

Discovering Psychology Series

Fundamentals of Psychological Disorders

(formerly Abnormal Psychology)

3rd edition (5-TR)

Alexis Bridley, Ph.D.

Lee W. Daffin Jr., Ph.D.

Washington State University

Version 3.50

July 2023

Contact Information about this OER:

1. Dr. Lee Daffin, Associate Professor of Psychology – ldaffin@wsu.edu
2. Dr. Alexis Bridley - Adjunct Instructor – alexis.bridley@wsu.edu

Table of Contents

Preface

Record of Changes

Part I. Setting the Stage

- Module 1: What is Abnormal Psychology? 1-1
- Module 2: Models of Abnormal Psychology 2-1
- Module 3: Clinical Assessment, Diagnosis, and Treatment 3-1

Part II. Mental Disorders – Block 1

- Module 4: Mood Disorders 4-1
- Module 5: Trauma- and Stressor-Related Disorders 5-1
- Module 6: Dissociative Disorders 6-1

Part III. Mental Disorders – Block 2

- Module 7: Anxiety Disorders 7-1
- Module 8: Somatic Symptom and Related Disorders 8-1
- Module 9: Obsessive-Compulsive and Related Disorders 9-1

Part IV. Mental Disorders – Block 3

- Module 10: Feeding and Eating Disorders 10-1
- Module 11: Substance-Related and Addictive Disorders 11-1

Part V. Mental Disorders – Block 4

- Module 12: Schizophrenia Spectrum and Other Psychotic Disorders 12-1
- Module 13: Personality Disorders 13-1

Part VI. Mental Disorders – Block 5

- Module 14: Neurocognitive Disorders 14-1
- Module 15: Contemporary Issues in Psychopathology 15-1
- Module 16: Disorders of Childhood Overview 16-1

Glossary

References

Index

Record of Changes

Edition	As of Date	Changes Made
1.0	Fall 2017	Initial writing; feedback pending
1.01	Spring 2018	Addition of Modules 2, 3, and 15
1.02	Summer 2018	Addition of Index, Glossary, and Preface; made minor edits based on student feedback.
1.03	Summer 2019	Proofreading edits
2.00	August 2020	Proofreading edits and overall improvements such as end of section summaries and review questions. Added a Tokens of Appreciation page. Added lecture slides courtesy of Arizona State University.
2.05	November 2021	Section 1.1.1. changed the following: “Psychology worked with the disease model for over 60 years, from about the late 1800s into the middle part of the 20th century.” It previously indicated 19 th century when it should have said 20 th which was the mid-1900s. Thank you to Dr. Irving Herman of Columbia University for pointing this out.
3.00	August 2022	<p>NAME CHANGE – The name of the book has changed from Abnormal Behavior to Fundamentals of Psychological Disorders. We have continued with the numerical progression of edition numbers, making this three, despite the shift in name. We do not want instructors using the book to believe the book is different.</p> <p>Additional round of text revisions but main changes were to update the book to the newly released DSM 5-TR in March 2022. Some references have been updated as well.</p>
3.5	July 2023	Addition of Module 16: Disorders of Childhood Overview; updating of the references, index, glossary, and front matter, accordingly. Creation of new PDF of the entire book. Modified text related to eating disorders.

Tokens of Appreciation

August 2022

Alexis and I want to offer a special thank you to Ms. Celeste Ernst, undergraduate within the online Bachelor of Science degree in Psychology program, for her edits of the 1st edition during the spring 2020. Her changes, and our own, were integrated into the 2nd edition of the book and are a dramatic improvement over the 1st edition. Thank you, Celeste. Many of those changes will be present in the 3rd edition and improved upon as we transition to DSM 5-TR.

We would also like to extend a special thank you to Madeleine Stewart and Matt Meier, PsyD., of the Department of Psychology at Arizona State University for the development of the lecture slides for this book. They did this work unsolicited and produced top quality presentations which we will include in a password protected page, along with additional ancillaries such as an Instructor's Manual and test banks, in the very near future (i.e. hopefully by mid fall semester at the latest but the slides in August) and for Instructors (Not students. Sorry). Thank you again for your excellent work, Madeleine and Matt. It is more appreciated than you could ever imagine.

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

Enjoy the 3rd edition of Fundamentals of Psychological Disorders (formerly Abnormal Psychology).

Lee Daffin

On behalf of, Alexis Bridley

Part I. Setting the Stage

Topics Covered:

1. What is Abnormal Psychology?
2. Models of Abnormal Psychology
3. Clinical Assessment, Diagnosis, and Treatment

Part I. Setting the Stage

Module 1: What is Abnormal Psychology?

Module 1: What is Abnormal Psychology?

Module Overview

Cassie is an 18-year-old female from suburban Seattle, WA. She was a successful student in high school, graduating valedictorian and obtaining a National Merit Scholarship for her performance on the PSAT during her junior year. She was accepted to a university on the opposite side of the state, where she received additional scholarships giving her a free ride for her entire undergraduate education. Excited to start this new chapter in her life, Cassie's parents begin the 5-hour commute to Pullman, where they will leave their only daughter for the first time in her life.

The semester begins as it always does in mid to late August. Cassie meets the challenge with enthusiasm and does well in her classes for the first few weeks of the semester, as expected. Sometime around Week 6, her friends notice she is despondent, detached, and falling behind in her work. After being asked about her condition, she replies that she is "just a bit homesick," and her friends accept this answer as it is a typical response to leaving home and starting college for many students. A month later, her condition has not improved but worsened. She now regularly shirks her responsibilities around her apartment, in her classes, and on her job. Cassie does not hang out with friends like she did when she first arrived for college and stays in bed most of the day. Concerned, Cassie's friends contact Health and Wellness for help.

Cassie's story, though hypothetical, is true of many Freshmen leaving home for the first time to earn a higher education, whether in rural Washington state or urban areas such as Chicago and Dallas. Most students recover from this depression and go on to be functional members of their collegiate environment and accomplished scholars. Some students learn to cope

on their own while others seek assistance from their university's health and wellness center or from friends who have already been through the same ordeal. These are normal reactions.

However, in cases like Cassie's, the path to recovery is not as clear. Instead of learning how to cope, their depression increases until it reaches clinical levels and becomes an impediment to success in multiple domains of life such as home, work, school, and social circles.

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

Module Outline

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

Module Learning Outcomes

- Explain what it means to display abnormal behavior.
- Clarify how mental health professionals classify mental disorders.
- Describe the effect of stigma on those who have a mental illness.

- Outline the history of mental illness.
- Describe the research methods used to study abnormal behavior and mental illness.
- Identify types of mental health professionals, societies they may join, and journals they can publish their work in.

1.1. Understanding Abnormal Behavior

Section Learning Objectives

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

1.1.1. Understanding Abnormal Behavior

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

Psychology worked with the disease model for over 60 years, from about the late 1800s into the middle part of the 20th century. The focus was simple – curing mental disorders - and included such pioneers as Freud, Adler, Klein, Jung, and Erickson. These names are synonymous with the psychoanalytical school of thought. In the 1930s, behaviorism, under B.F. Skinner, presented a new view of human behavior. Simply, human behavior could be modified if the correct combination of reinforcements and punishments were used. This viewpoint espoused the dominant worldview of the time – mechanism – which presented the world as a great machine explained through the principles of physics and chemistry. In it, human beings serve as smaller machines in the larger machine of the universe.

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

- “The first was moral; that psychologists and psychiatrists became victimologists, pathologizers; that our view of human nature was that if you were in trouble, bricks fell on you. And we forgot that people made choices and decisions. We forgot responsibility. That was the first cost.”

- “The second cost was that we forgot about you people. We forgot about improving normal lives. We forgot about a mission to make relatively untroubled people happier, more fulfilled, more productive. And "genius," "high-talent," became a dirty word. No one works on that.”
- “And the third problem about the disease model is, in our rush to do something about people in trouble, in our rush to do something about repairing damage, it never occurred to us to develop interventions to make people happier -- positive interventions.”

Starting in the 1960s, figures such as Abraham Maslow and Carl Rogers sought to overcome the limitations of psychoanalysis and behaviorism by establishing a "third force" psychology, also known as humanistic psychology. As Maslow said,

“The science of psychology has been far more successful on the negative than on the positive side; it has revealed to us much about man’s shortcomings, his illnesses, his sins, but little about his potentialities, his virtues, his achievable aspirations, or his full psychological height. It is as if psychology had voluntarily restricted itself to only half its rightful jurisdiction, and that the darker, meaner half.” (Maslow, 1954, p. 354).

Humanistic psychology instead addressed the full range of human functioning and focused on personal fulfillment, valuing feelings over intellect, hedonism, a belief in human perfectibility, emphasis on the present, self-disclosure, self-actualization, positive regard, client centered therapy, and the hierarchy of needs. Again, these topics were in stark contrast to much of the work being done in the field of psychology up to and at this time.

In 1996, Martin Seligman became the president of the American Psychological Association (APA) and called for a **positive psychology** or one that had a more positive

conception of human potential and nature. Building on Maslow and Roger's work, he ushered in the scientific study of such topics as happiness, love, hope, optimism, life satisfaction, goal setting, leisure, and subjective well-being. Though positive and humanistic psychology have similarities, their methodology was much different. While humanistic psychology generally relied on qualitative methods, positive psychology utilizes a quantitative approach and aims to help people make the most out of life's setbacks, relate well to others, find fulfillment in creativity, and find lasting meaning and satisfaction

(<https://www.positivepsychologyinstitute.com.au/what-is-positive-psychology>).

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

1.1.2. How Do We Determine What Abnormal Behavior Is?

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

Disorders 5th edition, Text Revision (DSM-5-TR; 2022), states that, “Although no definition can capture all aspects of the range of disorders contained in DSM-5” (pg. 13) certain aspects are required. These include:

- **Dysfunction** – Includes “clinically significant disturbance in an individual’s cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning” (pg. 14). Abnormal behavior, therefore, has the capacity to make well-being difficult to obtain and can be assessed by looking at an individual’s current performance and comparing it to what is expected in general or how the person has performed in the past. As such, a good employee who suddenly demonstrates poor performance may be experiencing an environmental demand leading to stress and ineffective coping mechanisms. Once the demand resolves itself, the person’s performance should return to normal according to this principle.
- **Distress** – When the person experiences a disabling condition “in social, occupational, or other important activities” (pg. 14). Distress can take the form of psychological or physical pain, or both concurrently. Alone though, distress is not sufficient enough to describe behavior as abnormal. Why is that? The loss of a loved one would cause even the most “normally” functioning individual pain. An athlete who experiences a career-ending injury would display distress as well. Suffering is part of life and cannot be avoided. And some people who exhibit abnormal behavior are generally positive while doing so.
- **Deviance** – Closer examination of the word *abnormal* indicates a move away from what is normal, or the mean (i.e., what would be considered average and in this case

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

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

themselves. It is important to point out that people seen as dangerous are also not automatically mentally ill.

1.1.3. The Costs of Mental Illness

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

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

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

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

1.1.4. Defining Key Terms

Our discussion so far has concerned what normal and abnormal behavior is. We saw that the study of normal behavior falls under the providence of positive psychology. Similarly, the scientific study of abnormal behavior, with the intent to be able to predict reliably, explain, diagnose, identify the causes of, and treat maladaptive behavior, is what we refer to as **abnormal psychology**. Abnormal behavior can become pathological and has led to the scientific study of psychological disorders, or **psychopathology**. From our previous discussion we can fashion the following definition of a psychological or mental disorder: **mental disorders** are characterized

by psychological dysfunction, which causes physical and/or psychological distress or impaired functioning, and is not an expected behavior according to societal or cultural standards.

You should have learned the following in this section:

- Abnormal behavior is a combination of personal distress, psychological dysfunction, deviance from social norms, dangerousness to self and others, and costliness to society.
- Abnormal psychology is the scientific study of abnormal behavior, with the intent to be able to predict reliably, explain, diagnose, identify the causes of, and treat maladaptive behavior.
- The study of psychological disorders is called psychopathology.
- Mental disorders are characterized by psychological dysfunction, which causes physical and/or psychological distress or impaired functioning, and is not an expected behavior according to societal or cultural standards

Section 1.1 Review Questions

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

1.2. Classifying Mental Disorders

Section Learning Objectives

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

1.2.1. Classification

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

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

1.2.2. Determining Occurrence of a Disorder

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

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

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

- **Period prevalence** indicates the proportion of a population that has the characteristic at any point during a given period of time, typically the past year.
- **Lifetime prevalence** indicates the proportion of a population that has had the characteristic at any time during their lives.

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

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

Incidence indicates the number of new cases in a population over a specific period. This measure is usually lower since it does not include existing cases as prevalence does. If you wish to know the number of new cases of social phobia during the past year (going from say Aug 21, 2015 to Aug 20, 2016), you would only count cases that began during this time and ignore cases

before the start date, even if people are currently afflicted with the mental disorder. Incidence is often studied by medical and public health officials so that causes can be identified, and future cases prevented.

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

1.2.3. Other Key Factors Related to Mental Disorders

The **etiology** is the cause of the disorder. There may be social, biological, or psychological explanations for the disorder which need to be understood to identify the appropriate treatment. Likewise, the effectiveness of a treatment may give some hint at the cause of the mental disorder. More on this in Module 2.

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

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

Finally, we will discuss several treatment strategies in this book in relation to specific disorders, and in a general fashion in Module 3. **Treatment** is any procedure intended to modify abnormal behavior into normal behavior. The person suffering from the mental disorder seeks the assistance of a trained professional to provide some degree of relief over a series of therapy sessions. The trained mental health professional may prescribe medication or utilize psychotherapy to bring about this change. Treatment may be sought from the primary care provider, in an outpatient facility, or through inpatient care or hospitalization at a mental hospital or psychiatric unit of a general hospital. According to NAMI, the average delay between symptom onset and treatment is 11 years with 45% of adults with mental illness, 66% of adults with serious mental illness, and 51% of youth with a mental health condition seeking treatment in a given year. They also report that 50% of white, 49% of lesbian/gay and bisexual, 43% of mixed/multiracial, 34% of Hispanic or Latinx, 33% of black, and 23% of Asian adults with a mental health diagnosis received treatment or counseling in the past year (Source: Mental Health Care Matters infographic, <https://www.nami.org/mhstats>).

You should have learned the following in this section:

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

Section 1.2 Review Questions

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

1.3. The Stigma of Mental Illness

Section Learning Objectives

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

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

Social cognition is the process through which we collect information from the world around us and then interpret it. The collection process occurs through what we know as *sensation* – or detecting physical energy emitted or reflected by physical objects. Detection occurs courtesy

of our eyes, ears, nose, skin and mouth; or via vision, hearing, smell, touch, and taste, respectfully. Once collected, the information is relayed to the brain through the neural impulse where it is processed and interpreted, or meaning is added to this raw sensory data which we call *perception*.

