is low compared to the other medication options. Possible side effects from SSRI's include but are not limited to: nausea, insomnia, and reduced sex drive.

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

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 similar to SSRIs in that they work by affecting the brain chemistry,

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

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

12.5.1.4. Psychopharmacology – Monoamine oxidase inhibitors (MAOIs). While the use of MAOI's were found serendipitously after it produced antidepressant effects in a tuberculosis patient in the early 1950's, it has been effective in treating depression in adults. Although they are still prescribed, they are not typically first line medications due to their safety concerns with hypertensive crises. Because of this, individuals on MAOI's have strict diet restrictions in efforts to reduce their risk of hypertensive crises (Shulman, Herrman & Walker, 2013).

How do MAOI's work? In basic terms, monoamine oxidase is released in the brain to remove excess neurotransmitters, norepinephrine, serotonin, and dopamine. MAOI's essentially prevent the monoamine oxidase (hence the name monoamine oxidase *inhibitors*) from removing these neurotransmitters, thus having an increase in these brain chemicals (Shulman, Herman & Walker, 2013). As previously discussed, norepinephrine, serotonin, and dopamine are all involved in the biological mechanisms of maintaining depressive symptoms.

While these drugs are effective, they come with serious side effects. In addition to the hypertensive episodes, they can also cause nausea, headaches, drowsiness, involuntary muscle jerks, reduced sexual desire, and weight gain to name a few (American Psychiatric Association, 2010). Despite these side effects, studies have shown that individual's prescribed MAOI's for depression have a treatment response rate of 50-70% (Krishnan, 2007). Overall, despite their effectiveness, MAOIs are likely the best treatment for later staged, treatment resistant depression in 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 1960's and is a widely practiced therapeutic tool used to treat depression (and other disorders as well). The basics of CBT involve what Beck called the **cognitive triad-** cognitions (thoughts), behaviors, and emotions. Beck believed that these three components are interconnected, and therefore, affect one another. It is believed that CBT can improve emotions in depressed patients by changing both cognitions (thoughts) and behaviors, which in return will improve mood. Common cognitive

interventions with CBT include thought monitoring and recording, identifying cognitive errors, examining evidence supporting/negating cognitions, and creating rational alternatives to maladaptive thought patterns. Behavioral interventions of CBT include activity planning, pleasant event scheduling, task assignments, and coping-skills training.

Cognitive behavioral therapy generally follows four phases of treatment:

- **Phase 1: Increasing pleasurable activities.** Similar to behavioral activation (read below), the clinician encourages the patient to identify and engage in activities that are pleasurable to the individual. The clinician is able to help the patient during session to identify the activity, as well as help them plan out during the week when they will engage in that activity.
- **Phase 2: Challenging automatic thoughts.** During this stage, the clinician provides psychoeducation about the negative automatic thoughts that can maintain depressive symptoms. The patient will learn to identify these thoughts on their own during the week, and maintain a thought journal of these cognitions to review with the clinician in session.
- **Phase 3: Identifying negative thoughts.** Once the individual is consistently able to identify these negative thoughts on a daily basis, the clinician is able to help the patient identify *how* these thoughts are maintaining their depressive symptoms. It is at this point that the patient begins to have direct insight as to how their cognitions contribute to their disorder.
- **Phase 4: Changing thoughts.** The final stage of treatment involves challenging the negative thoughts the patient has been identifying in the last two phases of treatment and replacing them with positive thoughts.

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 both Ferster (1973) and Lewinsohn and colleagues (Lewinsohn, 1974; Lewinsohn, Biglan, & Zeiss, 1976) the goal of BA is to increase the frequency of behaviors so that individuals have opportunities to experience greater contact with sources of reward in their lives. In order to do this, the clinician assists the 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. The thought is if the negative behaviors are minimized and the pleasurable activities are maximized, the individual will receive more positive rewards or reinforcement from others and their environment, thus improving their overall mood.

12.5.1.7. Psychotherapy – Interpersonal therapy (IPT). IPT was developed by Klerman, Weissman, and colleagues in the 1970's as a treatment arm for a pharmacotherapy study of depression (Weissman,

1995). The treatment was created based off data from post-World War II individuals who expressed a significant impact on their psychosocial life events. Klerman and colleagues noticed a significant relationship between the development of depression and complicated bereavement, role disputes, role transitions, and interpersonal deficits in these individuals (Weissman, 1995). The idea behind IPT therapy is that depressive episodes compromise interpersonal functioning, which in return, makes it difficult to manage stressful life events. The basic mechanism of IPT is to establish effective strategies to manage interpersonal issues, which in return, will ameliorate depressive symptoms.

There are two main principles of IPT. First, depression is a common, medical illness, with a complex and multi-determined etiology. Since depression is a medical illness, it is also treatable and *not* the patient's fault. Second, depression is connected to a current or recent life event. The goal of IPT is to identify the interpersonal problem that is connected to the depressive symptoms and solving this crisis so the patient can improve their life situation while relieving depressive symptoms.

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

12.5.1.9. A note about treating DMDD. Specific treatment guidelines have not been established for DMDD. Given its relatively new emergence, we still have a long way to go before we fully understand treatment options. However, some research that examine presentations closely related to DMDD 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). Some 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). However, again, these studies are not specifically examining children with DMDD; and as such, we cannot necessarily translate these findings directly to children with DMDD.

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

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

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. The 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. 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 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 in 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 this 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)

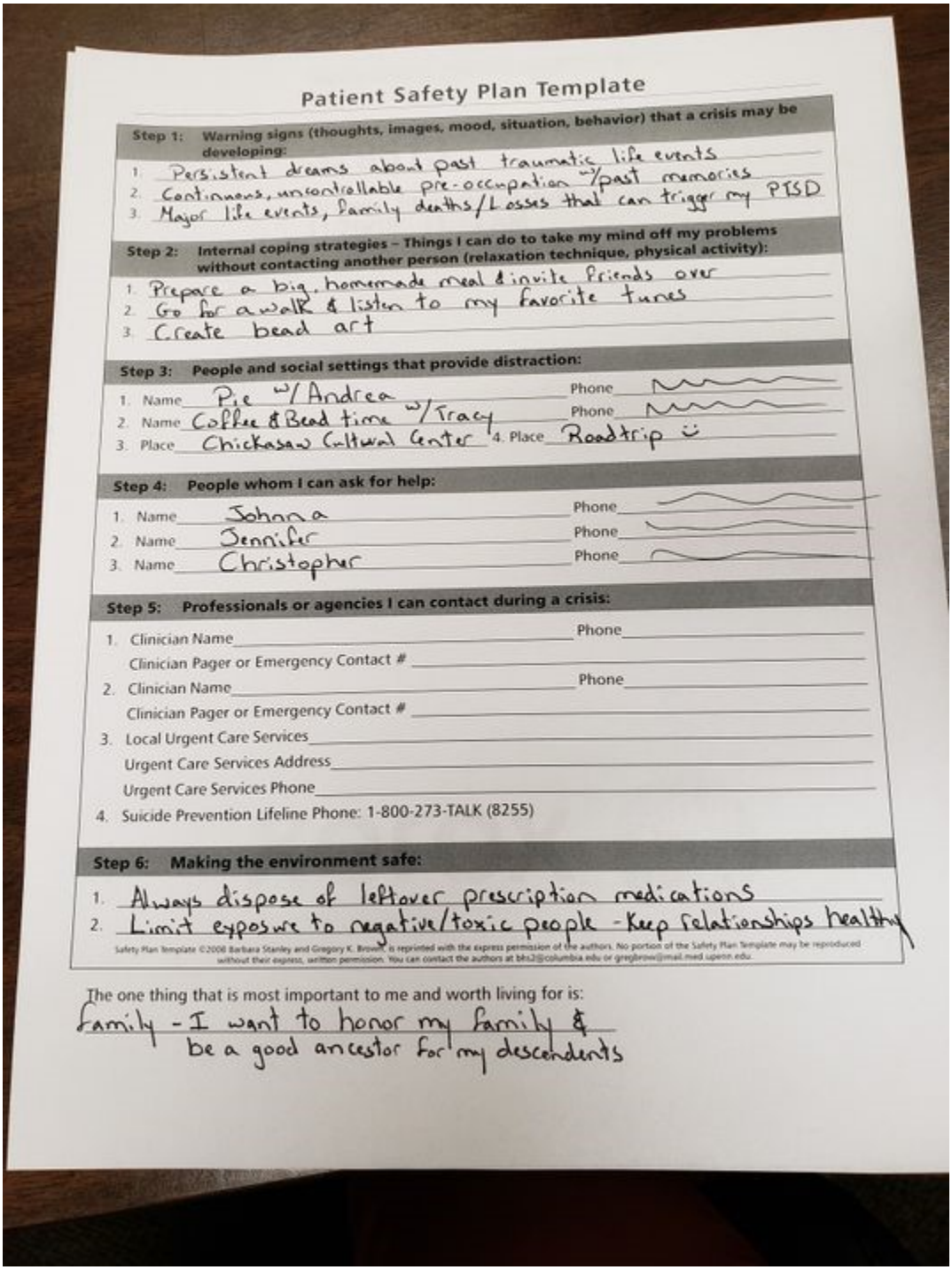
Family-focused treatment (FFT) for adolescents with bipolar has also been found to be beneficial (Mikolwitz et al., 2000). It is the only family-related treatment that has been shown to have benefits for adolescents with bipolar. This therapy, originally designed for schizophrenia, includes all immediate family members in therapy. It also focuses on several areas including psychoeducation about bipolar and the symptoms of bipolar, the causes of bipolar, 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, 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. An example of a coping card is provided in Figure 12.3. There are also several phone applications, such as Virtual Hope Box, that can be downloaded that facilitate creating a coping card. Also, a helpful number and agency to utilize is the National Suicide Prevention Lifeline (1.800.273.8255).