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

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

Social identity theory (Tajfel, 1982; Turner, 1987) states that people categorize their social world into meaningfully simplistic representations of groups of people. These representations are then organized as *prototypes*, or “fuzzy sets of a relatively limited number of

category-defining features that not only define one category but serve to distinguish it from other categories” (Foddy and Hogg, as cited in Foddy et al., 1999). We construct in-groups and out-groups and categorize the self as an in-group member. The self is assimilated into the salient in-group prototype, which indicates what cognitions, affect, and behavior we may exhibit. Stereotyping, out-group homogeneity, in-group/out-group bias, normative behavior, and conformity are all based on self-categorization.

How so? *Out-group homogeneity* occurs when we see all members of an outside group as the same. This leads to a tendency to show favoritism to, and exclude or hold a negative view of, members outside of, one’s immediate group, called the *in-group/out-group bias*. The negative view or set of beliefs about a group of people is what we call *prejudice*, and this can result in acting in a way that is negative against a group of people, called *discrimination*. It should be noted that a person can be prejudicial without being discriminatory since most people do not act on their attitudes toward others due to social norms against such behavior. Likewise, a person or institution can be discriminatory without being prejudicial. For example, when a company requires that an applicant have a certain education level or be able to lift 80 pounds as part of typical job responsibilities. Individuals without a degree or ability to lift will be removed from consideration for the job, but this discriminatory act does not mean that the company has negative views of people without degrees or the inability to lift heavy weight. You might even hold a negative view of a specific group of people and not be aware of it. An attitude we are unaware of is called an *implicit attitude*, which stands in contrast to *explicit attitudes*, which are the views within our conscious awareness.

We have spent quite a lot of space and time understanding how people gather information about the world and people around them, process this information, use it to make snap

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

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

- *Public stigma* – When members of a society endorse negative stereotypes of people with a mental disorder and discriminate against them. They might avoid them altogether, resulting in social isolation. An example is when an employer intentionally does not hire a person because their mental illness is discovered.
- *Label avoidance* – To avoid being labeled as “crazy” or “nuts” people needing care may avoid seeking it altogether or stop care once started. Due to these labels, funding for mental health services could be restricted and instead, physical health services funded.
- *Self-stigma* – When people with mental illnesses internalize the negative stereotypes and prejudice, and in turn, discriminate against themselves. They may experience shame, reduced self-esteem, hopelessness, low self-efficacy, and a reduction in

coping mechanisms. An obvious consequence of these potential outcomes is the *why try* effect, or the person saying ‘Why should I try and get that job? I am not worthy of it’ (Corrigan, Larson, & Rusch, 2009; Corrigan, et al., 2016).

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

Other effects of stigma include experiencing work-related discrimination resulting in higher levels of self-stigma and stress (Rusch et al., 2014), higher rates of suicide especially when treatment is not available (Rusch, Zlati, Black, and Thornicroft, 2014; Rihmer & Kiss, 2002), and a decreased likelihood of future help-seeking intention (Lally et al., 2013). The results of the latter study also showed that personal contact with someone with a history of mental

illness led to a decreased likelihood of seeking help. This is important because 48% of the university sample stated that they needed help for an emotional or mental health issue during the past year but did not seek help. Similar results have been reported in other studies (Eisenberg, Downs, Golberstein, & Zivin, 2009). It is also important to point out that social distance, a result of stigma, has also been shown to increase throughout the life span, suggesting that anti-stigma campaigns should focus on older people primarily (Schomerus, et al., 2015).

One potentially disturbing trend is that mental health professionals have been shown to hold negative attitudes toward the people they serve. Hansson et al. (2011) found that staff members at an outpatient clinic in the southern part of Sweden held the most negative attitudes about whether an employer would accept an applicant for work, willingness to date a person who had been hospitalized, and hiring a patient to care for children. Attitudes were stronger when staff treated patients with a psychosis or in inpatient settings. In a similar study, Martensson, Jacobsson, and Engstrom (2014) found that staff had more positive attitudes towards persons with mental illness if their knowledge of such disorders was less stigmatized; their workplaces were in the county council where they were more likely to encounter patients who recover and return to normal life in society, rather than in municipalities where patients have long-term and recurrent mental illness; and they have or had one close friend with mental health issues.

To help deal with stigma in the mental health community, Papish et al. (2013) investigated the effect of a one-time contact-based educational intervention compared to a four-week mandatory psychiatry course on the stigma of mental illness among medical students at the University of Calgary. The curriculum included two methods requiring contact with people diagnosed with a mental disorder: patient presentations, or two one-hour oral presentations in

which patients shared their story of having a mental illness, and "clinical correlations" in which a psychiatrist mentored students while they interacted with patients in either inpatient or outpatient settings. Results showed that medical students held a stigma towards mental illness and that comprehensive medical education reduced this stigma. As the authors stated, "These results suggest that it is possible to create an environment in which medical student attitudes towards mental illness can be shifted in a positive direction." That said, the level of stigma was still higher for mental illness than it was for the stigmatized physical illness, type 2 diabetes mellitus.

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

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

Stigma leads to health inequities (Hatzenbuehler, Phelan, & Link, 2013), prompting calls for stigma change. Targeting stigma involves two different agendas: The *services agenda* attempts to remove stigma so people can seek mental health services, and the *rights agenda* tries

to replace discrimination that “robs people of rightful opportunities with affirming attitudes and behavior” (Corrigan, 2016). The former is successful when there is evidence that people with mental illness are seeking services more or becoming better engaged. The latter is successful when there is an increase in the number of people with mental illnesses in the workforce who are receiving reasonable accommodations. The federal government has tackled this issue with landmark legislation such as the Patient Protection and Affordable Care Act of 2010, Mental Health Parity and Addiction Equity Act of 2008, and the Americans with Disabilities Act of 1990. However, protections are not uniform across all subgroups due to “1) explicit language about inclusion and exclusion criteria in the statute or implementation rule, 2) vague statutory language that yields variation in the interpretation about which groups qualify for protection, and 3) incentives created by the legislation that affect specific groups differently” (Cummings, Lucas, and Druss, 2013). More on this in Module 15.

You should have learned the following in this section:

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

Section 1.3 Review Questions

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

1.4. The History of Mental Illness

Section Learning Objectives

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

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

1.4.1. Prehistoric and Ancient Beliefs

Prehistoric cultures often held a supernatural view of abnormal behavior and saw it as the work of evil spirits, demons, gods, or witches who took control of the person. This form of

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

1.4.2. Greco-Roman Thought

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

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

responsibility of the community and their families to care for them. The Greek physician Galen (A.D. 129-199) said mental disorders had either physical or psychological causes, including fear, shock, alcoholism, head injuries, adolescence, and changes in menstruation.

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

1.4.3. The Middle Ages – 500 AD to 1500 AD

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

Group hysteria, or **mass madness**, was also seen when large numbers of people displayed similar symptoms and false beliefs. This included the belief that one was possessed by wolves or other animals and imitated their behavior, called **lycanthropy**, and a mania in which large numbers of people had an uncontrollable desire to dance and jump, called **tarantism**. The latter

was believed to have been caused by the bite of the wolf spider, now called the tarantula, and spread quickly from Italy to Germany and other parts of Europe where it was called **Saint Vitus's dance**.

Perhaps the return to supernatural explanations during the Middle Ages makes sense given events of the time. The black death (bubonic plague) killed up to a third, or according to other estimates almost half, of the population. Famine, war, social oppression, and pestilence were also factors. The constant presence of death led to an epidemic of depression and fear. Near the end of the Middle Ages, mystical explanations for mental illness began to lose favor, and government officials regained some of their lost power over nonreligious activities. Science and medicine were again called upon to explain psychopathology.

1.4.4. The Renaissance - 14th to 16th centuries

The most noteworthy development in the realm of philosophy during the Renaissance was the rise of **humanism**, or the worldview that emphasizes human welfare and the uniqueness of the individual. This perspective helped continue the decline of supernatural views of mental illness. In the mid to late 1500s, German physician Johann Weyer (1515-1588) published his book, *On the Deceits of the Demons*, that rebutted the Church's witch-hunting handbook, the *Malleus Maleficarum*, and argued that many accused of being witches and subsequently imprisoned, tortured, and/or burned at the stake, were mentally disturbed and not possessed by demons or the Devil himself. He believed that like the body, the mind was susceptible to illness. Not surprisingly, the book was vehemently protested and banned by the Church. It should be noted that these types of acts occurred not only in Europe, but also in the United States. The most

famous example, the Salem Witch Trials of 1692, resulted in more than 200 people accused of practicing witchcraft and 20 deaths.

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

1.4.5. Reform Movement – 18th to 19th centuries

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

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

Reform in the United States started with the figure largely considered to be the father of American psychiatry, Benjamin Rush (1745-1813). Rush advocated for the humane treatment of the mentally ill, showing them respect, and even giving them small gifts from time to time.

Despite this, his practice included treatments such as bloodletting and purgatives, the invention of the "tranquilizing chair," and reliance on astrology, showing that even he could not escape from the beliefs of the time.

Due to the rise of the moral treatment movement in both Europe and the United States, asylums became habitable places where those afflicted with mental illness could recover. Regrettably, its success was responsible for its decline. The number of mental hospitals greatly increased, leading to staffing shortages and a lack of funds to support them. Though treating patients humanely was a noble endeavor, it did not work for some patients and other treatments were needed, though they had not been developed yet. Staff recognized that the approach worked best when the facility had 200 or fewer patients, but waves of immigrants arriving in the U.S. after the Civil War overwhelmed the facilities, and patient counts soared to 1,000 or more. Prejudice against the new arrivals led to discriminatory practices in which immigrants were not

afforded the same moral treatments as native citizens, even when the resources were available to treat them.

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

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

“In the early 1950s, Mental Health America issued a call to asylums across the country for their discarded chains and shackles. On April 13, 1953, at the McShane Bell Foundry in Baltimore, Md., Mental Health

America melted down these inhumane bindings and recast them into a sign of hope: the Mental Health Bell.

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

For more information on MHA, please visit: <https://www.mhanational.org/>

1.4.6. 20th – 21st Centuries

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

1.4.6.1. Biological or Somatogenic Perspective. Recall that Greek physicians Hippocrates and Galen said that mental disorders were akin to physical disorders and had natural causes. Though the idea fell into oblivion for several centuries, it re-emerged in the late 19th century for two reasons. First, German psychiatrist Emil Kraepelin (1856-1926) discovered that symptoms occurred regularly in clusters, which he called **syndromes**. These syndromes represented a unique mental disorder with a distinct cause, course, and prognosis. In 1883 he published his textbook, *Compendium der Psychiatrie* (Textbook of Psychiatry), and described a system for classifying mental disorders that became the basis of the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders (DSM)* that is currently in its 5th edition Text Revision (published in 2022).

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

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

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

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

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

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

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

By the end of the 19th century, it had become evident that mental disorders were caused by a combination of biological and psychological factors, and the investigation of how they develop began. Sigmund Freud's development of psychoanalysis followed on the heels of the work of Bruner, and others who came before him.

1.4.7. Current Views/Trends

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

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

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

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

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

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

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

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

Frank (2006) found that by 1996, psychotropic drugs were used in 77% of mental health cases and spending on these drugs grew from \$2.8 billion in 1987 to about \$18 billion in 2001 (Coffey et al., 2000; Mark et al., 2005), representing over a sixfold increase. The largest classes of psychotropic drugs are anti-psychotics and anti-depressants, followed closely by anti-anxiety medications. Frank, Conti, and Goldman (2005) point out, “The expansion of insurance coverage for prescription drugs, the introduction and diffusion of managed behavioral health care techniques, and the conduct of the pharmaceutical industry in promoting their products all have influenced how psychotropic drugs are used and how much is spent on them.” Is it possible then that we are overprescribing these medications? Davey (2014) provides ten reasons why this may be so, including leading suffers from believing that recovery is in their hands but instead in the hands of their doctors; increased risk of relapse; drug companies causing the “medicalization of perfectly normal emotional processes, such as bereavement” to ensure their survival; side effects; and a failure to change the way the person thinks or the socioeconomic environments that may be the cause of the disorder. For more on this article, please see:

<https://www.psychologytoday.com/blog/why-we-worry/201401/overprescribing-drugs-treat-mental-health-problems>. Smith (2012) echoed similar sentiments in an article on inappropriate

prescribing. He cites the approval of Prozac by the Food and Drug Administration (FDA) in 1987 as when the issue began and the overmedication/overdiagnosis of children with ADHD as a more recent example.

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

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

- *Health Maintenance Organizations (HMO)* – Typically only pay for care within the network. The subscriber chooses a primary care physician (PCP) who coordinates most of their care. The PCP refers the subscriber to specialists or other health care providers as is necessary. This is the most restrictive option.
- *Preferred Provider Organizations (PPO)* - Usually pay more if the subscriber obtains care within the network, but if care outside the network is sought, they cover part of the cost.

- *Point of Service (POS)* – These plans provide the most flexibility and allow the subscriber to choose between an HMO or a PPO each time care is needed.

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

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

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

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

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

This type of sensitivity training is vital because bias based on ethnicity, race, and culture has been found in the diagnosis and treatment of autism (Harrison et al., 2017; Burkett, 2015), borderline personality disorder (Jani et al., 2016), and schizophrenia (Neighbors et al., 2003; Minsky et al., 2003). Despite these findings, Schwartz and Blankenship (2014) state, “It should also be noted that although clear evidence supports a longstanding trend in differential diagnoses

according to consumer race, this trend does not imply that one race (*e.g.*, African Americans) actually demonstrate more severe symptoms or higher prevalence rates of psychosis compared with other races (*e.g.*, Euro-Americans). Because clinicians are the diagnosticians and misinterpretation, bias or other factors may play a role in this trend caution should be used when making inferences about actual rates of psychosis among ethnic minority persons.” Additionally, white middle-class help seekers were offered appointments with psychotherapists almost three times as often as their black working-class counterparts. Women were offered an appointment time in their preferred time range more than men were, though average appointment offer rates were similar between genders (Kugelmass, 2016). These findings collectively show that though we are becoming more culturally sensitive, we have a lot more work to do.

1.4.7.5. Prescription rights for psychologists. To reduce inappropriate prescribing as described in 1.4.7.2, it has been proposed to allow appropriately trained psychologists the right to prescribe. Psychologists are more likely to utilize both therapy and medication, and so can make the best choice for their patient. The right has already been granted in New Mexico, Louisiana, Guam, the military, the Indian Health Services, and the U.S. Public Health Services. Measures in other states “have been opposed by the American Medical Association and American Psychiatric Association over concerns that inadequate training of psychologists could jeopardize patient safety. Supporters of prescriptive authority for psychologists are quick to point out that there is no evidence to support these concerns” (Smith, 2012).

1.4.7.6. Prevention science. As a society, we used to wait for a mental or physical health issue to emerge, then scramble to treat it. More recently, medicine and science has taken a **prevention** stance, identifying the factors that cause specific mental health issues and implementing interventions to stop them from happening, or at least minimize their deleterious

effects. Our focus has shifted from individuals to the population. Mental health promotion programs have been instituted with success in schools (Shoshani & Steinmetz, 2014; Weare & Nind, 2011; Berkowitz & Beer, 2007), in the workplace (Czabała, Charzyńska, & Mroziak, B., 2011), with undergraduate and graduate students (Conley et al., 2017; Bettis et al., 2016), in relation to bullying (Bradshaw, 2015), and with the elderly (Forsman et al., 2011). Many researchers believe it is the ideal time to move from knowledge to action and to expand public mental health initiatives (Wahlbeck, 2015). The growth of positive psychology in the late 1990s has further propelled this movement forward. For more on positive psychology, please see Section 1.1.1.

You should have learned the following in this section:

- Some of the earliest views of mental illness saw it as the work of evil spirits, demons, gods, or witches who took control of the person, and in the Middle Ages it was seen as possession by the Devil and methods such as exorcism, flogging, prayer, the touching of relics, chanting, visiting holy sites, and holy water were used to rid the person of demonic influence.
- During the Renaissance, humanism was on the rise which emphasized human welfare and the uniqueness of the individual and led to an increase in the number of asylums as places of refuge for the mentally ill.
- The 18th to 19th centuries saw the rise of the moral treatment movement followed by the mental hygiene movement.
- The psychological or psychogenic perspective states that emotional or psychological factors are the cause of mental disorders and represented a challenge to the biological perspective which said that mental disorders were akin to physical disorders and had natural causes.
- Psychiatric or psychotropic drugs used to treat mental illness became popular beginning in the 1950s and led to deinstitutionalization or a shift from inpatient to outpatient care.

Section 1.4 Review Questions

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

1.5. Research Methods in Psychopathology

Section Learning Objectives

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

1.5.1. The Scientific Method

Psychology is the “scientific study of behavior and mental processes.” We will spend quite a lot of time on the behavior and mental processes part throughout this book and in relation to mental disorders. Still, before we proceed, it is prudent to further elaborate on what makes psychology scientific. It is safe to say that most people outside of our discipline or a sister science would be surprised to learn that psychology utilizes the scientific method at all. That may be even truer of clinical psychology, especially in light of the plethora of self-help books found at any bookstore. But yes, the treatment methods used by mental health professionals are based on empirical research and the scientific method.

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

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

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

Table 1.1: The Steps of the Scientific Method

Step	Name	Description
0	Ask questions and be willing to wonder.	To study the world around us, you have to wonder about it. This inquisitive nature is the hallmark of critical thinking — our ability to assess claims made by others and make objective judgments that are independent of emotion and anecdote and based on hard evidence —and a requirement to be a scientist.
1	Generate a research question or identify a problem to investigate.	Through our wonderment about the world around us and why events occur as they do, we begin to ask questions that require further investigation to arrive at an answer. This investigation usually starts with a literature review , or when we conduct a literature search through our university library or a search engine such as Google Scholar to see what questions have been investigated already and what answers have been found, so that we can identify gaps or holes in this body of work.
2	Attempt to explain the phenomena we wish to study.	We now attempt to formulate an explanation of why the event occurs as it does. This systematic explanation of a phenomenon is a theory and our specific, testable prediction is the hypothesis . We will know if our theory is correct because we have formulated a hypothesis that we can now test.
3	Test the hypothesis.	It goes without saying that if we cannot test our hypothesis, then we cannot show whether our prediction is correct or not. Our plan of action of how we will go about testing the hypothesis is called our research design . In the planning

		stage, we will select the appropriate research method to answer our question/test our hypothesis.
4	Interpret the results.	With our research study done, we now examine the data to see if the pattern we predicted exists. We need to see if a cause and effect statement can be made, assuming our method allows for this inference. More on this in Section 2.3. For now, it is essential to know that statistics have two forms. First, there are descriptive statistics which provide a means of summarizing or describing data and presenting the data in a usable form. You likely have heard of mean or average, median, and mode. Along with standard deviation and variance, these are ways to describe our data. Second, there are inferential statistics that allow for the analysis of two or more sets of numerical data to determine the statistical significance of the results. Significance is an indication of how confident we are that our results are due to our manipulation or design and not chance.
5	Draw conclusions carefully.	We need to interpret our results accurately and not overstate our findings. To do this, we need to be aware of our biases and avoid emotional reasoning so that they do not cloud our judgment. How so? In our effort to stop a child from engaging in self-injurious behavior that could cause substantial harm or even death, we might overstate the success of our treatment method.
6	Communicate our findings to the broader scientific community.	Once we have decided on whether our hypothesis was correct or not, we need to share this information with others so that they might comment critically on our methodology, statistical analyses, and conclusions. Sharing also allows for replication or repeating the study to confirm its results. Communication occurs via scientific journals, conferences, or newsletters released by many of the organizations mentioned in Module 1.6.

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

1. *Observation* – To know about the world around us, we have to be able to see it firsthand. When a mental disorder afflicts an individual, we can see it through their overt behavior. An individual with depression may withdraw from activities he/she enjoys, those with

social anxiety disorder will avoid social situations, people with schizophrenia may express concern over being watched by the government, and individuals with dependent personality disorder may leave major decisions to trusted companions. In these examples and numerous others, the behaviors that lead us to a diagnosis of a specific disorder can easily be observed by the clinician, the patient, and/or family and friends.

2. *Experimentation* – To be able to make *causal* or cause and effect statements, we must isolate variables. We must manipulate one variable and see the effect of doing so on another variable. Let's say we want to know if a new treatment for bipolar disorder is as effective as existing treatments, or more importantly, better. We could design a study with three groups of bipolar patients. One group would receive no treatment and serve as a control group. A second group would receive an existing and proven treatment and would also be considered a control group. Finally, the third group would receive the new treatment and be the experimental group. What we are manipulating is what treatment the groups get – no treatment, the older treatment, and the newer treatment. The first two groups serve as controls since we already know what to expect from their results. There should be no change in bipolar disorder symptoms in the no-treatment group, a general reduction in symptoms for the older treatment group, and the same or better performance for the newer treatment group. As long as patients in the newer treatment group do not perform worse than their older treatment counterparts, we can say the new drug is a success. You might wonder why we would get excited about the performance of the new drug being the same as the old drug. Does it really offer any added benefit? In terms of a reduction of symptoms, maybe not, but it could cost less money than the older drug and that would be of value to patients.

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

1.5.2. Research Methods

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

1.5.2.1. Naturalistic and laboratory observation. In terms of **naturalistic observation**, the scientist studies human or animal behavior in its natural environment, which could include the home, school, or a forest. The researcher counts, measures, and rates behavior in a systematic way and, at times, uses multiple judges to ensure accuracy in how the behavior is being measured. The advantage of this method is that you see behavior as it happens, and the experimenter does not taint the data. The disadvantage is that it could take a long time for the behavior to occur, and if the researcher is detected, then this may influence the behavior of those being observed.

Laboratory observation involves observing people or animals in a laboratory setting. The researcher might want to know more about parent-child interactions, and so, brings a mother and her child into the lab to engage in preplanned tasks such as playing with toys, eating a meal, or the mother leaving the room for a short time. The advantage of this method over the naturalistic method is that the experimenter can use sophisticated equipment to record the session and examine it later. The problem is that since the subjects know the experimenter is watching

them, their behavior could become artificial. Clinical observation is a commonly employed research method to study psychopathology; we will talk about it more throughout this book.

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

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

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

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

1.5.2.5. Experiments. This is a controlled test of a hypothesis in which a researcher manipulates one variable and measures its effect on another variable. The manipulated variable is called the **independent variable (IV)**, and the one that is measured is called the **dependent variable (DV)**. In the example under Experimentation in Section 1.5.1, the treatment for bipolar disorder was the IV, while the actual intensity or number of symptoms serve as the DV. A

common feature of experiments is a **control group** that does not receive the treatment or is not manipulated and an **experimental group** that does receive the treatment or manipulation. If the experiment includes **random assignment**, participants have an equal chance of being placed in the control or experimental group. The control group allows the researcher (or teacher) to make a *comparison* to the experimental group and make a causal statement possible, and stronger. In our experiment, the new treatment should show a marked reduction in the intensity of bipolar symptoms compared to the group receiving no treatment, and perform either at the same level as, or better than, the older treatment. This would be the initial hypothesis made before starting the experiment.

In a drug study, to ensure the participants' expectations do not affect the final results by giving the researcher what he/she is looking for (in our example, symptoms improve whether the participant is receiving treatment or not), we might use what is called a **placebo**, or a sugar pill made to look exactly like the pill given to the experimental group. This way, participants all are given something, but cannot figure out what exactly it is. You might say this keeps them honest and allows the results to speak for themselves.

Finally, the study of mental illness does not always afford us a large sample of participants to study, so we have to focus on one individual using a **single-subject experimental design**. This differs from a case study in the sheer number of strategies available to reduce potential **confounding variables**, or variables not originally part of the research design but contribute to the results in a meaningful way. One type of single-subject experimental design is the **reversal** or **ABAB design**. Kuttler, Myles, and Carson (1998) used social stories to reduce tantrum behavior in two social environments in a 12-year old student diagnosed with autism, Fragile-X syndrome, and intermittent explosive disorder. Using an ABAB design, they found

that precursors to tantrum behavior decreased when the social stories were available (B) and increased when the intervention was withdrawn (A). A more recent study (Balakrishnan & Alias, 2017) also established the utility of social stories as a social learning tool for children with autism spectrum disorder (ASD) using an ABAB design. During the baseline phase (A), the four student participants were observed, and data recorded on an observation form. During the treatment phase (B), they listened to the social story and data was recorded in the same manner. Upon completion of the first B, the students returned to A, which was followed one more time by B and the reading of the social story. Once the second treatment phase ended, the participation was monitored again to obtain the outcome. All students showed improvement during the treatment phases in terms of the number of positive peer interactions, but the number of interactions reduced in the absence of social stories. From this, the researchers concluded that the social story led to the increase in positive peer interactions of children with ASD.

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

You should have learned the following in this section:

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

Section 1.5 Review Questions

1. What is the scientific method and what steps make it up?
2. Differentiate theory and hypothesis.
3. What are the three cardinal features of science and how do they relate to the study of mental disorders?
4. What are the five main research designs used by psychologists? Define each and then state its strengths and limitations.
5. What is the advantage of multi-method research?

1.6. Mental Health Professionals, Societies, and Journals

Section Learning Objectives

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

1.6.1. Types of Professionals

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

Table 1.2: Types of Mental Health Professionals

Name	Degree Required	Function/Training	Can they prescribe medications?
Clinical Psychologist	Ph.D.	Trained to make diagnoses and can provide individual and group therapy	Only in select states
School Psychologist	Masters or Ph.D.	Trained to make diagnoses and can provide individual and group therapy but also works with school staff	No
Counseling Psychologist	Ph.D.	Deals with adjustment issues primarily and less with mental illness	No
Clinical Social Worker	M.S.W. or Ph.D.	Trained to make diagnoses and can provide individual and group therapy and is involved in advocacy and case management. Usually in hospital	No

		settings.	
Psychiatrist	M.D.	Has specialized training in the diagnosis and treatment of mental disorders	Yes
Psychiatric Nurse Practitioner	M.S.N.	Has specialized treatment in the care and treatment of psychiatric patients	Yes
Occupational Therapist	B.S.	Trained to assist individuals suffering from physical or psychological handicaps and help them acquire needed resources	No
Pastoral Counselor	Clergy	Trained in pastoral education and can make diagnoses and can provide individual and group therapy	No
Drug Abuse and/or Alcohol Counselor	B.S. or higher	Trained in alcohol and drug abuse and can make diagnoses and can provide individual and group therapy	No
Child/Adolescent Psychiatrist	M.D. or Ph.D.	Specialized training in the diagnosis and treatment of mental illness in children	Yes
Marital and Family Therapist	Masters	Specialized training in marital and family therapy; Can make diagnoses and can provide individual and group therapy	No

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

<https://www.mhanational.org/types-mental-health-professionals>

1.6.2. Professional Societies and Journals

One of the functions of science is to communicate findings. Testing hypotheses, developing sound methodology, accurately analyzing data, and drawing sound conclusions are

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

1.6.2.1. Professional Societies

- **Society of Clinical Psychology – Division 12 of the American Psychological**

Association

- Website – <https://div12.org/>
 - Mission Statement – “The mission of the Society of Clinical Psychology is to represent the field of Clinical Psychology through encouragement and support of the integration of clinical psychological science and practice in education, research, application, advocacy and public policy, attending to the importance of diversity.”
 - Publications – Clinical Psychology: Science and Practice and the newsletter Clinical Psychology: Science and Practice (quarterly)
 - Other Information – Members and student affiliates may join one of eight sections such as clinical emergencies and crises, clinical psychology of women, assessment psychology, and clinical geropsychology
- **Society of Clinical Child and Adolescent Psychology – Division 53 of the**

American Psychological Association

- Website – <https://www.clinicalchildpsychology.org/>
- Mission Statement – “Our mission is to serve children, adolescents and families with the best possible clinical care based on psychological science. SCCAP strives to integrate scientific and professional aspects of

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

- Publication – Journal of Clinical Child and Adolescent Psychology
- **American Academy of Clinical Psychology**
 - Website – <https://www.aacpsy.org/>
 - Mission Statement – The American Academy of Clinical Psychology seeks to “recognize and promote advanced competence within Professional Psychology,” “provide a professional community that encourages communication between and among Members and Fellows of the Academy,” “provide opportunities for advanced education in Professional Psychology,” and “expand awareness and availability of AACCP Members and Fellows to the public through promotion and education.”
 - Publication – Bulletin of the American Academy of Clinical Psychology (newsletter)
- **The Society for a Science of Clinical Psychology (SSCP)**
 - Website – <http://www.sscpweb.org/>
 - Mission Statement – “The Society for a Science of Clinical Psychology (SSCP) was established in 1966. Its purpose is to affirm and continue to promote the integration of the scientist and the practitioner in training, research, and applied endeavors. Its members represent a diversity of interests and theoretical orientations across clinical psychology. The

common bond of the membership is a commitment to empirical research and the ideal that scientific principles should play a role in training, practice, and establishing public policy for health and mental health concerns. SSCP has organizational affiliations with both the American Psychological Association (Section III of Division 12) and the Association for Psychological Science.”