Figure 12.2. National Suicide Prevention Lifeline Information

Figure 12.3. Coping Card Example



Patient Safety Plan Template

Step 1: Warning signs (thoughts, images, mood, situation, behavior) that a crisis may be developing:

1. Persistent dreams about past traumatic life events
2. Continuous, uncontrollable pre-occupation w/ past memories
3. Major life events, family deaths/Losses that can trigger my PTSD

Step 2: Internal coping strategies – Things I can do to take my mind off my problems without contacting another person (relaxation technique, physical activity):

1. Prepare a big, homemade meal & invite friends over
2. Go for a walk & listen to my favorite tunes
3. Create bead art

Step 3: People and social settings that provide distraction:

1. Name Pie w/ Andrea Phone
2. Name Coffee & Bead time w/ Tracy Phone
3. Place Chickasaw Cultural Center 4. Place Roadtrip :)

Step 4: People whom I can ask for help:

1. Name Johnna Phone
2. Name Jennifer Phone
3. Name Christopher Phone

Step 5: Professionals or agencies I can contact during a crisis:

1. Clinician Name Phone
   Clinician Pager or Emergency Contact #
2. Clinician Name Phone
   Clinician Pager or Emergency Contact #
3. Local Urgent Care Services
   Urgent Care Services Address
   Urgent Care Services Phone
4. Suicide Prevention Lifeline Phone: 1-800-273-TALK (8255)

Step 6: Making the environment safe:

1. Always dispose of leftover prescription medications
2. Limit exposure to negative/toxic people - Keep relationships healthy

The one thing that is most important to me and worth living for is:
family - I want to honor my family & be a good ancestor for my descendents

Note: Picture directly pulled from USA TODAY at https://www.usatoday.com/story/news/investigations/2018/11/28/suicide-prevention-if-youve-had-suicidal-thoughts-do-safety-plan/2018051002/?fbclid=IwAR1SpVW20HKE1kl7tZ3-m9Xrc_kXzKrsb2_9lMWhWsTWxB86zAzdi0oILt8

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 efficacious; however, research supports that while psychopharmacological interventions are more effective in rapidly reducing symptoms, psychotherapy or even a combination treatment approach are more effective in establishing long-term relief of symptoms.

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

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

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. 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.

2nd edition

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, selective mutism, generalized anxiety, specific phobia, agoraphobia, social anxiety, and panic disorders. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

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

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

13.1.1. Separation Anxiety Disorder

Separation anxiety disorder is 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. They may refuse to go to school or other places due to, by nature, forced separation. They may refuse to sleep in their own bed or have recurring nightmares that involve separation from their caregiver. These children may also have several physical manifestations of anxiety including headaches and stomachaches when they are separated from their caregiver.

13.1.2. Selective Mutism

This disorder is characterized by an absence of speech in particular social situations in which a person is expected to speak. This lack of speech is not due to a language disorder or a lack of understanding that speaking is expected in the particular situation; rather, the lack of speech is due to excessive

anxiety (APA, 2013). 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.

13.1.3. Generalized Anxiety (GAD)

Generalized anxiety disorder, commonly referred to as GAD, is a disorder characterized by an underlying excessive worry related to a wide range of events or activities. While many individuals experience some levels of worry throughout the day, individuals with GAD experience worry of a greater intensity and for longer periods of times than the average person. Additionally, they are often unable to control their worry through various coping strategies, which directly interferes with their ability to engage in daily social and academic tasks. Individuals with GAD will also experience somatic symptoms during intensive periods of anxiety as listed below.

13.1.4. Specific Phobia

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

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

13.1.5. Agoraphobia

Similar to GAD, **agoraphobia** is defined as an intense fear triggered by a wide range of situations; however, unlike GAD, agoraphobia's fears are related to situations in which the individual is in public situations where escape may be difficult. In order to receive a diagnosis of agoraphobia, there must be a presence of fear in at least two of the following situations: using public transportation such as planes, trains, ships, buses; being in large, open spaces such as parking lots or on bridges; being in enclosed

spaces like stores or movie theaters; being in a large crowd similar to those at a concert; or being outside of the home in general (APA, 2013). When an individual is in one (or more) of these situations, they experience significant fear, often reporting panic-like symptoms (see Panic Disorder). It should be noted that fear and anxiety related symptoms are present *every time* the individual is presented with these situations. Should symptoms only occur occasionally, a diagnosis of agoraphobia is not warranted.

Due to the intense fear and somatic symptoms, individuals will go to great lengths to avoid these situations, often preferring to remain within their home where they feel safe, thus causing significant impairment in one's daily functioning. They may also engage in active avoidance, where the individual will intentionally avoid agoraphobic situations. These avoidance behaviors may be behavioral or cognitive. 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. Cognitive avoidance may include using distraction and various other cognitive techniques to successfully get through the agoraphobic situation.

13.1.6. Social Anxiety Disorder

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

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

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

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 or fear or impending doom along with at least four physical or cognitive symptoms (listed below). The symptoms generally peak within a few minutes, although it seems much longer for the individual experiencing the panic attack.

There are two key components to panic disorder—the attacks are *unexpected* meaning there is nothing that triggers them, and they are *recurrent* meaning they occur multiple times. Because these panic attacks occur frequently and essentially "out of the blue," they cause significant worry or anxiety in the individual as they are unsure of when the next attack will occur. In some individuals, significant behavioral changes such as fear of leaving their home or attending large events occurs as the individual is fearful an attack will happen in one of these situations, causing embarrassment. Additionally, individuals report worry that 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 (see more below).

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

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 Phobias.
- 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 is relatively common, in comparison to other disorders, occurring in 4% of children. In adolescence, it is less common, occurring in only 1.6% of adolescents. Separation disorders is highly comorbid with other anxiety disorders (APA, 2013).

13.2.2. Selective Mutism

This disorder is relatively rare. It occurs in only .3 to 1% of the population and is more common in young children. Again, selective mutism is highly comorbid with other anxiety disorders, particularly social anxiety disorder (APA, 2013).

13.2.3. Generalized anxiety disorder

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

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

13.2.4. Specific phobia

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

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

substance related disorders and somatic symptom disorders.

13.2.5. Agoraphobia

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

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

13.2.6. Social anxiety disorder

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

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

13.2.7. Panic disorder

Prevalence rates for panic disorder are estimated at around 2-3% in adults and adolescents. Higher rates of panic disorder are found in American Indians and non-Latino whites. Females are more commonly diagnosed than males with a 2:1 diagnosis rate—this gender discrepancy is seen throughout

the lifespan. Although panic disorder can occur in young children, it is generally not observed in individuals younger than 14 years of age.

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

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

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

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 in efforts to prepare the body for a response. First, the amygdala triggers the hypothalamic-pituitary-adrenal (HPA) axis to prepare for immediate action— either to fight or flight. The second pathway is activated by the feared stimulus itself, by sending a sensory signal to the **hippocampus** and **prefrontal cortex**, for determination if the threat is real or imagined. If it is determined that no threat is present, the amygdala sends a calming response to the HPA axis, thus reducing the level of fear. If there is a threat present, the amygdala is activated, producing a fear response.

Specific to *panic disorder* is the implication of the **locus coeruleus**, the brain structure that serves as an "on-off" switch for norepinephrine neurotransmitters. It is believed that increased activation of the locus coeruleus results in panic like symptoms; therefore, individuals with panic disorder may have a hyperactive locus coeruleus, leaving them more susceptible to experience more intense and frequent physiological arousal than the general public (Gorman, Kent, Sullivan, & Coplan, 2000). This theory is supported by studies in which individuals experienced increased panic symptoms following injection of norepinephrine (Bourin, Malinge, & Guitton, 1995).

Unfortunately, norepinephrine and the locus coeruleus fail to fully explain the development of panic disorder, as treatment would be much easier if *only* norepinephrine was implicated. Therefore, researchers argue that a more complex neuropathway is likely implicated in the development of panic disorder. More specifically, the **corticostriatal-thalamocortical (CSTC) circuit**, also known as the fear-specific circuit, is theorized as a major contributor to panic symptoms (Gutman, Gorman, & Hirsch, 2004). When an individual is presented with a frightening object or situation, the amygdala is activated, sending a fear response to the anterior cingulate cortex and the orbitofrontal cortex. Additional projection from the amygdala to the hypothalamus activates endocrinologic responses to fear- releasing adrenaline and cortisol to help prepare the body to fight or flight (Gutman, Gorman, & Hirsch, 2004). This complex pathway supports the theory that panic disorder is mediated by several neuroanatomical structures and their associated neurotransmitters.

13.3.2. Psychological

13.3.2.1. Psychological – Cognitive. The cognitive perspective on the development of anxiety related disorders centers around dysfunctional thought patters. As seen in depression, **maladaptive assumptions** are routinely observed in individuals with anxiety related disorders, as they often engage in interpreting events as dangerous or overreacting to potential stressful events, which contributes to a heightened overall anxiety level. These **negative appraisals**, in combination with a biological predisposition to anxiety likely contribute to the development of anxiety symptoms (Gallagher et al., 2013).

Sensitivity to physiological arousal not only contributes to anxiety disorders in general, but also for panic disorder where individuals experience various physiological sensations and misinterpret them as catastrophic. One explanation for this theory is that individuals with panic disorder are actually more susceptible to more frequent and intensive physiological symptoms than the general public (Nillni, Rohan, & Zvolensky, 2012). Others argue that these individuals have had more trauma-related experiences in the past, and therefore, are quick to misevaluate their symptoms as a potential treat. This misevaluation of symptoms as impending disaster likely maintain symptoms as the cognitive misinterpretations to physiological arousal creates a negative feedback loop, leading to more physiological changes.

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

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

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

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

13.3.4. Sociocultural

Seeing how prominent the biological and psychological constructs are in explaining the development of anxiety related disorders, we also need to review the social constructs that contribute and maintain anxiety disorders. While characteristics such as living in poverty, experiencing significant daily stressors, and increased exposure to traumatic events are all identified as major contributors to anxiety disorders, additional sociocultural influences such as gender and discrimination have also received great attention, particularly due to the epidemiological nature of the disorder.

Gender has largely been researched within anxiety disorders due to the consistent discrepancy in diagnosis rate between males and females. As previously discussed, females are routinely diagnosed with anxiety disorders more often than males, a trend that is observed throughout the entire lifespan. One potential explanation for this discrepancy is the influence of social pressures on females. Females are more susceptible to experience traumatic experiences throughout their life, which may contribute to anxious appraisals of future events. Furthermore, females 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 females that leave them susceptible to develop symptoms of anxiety. Therefore, it appears a combination of genetic, environmental, and social factors may explain why females tend to be diagnosed more often with anxiety related disorders.

Exposure to discrimination and prejudice, particularly relevant to ethnic minority and other marginalized groups, can also impact an individual’s anxiety level. Discrimination and prejudice contribute to negative interactions, which is directly related to negative affect and an overall decline in mental health (Gibbons et al., 2014). The repeated exposure to discrimination and prejudice over time can lead to fear responses in individuals, along with subsequent avoidance of social situations in efforts to protect themselves emotionally.

13.4. ASSESSMENT

Section Learning Objectives

- Describe assessment tools commonly used

13.4.1. Assessment

Similar to assessing for depression, assessment is going to include a thorough interview with a caregiver and the child/adolescent. The Diagnostic Interview Schedule for Children (DISC) may be utilized. It 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 assess for childhood psychological disorders.

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

Objective measures are also 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 anxiety are utilized. The Revised Children's Manifest Anxiety Scale (RCMAS) and
Multidimensional Anxiety Scale for Children, 2nd Edition (MASC 2) are frequently used narrow-band (i.e., specifically looking at anxiety) measures of anxiety.

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 Phobias.
- 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. Described in detail in this section, exposure therapy is often utilized to reduce anxiety reactions. 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. Similar to that in exposure-based treatment for phobias, prior to engaging in exposure training, the individual must learn a relaxation technique to apply during onset of panic attacks. 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 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).

13.5.2. 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 progressing to increasing speech in the real-world setting. In addition to the above strategies listed, social skills training may also help children with selective mutism, especially given the high comorbidity of social anxiety disorder. Social skills training is discussed in 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 chapter, originally replaced barbiturates as the leading

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

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

13.5.3.3. Cognitive Behavioral Therapy (CBT). CBT is discussed in great detail in the Depression chapter; however, it is also among the most effective treatment options for a variety of anxiety related disorders, including GAD. In fact, findings suggest 60 percent of individuals report a significant reduction/elimination in anxious thoughts one-year post treatment (Hanrahan, Field, Jones, & Davy, 2013). The fundamental goal of CBT is a combination of cognitive and behavioral strategies aimed to identify and restructure maladaptive thoughts while also providing opportunities to utilize these more effective thought patterns through exposure-based experiences. Through repetition, the individual will be able to identify and replace anxious thoughts outside of therapy sessions, ultimately reducing their overall anxiety levels (Borkovec, & Ruscio, 2001).

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 of their physiological states. There are several ways a patient can be connected to the computer. Among the most common is electromyography (EMG). **EMG** measures the amount of muscle activity currently experienced by the individual. An electrode is placed on a patient's skin just above a major muscle group- commonly the forearm or the forehead. Other common areas of measurement are **electroencephalography** (EEG) which measures the neurofeedback or brain activity; **heart rate variability** (HRV) which measures autonomic activity such as heart rate or blood pressure; and **galvanic skin response** (GSR) which measures sweat.

Once the patient is connected to the biofeedback machine, the clinician is able to walk the patient through a series of relaxation scripts or techniques as the computer simultaneously measures the

changes in muscle tension. The theory behind biofeedback is that in providing a patient with a visual representation of changes in their physiological state, they become more skilled at voluntarily reducing their physiological arousal, and thus, their overall sense of anxiety or stress. While research has identified only a modest effect of biofeedback on anxiety levels, patients do report a positive experience with the treatment due to the visual feedback of their physiological arousal (Brambrink, 2004).

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 are developed via classical conditioning, the treatment approach revolves around breaking the maladaptive association developed between the object and fear. This is generally accomplished through **exposure treatments**. As the name implies, the individual is *exposed* to their feared stimuli. This can be done in several different approaches: *systematic desensitization, flooding, and modeling.*

Systematic desensitization is an exposure technique that utilizes relaxation strategies to help calm the individual as they are presented with the fearful object. The notion behind this technique is that both fear and relaxation cannot exist at the same time; therefore, the individual is taught how to replace their fearful reaction with a calm, relaxing reaction.

To begin, the patient, with assistance from the clinician, will identify a *fear hierarchy*, or a list of feared objects/situations ordered from least fearful to most fearful. After learning intensive relaxation techniques, the clinician will present items from the fear hierarchy- starting from the least fearful object/subject- while the patient practices using the learned relaxation techniques. The presentation of the feared object/situation can be in person- **in vivo exposure** or it can be imagined- **imaginal exposure.** Imaginal exposure tends to be less intensive than in vivo exposure; however, it is less effective than in vivo exposure in eliminating the phobia. Depending on the phobia, in vivo exposure may not be an option, such as with a fear of a tornado. Once the patient is able to effectively employ relaxation techniques to reduce their fear/anxiety to a manageable level, the clinician will slowly move up the fear hierarchy until the individual does not experience excessive fear of all objects on the list.

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

13.5.4.2. Modeling. Finally, **modeling** is another common technique that is used to treat phobia disorders (Kelly, Barker, Field, Wilson, & Reynolds, 2010). In this technique, the clinician approaches

the feared object/subject while the patient observes. Like the name implies, the clinician models appropriate behaviors when exposed to the feared stimulus, implying that the phobia is irrational. After modeling several times, the clinician encourages the patient to confront the feared stimulus with the clinician, and then ultimately, without the clinician.

13.5.5. Agoraphobia

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

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

13.5.6. Social Anxiety Disorder

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

13.5.6.2. Social skills training. This treatment is specific to social anxiety disorder as it focuses on

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

13.5.6.3. Cognitive restructuring. While exposure and social skills training are helpful treatment options, research routinely supports the need to incorporate cognitive restructuring as an additive component in treatment to provide substantial symptom reduction. Similar to cognitive restructuring previously discussed in the Depression chapter, the clinician will work with the patient 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).

13.5.7.2. Psychoeducation. Treatment begins by educating the patient on the nature of panic disorder, the underlying causes of panic disorder, as well as the mechanisms that maintain the disorder such as the physical, cognitive, and behavioral response systems (Craske & Barlow, 2014). This part of treatment is fundamental in correcting any myths or misconceptions about panic symptoms, as they often contribute to the exacerbation of panic symptoms.

13.5.7.3. Self-monitoring. Self-monitoring, or the awareness of self-observation, is essential to the CBT treatment process for panic disorder. In this part of treatment, the individual is taught to identify the physiological cues immediately leading up to and during a panic attack. The patient is then encouraged to identify and document the thoughts and behaviors associated with these physiological symptoms. By bringing awareness to the symptoms, as well as the relationship between physical arousal and cognitive/behavioral responses, the patient is learning the fundamental processes in which they can manage their panic symptoms (Craske & Barlow, 2014).

13.5.7.4. Relaxation training. Similar to that in exposure-based treatment for phobias, prior to

engaging in exposure training, the individual must learn a relaxation technique to apply during onset of panic attacks. While breathing training was once included as the relaxation training technique of choice for panic disorder due to the high report of hyperventilation during panic attacks, more recent research has failed to support this technique as effective in the use of panic disorder (Schmidt et al., 2000). Findings suggest that breathing retraining is more commonly misused as a means for avoiding physical symptoms as opposed to an effective physiological response to stress (Craske & Barlow, 2014).

13.5.7.5. Progressive muscle relaxation. To replace the breathing retraining, Craske & Barlow (2014) suggest **progressive muscle relaxation** (PMR). In progressive muscle relaxation, the patient learns to tense and relax various large muscle groups throughout the body. Generally speaking, the patient is encouraged to start at either the head or the feet, and gradually work their way up through the entire body, holding the tension for roughly 10 seconds before relaxing. The theory behind PMR is that in tensing the muscles for a prolonged period of time, the individual exhausts those muscles, forcing them (and eventually) the entire body to engage in relaxation (McCallie, Blum, & Hood, 2006).