- Other Information – Offers ten awards ranging from early career award, outstanding mentor award, outstanding student teacher award, and outstanding student clinician award.

- **American Society of Clinical Hypnosis**
 - Website – <http://www.asch.net/>
 - Mission Statement – “To provide and encourage education programs to further, in every ethical way, the knowledge, understanding, and application of hypnosis in health care; to encourage research and scientific publication in the field of hypnosis; to promote the further recognition and acceptance of hypnosis as an important tool in clinical health care and focus for scientific research; to cooperate with other professional societies that share mutual goals, ethics and interests; and to provide a professional community for those clinicians and researchers who use hypnosis in their work.”
 - Publication – American Journal of Clinical Hypnosis
 - Other Information – Offers certification in clinical hypnosis

1.6.2.2. Professional Journals

- **Clinical Psychology: Science and Practice**
 - Website – [http://onlinelibrary.wiley.com/journal/10.1111/\(ISSN\)1468-2850](http://onlinelibrary.wiley.com/journal/10.1111/(ISSN)1468-2850)
 - Published by – American Psychological Association, Division 12
 - Description – “*Clinical Psychology: Science and Practice* presents cutting-edge developments in the science and practice of clinical psychology and related mental health fields by publishing scholarly articles, primarily involving narrative and systematic reviews as well as meta-analyses related to assessment, intervention, and service delivery.”

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

- **American Journal of Clinical Hypnosis**

- Website -

<http://www.asch.net/Public/AmericanJournalofClinicalHypnosis.aspx>

- Published by – American Society of Clinical Hypnosis
- Description – “The *Journal* publishes original scientific articles and clinical case reports on hypnosis, as well as reviews of related books and abstracts of the current hypnosis literature.”

You should have learned the following in this section:

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

Section 1.6 Review Questions

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

Module Recap

In Module 1, we undertook a relatively lengthy discussion of what abnormal behavior is by first looking at what normal behavior is. What emerged was a general set of guidelines focused on mental illness as causing dysfunction, distress, deviance, and at times, being dangerous for the afflicted and others around him/her. Then we classified mental disorders in terms of their occurrence, cause, course, prognosis, and treatment. We acknowledged that mental illness is stigmatized in our society and provided a basis for why this occurs and what to do about it. This involved a discussion of the history of mental illness and current views and trends.

Psychology is the scientific study of behavior and mental processes. The word *scientific* is key as psychology adheres to the strictest aspects of the scientific method and uses five main research designs in its investigation of mental disorders – observation, case study, surveys, correlational research, and experiments. Various mental health professionals use these designs, and societies and journals provide additional means to communicate findings or to be good consumers of psychological inquiry.

It is with this foundation in mind that we move to examine models of abnormality in Module 2.

Part I. Setting the Stage

Module 2: Models of Abnormal Psychology

Module 2: Models of Abnormal Psychology

Module Overview

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

Module Outline

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

Module Learning Outcomes

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

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

Section Learning Objectives

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

2.1.1. Uni-Dimensional

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

2.1.2. Multi-Dimensional

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

Before introducing the current main models, it is crucial to understand what a model is. In a general sense, a **model** is defined as a representation or imitation of an object (dictionary.com). For mental health professionals, models help us to understand mental illness since diseases such as depression cannot be touched or experienced firsthand. To be considered distinct from other conditions, a mental illness must have its own set of symptoms. But as you will see, the individual does not have to present with the entire range of symptoms. For example, to be diagnosed with separation anxiety disorder, you must present with three of eight symptoms for criteria A whereas for a major depressive episode as part of Bipolar II disorder, you have to display five (or more) symptoms for criteria A. There will be some variability in terms of what symptoms are displayed, but in general, all people with a specific psychopathology have symptoms from that group.

We can also ask the patient probing questions, seek information from family members, examine medical records, and in time, organize and process all this information to better understand the person's condition and potential causes. Models aid us with doing all of this. Still, we must remember that the model is a starting point for the researcher, and due to this, it determines what causes might be investigated at the exclusion of other causes. Often, proponents of a given model find themselves in disagreement with proponents of other models. All forget that there is no individual model that completely explains human behavior, or in this case, abnormal behavior, and so each model contributes in its own way. Here are the models we will examine in this module:

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

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

You should have learned the following in this section:

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

Section 2.1 Review Questions

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

2.2. The Biological Model

Section Learning Objectives

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

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

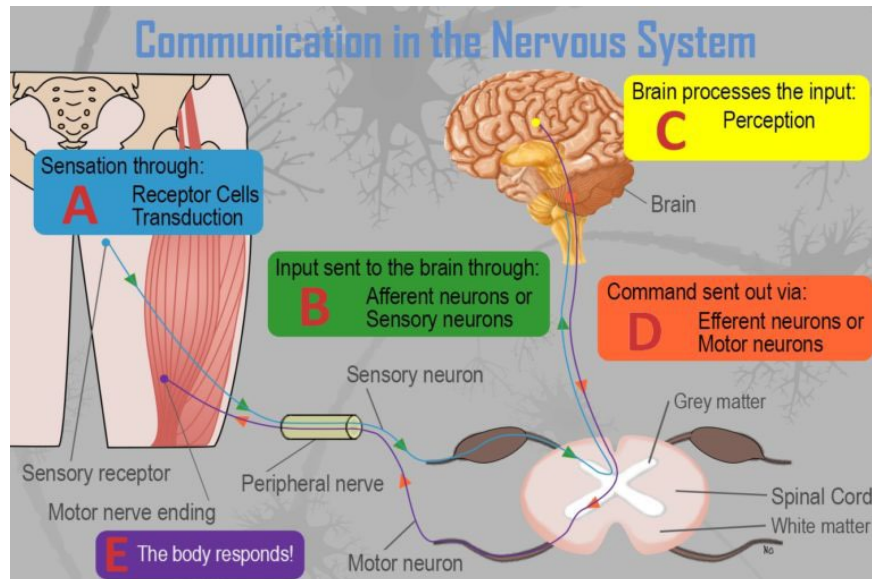
2.2.1. Brain Structure and Chemistry

2.2.1.1. Communication in the nervous system. To truly understand brain structure and chemistry, it is a good idea to understand how communication occurs within the nervous system.

See Figure 2.1 below. Simply:

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

Figure 2.1. Communication in the Nervous System

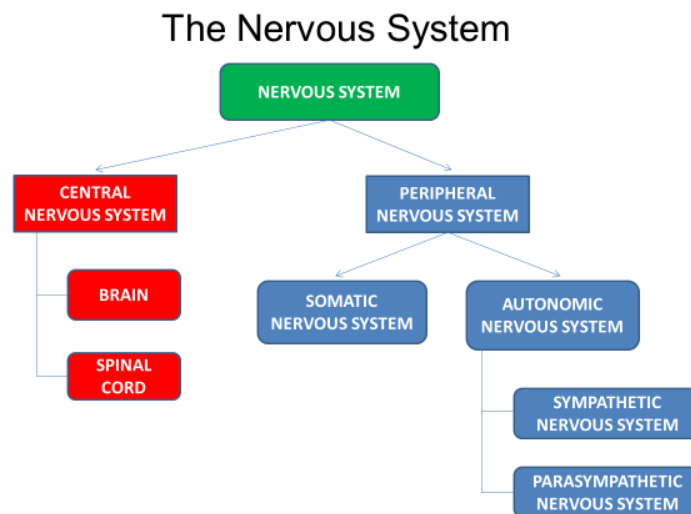


Please note that we will not cover this process in full, but just the parts relevant to our topic of psychopathology.

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

Eventually, the response brought about by the sympathetic nervous system must end. The **parasympathetic nervous system** calms the body.

Figure 2.2. *The Structure of the Nervous System*



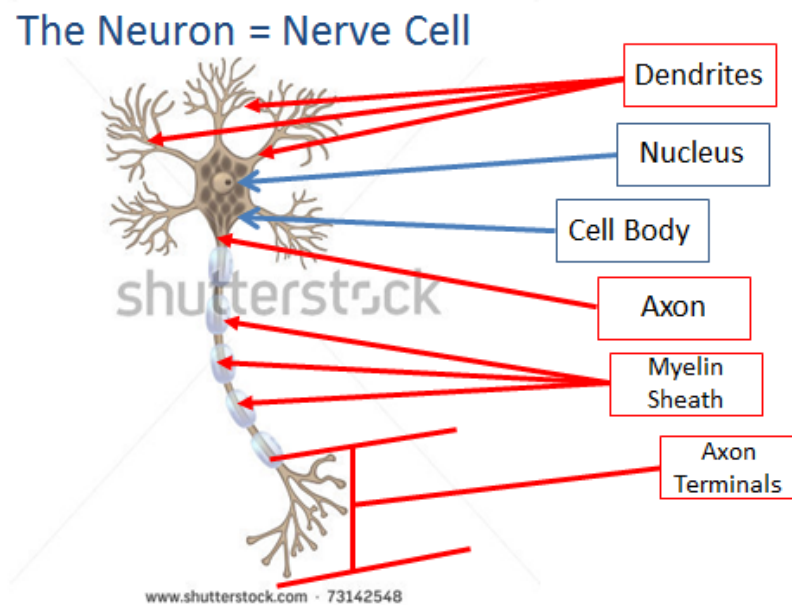
2.2.1.3. The neuron. The fundamental unit of the nervous system is the neuron, or nerve cell (See Figure 2.3). It has several structures in common with all cells in the body. The **nucleus** is the control center of the neuron, and the **soma** is the cell body. In terms of distinctive structures, these focus on the ability of a neuron to send and receive information. The **axon** sends signals/information to neighboring neurons while the **dendrites**, which resemble little trees, receive information from neighboring neurons. Note the plural form of dendrite and the singular form of axon; there are many dendrites but only one axon. Also of importance to the neuron is the **myelin sheath** or the white, fatty covering which: 1) provides insulation so that signals from adjacent neurons do not affect one another and, 2) increases the speed at which signals are transmitted. The **axon terminals** are the end of the axon where the electrical impulse becomes a chemical message and passes to an adjacent neuron.

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

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

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

Figure 2.3. The Structure of the Neuron



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

Part 1. The Axon and Neural Impulse

The neural impulse proceeds across the following steps:

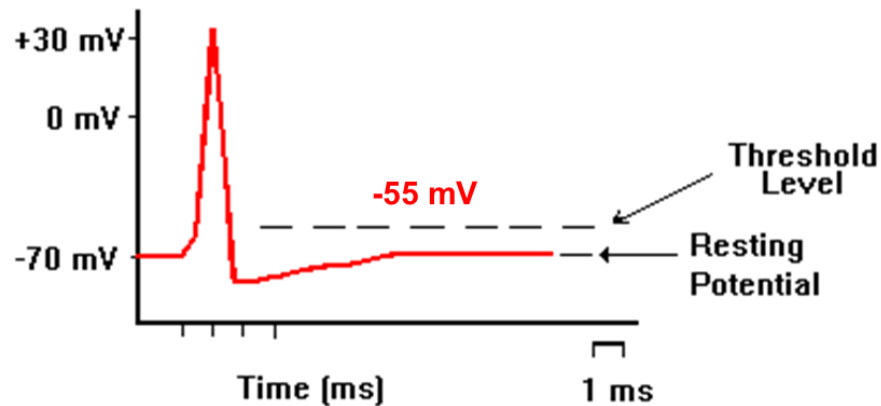
- Step 1 – Neurons waiting to fire are said to be in **resting potential** and **polarized**, or having a negative charge inside the neuron and a positive charge outside.
- Step 2 – If adequately stimulated, the neuron experiences an **action potential** and becomes **depolarized**. When this occurs,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

- Step 6 - Please note that this process is cyclical. We started at resting potential in Step 1 and end at resting potential in Step 6.

Part 2. The Action Potential

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

Figure 2.4. The Action Potential



- Recall that a neuron is usually at resting potential and polarized. The charge inside is -70mV at rest.
- If it receives sufficient stimulation, causing the polarity inside the neuron to rise from -70 mV to -55mV (**threshold of excitation**), the neuron will **fire** or send an electrical impulse down the length of the axon (the action potential or depolarization). It should be noted that it either hits -55mV and fires, or it does not fire at all. This is the **all-or-nothing principle**. The threshold must be reached.
- Once the electrical impulse has passed from one segment of the axon to the next, the neuron begins the process of resetting called repolarization.

- During repolarization the neuron will not fire no matter how much stimulation it receives. This is called the absolute refractory period.
- The neuron next moves into a relative refractory period, meaning it can fire but needs higher than normal levels of stimulation. Notice how the line has dropped below -70mV. Hence, to reach -55mV and fire, it will need more than the normal gain of +15mV (-70 to -55 mV).
- And then we return to resting potential, as you saw in Figure 2.4

Part 3. The Synapse

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

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

- **Dopamine** – controls voluntary movements and is associated with the reward mechanism in the brain
- **Serotonin** – regulates pain, sleep cycle, and digestion; leads to a stable mood, so low levels lead to depression
- **Endorphins** – involved in reducing pain and making the person calm and happy
- **Norepinephrine** – increases the heart rate and blood pressure and regulates mood
- **GABA** – blocks the signals of excitatory neurotransmitters responsible for anxiety and panic
- **Glutamate** – associated with learning and memory

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

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

- **Medulla** – regulates breathing, heart rate, and blood pressure
- **Pons** – acts as a bridge connecting the cerebellum and medulla and helps to transfer messages between different parts of the brain and spinal cord
- **Reticular formation** – responsible for alertness and attention
- **Cerebellum** – involved in our sense of balance and for coordinating the body's muscles so that movement is smooth and precise. Involved in the learning of certain kinds of simple responses and acquired reflexes.
- **Thalamus** – the major sensory relay center for all senses except smell

- **Hypothalamus** – involved in drives associated with the survival of both the individual and the species. It regulates temperature by triggering sweating or shivering and controls the complex operations of the autonomic nervous system
- **Amygdala** – responsible for evaluating sensory information and quickly determining its emotional importance
- **Hippocampus** – our “gateway” to memory. Allows us to form spatial memories so that we can accurately navigate through our environment and helps us to form new memories about facts and events
- The **cerebrum** has four distinct regions in each cerebral hemisphere. First, the **frontal lobe** contains the motor cortex, which issues orders to the muscles of the body that produce voluntary movement. The frontal lobe is also involved in emotion and in the ability to make plans, think creatively, and take initiative. The **parietal lobe** contains the somatosensory cortex and receives information about pressure, pain, touch, and temperature from sense receptors in the skin, muscles, joints, internal organs, and taste buds. The occipital lobe contains the **visual** cortex for receiving and processing visual information. Finally, the temporal lobe is involved in memory, perception, and emotion. It contains the **auditory** cortex which processes sound.