13.5.7.6. Cognitive restructuring. Cognitive restructuring, or the ability to recognize cognitive errors and replace them with alternate, more appropriate thoughts, is likely the most powerful part of CBT treatment for panic disorder, aside from the exposure part. Similar to the discussion in the Depression chapter, cognitive restructuring involves identifying the role of thoughts in generating and maintaining emotions. The clinician encourages the patient to view these thoughts as "hypotheses" as opposed to fact, which allows the thoughts to be questioned and challenged. This is where the detailed recordings in the self-monitoring section of treatment is helpful. By discussing specifically what the patient has recorded for the relationship between physiological arousal and thoughts/behaviors, the clinician is able to help the patient restructure the maladaptive thought processes to more positive thought processes which in return, helps to reduce fear and anxiety.

13.5.7.7. Exposure. As discussed in detail above in the specific phobia section, the patient is next encouraged to engage in a variety of exposure techniques such as in vivo exposure and *interoceptive exposure*, while also incorporating the cognitive restructuring and relaxation techniques previously learned in efforts to reduce and eliminate ongoing distress. **Interoceptive exposure** involves inducing panic specific symptoms to the individual repeatedly, for a prolonged time period, so that maladaptive thoughts about the sensations can be disconfirmed and conditional anxiety responses are extinguished (Craske & Barlow, 2014). Some examples of these exposure techniques are spinning a patient repeatedly in a chair to induce dizziness and breathing in a paper bag to induce hyperventilation. These treatment approaches can be presented in a gradual manner; however, the patient must endure the physiological sensations for at least 30 seconds to 1 minute to ensure adequate time for applying cognitive strategies to misappraisal of cognitive symptoms (Craske & Barlow, 2014).

Interoceptive exposure is continued both in and outside of treatment until panic symptoms remit. Over time, the habituation of fear within an exposure session will ultimately lead to habituation across treatment, which leads to long-term remission of panic symptoms (Foa & McNally, 1996). Occasionally, panic symptoms will return in individuals who report complete remission of panic disorder. Follow-up

booster sessions reviewing the steps above is generally effective in eliminating symptoms again.

13.5.7.8. Pharmacological interventions. According to Craske & Barlow (2014), nearly half of patients with panic disorder present to psychotherapy already on medication, likely prescribed by their primary care physician. Some researchers argue that anti-anxiety medications impede the progress of CBT treatment as the individual is not able to fully experience the physiological sensations during exposure sessions, thus limiting their ability to modify maladaptive thoughts maintaining the panic symptoms. Results from large clinical trials suggest *no advantage* during or immediately after treatment of combining CBT and medication (Craske & Barlow, 2014). Additionally, when medication was discontinued post treatment, the CBT+ medication groups fared worse than the CBT treatment alone groups, thus supporting the theory that immersion in interoceptive exposure is limited due to the use of medication. Therefore, it is suggested that medications are reserved for those who do not respond to CBT therapy alone (Kampman, Keijers, Hoogduin & Hendriks, 2002).

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 [mother] 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 trying to get her in the car or during the car drive. 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 her school nurse she was feeling ill while at school, but as soon as she gets home, she is feeling 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 sleep over 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 specific disorders. This discussion included Separation Anxiety Disorder, Selective Mutism, Generalized Anxiety Disorder, Specific Phobias, Agoraphobia, Social Anxiety Disorder, and Panic Disorder. As with other modules in this book, we discussed the clinical presentation, epidemiology, comorbidity, and etiology of the anxiety disorders. Treatment options were also given and included biological, psychological, and sociocultural options. In Module 14 we will discuss obsessive-compulsive disorders.

2nd edition

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, body dysmorphic, muscle dysmorphia, trichotillomania, and excoriation 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

- 14.1. Clinical Presentation
- 14.2. Prevalence and Comorbidity
- 14.3. Etiology
- 14.4. Assessment and Treatment

Module Learning Outcomes

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

14.1. CLINICAL PRESENTATION

Section Learning Objectives

- Describe how OCD presents itself.
- Describe how BDD presents itself.
- Describe how trichotillomania and excoriation disorder presents itself.

14.1.1. Obsessive Compulsive Disorder

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

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

These obsessions and compulsions are more excessive than the typical "cleanliness" as they consume a large part of the individual's day. Additionally, they cause significant impairment in one's daily functioning. Given the example above, an individual with a fear of contamination may refuse to eat out at restaurants or may bring their own utensils with them and insist on using them when they are not eating at home.

14.1.2. Body Dysmorphic Disorder

Body dysmorphic disorder (BDD) is another obsessive disorder, however, the focus of these obsessions are with one's perceived defects or flaws in their physical appearance. A key feature of these obsessions

is that they are *not* observable to others. An individual who has a congenital facial defect or a burn victim who is concerned about their scars are ***not*** examples of an individual with BDD. The obsessions related to one's appearance can run the spectrum from feeling "unattractive" to "looking hideous." While any part of the body can be a concern for an individual with BDD, the most commonly reported areas are skin- such as acne, wrinkles, skin color, hair-particularly thinning, excessive body hair, or nose size.

Due to the distressing nature of the obsessions regarding one's body, individuals with BDD also engage in compulsive behaviors that take up a considerable amount of time in one's day. For example, one may repeatedly compare their body to other people's bodies in the general public; repeatedly look at themselves in the mirror; engage in excessive grooming which includes using make-up to modify their appearance. Some individuals with BDD will go as far as having numerous plastic surgeries in attempts to obtain their "perfect" appearance. While most of us are guilty of engaging in some of these behaviors, to meet criteria for BDD, one must spend a considerable amount of time preoccupied with their appearance (i.e on average 3-8 hours a day), as well as display significant impairment in social, home, school, or other areas of functioning.

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 appropriate amount of muscle definition (Ahmed, Cook, Genen & Schwartz, 2014). While severity of BDD between individuals with and without muscle dysmorphia appears to be the same, some studies have found a higher use of substance abuse (i.e. steroid use), poorer quality of life, and increased reports of suicide attempts in those with muscle dysmorphia (Pope, Pope, Menard, Fay Olivardia, & Philips, 2005).

14.1.3. Trichotillomania and Excoriation

Trichotillomania "Hair-Pulling" disorder is characterized by an individual recurrently pulling their hair our and results in hair loss. Despite attempts to cease the behavior, the individual continues to pull their hair out. These behaviors cause distress and impairment and may also lead to other dermatological/medical conditions (APA, 2103).

Excoriation "Skin Picking" Disorder is similar to trichotillomania, except it involves skin picking rather than hair pulling. The individual regularly pulls at skin lesions despite frequent attempts to stop. The skin picking causes impairment and may also lead to other medical consequences (APA, 2013).

14.2. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe prevalence and comorbidity of Obsessive-Compulsive Disorder.
- Describe prevalence and comorbidity of Body Dysmorphia Disorder.
- Describe prevalence and comorbidity of Trichotillomania and Excoriation Disorder.

14.2.1. OCD

The prevalence rate for OCD is approximately 1.2% both in the US, and worldwide (APA, 2013). Specific prevalence rates for children and adolescents are estimated to be anywhere between .25 to 4% (Krebs & Heyman, 2015). Similar to other anxiety related disorders, females are diagnosed with OCD more often than males; however, in childhood, boys are actually diagnosed more frequently than girls (APA, 2013). With respect to gender and symptoms, females are more likely to be diagnosed with cleaning-related obsessions and compulsions, whereas males are more likely to display symptoms related to forbidden thoughts and symmetry (APA, 2013). Additionally, males have an earlier age of onset (5-15 years) compared to females (20-24 years; Rasmussen & Eisen, 1990). Approximately two-thirds of all individuals with OCD had some symptoms present before the age of 15 (Rasmussen & Eisen, 1990).

There is a high comorbidity rate between OCD and other anxiety disorders. Nearly 76% of individuals with OCD will be diagnosed with another anxiety disorder, most commonly panic disorder, social anxiety disorder, generalized anxiety disorder, or a specific phobia (APA, 2013). Additionally, due to the nature of OCD and its symptoms, nearly 41% of those with OCD will also be diagnosed with a major depressive episode (APA, 2013).

There is a high comorbidity rate between OCD and tic disorder, particularly in males with an onset of OCD in childhood. Children presenting with early onset OCD typically have a different presentation of symptoms than traditional OCD. Specifically early onset OCD is typically more common in males, associated with more severe presentations and higher prevalence rates, is associated with a higher likelihood of comorbid tics and other obsessive-compulsive disorders, and is associated with a familial history of OCD. Research has also indicated a strong triad of OCD, tic disorder, and attention-deficit/hyperactivity disorder in children. Due to this psychological disorder triad, it is believed there is a neurobiological mechanism at fault for development and maintenance of the disorders.

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

14.2.2. BDD

The point prevalence rate for BDD among US adults is 2.4% (APA, 2013). Internationally, this rate drops to 1.7%-1.8% (APA, 2013). Despite the difference between the national and international prevalence rates, the symptoms across races and cultures appears similar. Specific prevalence rates for youth are not as well known. The unknown prevalence in youth is interesting given that it is reported that most individuals (i.e., adults) with BDD experienced their first symptoms of the disorder in adolescence (Phillips, 2001) with age of first onset of symptoms as early as age 11 (Phillips, 2006). Although in adults there doesn't seem to be a specific difference in prevalence regarding ethnic and racial identify, when examining prevalence in adolescents, BDD presents at a higher rate in ethnic and racial minority adolescents. Regarding the presentation of BDD, the area in which adolescents and adults tend to focus on most commonly are on their skin, hair, stomach, weight, or teeth (Phillips, 2006).

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

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

14.2.3. Trichotillomania and Excoriation

Trichotillomania disorder 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 by this disorder (APA, 2013).