Of course, this is not an exhaustive list of structures found in the brain but gives you a pretty good idea of function and which structure is responsible for it. What is important to mental health professionals is some disorders involve specific areas of the brain. For instance, Parkinson’s disease is a brain disorder that results in a gradual loss of muscle control and arises when cells in the **substantia nigra**, a long nucleus considered to be part of the basal ganglia, stop making dopamine. As these cells die, the brain fails to receive messages about when and

how to move. In the case of depression, low levels of serotonin are responsible, at least partially. New evidence suggests “nerve cell connections, nerve cell growth, and the functioning of nerve circuits have a major impact on depression... and areas that play a significant role in depression are the amygdala, the thalamus, and the hippocampus.” Also, individuals with borderline personality disorder have been shown to have structural and functional changes in brain areas associated with impulse control and emotional regulation, while imaging studies reveal differences in the frontal cortex and subcortical structures for those suffering from OCD.

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

2.2.2. Genes, Hormonal Imbalances, and Viral Infections

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

Recent research has discovered that autism, ADHD, bipolar disorder, major depression, and schizophrenia all share genetic roots. They “were more likely to have suspect genetic variation at the same four chromosomal sites. These included risk versions of two genes that regulate the flow of calcium into cells.” Likewise, twin and family studies have shown that people with first-degree relatives suffering from OCD are at higher risk to develop the disorder themselves. The same is true of borderline personality disorder.

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

(<https://www.webmd.com/mental-health/mental-health-causes-mental-illness#1>).

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

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

2.2.2.2. Hormonal imbalances. The body has two coordinating and integrating systems, the nervous system and the endocrine system. The main difference between these two systems is the speed with which they act. The nervous system moves quickly with nerve impulses moving in a few hundredths of a second. The endocrine system moves slowly with hormones, released by endocrine glands, taking seconds, or even minutes, to reach their target. Hormones are important to psychologists because they manage the nervous system and body tissues at certain

stages of development and activate behaviors such as alertness or sleepiness, sexual behavior, concentration, aggressiveness, reaction to stress, and a desire for companionship. The **pituitary gland** is the “master gland” which regulates other endocrine glands. It influences blood pressure, thirst, contractions of the uterus during childbirth, milk production, sexual behavior and interest, body growth, the amount of water in the body’s cells, and other functions as well. The **pineal gland** helps regulate the sleep-wake cycle while the **thyroid gland** regulates the body’s energy levels by controlling metabolism and the basal metabolic rate (BMR). It regulates the body’s rate of metabolism and so how energetic people are.

Of importance to mental health professionals are the **adrenal glands**, located on top of the kidneys, and which release *cortisol* to help the body deal with stress. Elevated levels of this hormone can lead to several problems, including increased weight gain, interference with learning and memory, reduced bone density, high cholesterol, and an increased risk of depression. Similarly, the overproduction of the hormone melatonin can lead to SAD.

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

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

2.2.2.3. Bacterial and viral infections. Infections can cause brain damage and lead to the development of mental illness or exacerbate existing symptoms. For instance, evidence suggests that contracting strep throat, “an infection in the throat and tonsils caused by bacteria called group A *Streptococcus*” (for more on strep throat, please visit <https://www.cdc.gov/groupastrep/diseases-public/strep-throat.html>), can lead to the development of OCD, Tourette’s syndrome, and tic disorder in children (Mell, Davis, & Owens, 2005; Giedd

et al., 2000; Allen et al., 1995; <https://www.psychologytoday.com/blog/the-perfectionists-handbook/201202/can-infections-result-in-mental-illness>). Influenza epidemics, caused by viral infections (for more on influenza, please see the following: <https://www.mayoclinic.org/diseases-conditions/flu/symptoms-causes/syc-20351719>), have also been linked to schizophrenia (Brown et al., 2004; McGrath and Castle, 1995; McGrath et al., 1994; O'callaghan et al., 1991) though more recent research suggests this evidence is weak at best (Selten & Termorshuizen, 2017; Ebert & Kotler, 2005).

2.2.3. Treatments

2.2.3.1. Psychopharmacology and psychotropic drugs. One option to treat severe mental illness is psychotropic medications. These medications fall under five major categories. *Antidepressants* are used to treat depression, but also anxiety, insomnia, and pain. The most common types of antidepressants are SSRIs or selective serotonin reuptake inhibitors and include Citalopram, Paroxetine, and Fluoxetine (Prozac). Possible side effects include weight gain, sleepiness, nausea and vomiting, panic attacks, or thoughts about suicide or dying.

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

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

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

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

For more information on psychotropic medications, please visit:

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

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

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

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

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

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

2.2.4. Evaluation of the Model

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

More on that over the next two sections.

You should have learned the following in this section:

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

Section 2.2 Review Questions

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

2.3. Psychological Perspectives

Section Learning Objectives

- Describe the psychodynamic theory.
- Outline the structure of personality and how it develops over time.
- Describe ways to deal with anxiety.
- Clarify what psychodynamic techniques are used.
- Evaluate the usefulness of psychodynamic theory.
- Describe learning.
- Outline respondent conditioning and the work of Pavlov and Watson.
- Outline operant conditioning and the work of Thorndike and Skinner.
- Outline observational learning/social-learning theory and the work of Bandura.
- Evaluate the usefulness of the behavioral model.
- Define the cognitive model.
- Exemplify the effect of schemas on creating abnormal behavior.
- Exemplify the effect of attributions on creating abnormal behavior.
- Exemplify the effect of maladaptive cognitions on creating abnormal behavior.
- List and describe cognitive therapies.
- Evaluate the usefulness of the cognitive model.
- Describe the humanistic perspective.
- Describe the existential perspective.
- Evaluate the usefulness of humanistic and existential perspectives.

2.3.1. Psychodynamic Theory

In 1895, the book, *Studies on Hysteria*, was published by Josef Breuer (1842-1925) and Sigmund Freud (1856-1939), and marked the birth of psychoanalysis, though Freud did not use this actual term until a year later. The book published several case studies, including that of Anna O., born February 27, 1859 in Vienna to Jewish parents Siegmund and Recha Pappenheim, strict Orthodox adherents who were considered millionaires at the time. Bertha, known in published case studies as Anna O., was expected to complete the formal education typical of upper-middle-class girls, which included foreign language, religion, horseback riding, needlepoint, and piano. She felt confined and suffocated in this life and took to a fantasy world she called her “private theater.” Anna also developed hysteria, including symptoms such as memory loss, paralysis, disturbed eye movements, reduced speech, nausea, and mental deterioration. Her symptoms appeared as she cared for her dying father, and her mother called on Breuer to diagnosis her condition (note that Freud never actually treated her). Hypnosis was used at first and relieved her symptoms, as it had done for many patients (See Module 1). Breuer made daily visits and allowed her to share stories from her private theater, which she came to call “talking cure” or “chimney sweeping.” Many of the stories she shared were actually thoughts or events she found troubling and reliving them helped to relieve or eliminate the symptoms. Breuer’s wife, Mathilde, became jealous of her husband’s relationship with the young girl, leading Breuer to terminate treatment in June of 1882 before Anna had fully recovered. She relapsed and was admitted to Bellevue Sanatorium on July 1, eventually being released in October of the same year. With time, Anna O. did recover from her hysteria and went on to become a prominent member of the Jewish Community, involving herself in social work, volunteering at soup kitchens, and becoming ‘House Mother’ at an orphanage for Jewish girls in 1895. Bertha (Anna

O.) became involved in the German Feminist movement, and in 1904 founded the League of Jewish Women. She published many short stories; a play called *Women's Rights*, in which she criticized the economic and sexual exploitation of women; and wrote a book in 1900 called *The Jewish Problem in Galicia*, in which she blamed the poverty of the Jews of Eastern Europe on their lack of education. In 1935, Bertha was diagnosed with a tumor, and in 1936, she was summoned by the Gestapo to explain anti-Hitler statements she had allegedly made. She died shortly after this interrogation on May 28, 1936. Freud considered the talking cure of Anna O. to be the origin of psychoanalytic therapy and what would come to be called the cathartic method.

For more on Anna O., please see:

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

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

According to Freud, our personality has three parts – the id, superego, and ego, and from these our behavior arises. First, the **id** is the impulsive part that expresses our sexual and aggressive instincts. It is present at birth, completely unconscious, and operates on the *pleasure*

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

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

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

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

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

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

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

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

For more on defense mechanisms, please visit:

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

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

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

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

2.3.1.5. Evaluating psychodynamic theory. Freud's psychodynamic theory made a lasting impact on the field of psychology but also has been criticized heavily. First, Freud made most of his observations in an unsystematic, uncontrolled way, and he relied on the case study method. Second, the participants in his studies were not representative of the broader population. Despite Freud's generalization, his theory was based on only a few patients. Third, he relied solely on the reports of his patients and sought no observer reports. Fourth, it is difficult to empirically study psychodynamic principles since most operate unconsciously. This begs the question of how we can really know that they exist. Finally, psychoanalytic treatment is expensive and time

consuming, and since Freud's time, drug therapies have become more popular and successful. Still, Sigmund Freud developed useful therapeutic tools for clinicians and raised awareness about the role the unconscious plays in both normal and abnormal behavior.

2.3.2. The Behavioral Model

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

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

Behaviorism is the school of thought associated with learning that began in 1913 with the publication of John B. Watson's article, "Psychology as the Behaviorist Views It," in the journal *Psychological Review* (Watson, 1913). Watson believed that the subject matter of psychology was to be observable behavior, and to that end, psychology should focus on the prediction and

control of behavior. Behaviorism was dominant from 1913 to 1990 before being absorbed into mainstream psychology. It went through three major stages – behaviorism proper under Watson and lasting from 1913-1930 (discussed as classical/respondent conditioning), neobehaviorism under Skinner and lasting from 1930-1960 (discussed as operant conditioning), and sociobehaviorism under Bandura and Rotter and lasting from 1960-1990 (discussed as social learning theory).

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

The important thing to understand is that not all behaviors occur due to reinforcement and punishment as operant conditioning says. In the case of respondent conditioning, stimuli exert complete and automatic control over some behaviors. We see this in the case of reflexes. When a doctor strikes your knee with that little hammer, your leg extends out automatically. Another example is how a baby will root for a food source if the mother's breast is placed near their mouth. And if a nipple is placed in their mouth, they will also automatically suck via the sucking

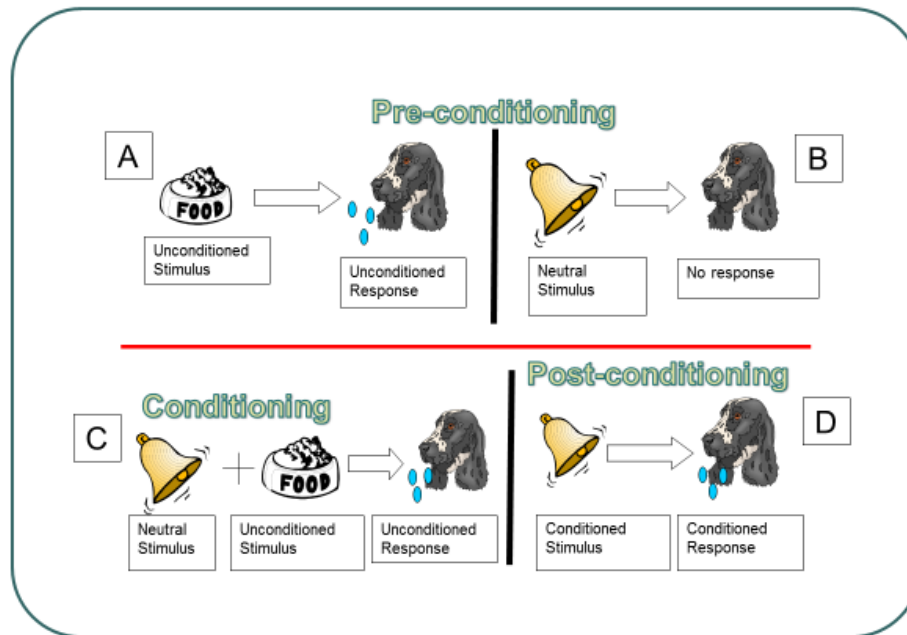
reflex. Humans have several of these reflexes, though not as many as other animals due to our more complicated nervous system.

Respondent conditioning (also called classical or Pavlovian conditioning) occurs when we link a previously neutral stimulus with a stimulus that is unlearned or inborn, called an unconditioned stimulus. In respondent conditioning, learning happens in three phases: preconditioning, conditioning, and postconditioning. See Figure 2.5 for an overview of Pavlov's classic experiment.

Preconditioning. Notice that preconditioning has both an A and a B panel. All this stage of learning signifies is that some learning is already present. There is no need to learn it again, as in the case of primary reinforcers and punishers in operant conditioning. In Panel A, food makes a dog salivate. This response does not need to be learned and shows the relationship between an unconditioned stimulus (UCS) yielding an unconditioned response (UCR). Unconditioned means unlearned. In Panel B, we see that a neutral stimulus (NS) produces no response. Dogs do not enter the world knowing to respond to the ringing of a bell (which it hears).