Excoriation disorder occurs in 1.4% of the population. Over 75% of individuals with this disorder are female (APA, 2013).

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, obsessions were more specific to the family members than that of the disorder. This suggests that there is a stronger heritability association for obsessions than compulsions.

This study also found a relationship between age of onset of OCD symptoms and family heritability. Individuals who experienced an earlier age of onset, particularly before age 17, were found to have more first-degree relatives diagnosed with OCD. In fact, after the age of 17, there was no relationship between family diagnoses, suggesting those who develop OCD at an older age may have a different diagnostic origin (Nestadt, et al., 2000).

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

14.3.1.2. Neurotransmitters. Neurotransmitters, particularly serotonin have been identified as a contributing factor to obsessive and compulsive behaviors. This discovery was actually on accident, when individuals with depression and comorbid OCD were given antidepressant medications clomipramine and/or fluoxetine- both of which increase levels of serotonin- to mediate symptoms of depression. Not only did these patients report a significant reduction in their depressive symptoms, but also significant improvement in their OCD symptoms (Bokor & Anderson, 2014). Interestingly enough, antidepressant medications that do not 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 a direct involvement in the development of obsessive-compulsive behaviors, it's only logical that brain structures that house these neurotransmitters also likely play a role in symptom development. Neuroimaging studies implicate the brain structures and circuits in the frontal lobe, more specifically, the orbitofrontal cortex, which is located just above each eye (Marsh et al., 2014). This brain region is responsible for mediating strong emotional responses and converts them into behavioral responses. Once the orbitofrontal cortex receives sensory/emotional information via sensory inputs, it transmits this information through impulses. These impulses are then passed on to the caudate nuclei which filters through the many impulses received, passing along only the strongest impulses to the thalamus. Once the impulses reach the thalamus, the individual essentially reassesses the emotional response and decides whether or not to act behaviorally (Beucke et al., 2013). It is believed that individuals with obsessive compulsive behaviors experience over activity of the orbitofrontal cortex and a lack of filtering in the caudate nuclei, thus causing too many impulses transferred to the thalamus (Endrass et al., 2011). Further support for this theory has been shown when individuals with OCD experience brain damage to the orbitofrontal cortex or caudate nuclei and experience remission of OCD symptoms (Hofer et al., 2013).

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, control, or uncertainty in their life, thus leading them to over interpret potential negative outcomes of events. Additionally, some research has indicated that those with OCD also experience disconfirmatory bias, which causes the individual to seek out evidence that proves they failed to perform the ritual or compensatory behavior incorrectly (Sue, Sue, Sue, & Sue, 2017). Finally, individuals with OCD often report the inability to trust themselves and their instincts, and therefore, feel 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).

Now that we have identified that individuals with OCD experience cognitive biases, and that these biases contribute to the obsessive and compulsive behaviors, we have yet to identify why these cognitive biases occur so often and why does this happen? Everyone has times when they have repetitive or intrusive thoughts such as: "Did I shut the oven off after cooking dinner?" or "Did I

remember to lock the door before I left home?" Fortunately, most individuals are able to either check up on their thoughts once, or even forgo checking their thoughts after they confidently talk themselves through their actions, ensuring that the behavior in question was or was not completed. Unfortunately, individuals with OCD are unable to neutralize these thoughts without performing a ritual as a way to put themselves at ease. As you will see in more detail in the behavioral section below, the behaviors (compulsions) used to neutralize the thoughts (obsessions) provide a temporary relief to the individual. As the individual is continually exposed to the obsession and repeatedly engages in the compulsive behaviors to neutralize their anxiety, the behavior is repeatedly reinforced, thus becoming a compulsion. This theory is supported by studies where individuals with OCD report using more neutralizing strategies and report significant reductions in anxiety after employing these neutralizing techniques (Jacob, Larson, & Storch, 2014; Salkovskis, et al., 2003).

14.3.3. Behavioral

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

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

14.4. ASSESSMENT AND TREATMENT

Section Learning Objectives

- Provide an overview of assessment options.
- Describe treatment options for OCD.
- Describe treatment options for BDD.

14.4.1 Assessment

Overall, assessing for obsessive-compulsive related disorders is largely based on interviewing and objective measures. Very specific information about the various behaviors and thoughts occurring, the frequency, the context, and the severity is obtained. Thorough understanding of the presence of various obsessions and compulsions is important to obtain. 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 utilize and understand the presence of a wide variety of obsessions and compulsions as well as the severity of these symptoms.

14.4.2 Treatment

14.4.2.1. OCD. Exposure and Response Prevention is one of the most commonly used and supported evidenced-based treatment for OCD. Treatment of OCD has come a long way in the recent years. Among the most effective treatment options is exposure and response prevention (March, Frances, Kahn, & Carpenter, 1997). First developed by psychiatrist Victor Meyer (1966), as you might infer from the name, individuals are repeatedly exposed to their obsession, thus causing anxiety/fears, while simultaneously prevented from engaging in their compulsive behaviors. Exposure sessions are often done *in vivo*, or in real life, via videos, or even imaginary, depending on the type of obsession. For example, a fear that one's house would burn down if their compulsion was not carried out would obviously be done via imaginary exposure, as it would not be ethical to have a person burn their house down.

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

Within the hierarchy of obsessions, the individual is also gradually exposed to their obsession. For example, an individual obsessed with germs, may first watch a person sneeze on the computer in session. Once anxiety is managed and compulsions are refrained at this level of exposure, the individual would move on to being present in the same room as a sick individual, to eventually shaking hands with

someone obviously sick, each time preventing them from engaging in their compulsive behavior. Once this level of their hierarchy was managed, they would move on to the next obsession and so forth until the entire list was complete.

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

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

14.4.2.2. BDD. Seeing as though there are strong similarities between OCD and BDD, it should not come as a surprise that the only two effective treatments for BDD are those that are effective in OCD. Exposure and response prevention have been successful in treating symptoms of BDD, as patients are repeatedly exposed to their body imperfections/obsessions and prevented from engaging in compulsions used to reduce their anxiety. (Veale, Gournay, et al., 1996; Wilhelm, Otto, Lohr, & Deckersbach, 1999). The other treatment option, psychopharmacology, has also been shown to reduce symptoms in patients with BDD. Similar to OCD, medications such as clomipramine and other SSRIs are generally prescribed. While these are effective in reducing BDD symptoms, once medication is discontinued, symptoms resume nearly immediately suggesting this is not an effective long-term treatment option for those with BDD.

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

14.4.2.3. 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 improvement with these disorders. The individual learns to accept that negative thoughts and feelings are part of being human. The individual learns to respond in new ways to these negative thoughts and feelings that is more in line with their overall values and goals (e.g., not skin picking or hair pulling; Lochner, Ross, & Stein,

2017).

Pharmacological options may include SSRIs and glutamatergic medication options. SSRIs and glutamatergic agents have shown promising results in studies. Other, less common drugs such as opioid antagonist and anti-epileptic drugs have been theorized to be beneficial. However, studies regarding opioid antagonist and anti-epileptic drugs either show no benefit or are not in-depth random control trials (RCTs), and as such, despite some promising results, cannot be used to draw overarching conclusions on benefits (Lochner, Ross, & Stein, 2017).

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 becoming 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 modules past, we have discussed the clinical presentation, epidemiology, comorbidity, etiology, and treatment options for a specific class of disorders – the obsessive compulsive and related disorders. This concludes our discussions of mood and anxiety-related disorders. In our next block of disorders, we will cover Trauma, Eating, and Substance-related Disorders. Specifically, in our next chapter, we will discuss trauma-related disorders.

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VI

PART VI. TRAUMA, EATING, AND SUBSTANCE-RELATED DISORDERS

Part VI. Trauma, Eating, and Substance-related Disorders

Module 15 - Trauma-related Disorders

Module Overview

In Module 15, we will discuss matters related to trauma-related disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include PTSD, acute stress, and adjustment disorders. We will also distinguish between stressors and trauma and list common types of both. Adverse childhood events will be described. 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

- 15.1. Stressors and CACs
- 15.2. Clinical Presentation
- 15.3. Prevalence and Comorbidity
- 15.4. Etiology
- 15.5. Assessment
- 15.6. Treatment

Module Learning Outcomes

- Explain what stressors are.
- Describe how trauma- and stressor-related disorders present.
- Describe the prevalence and comorbidity in relation to 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, TRAUMAS, AND CACS

Section Learning Objectives

- Define stressor and trauma.
- Describe common stressors and traumas.
- Describe Adverse Childhood Events (ACE).
- Describe CACs

15.1.1. Stressors and Traumas

Before we get into clinical presentations for the three most common trauma and stress related disorders, let's discuss a little about common events that precipitate a stress related diagnosis. A stress disorder occurs when an individual has difficulty coping with or adjusting to a recent **stressor**. Stressors can be any event- either witnessed firsthand, experienced personally, or experienced by a close family member- that increases physical or psychological demands on an individual. Some stressful events are not considered traumatic. For example, having a minor fender-bender while driving is not likely. However, when a stressor is significant enough that it poses a threat, whether real or imagined, to the individual or loved one, we reach a point of potential **trauma**. That is, a traumatic event is considered to be a "frightening, dangerous, or violent event that poses a threat to a child's [or individual's] life or bodily integrity. Witnessing a traumatic event that threatens life or physical security of a loved one…" is also considered to be defined as a potentially traumatic event (NCTSN, n.d.a). While many people experience similar stressors throughout their lives, only a small percentage of individuals experience significant maladjustment to the event that psychological intervention is warranted.