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

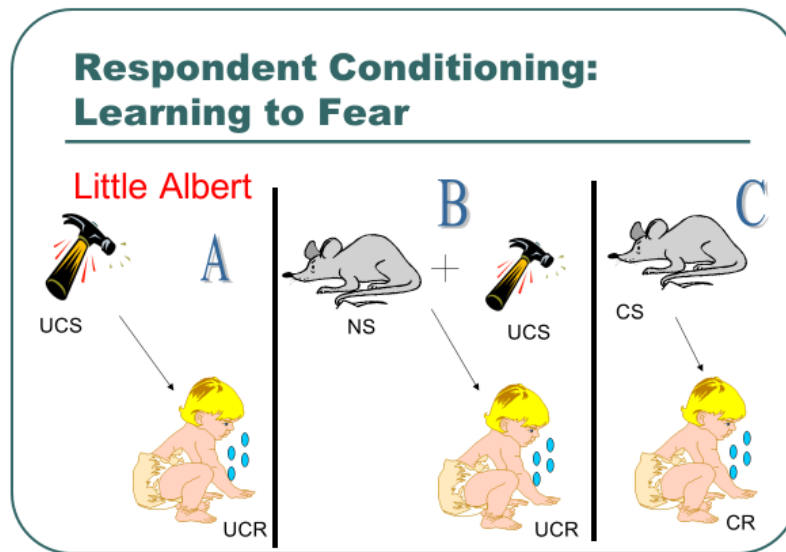
Figure 2.5. Pavlov's Classic Experiment



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

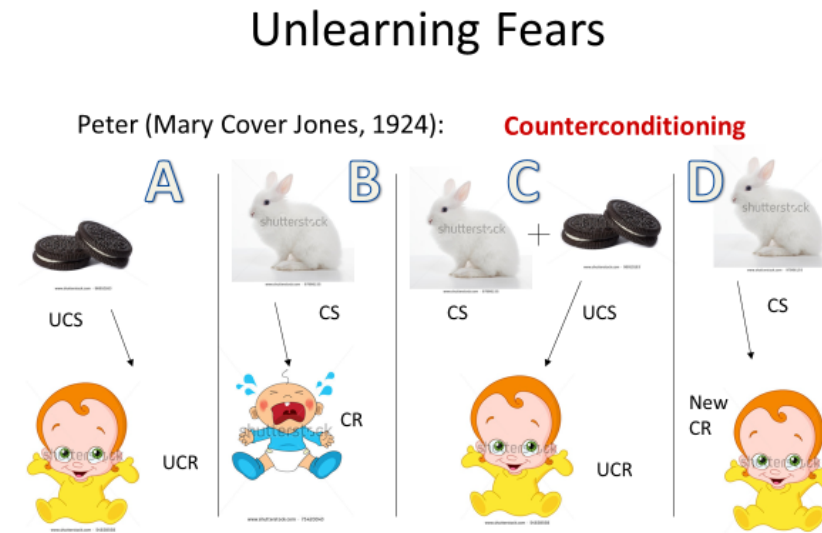
Watson and Rayner (1920) conducted one of the most famous studies in psychology. Essentially, they wanted to explore “the possibility of conditioning various types of emotional response(s).” The researchers ran a series of trials in which they exposed a 9-month-old child, known as Little Albert, to a white rat. Little Albert made no response outside of curiosity (NS–NR not shown). Panel A of Figure 2.6 shows the naturally occurring response to the stimulus of a loud sound. On later trials, the rat was presented (NS) and followed closely by a loud sound (UCS; Panel B). After several conditioning trials, the child responded with fear to the mere presence of the white rat (Panel C).

Figure 2.6. Learning to Fear



As fears can be learned, so too they can be unlearned. Considered the follow-up to Watson and Rayner (1920), Jones (1924; Figure 2.7) wanted to see if a child who learned to be afraid of white rabbits (Panel B) could be conditioned to become unafraid of them. Simply, she placed the child in one end of a room and then brought in the rabbit. The rabbit was far enough away so as not to cause distress. Then, Jones gave the child some pleasant food (i.e., something sweet such as cookies [Panel C]; remember the response to the food is unlearned, i.e., Panel A). The procedure in Panel C continued with the rabbit being brought a bit closer each time until, eventually, the child did not respond with distress to the rabbit (Panel D).

Figure 2.7. Unlearning Fears



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

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

Finally, respondent conditioning has several properties:

- **Respondent Generalization** – When many similar CSs or a broad range of CSs elicit the same CR. An example is the sound of a whistle eliciting salivation much the same as a ringing bell, both detected via audition.
- **Respondent Discrimination** – When a single CS or a narrow range of CSs elicits a CR, i.e., teaching the dog to respond to a specific bell and ignore the whistle. The whistle would not be followed by food, eventually leading to....

- **Respondent Extinction** – When the CS is no longer paired with the UCS. The sound of a school bell ringing (new CS that was generalized) is not followed by food (UCS), and so eventually, the dog stops salivating (the CR).
- **Spontaneous Recovery** – When the CS elicits the CR after extinction has occurred. Eventually, the school bell will ring, making the dog salivate. If no food comes, the behavior will not continue. If food appears, the salivation response will be re-established.

2.3.2.3. Operant conditioning. Influential on the development of Skinner's operant conditioning, Thorndike (1905) proposed the **law of effect** or the idea that if our behavior produces a favorable consequence, in the future when the same stimulus is present, we will be more likely to make the response again, expecting the same favorable consequence. Likewise, if our action leads to dissatisfaction, then we will not repeat the same behavior in the future. He developed the law of effect thanks to his work with a puzzle box. Cats were food deprived the night before the experimental procedure was to occur. The next morning, researchers placed a hungry cat in the puzzle box and set a small amount of food outside the box, just close enough to be smelled. The cat could escape the box and reach the food by manipulating a series of levers. Once free, the cat was allowed to eat some food before being promptly returned to the box. With each subsequent escape and re-insertion into the box, the cat became faster at correctly manipulating the levers. This scenario demonstrates **trial and error learning** or making a response repeatedly if it leads to success. Thorndike also said that stimulus and responses were connected by the organism, and this led to learning. This approach to learning was called **connectionism**.

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

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

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

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

Figure 2.8. Contingencies in Operant Conditioning

	Some “ Bad ” Thing	Some “ Good ” Thing
Giving	Positive Punishment	Positive Reinforcement
Taking Away	Negative Reinforcement	Negative Punishment

Let’s go through each:

- **Positive Punishment (PP)** – If something bad or aversive is given or added, then the behavior is less likely to occur in the future. If you talk back to your mother and she slaps your mouth, this is a PP. Your response of talking back led to the consequence of the aversive slap being given to your face. Ouch!!!
- **Positive Reinforcement (PR)** – If something good is given or added, then the behavior is more likely to occur in the future. If you study hard and receive an A on your exam, you will be more likely to study hard in the future. Similarly, your parents may give you money for your stellar performance. Cha Ching!!!
- **Negative Reinforcement (NR)** – This is a tough one for students to comprehend because the terms seem counterintuitive, even though we experience NR all the time. NR is when something bad or aversive is taken away or subtracted due to your actions, making it that you will be more likely to make the same behavior in the

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

- **Negative Punishment (NP)** – This is when something good is taken away or subtracted, making a behavior less likely in the future. If you are late to class and your professor deducts 5 points from your final grade (the points are something good and the loss is negative), you will hopefully be on time in all subsequent classes.

The type of reinforcer or punisher we use is crucial. Some are naturally occurring, while others need to be learned. We describe these as primary and secondary reinforcers and punishers. *Primary* refers to reinforcers and punishers that have their effect without having to be learned. Food, water, temperature, and sex, for instance, are primary reinforcers, while extreme cold or hot or a punch on the arm are inherently punishing. A story will illustrate the latter. When I was about eight years old, I would walk up the street in my neighborhood, saying, “I’m Chicken Little and you can’t hurt me.” Most ignored me, but some gave me the attention I was seeking, a positive reinforcer. So I kept doing it and doing it until one day, another kid grew tired of hearing

about my other identity and punched me in the face. The pain was enough that I never walked up and down the street echoing my identity crisis for all to hear. This was a positive punisher that did not have to be learned, and definitely not one of my finer moments in life.

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

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

Figure 2.9. Key Components of Reinforcement Schedules

Two Key Components

1. Fixed Variable	or	Reinforcement occurs at a set rate Rate of reinforcement changes
2. Ratio Interval	or	The number of correct responses Time elapsed between correct responses

Figure 2.9 shows that there are two main components that make up a reinforcement schedule – when you will reinforce and what is being reinforced. In the case of when, it will be either fixed or at a set rate, or variable and at a rate that changes. In terms of what is being reinforced, we will either reinforce responses or time. These two components pair up as follows:

- **Fixed Ratio schedule (FR)** – With this schedule, we reinforce some set number of responses. For instance, every twenty problems (fixed) a student gets correct (ratio), the teacher gives him an extra credit point. A specific behavior is being reinforced – getting problems correct. Note that if we reinforce each occurrence of the behavior, the definition of continuous reinforcement, we could also describe this as an FR1 schedule. The number indicates how many responses have to be made, and in this case, it is one.
- **Variable Ratio schedule (VR)** – We might decide to reinforce some varying number of responses, such as if the teacher gives him an extra credit point after finishing

between 40 and 50 correct problems. This approach is useful if the student is learning the material and does not need regular reinforcement. Also, since the schedule changes, the student will keep responding in the absence of reinforcement.

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

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

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

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


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

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

Albert Bandura conducted pivotal research on observational learning, and you likely already know all about it. Check out Figure 2.10 to see if you do. In Bandura's experiment, children were first brought into a room to watch a video of an adult playing nicely or aggressively with a Bobo doll, which provided a model. Next, the children are placed in a room with several toys in it. The room contains a highly prized toy, but they are told they cannot play with it. All other toys are allowed, including a Bobo doll. Children who watched the aggressive model behaved aggressively with the Bobo doll while those who saw the gentle model, played nice. Both groups were frustrated when deprived of the coveted toy.

Figure 2.10. Bandura's Classic Experiment

Observational Learning: learning through watching others



In Bandura's (1965) experiment, most children who watched an aggressive model attack a Bobo doll later imitated that behavior.

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

Modeling techniques change behavior by having subjects observe a model in a situation that usually causes them some anxiety. By seeing the model interact nicely with the fear evoking stimulus, their fear should subside. This form of behavior therapy is widely used in clinical, business, and classroom situations. In the classroom, we might use modeling to demonstrate to a student how to do a math problem. In fact, in many college classrooms, this is exactly what the instructor does. In the business setting, a model or trainer demonstrates how to use a computer program or run a register for a new employee.

However, keep in mind that we do not model everything we see. Why? First, we cannot pay attention to everything going on around us. We are more likely to model behaviors by

someone who commands our attention. Second, we must remember what a model does to imitate it. If a behavior is not memorable, it will not be imitated. We must try to convert what we see into action. If we are not motivated to perform an observed behavior, we probably will not show what we have learned.

2.3.2.5. Evaluating the behavioral model. Within the context of psychopathology, the behavioral perspective is useful because explains maladaptive behavior in terms of learning gone awry. The good thing is that what is learned can be unlearned or relearned through **behavior modification**, the process of changing behavior. To begin, an applied behavior analyst identifies a target behavior, or behavior to be changed, defines it, works with the client to develop goals, conducts a functional assessment to understand what the undesirable behavior is, what causes it, and what maintains it. With this knowledge, a plan is developed and consists of numerous strategies to act on one or all these elements – antecedent, behavior, and/or consequence. The strategies arise from all three learning models. In terms of operant conditioning, strategies include antecedent manipulations, prompts, punishment procedures, differential reinforcement, habit reversal, shaping, and programming. Flooding and desensitization are typical respondent conditioning procedures used with phobias, and modeling arises from social learning theory and observational learning. Watson and Skinner defined behavior as what we do or say, but later behaviorists added what we think or feel. In terms of the latter, cognitive behavior modification procedures arose after the 1960s and with the rise of cognitive psychology. This led to a cognitive-behavioral perspective that combines concepts from the behavioral and cognitive models, the latter discussed in the next section.

Critics of the behavioral perspective point out that it oversimplifies behavior and often ignores inner determinants of behavior. Behaviorism has also been accused of being mechanistic

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

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

2.3.3. The Cognitive Model

2.3.3.1. What is it? As noted earlier, the idea of people being machines, called **mechanism**, was a key feature of behaviorism and other schools of thought in psychology until about the 1960s or 1970s. In fact, behaviorism said psychology was to be the study of observable behavior. Any reference to cognitive processes was dismissed as this was not overt, but covert

according to Watson and later Skinner. Of course, removing cognition from the study of psychology ignored an important part of what makes us human and separates us from the rest of the animal kingdom. Fortunately, the work of George Miller, Albert Ellis, Aaron Beck, and Ulrich Neisser demonstrated the importance of cognitive abilities in understanding thoughts, behaviors, and emotions, and in the case of psychopathology, show that people can create their problems by how they come to interpret events experienced in the world around them. How so?

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

Can our schemas lead us astray or be false? Consider where students sit in a class. It is generally understood that the students who sit in the front of the class are the overachievers and want to earn an A in the class. Those who sit in the back of the room are underachievers who don't care. Right? Where do you sit in class, if you are on a physical campus and not an online student? Is this correct? What about other students in the class that you know? What if you found out that a friend who sits in the front row is a C student but sits there because he cannot see the screen or board, even with corrective lenses? What about your friend or acquaintance in the

back? This person is an A student but does not like being right under the nose of the professor, especially if he/she tends to spit when lecturing. The person in the back could also be shy and prefer sitting there so that s/he does not need to chat with others as much. Or, they are easily distracted and sits in the back so that all stimuli are in front of him/her. Again, your schema about front row and back row students is incorrect and causes you to make certain assumptions about these individuals. This might even affect how you interact with them. Would you want notes from the student in the front or back of the class?

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

2.3.3.4. Maladaptive cognitions. Irrational thought patterns can be the basis of psychopathology. Throughout this book, we will discuss several treatment strategies used to change unwanted, maladaptive cognitions, whether they are present as an *excess* such as with

paranoia, suicidal ideation, or feelings of worthlessness; or as a *deficit* such as with self-confidence and self-efficacy. More specifically, cognitive distortions/maladaptive cognitions can take the following forms:

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

2.3.3.5. Cognitive therapies. According to the National Alliance on Mental Illness (NAMI), **cognitive behavioral therapy** “focuses on exploring relationships among a person's thoughts, feelings and behaviors. During CBT a therapist will actively work with a person to uncover unhealthy patterns of thought and how they may be causing self-destructive behaviors and beliefs.” CBT attempts to identify negative or false beliefs and restructure them. They add, “Oftentimes someone being treated with CBT will have homework in between sessions where

they practice replacing negative thoughts with more realistic thoughts based on prior experiences or record their negative thoughts in a journal.” For more on CBT, visit:

<https://www.nami.org/About-Mental-Illness/Treatments/Psychotherapy>. Some commonly used strategies include cognitive restructuring, cognitive coping skills training, and acceptance techniques.

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

1. Notice when you are having a maladaptive cognition, such as making “negative predictions.” Figure out what is the worst thing that could happen and what alternative outcomes are possible.
2. Track the accuracy of the thought. If you believe focusing on a problem generates a solution, then write down each time you ruminate and the result. You can generate a percentage of times you ruminated to the number of successful problem-solving strategies you generated.
3. Behaviorally test your thought. Try figuring out if you genuinely do not have time to go to the gym by recording what you do each day and then look at open times of the day. Add them up and see if making some minor, or major, adjustments to your schedule will free an hour to get in some valuable exercise.

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

The article also suggested a few non-cognitive restructuring techniques, including mindfulness meditation and self-compassion. For more on these, visit:

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

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

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

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

2.3.4. The Humanistic and Existential Perspectives

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

Rogers said that all people want to have *positive regard* from significant others in their life. When the individual is accepted as they are, they receive *unconditional positive regard* and become a *fully functioning person*. They are open to experience, live every moment to the fullest, are creative, 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 becomes distorted, now seen as having worth only when these significant others approve, leading to a disharmonious state and psychopathology. Individuals in this situation are unsure of what they feel, value, or need leading to dysfunction and the need for therapy. Rogers stated that the humanistic therapist should be warm, understanding, supportive, respectful, and accepting of his/her clients. This approach came to be called **client-centered therapy**.

2.3.4.2. The existential perspective. This approach stresses the need for people to re-create themselves continually and be self-aware, acknowledges that anxiety is a normal part of life, focuses on free will and self-determination, emphasizes that each person has a unique identity known only through relationships and the search for meaning, and finally, that we develop to our maximum potential. Abnormal behavior arises when we avoid making choices, do not take responsibility, and fail to actualize our full potential. Existential therapy is used to treat substance abuse, “excessive anxiety, apathy, alienation, nihilism, avoidance, shame, addiction, despair, depression, guilt, anger, rage, resentment, embitterment, purposelessness, psychosis, and violence. They also focus on life-enhancing experiences like relationships, love, caring, commitment, courage, creativity, power, will, presence, spirituality, individuation, self-actualization, authenticity, acceptance, transcendence, and awe.” For more information, please visit: <https://www.psychologytoday.com/therapy-types/existential-therapy>

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

You should have learned the following in this section:

- According to Freud, consciousness had three levels (consciousness, preconscious, and the unconscious), personality had three parts (the id, ego, and superego), personality developed over five stages (oral, anal, phallic, latency, and genital), there are ten defense mechanisms to protect the ego such as repression and sublimation, and finally three assessment techniques (free association, transference, and dream analysis) could be used to understand the personalities of his patients and expose repressed material.
- The behavioral model concerns the cognitive process of learning, which is any relatively permanent change in behavior due to experience and practice and has two main forms – associative learning to include classical and operant conditioning and observational learning.
- Respondent conditioning (also called classical or Pavlovian conditioning) occurs when we link a previously neutral stimulus with a stimulus that is unlearned or inborn, called an unconditioned stimulus.
- Operant conditioning is a type of associate learning which focuses on consequences that follow a response or behavior that we make (anything we do, say, or think/feel) and whether it makes a behavior more or less likely to occur.
- Observational learning is learning by watching others and modeling techniques change behavior by having subjects observe a model in a situation that usually causes them some anxiety.
- The cognitive model focuses on schemas, cognitive errors, attributions, and maladaptive cognitions and offers strategies such as CBT, cognitive restructuring, cognitive coping skills training, and acceptance.
- The humanistic perspective focuses on positive regard, conditions of worth, and the fully functioning person while the existential perspective stresses the need for people to re-create themselves continually and be self-aware, acknowledges that anxiety is a normal part of life, focuses on free will and self-determination, emphasizes that each person has a unique identity known only through relationships and the search for meaning, and finally, that we develop to our maximum potential.

Section 2.3 Review Questions

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

2.4. The Sociocultural Model

Section Learning Objectives

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

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

2.4.1. Socioeconomic Factors

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

income brackets (Tomko et al., 2012) and group differences for personality disorders have been found between African and European Americans (Ryder, Sunohara, and Kirmayer, 2015).

2.4.2. Gender Factors

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

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

Consider this...

In relation to men: “While mental illnesses affect both men and women, the prevalence of mental illnesses in men is often lower than women. Men with mental illnesses are also less likely to have received mental health treatment than women in the past year. However, men are more likely to die by suicide than women, according to the [Centers for Disease Control and Prevention](#). Recognizing the signs that you or someone you love may have a mental disorder is the first step toward getting treatment. The earlier that treatment begins, the more effective it can be.”

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

In relation to women: “Some disorders are more common in women such as [depression](#) and [anxiety](#). There are also certain types of disorders that are unique to women. For example, some women may experience symptoms of mental disorders at times of hormone change, such as perinatal depression, premenstrual dysphoric disorder, and perimenopause-related depression. When it comes to other mental disorders such as [schizophrenia](#) and [bipolar disorder](#), research has not found differences in the rates at which men and women experience these illnesses. But women may experience these illnesses differently – certain symptoms may be more common in women than in men, and the course of the illness can be affected by the sex of the individual. Researchers are only now beginning to tease apart the various biological and psychosocial factors that may impact the mental health of both women and men.”

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

2.4.3. Environmental Factors

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

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

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

2.4.4. Multicultural Factors

Racial, ethnic, and cultural factors are also relevant to understanding the development and course of mental illness. Multicultural psychologists assert that both normal behavior and abnormal behavior need to be understood in the context of the individual's unique culture and the group's value system. Racial and ethnic minorities must contend with prejudice, discrimination, racism, economic hardships, etc. as part of their daily life and this can lead to disordered behavior (Lo & Cheng, 2014; Jones, Cross, & DeFour, 2007; Satcher, 2001), though

some research suggests that ethnic identity can buffer against these stressors and protect mental health (Mossakowski, 2003). To address this unique factor, **culture-sensitive therapies** have been developed and include increasing the therapist's awareness of cultural values, hardships, stressors, and/or prejudices faced by their client; the identification of suppressed anger and pain; and raising the client's self-worth (Prochaska & Norcross, 2013). These therapies have proven efficacy for the treatment of depression (Kalibatseva & Leong, 2014) and schizophrenia (Naeem et al., 2015).

2.4.5. Evaluation of the Model

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

You should have learned the following in this section:

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

Section 2.4 Review Questions

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

Module Recap

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

Part I. Setting the Stage

Module 3: Clinical Assessment, Diagnosis, and Treatment

Module 3: Clinical Assessment, Diagnosis, and Treatment

Module Overview

Module 3 covers the issues of clinical assessment, diagnosis, and treatment. We will define assessment and then describe key issues such as reliability, validity, standardization, and specific methods that are used. In terms of clinical diagnosis, we will discuss the two main classification systems used around the world – the DSM-5-TR and ICD-11. Finally, we discuss the reasons why people may seek treatment and what to expect when doing so.

Module Outline

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

Module Learning Outcomes

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

3.1. Clinical Assessment of Abnormal Behavior

Section Learning Objectives

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

3.1.1. What is Clinical Assessment?

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

Consider this. First, we need to determine if a treatment is even needed. By having a clear accounting of the person's symptoms and how they affect daily functioning, we can decide to what extent the individual is adversely affected. Assuming a treatment is needed, our second

reason to engage in clinical assessment will be to determine what treatment will work best. As you will see later in this module, there are numerous approaches to treatment. These include Behavior Therapy, Cognitive and Cognitive-Behavioral Therapy (CBT), Humanistic-Experiential Therapies, Psychodynamic Therapies, Couples and Family Therapy, and biological treatments (psychopharmacology). Of course, for any mental disorder, some of the aforementioned therapies will have greater efficacy than others. Even if several can work well, it does not mean a particular therapy will work well for that specific client. Assessment can help figure this out. Finally, we need to know if the treatment we employed worked. This will involve measuring before any treatment is used and then measuring the behavior while the treatment is in place. We will even want to measure after the treatment ends to make sure symptoms of the disorder do not return. Knowing what the person's baselines are for different aspects of psychological functioning will help us to see when improvement occurs.

In recap, obtaining the baselines happens in the beginning, implementing the treatment plan that is agreed upon happens more so in the middle, and then making sure the treatment produces the desired outcome occurs at the end. It should be clear from this discussion that clinical assessment is an *ongoing* process.

3.1.2. Key Concepts in Assessment

The assessment process involves three critical concepts – reliability, validity, and standardization. These three are important to science in general. First, we want the assessment to be **reliable** or consistent. Outside of clinical assessment, when our car has an issue and we take it to the mechanic, we want to make sure that what one mechanic says is wrong with our car is the same as what another says, or even two others. If not, the measurement tools they use to assess

cars are flawed. The same is true of a patient who is suffering from a mental disorder. If one mental health professional says the person suffers from major depressive disorder and another says the issue is borderline personality disorder, then there is an issue with the assessment tool being used. Ensuring that two different raters are consistent in their assessment of patients is called *interrater reliability*. Another type of reliability occurs when a person takes a test one day, and then the same test on another day. We would expect the person's answers to be consistent, which is called *test-retest reliability*. For example, let's say the person takes the MMPI on Tuesday and then the same test on Friday. Unless something miraculous or tragic happened over the two days in between tests, the scores on the MMPI should be nearly identical to one another. What does identical mean? The score at test and the score at retest are correlated with one another. If the test is reliable, the correlation should be very high (remember, a correlation goes from -1.00 to +1.00, and positive means as one score goes up, so does the other, so the correlation for the two tests should be high on the positive side).

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

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

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

3.1.3. Methods of Assessment

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

3.1.3.1. Observation. In Section 1.5.2.1 we talked about two types of observation – *naturalistic*, or observing the person or animal in their environment, and *laboratory*, or observing the organism in a more controlled or artificial setting where the experimenter can use sophisticated equipment and videotape the session to examine it later. One-way mirrors can also be used. A limitation of this method is that the process of recording a behavior causes the

behavior to change, called **reactivity**. Have you ever noticed someone staring at you while you sat and ate your lunch? If you have, what did you do? Did you change your behavior? Did you become self-conscious? Likely yes, and this is an example of reactivity. Another issue is that the behavior made in one situation may not be made in other situations, such as your significant other only acting out at the football game and not at home. This form of validity is called **cross-sectional validity**. We also need our raters to observe and record behavior in the same way or to have high inter-rater reliability.

3.1.3.2. The clinical interview. A clinical interview is a face-to-face encounter between a mental health professional and a patient in which the former observes the latter and gathers data about the person's behavior, attitudes, current situation, personality, and life history. The interview may be *unstructured* in which open-ended questions are asked, *structured* in which a specific set of questions according to an interview schedule are asked, or *semi-structured*, in which there is a pre-set list of questions, but clinicians can follow up on specific issues that catch their attention. A **mental status examination** is used to organize the information collected during the interview and systematically evaluates the patient through a series of questions assessing appearance and behavior. The latter includes grooming and body posture, thought processes and content to include disorganized speech or thought and false beliefs, mood and affect such that whether the person feels hopeless or elated, intellectual functioning to include speech and memory, and awareness of surroundings to include where the person is and what the day and time are. The exam covers areas not normally part of the interview and allows the mental health professional to determine which areas need to be examined further. The limitation of the interview is that it lacks reliability, especially in the case of the unstructured interview.

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

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

3.1.3.4. Neurological tests. Neurological tests are used to diagnose cognitive impairments caused by brain damage due to tumors, infections, or head injuries; or changes in

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

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

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

our own behavior, which is called self-modification. The person does their own measuring and recording of the ABCs, which is called **self-monitoring**. In the context of psychopathology, behavior modification can be useful in treating phobias, reducing habit disorders, and ridding the person of maladaptive cognitions.

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

You should have learned the following in this section:

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

Section 3.1 Review Questions

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

3.2. Diagnosing and Classifying Abnormal Behavior

Section Learning Objectives

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

3.2.1. Clinical Diagnosis and Classification Systems

Before starting any type of treatment, the client/patient must be clearly diagnosed with a mental disorder. **Clinical diagnosis** is the process of using assessment data to determine if the pattern of symptoms the person presents with is consistent with the diagnostic criteria for a specific mental disorder outlined in an established classification system such as the DSM-5-TR or ICD-11 (both will be described shortly). Any diagnosis should have *clinical utility*, meaning it aids the mental health professional in determining prognosis, the treatment plan, and possible outcomes of treatment (APA, 2022). Receiving a diagnosis does not necessarily mean the person requires treatment. This decision is made based upon how severe the symptoms are, level of distress caused by the symptoms, symptom salience such as expressing suicidal ideation, risks

and benefits of treatment, disability, and other factors (APA, 2022). Likewise, a patient may not meet the full criteria for a diagnosis but demonstrate a clear need for treatment or care, nonetheless. As stated in the DSM, “The fact that some individuals do not show all symptoms indicative of a diagnosis should not be used to justify limiting their access to appropriate care” (APA, 2022).

Symptoms that cluster together regularly are called a **syndrome**. If they also follow the same, predictable course, we say that they are characteristic of a *specific disorder*.

Classification systems provide mental health professionals with an agreed-upon list of disorders falling into distinct categories for which there are clear descriptions and criteria for making a diagnosis. Distinct is the keyword here. People suffering from delusions, hallucinations, disorganized thinking (speech), grossly disorganized or abnormal motor behavior, and/or negative symptoms are different from people presenting with a primary clinical deficit in cognitive functioning that is not developmental but acquired (i.e., they have shown a decline in cognitive functioning over time). The former suffers from a schizophrenia spectrum disorder while the latter suffers from a neurocognitive disorder (NCD). The latter can be further distinguished from neurodevelopmental disorders which manifest early in development and involve developmental deficits that cause impairments in social, personal, academic, or occupational functioning (APA, 2022). These three disorder groups or categories can be clearly distinguished from one another. Classification systems also permit the gathering of statistics to determine incidence and prevalence rates and conform to the requirements of insurance companies for the payment of claims.

The most widely used classification system in the United States is the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) which is a “medical classification of disorders

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

3.2.2. The DSM Classification System

3.2.2.1. A brief history of the DSM. The DSM-5 was published in 2013 and took the place of the DSM IV-TR (TR means Text Revision; published in 2000). In March 2022, a Text-Revision was published for the DSM-5, making it the DSM-5-TR.

The history of the DSM goes back to 1952 when the American Psychiatric Association published the first edition of the DSM which was “...the first official manual of mental disorders to contain a glossary of descriptions of the diagnostic categories” (APA, 2022, p. 5). The DSM evolved through four major editions after World War II into a diagnostic classification system to be used by psychiatrists and physicians, but also other mental health professionals. The Herculean task of revising the DSM began in 1999 when the APA embarked upon an evaluation of the strengths and weaknesses of the DSM in coordination with the World Health Organization (WHO) Division of Mental Health, the World Psychiatric Association, and the National Institute of Mental Health (NIMH). This collaboration resulted in the publication of a monograph in 2002 called *A Research Agenda for DSM-V*. From 2003 to 2008, the APA, WHO, NIMH, the National

Institute on Drug Abuse (NIDA), and the National Institute on Alcoholism and Alcohol Abuse (NIAAA) convened 13 international DSM-5 research planning conferences “to review the world literature in specific diagnostic areas to prepare for revisions in developing both DSM-5 and the International Classification of Disease, 11th Revision (ICD-11)” (APA, 2022, pg. 6).