Among the most commonly studied triggers for trauma-related disorders are combat and physical/sexual assault. This is where the emergence of PTSD came from. Symptoms of combat related trauma date back to World War I, when soldiers would return home with "shell shock" (Figley, 1978). Unfortunately, it wasn't until after the Vietnam War that significant progress was made in both identifying and treating war-related psychological difficulties (Roy-Byrne et al., 2004). Physical assault, more specifically sexual assault, is another commonly studied traumatic event. **Rape,** or forced sexual intercourse or other sexual act committed without an individual's consent, occurs in one out of every five women and one in every 71 men (Black et al., 2011). Unfortunately, this statistic is likely an underestimate of the actual number of cases that occur due to the reluctance of many individuals to report their sexual assault. Of the reported cases, it is estimated that nearly 81% of female and 35% of male rape victims report both acute stress disorder and posttraumatic stress disorder symptoms (Black et al., 2011).

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. 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 specific maltreatment 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 (Figure 15.1 provides an overview of various childhood maltreatment and prevalence). 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 discussed in general societal conversations of 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 misperception that if trauma occurs before a child can remember it, then it does not impact them. However, this is incorrect. Moreover, children's brain structures may even be impacted. For example, their brain cortex may be reduced in size. It can also lead to significant disruptions in the attachment a child forms to 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 has to 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 compromised immune systems (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 as a way 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

Maltreatment Type	Definition	Prevalence
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 where 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%
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%
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%
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%
Other	Parental substance abuse, threatened abuse, or any other maltreatment	6.9%

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 experience two separate types of adverse events. With increased adverse events, adult outcomes were increasingly negative (e.g., substance abuse, serious mental illness). Moreover, 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 everts 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 videoed 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 from, on average to 8 different people, to 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.

15.2. CLINICAL PRESENTATION

Section Learning Objectives

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

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

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

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

The third category experienced by individuals with PTSD is *negative alterations in cognitions or mood*. This is often reported as difficulty remembering an important aspect of the traumatic event. It should be noted that this amnesia is not due to a head injury, loss of consciousness, or substances, but rather, due to the traumatic nature of the event. The impaired memory may also lead individuals to have false beliefs about the cause 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 participation of previously enjoyable activities, as well as the desire to socially engage with others.

The fourth and final category is *alterations in arousal and reactivity*. Because of the negative mood and increased irritability, individuals with PTSD may be quick tempered and act out in aggressive manners, both verbally and physically. While these aggressive responses may be provoked, they are also sometimes unprovoked. It is believed these behaviors occur due to the heightened sensitivity to

potential threats, especially if the threat is similar in nature to their traumatic event. More specifically, individuals with PTSD have a heightened startle response and easily jump or respond to unexpected noises such as a telephone ringing or a car backfiring. Given this heightened arousal state, it should not be surprising that individuals with PTSD also experience significant sleep disturbances, with difficulty falling asleep, as well as staying asleep due to nightmares.

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

15.2.1.1. Diagnosing PTSD in children. 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 is the same, such as experiencing a traumatic event, other components are different. The biggest difference is the absence of requiring the presence of negative cognitions – which, historically, was one of the hardest to assess in children (APA, 2013).

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 1 month, the individual would then meet criteria for PTSD. Additionally, if symptoms present immediately following the traumatic event but resolve by day 3, an individual would not meet criteria for acute stress disorder.

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

15.2.3. Adjustment Disorder

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

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

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

15.3. PREVALENCE AND COMORBIDITY

Section Learning Objectives

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

15.3.1. PTSD

The prevalence rate for PTSD in the US is 8.7% (APA, 2013). Specifically, 5% of adolescents are diagnosed with PTSD (NIH, 2017, November). Think about this for a moment, although approximately 66% (2/3rds) of youth experience a trauma, 5% of adolescence (8.7% of individuals overall) go on to develop PTSD. These statistics help illustrate that, simply because someone experiences a traumatic event does not mean they will certainly develop PTSD. In fact, only a small percentage go on to develop

PTSD.

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

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

There is also a strong relationship between PTSD and major neurocognitive disorders, which may be due to the overlapping symptoms between these disorders.

15.3.2. Acute Stress Disorder

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

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

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

While some researchers indicated ASD is a good predictor of PTSD, others argue further research between the two and confounding variables should be further explored to determine more consistent findings.

15.3.3. Adjustment Disorder

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

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

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

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

15.4.2. Cognitive

Preexisting conditions of depression and/or anxiety may predispose an individual to develop PTSD or other stress disorders. One theory is that these individuals may ruminate, or over analyze the traumatic event, thus bringing more attention to the traumatic event 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 true for many psychological disorders, social and family support have been identified as protective factors for individuals prone to develop PTSD. More specifically, rape victims who are loved and cared for by their friends and family members as opposed to judged for their actions prior to the rape, report fewer trauma symptoms and faster psychological improvement (Street et al., 2011).

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 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).

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

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. This means, if I see a child that is being assessed for ADHD, I should still screen for trauma. This is because we never know when a child may disclose a trauma, and trauma and PTSD reactions may actually 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? I cannot answer that if I do not screen for trauma.

When assessing for PTSD, two components 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 have to 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 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 live to understand when traumatic events occurred and when symptoms started. This helps us understand if the symptoms are related to a trauma or not. For example, if a child that 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; however, following a trauma, they 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.

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 appear to follow relatively the same format:

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

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

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 assistance in PTSD related symptoms as it helps individuals extinguish fears associated with the traumatic event. There are several different types of exposure techniques- **imaginal, in vivo,** and **flooding** are among the most common types of exposure (Cahill, Rothbaum, Resick, & Follette, 2009).

In imaginal exposure, the individual is asked to re-create, or imagine, specific details of the traumatic event. The patient is then asked to repeatedly discuss the event in more and more detail, providing more information regarding their thoughts and feelings at each step of the event. With in-vivo exposure, the individual is reminded of the traumatic event through the use of videos, images, or other tangible

objects related to the traumatic event, that induce a heightened arousal response. While the patient is re-experiencing cognitions, emotions, and physiological symptoms related to the traumatic experience, they are encouraged to utilize positive coping strategies, such as relaxation techniques to reduce their overall level of anxiety.

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

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 replacing them with positive, more adaptive cognitions (Foa et al., 2005).

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

- **P:** Psycho-education about the traumatic event. This includes discussion about the event itself, as well as typical emotional and/or behavioral responses to the event.
- **R**: Relaxation Training. Teaching the patient how to engage in various types of relaxation techniques such as deep breathing and progressive muscle relaxation.
- **A:** Affect. Discussing ways for the patient to effectively express their emotions/fears related to the traumatic event.
- **C:** Correcting negative or maladaptive thoughts.
- **T:** Trauma Narrative. This involves having the patient relive the traumatic event (verbally or written), including as many specific details as possible. This can be a straight forward as writing

the event out with paper and pencil, or as elaborate as creating a song, “story book” or other project. It is important that the child is involved in selecting how they would like to express the trauma narrative.

- **I:** In vivo exposure (see above discussion of exposure). The main goal of this component is to reduce reactions to triggers. The child may have to repeat certain words that are triggers, smell particular scents, etc. This will be specific to any identified triggers the child has.
- **C:** Co-joint family session. This is to provide the patient with a strong social support and sense of security. It also allows family members to learn about the treatment so that they are able to assist the patient if necessary.
- **E:** Enhancing Security. Patients are encouraged to practice the coping strategies they learn in TF-CBT so that they are prepared when they experience these triggers out in the real world, as well as any future challenges that may come their way.

TF-CBT is also beneficial for children that have experienced traumatic grief. The same principals 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 experience trauma. It is composed of 3 different general components: child-directed components, caregiver-directed components, and parent-child/family-system directed components. Child-directed components focus on similar concepts of 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 caregivers are noticed to engage in somewhat 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 (see discussion above). 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. However, the intervention has proven to be beneficial for children that have experienced trauma. The therapy focuses on increasing the positive interaction between a parent and child. The parent learns how to interact and impose appropriate consequences to 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 "ear piece" 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 ear piece 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. In 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. Parent's 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, SSRI's work by increasing the amount of serotonin available to neurotransmitters. Tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs) are also recommended as second-line treatments. Their effectiveness is most often observed in individuals who report co-occurring major depressive disorder symptoms, as well as those who do not respond to SSRIs (Forbes et al., 2010). Unfortunately, due to the effective CBT and EMDR treatment options, research on psychopharmacological interventions have been limited. Future studies exploring other medication options are needed to determine if there are alternative medication options for stress/trauma disorder patients.

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.

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 was called, 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.

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 outburst 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 themselves 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. Our discussion in Module 16 moves to eating disorders.

2nd edition

Module 16 - Eating Disorders

Module Overview

In Module 16, we will discuss matters related to eating disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include anorexia nervosa, bulimia nervosa, and binge-eating disorders. 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

- 16.1. Clinical Presentation
- 16.2. EDNOS and Changes from DSM IV-TR to DSM-5
- 16.3. Prevalence and Comorbidity
- 16.4. Etiology
- 16.5. Assessment
- 16.6. 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

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

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

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

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

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

16.1.1. Anorexia Nervosa

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

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

fine hair/hair loss, and muscle weakness to name a few.

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

Anorexia is categorized into two types: restricting type and binge-eating/purging type. In restricting type, no binge eating or purging occurs and weight loss is predominately achieved through dieting or fasting (APA, 2013). Binge-eating/purging indicates that there are some occurrences of binging following by purging. It should be noted that purging might follow periods of typical food consumption, not necessarily binging, as well.

16.1.2. Bulimia Nervosa

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

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 binging episode, feeling uncomfortable eating in public, developing food rituals/limited diet, disappearing to a 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. One difference in the two disorder is that a diagnosis of bulimia does not require significantly low body weight.

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

reduction often occurs for years prior to seeking help.

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; however, these episodes are *not* followed by a compensatory behavior to rid the body of calories. Despite the feelings of shame and guilt post binge, individual's with BED will not engage in vomiting, excessive exercises or other compensatory behaviors. These binge eating episodes occur on average, at least once a week for 3 months.

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

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

16.1.4. Anorexia versus Bulimia versus Binging-Eating Disorder

These three disorders can seem similar so outlining the differences is important. To start, while anorexia and bulimia may include behaviors utilized to prevent weight gain and negative self-evaluations of their bodies, binge eating disorder does not. To differentiate between BED from anorexia and bulimia, consider if there are efforts to prevent weight gain or significant distress about body image. To differentiate between anorexia and bulimia, the biggest factor is if the individual has significantly low weight. anorexia requires significantly low weight and may only involve restriction of food but *may also* include binging/purging (thus anorexia may or may not involve binge/purging). Bulimia does *not* include significant periods of food restriction, although restriction may be present periodically, and *always* includes binging and purging behaviors.

16.2. EDNOS AND CHANGES FROM DSM IV-TR TO DSM-5

Section Learning Objectives

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

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

16.2.1. EDNOS

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

16.2.2. BED

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

16.2.3. Anorexia Nervosa

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

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

16.2.4. Bulimia Nervosa

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

16.3. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe the epidemiology of eating disorders.
- Describe the comorbidity of anorexia nervosa.
- Describe the comorbidity of bulimia nervosa.
- Describe the comorbidity of BED.

16.3.1. General Prevalence of Eating Disorders

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

16.3.2. Anorexia Comorbidity

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

16.3.3. Bulimia Comorbidity

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

16.3.4. BED Comorbidity

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

16.4. ETIOLOGY

Section Learning Objectives

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

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

16.4.1. Biological

There is some evidence of a genetic predisposition to eating disorders, in which relatives of those diagnosed with an eating disorder are up to six times more likely than other individuals to be diagnosed with an eating disorder (APA, 2013). Twin concordance studies also support the gene theory. If an

identical twin is diagnosed with anorexia, there is a 70 percent chance the other twin will develop an anorexia in their lifetime (APA, 2013). The concordance rate for fraternal twins (who share less genes) is 20 percent. While not as strong for bulimia cases, identical twins still display a 23 percent concordance rate, compared to the 9 percent fraternal twins rate (APA, 2013).

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

16.4.2. Cognitive

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

16.4.3. Sociocultural

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

16.4.3.1. Media. One commonly discussed contributor to eating disorders is the media. Idealizing thin models and actresses sends the message to young women (and adolescents) that in order to be popular and attractive, you must be thin. These images are not isolated to magazines, but are also seen in television shows, movies, commercials, and large advertisements on billboards and hanging in store windows. With the immergence of social media (i.e. Facebook, Snapchat, Instagram), exposure to media

images and celebrities is even easier. Couple this with the ability to alter images to make individuals even thinner, it is no wonder many young women (and men) become dissatisfied with their own weight and shape (Polivy & Herman, 2004).

16.4.3.2. Ethnicity. While eating disorders are not solely a "white female" disorder, there are significant discrepancies when it comes to race, especially for anorexia nervosa. Why is this? Research indicates that black males prefer heavier females than do white males (Greenberg & Laporte, 1996). Given this preference, it should not be surprising that, frequently, black females and children have larger ideal physiques than their white peers (Polivy & Herman, 2000). Since black females may idealize thinness to a lesser degree, black females are less likely to develop anorexia; however, more recent studies suggest that this may not remain. Caldwell and colleagues (1997) found that high-income black females were equally as dissatisfied as high-income white females 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).

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

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

There is some speculation that males are not diagnosed as frequently as females 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).

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

While family involvement can help maintain the disorder, it can also contribute to the development of one as well. Families that emphasize thinness or place a large emphasis on physical appearance are more likely to have a child diagnosed with an eating disorder (Zerbe, 2008). In fact, mothers with eating disorders are more likely to have children who develop some type of feeding/eating disorder than mother's without eating disorders (Whelan & Cooper, 2000). Additional family characteristics that are

common in adolescents that present for treatment of eating disorders are: enmeshed, intrusive, critical, hostile, or overly concerned with parenting (Polivy & Herman, 2002). It should be noted that while there has been some correlation between these family dynamics and eating disorders, they are not evident in all families of people with eating disorders.

16.4.4. 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.

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

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

16.5. 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. Moreover, the treatment of the disorders usually requires several different professionals (e.g., medical doctor, dietician, nurse, psychologist, therapist).

16.5.1. Physical Assessment

A full medical and physical exam should occur. The medical should attempt to exclude inflammatory bowel disease, hyperthyroidism, chronic infection, diabetes, and other serious medical conditions. Moreover, a female's menstruation history should be obtained. If menarche has occurred, information about the individual's first menstrual period should be obtained. Additionally, information if their menstrual period has become irregular or completely stopped should also be obtained (Lask & Bryant-Waugh, 2013).

The physical exam should be done alone unless child requests otherwise. The provider will look for physical consequences of the disorder and rule out other serious disorders. Physical growth will be assessed and weight/body measurements will be obtained. Body mass index will be calculated. 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).

16.5.2. Psychological Assessment

A clinical interview is conducted not just with the individual, but also family members. During the interview, a timeline may be established which includes life events and also depicts the onset or progression of eating behaviors. Eating behaviors will be thoroughly assessed. Additionally, assessing for weight-controlling behaviors such as excessive exercise, induced vomiting, laxatives, etc. will occur. Moreover, obtaining an understanding of the individual's body image will be established. An overall assessment of their mental health, as well as concern for suicidality, should occur (Lask & Bryant-Waugh, 2013).

Although interviews can be conducted informally, structured interviews can also occur, and are preferred at times. The gold standard for assessing for eating disorders in children is the Child Eating Disorder Examination (ChEDE). Moreover, standardized objective measures to screen for levels of symptoms such as the Eating Disorder Examination Questionnaire (EDEQ) and Eating Disorder Inventory for Children (EDIC). Moreover, helpful screeners may include Children's Eating Attitudes Test (ChEAT) or the Kids' Eating Disorder Survey (KEDS; Lask & Bryant-Waugh, 2013).

16.6. TREATMENT

Section Learning Objectives

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

16.6.1. Anorexia

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

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

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

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

16.6.1.3. 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 shifts 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.

16.6.2. Bulimia

Just as anorexia nervosa treatment's initial focus is on weight gain, bulimia nervosa's initial goal of treatment is to eliminate binge eating episodes, as well as eliminate compensatory behaviors. The goal is to replace both of these negative behaviors with positive eating habits. One of the most effective ways to establish this is through Cognitive Behavioral Therapy (CBT).

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

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

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

individuals begin to control their mind, which in return, can help them control their behaviors.

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

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

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

Phase Three is the final stage. The goals of this phase are to ensure that the changes made in phase two are maintained. To achieve this, treatment sessions are spaced out, allowing patients more time to engage in their changed behavior. Additionally, relapse prevention (i.e. problem-solving ways *not* to relapse) is also discussed to ensure long term results. In doing this, the patient reviews the progress they have made over the course of treatment, as well as identifying potential interpersonal issues that may arise, and how their treatment can be adapted to address those issues.

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

16.6.3. Binge Eating Disorder

Given the similar presentations of BED and bulimia nervosa, it should not be surprising that the most effective treatments for BED is also 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 (a treatment that focuses on reducing harmful behaviors, increasing positive coping skills, and improving interpersonal and emotional regulation skills) have also been effective in reducing binge-eating episodes, however, they have not been effective in weight loss (Guerdjikova, Mori, Casuto,

& McElroy, 2017). Goals of treatment are of course to eliminate binge eating episodes, as well as reduce body weight as most individuals with BED are overweight. Seeing as BED has only recently been established as its own separate eating disorder, treatment research specific to this disorder is expected to grow.

16.6.3.1. 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.

16.6.4. Outcome of Treatment

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

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

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

Apply Your Knowledge

CASE VIGNETTE

Sally is a 1-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 that 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. We also discussed changes in the DSM from IV-TR to DSM-5. Our next and final chapter will cover substance use disorders.

2nd edition

Module 17 - Substance-Induced Disorders

Module Overview

In Module 17, we will discuss matters related to substance-induced disorders to include their clinical presentation, prevalence, comorbidity, etiology, assessment, and treatment options. Our discussion will include substance abuse and the various types of substances that can be abused. 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

- 17.1. Clinical Presentation
- 17.2. Prevalence and Comorbidity
- 17.3. Etiology
- 17.4. Assessment
- 17.5. Treatment

Module Learning Outcomes

- Describe how substance abuse disorders present.
- Describe the prevalence and comorbidity of substance abuse disorders.
- Describe the etiology of substance abuse disorders.
- Describe how substance abuse disorders are assessed, diagnosed, and treated.

17.1. CLINICAL PRESENTATION AND DSM-5 CRITERIA

Section Learning Objectives

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

17.1.1. Substance Abuse

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

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

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

Listed below are the diagnostic criteria for an individual with Substance Intoxication, Substance Abuse, and Substance Withdrawal. While some individual symptoms, particularly withdrawal symptoms, may vary depending on the substance abused and its physiological effects on the body, the behaviors and the impact to an individual's daily functioning are much the same across all substances commonly abused.

According to the DSM-5 (APA, 2013), an individual is diagnosed with *Substance Intoxication, Use*, and/or *Withdrawal* depending on the specific substance(s) the individual is ingesting. While there are

some subtle differences in symptoms among the specific substances (e.g. psychological and/or physical symptoms), the general diagnostic criteria for *Substance Intoxication, Use*, and *Withdrawal* remains the same across substances. Therefore, the general diagnostic criteria for *Substance Intoxication, Use*, and *Withdrawal* is reviewed below, with more specific details of behavioral, psychological, and/or physical symptoms in the Types of Substances Abused section.

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

In order to meet criteria for *Substance Use Disorder*, an individual must experience significant impairment or distress over the course of 12-months due to their use of a substance (APA, 2013). Distress or impairment can be described as any of the following: inability to complete or lack of participation in work, school or home obligations/activities; increased time spent on activities obtaining, using, or recovering from substance use; impairment in social or interpersonal relationships; use of substance in a potentially hazardous situation; psychological problems due to recurrent substance abuse; craving for substance; an increase in the amount of substance used over time (i.e. tolerance); difficulty reducing the amount of substance used despite desire to reduce/stop using the substance; and/or withdrawal symptoms. While the number of these symptoms may vary among individuals, only two symptoms must be present to meet criteria for a *Substance Use Disorder.*

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

17.1.2. Types of Substances Abused

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

17.1.2.1. Depressants. Depressant substances such as alcohol, sedative-hypnotic drugs, and opioids, are known to have a depressing, or inhibiting effect on one’s central nervous system; therefore, they are often used to alleviate tension and stress. Unfortunately, when used in large amounts, they can also

impair an individual's judgment and motor activity.

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

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

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

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

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

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

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

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

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

Opioids are unique in that they provide both euphoria and drowsiness. Tolerance to these drugs builds quickly, thus resulting in an increased need of the medication to produce desired effects. This rapid tolerance is also likely responsible for opioids highly addictive nature. Opioid withdrawal symptoms can range from restlessness, muscle pain, fatigue, anxiety, and insomnia. Unfortunately, these withdrawal symptoms, as well as intense cravings for the drug can persist for several months, with some reports up to years. Because of the intensity and longevity of these withdrawal symptoms, many individuals struggle to remain abstinent, and accidental overdoses are common (CDC, 2013).

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

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

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

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

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

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, n.d.).

Amphetamines are manufactured in a laboratory setting. Currently, the most common amphetamines are prescription medications such as Ritalin, Adderall, and Dexedrine (prescribed for sleep disorders).

These medications produce an increase in energy and alertness and reduce appetite when taken at clinical levels; however, when consumed at larger dosages, it can produce intoxication similar to psychosis, including violent behaviors. Due to the increased energy levels and appetite suppressant qualities, these medications are often abused by students studying for exams, athletes needing extra energy, and individuals seeking weight loss (Haile, 2012). Biologically, similar to cocaine, amphetamines effect the central nervous system by increasing the amount of dopamine, norepinephrine, and serotonin in the brain (Haile, 2012).

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

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

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

Hallucinogens come from natural sources and have been involved in cultural and religious ceremonies for thousands of years. Synthetic forms of hallucinogens have also been created – most common of which are *PCP*, *Ketamine, LSD* and *Ecstasy*. In general, hallucinogens produce powerful changes in sensory perception. Depending on the type of drug ingested, effects can range from hallucinations, changes in color perception, or distortion of objects. Additionally, some individuals report enhanced auditory, as well as changes in physical perception such as tingling or numbness of limbs and interchanging hot and cold sensations (Weaver & Schnoll, 2008). Interestingly, the effect of

hallucinogens can vary both between individuals, as well as *within* the same individual. This means that the same amount of the same drug may produce a positive experience one time, but a negative experience the next time.

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

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

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

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

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

another drug may lead to an accidental overdose. Finally, the use of one drug to counteract the effects of another drug—taking a depressant to combat the effects of a stimulant—is equally as dangerous as the body is unable to regulate homeostasis.

17.2. PREVALENCE AND COMORBIDITY

Section Learning Objectives

- Describe the prevalence of substance abuse.
- Outline comorbidity of substance abuse.

It has been estimated that nearly 9 percent of teens and adults in the United States have a substance abuse disorder (SAMHSA, 2014). Asian/Pacific Islander, Hispanic, and African American's are less likely to develop a lifetime substance abuse disorder compared to non-Hispanic white individuals (Grant et al., 2016). Native Americans actually have the highest rate of substance abuse at nearly 22 percent (NSDUH, 2013). Additional demographic variables also suggest that overall substance abuse is greater in males than females, 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). Within adolescence specifically, marijuana is the most commonly used illicit drug (SAMHSA, 2015, September).

17.2.1. Depressants

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

17.2.2. Stimulants

Nearly 1.1 percent of all high school seniors have used cocaine within the past month (Johnston et al., 2014). Due to the high cost of cocaine, it is more commonly found in suburban neighborhoods where consumers have the financial means to purchase the drugs. Methamphetamine is used by males and females equally.

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

17.2.3. Hallucinogens

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

17.2.4. General Comorbidity

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

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

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

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. The majority of research on the brain reward system has focused on the mesocorticolimbic dopamine system, as it appears this area is the primary reward system of most substances that are abused. As research has evolved in the field of substance abuse, five additional neurotransmitters have also been implicated in the reinforcing effect of addiction: dopamine, opioid peptides, GABA, serotonin, and endocannabinoids. More specifically, dopamine is less involved in opioid, alcohol, and cannabis. Alcohol and benzodiazepines lower the production of GABA, while cocaine and amphetamines are involved in the lowering of dopamine. Cannabis has been shown to reduce the

production of endocannabinoids.

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 expectations are thought to increase drug-seeking behavior, while negative experiences would decrease substance use (Oei & Morawska, 2004). Several alcohol studies have examined the expectancy effect on the use of alcohol. Those with alcohol abuse reported expectations of tension reduction, enhanced sexual experiences, and improved social pleasure (Brown, 1985). Additionally, observing positive experiences, both in person and through television or social media also shapes our drug use expectancies.

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

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 a reinforcement or punishment. Since we are talking about increasing substance use, behavioral theorists suggest that substance abuse is *positively and negatively reinforced* due to the effects of a substance.

Positive reinforcement occurs when the substance use is increased due to the positive or pleasurable experiences of the substance. More specifically, the rewarding effect or pleasurable experiences while under the influence of various substances directly impacts the likelihood that the individual will use the substance again (Wise & Koob, 2013). Studies of substance use on animals routinely 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). The second way negative reinforcement is involved in substance abuse is during symptoms of withdrawal. As previously mentioned, withdrawal from a substance often produces significant negative symptoms such as nausea, vomiting, uncontrollable

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

Additionally, research continually supports a strong relationship between second generation substance abusers (Wilens et al., 2014). The increased likelihood of family members substance abuse is likely related to both a genetic predisposition, as well as the accepting attitude of the familial environment (Chung et al., 2014). Not only does a child have early exposure to these substances if their parent has had 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 actually reduce substance use by promoting negative experiences (Galen & Rogers, 2004).

Another sociocultural view on substance abuse is stressful life events, particularly those related to financial stability. Prevalence rates of substance abuse is higher in poorer people (SAMHSA, 2014). Furthermore, additional stressors such as childhood abuse/trauma, negative home/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).

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.

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 that is 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 a withdrawal of a specified drug. While most detoxification programs are inpatient for increased supervision, there are some programs that allow for outpatient detoxification, particularly if the addiction is not as severe. There are two main theories of detoxification—gradually decreasing the amounts of the substance until the individual is off the drug completely, or, eliminate the substance entirely while providing additional medications to manage withdrawal symptoms (Bisaga et al., 2015). Unfortunately, relapse rates are

high for those engaging in detoxification programs, particularly if they lack any follow-up psychological treatment.

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

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

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

17.5.2. Behavioral

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

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

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

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/or physiological risks for relapse. Once these triggers are identified, the clinician works with the patient on cognitive and behavioral strategies such as learning effective coping strategies, enhancing self-efficacy, and encouraging mastery of outcomes. Additionally, psychoeducation about how substance abuse is maintained, as well as identifying maladaptive thoughts and learning cognitive restructuring techniques helps the patient make informed choices during high-risk situations. Finally, role-playing these high-risk situations in session allows patients to become comfortable engaging in these effective coping strategies that enhance their self-efficacy, and ultimately reduce 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 as a way to

help guide members in spiritual and character development. Due to the popularity of the treatment program, other programs such as Narcotics Anonymous and Cocaine Anonymous adopted and adapted the Twelve Steps for their respective substance abuse. Similarly, Al-Anon and Alateen are two groups that offer support for families and teenagers of individuals struggling with alcohol abuse.

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

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

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 environment while also attending daily individual, group, and family therapy. The types of treatment used within a residential program varies from program to program, with most focusing on cognitive-behavioral and behavioral techniques. Many also incorporate 12-step programs into treatment as well, as many patients transition from a residential treatment center to a 12-step program post discharge. As one would expect, residential treatment programs goals are abstinence and any evidence of substance abuse during the program is grounds for immediate termination.

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

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. For adolescents, this may include family-therapy and family support components.

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

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.

2nd edition

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

Q

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

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