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

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

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

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

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

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

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

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

- Diagnostic Criteria and Descriptors – Diagnostic criteria are the guidelines for making a diagnosis and should be informed by clinical judgment. When the full criteria are met, mental health professionals can add severity and course specifiers to indicate the patient’s current presentation. If the full criteria are not met, designators such as “other specified” or “unspecified” can be used. If applicable, an indication of severity (mild, moderate, severe, or extreme), descriptive features, and course (type of remission – partial or full – or recurrent) can be provided with the diagnosis. The final diagnosis is based on the clinical interview, text descriptions, criteria, and clinical judgment.
- Subtypes and Specifiers – *Subtypes* denote “mutually exclusive and jointly exhaustive phenomenological subgroupings within a diagnosis” (APA, 2022, pg. 22). For example, non-rapid eye movement (NREM) sleep arousal disorders can have either a sleepwalking or sleep terror type. Enuresis is nocturnal-only, diurnal-only, or both. *Specifiers* are not mutually exclusive or jointly exhaustive and so more than one

specifier can be given. For instance, binge eating disorder has remission and severity specifiers. Somatic symptom disorder has a specifier for severity, if with predominant pain, and/or if persistent. Again, the fundamental distinction between subtypes and specifiers is that there can be only one subtype but multiple specifiers. As the DSM-5-TR says, “Specifiers and subtypes provide an opportunity to define a more homogeneous subgrouping of individuals with the disorder who share certain features... and to convey information that is relevant to the management of the individual’s disorder” (pg. 22).

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

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

Table 3.1. DSM-5 Classification System of Mental Disorders

Disorder Category	Short Description	Module
Neurodevelopmental disorders	A group of conditions that arise in the developmental period and include intellectual disability, communication disorders, autism spectrum disorder, specific learning disorder, motor disorders, and ADHD	Not covered
Schizophrenia Spectrum	Disorders characterized by one or more of the following: delusions, hallucinations, disorganized thinking and speech, disorganized motor behavior, and negative symptoms	12
Bipolar and Related	Characterized by mania or hypomania and possibly depressed mood; includes Bipolar I and II and cyclothymic disorder	4
Depressive	Characterized by sad, empty, or irritable mood, as well as somatic and cognitive changes that affect functioning; includes major depressive, persistent depressive disorder, mood dysregulation disorder, and premenstrual dysphoric disorder	4
Anxiety	Characterized by excessive fear and anxiety and related behavioral disturbances; Includes phobias, separation anxiety, panic disorder, generalized anxiety disorder, social anxiety disorder, agoraphobia	7
Obsessive-Compulsive	Characterized by obsessions and compulsions and includes OCD, hoarding, body dysmorphic disorder, trichotillomania, and excoriation	9
Trauma- and Stressor- Related	Characterized by exposure to a traumatic or stressful event; PTSD, acute stress disorder, adjustment disorders, and prolonged grief disorder	5
Dissociative	Characterized by a disruption or discontinuity in memory, identity, emotion, perception, body representation,	6

	consciousness, motor control, or behavior; dissociative identity disorder, dissociative amnesia, and depersonalization/derealization disorder	
Somatic Symptom	Characterized by prominent somatic symptoms and/or illness anxiety associated with significant distress and impairment; includes illness anxiety disorder, somatic symptom disorder, and conversion disorder	8
Feeding and Eating	Characterized by a persistent disturbance of eating or eating-related behavior to include bingeing and purging; Includes pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia, bulimia, and binge-eating disorder	10
Elimination	Characterized by the inappropriate elimination of urine or feces; usually first diagnosed in childhood or adolescence; Includes enuresis and encopresis	Not covered
Sleep-Wake	Characterized by sleep-wake complaints about the quality, timing, and amount of sleep; includes insomnia, sleep terrors, narcolepsy, sleep apnea, hypersomnolence disorder, restless leg syndrome, and circadian-rhythm sleep-wake disorders	Not covered
Sexual Dysfunctions	Characterized by sexual difficulties and include premature or delayed ejaculation, female orgasmic disorder, and erectile disorder (to name a few)	Not covered
Gender Dysphoria	Characterized by distress associated with the incongruity between one's experienced or expressed gender and the gender assigned at birth	Not covered
Disruptive, Impulse-Control, Conduct	Characterized by problems in the self-control of emotions and behavior and involve the violation of the rights of others and cause the individual to violate societal norms; includes oppositional defiant disorder, antisocial personality disorder, kleptomania, intermittent explosive disorder, conduct disorder, and pyromania	Not covered
Substance-Related and Addictive	Characterized by the continued use of a substance despite significant problems related to its use	11
Neurocognitive	Characterized by a decline in cognitive functioning over time	14

	and the NCD has not been present since birth or early in life; Includes delirium, major and mild neurocognitive disorder, and Alzheimer’s disease	
Personality	Characterized by a pattern of stable traits which are inflexible, pervasive, and leads to distress or impairment; Includes paranoid, schizoid, borderline, obsessive- compulsive, narcissistic, histrionic, dependent, schizotypal, antisocial, and avoidant personality disorder	13
Paraphilic	Characterized by recurrent and intense sexual fantasies that can cause harm to the individual or others; includes exhibitionism, voyeurism, sexual sadism, sexual masochism, pedophilic, and fetishistic disorders	Not covered

3.2.3. The ICD-11

In 1893, the International Statistical Institute adopted the International List of Causes of Death which was the first international classification edition. The World Health Organization was entrusted with the development of the ICD in 1948 and published the 6th version (ICD-6). The ICD-11 went into effect January 1, 2022, though it was adopted in May 2019. The WHO states:

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

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

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

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

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

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

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

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

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

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

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

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

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

You should have learned the following in this section:

- Clinical diagnosis is the process of using assessment data to determine if the pattern of symptoms the person presents with is consistent with the diagnostic criteria for a specific mental disorder outlined in an established classification system such as the DSM-5-TR or ICD-11.
- Classification systems provide mental health professionals with an agreed-upon list of disorders falling into distinct categories for which there are clear descriptions and criteria for making a diagnosis.
- Elements of a diagnosis in the DSM include the diagnostic criteria and descriptors, subtypes and specifiers, the principle diagnosis, and a provisional diagnosis.

Section 3.2 Review Questions

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

3.3. Treatment of Mental Disorders – An Overview

Section Learning Objectives

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

3.3.1. Seeking Treatment

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

Journal of Consulting and Clinical Psychology, 88% of therapy-goers reported improvements after just one session.”

For more on this article, please visit:

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

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

3.3.1.3. What exactly is psychotherapy? According to the APA, in **psychotherapy** “psychologists apply scientifically validated procedures to help people develop healthier, more effective habits.” Several different approaches can be utilized to include behavior, cognitive and cognitive-behavior, humanistic-experiential, psychodynamic, couples and family, and biological treatments.

3.3.1.4. The client-therapist relationship. What is the ideal client-therapist relationship?

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

So how do you find a psychotherapist? Several strategies may prove fruitful. You could ask family and friends, your primary care physician (PCP), look online, consult an area community mental health center, your local university’s psychology department, state psychological association, or use APA’s Psychologist Locator Service (https://locator.apa.org/?_ga=2.160567293.1305482682.1516057794-1001575750.1501611950).

Once you find a list of psychologists or other practitioners, choose the right one for you by determining if you plan on attending alone or with family, what you wish to get out of your time with a psychotherapist, how much your insurance company pays for and if you have to pay out of pocket how much you can afford, when you can attend sessions, and how far you are willing to travel to see the mental health professional. Once you have done this, make your first appointment.

But what should you bring? APA suggests, “to make the most of your time, make a list of the points you want to cover in your first session and what you want to work on in psychotherapy. Be prepared to share information about what’s bringing you to the psychologist. Even a vague idea of what you want to accomplish can help you and your psychologist proceed

efficiently and effectively.” Additionally, they suggest taking report cards, a list of medications, information on the reasons for a referral, a notebook, a calendar to schedule future visits if needed, and a form of payment. What you take depends on the reason for the visit.

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

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

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

3.3.1.6. How do you know you are finished? “How long psychotherapy takes depends on several factors: the type of problem or disorder, the patient's characteristics and history, the patient's goals, what's going on in the patient's life outside psychotherapy and how fast the

patient is able to make progress.” It is important to note that psychotherapy is not a lifelong commitment, and it is a joint decision of client and therapist as to when it ends. Once over, expect to have a periodic check-up with your therapist. This might be weeks or even months after your last session. If you need to see him/her sooner, schedule an appointment. APA calls this a “mental health tune up” or a “booster session.”

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

<http://www.apa.org/helpcenter/understanding-psychotherapy.aspx>

You should have learned the following in this section:

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

Section 3.3 Review Questions

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

Module Recap

That's it. With the conclusion of Module 3, you now have the necessary foundation to understand each of the groups of disorders we discuss beginning in Module 4 and through Module 14.

In Module 3 we reviewed clinical assessment, diagnosis, and treatment. In terms of assessment, we covered key concepts such as reliability, validity, and standardization; and discussed methods of assessment such as observation, the clinical interview, psychological tests, personality inventories, neurological tests, the physical examination, behavioral assessment, and intelligence tests. In terms of diagnosis, we discussed the classification systems of the DSM-5-TR and ICD-11. For treatment, we discussed the reasons why someone may seek treatment, self-treatment, psychotherapy, the client-centered relationship, and how well psychotherapy works.

Part II. Mental Disorders – Block 1

Disorders Covered:

4. Mood Disorders
5. Trauma- and Stressor-Related Disorders
6. Dissociative Disorders

Part II. Mental Disorders – Block 1

Module 4: Mood Disorders

Module 4: Mood Disorders

Module Overview

In Module 4, we will discuss matters related to mood disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will cover major depressive disorder, persistent depressive disorder (formerly Dysthymia), bipolar I disorder, bipolar II disorder, and cyclothymic disorder. We will also cover major depressive, manic, and hypomanic episodes. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of several therapies (Module 3). Note that this module will cover two chapters from the DSM 5-TR; namely, Bipolar and Related Disorders and Depressive Disorders.

Module Outline

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

Module Learning Outcomes

- Describe how depressive disorders present.
 - Describe how bipolar and related disorders present.
 - Describe the epidemiology of mood disorders.
 - Describe comorbidity in relation to mood disorders.
 - Describe the etiology of mood disorders.
 - Describe treatment options for mood disorders.
-

4.1. Clinical Presentation – Depressive Disorders

Section Learning Objectives

- Distinguish the two distinct groups of mood disorders.
- Identify and describe the two types of depressive disorders.
- Classify symptoms of depression.
- Describe premenstrual dysphoric disorder.

4.1.1. Distinguishing Mood Disorders

Within mood disorders are two distinct groups—individuals with depressive disorders and individuals with bipolar disorders. **The key difference between the two mood disorder groups is episodes of mania/hypomania.** More specifically, in bipolar I disorder, the individual experiences a manic episode that “may have been preceded by and may be followed by hypomanic or major depressive episodes” (APA, 2022, pg. 139) whereas for bipolar II disorder,

the individual has experienced in the past or is currently experiencing a hypomanic episode and has experienced in the past or is currently experiencing a major depressive episode. In contrast, individuals presenting with a depressive disorder have never experienced a manic or hypomanic episode.

4.1.2. Types of Depressive Disorders

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

It should be noted that after a careful review of the literature, **premenstrual dysphoric disorder**, was moved from “Criteria Sets and Axes Provided for Future Study” in the DSM-IV to Section II of DSM-5 as the disorder was confirmed as a “specific and treatment-responsive form of depressive disorder that begins sometime following ovulation and remits within a few days of menses and has a marked impact on functioning” (APA, 2022, pg. 177).

The DSM-5 also added a new diagnosis, *disruptive mood dysregulation disorder* (DMDD), for children up to 12 years of age, to deal with the potential for overdiagnosis and treatment of bipolar disorder in children, both in the United States and internationally. Children with DMDD present with persistent irritability and frequent episodes of extreme behavioral dyscontrol and so develop unipolar, not bipolar, depressive disorders or anxiety disorders as they move into adolescence and adulthood.

For a discussion of DMDD, please visit our sister book, [Behavioral Disorders of Childhood](https://opentext.wsu.edu/behavioral-disorders-childhood/):

<https://opentext.wsu.edu/behavioral-disorders-childhood/>

4.1.3. Symptoms Associated with Depressive Disorders

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

4.1.3.1. Mood. While clinical depression can vary in its presentation among individuals, most, if not all individuals with depression will report significant mood disturbances such as a depressed mood for most of the day and/or feelings of *anhedonia*, which is the loss of interest in previously interesting activities.

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

4.1.3.3. Cognitive. It should not come as a surprise that there is a serious disruption in cognitions as individuals with depressive disorders typically hold a negative view of themselves and the world around them. They are quick to blame themselves when things go wrong, and

rarely take credit when they experience positive achievements. Individuals with depressive disorders often feel worthless, which creates a negative feedback loop by reinforcing their overall depressed mood. They also report difficulty concentrating on tasks, as they are easily distracted from outside stimuli. This assertion is supported by research that has found individuals with depression perform worse than those without depression on tasks of memory, attention, and reasoning (Chen et al., 2013). Finally, thoughts of suicide and self-harm do occasionally occur in those with depressive disorders (*Note - this will be discussed in more detail in Section 4.3*).

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

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

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

4.1.4. Diagnostic Criteria and Features for Depressive Disorders

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

4.1.4.2. Persistent depressive disorder (PDD). For a diagnosis of persistent depressive disorder, an individual must experience a depressed mood for most of the day, for more days than not, for *at least two years*. (APA, 2022). This feeling of a depressed mood is also accompanied by *two* or more additional symptoms, to include changes in appetite, insomnia or

hypersomnia, low energy or fatigue, low self-esteem, feelings of hopelessness, and poor concentration or difficulty with decision making. The symptoms taken together cause clinically significant distress or impairment in important areas of functioning such as social and occupational and these impacts can be as great as or greater than MDD. The individual may experience a temporary relief of symptoms; however, the individual will not be without symptoms for more than two months during this two-year period.

Making Sense of the Disorders

In relation to depressive disorders, note the following:

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

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

4.1.4.3. Premenstrual dysphoric disorder. In terms of premenstrual dysphoric disorder, the DSM-5-TR states in the majority of menstrual cycles, at least *five* symptoms must be present in the final week before the onset of menses, being improving with a few days after menses begins, and disappear or become negligible in the week postmenses. Individuals diagnosed with premenstrual dysphoric disorder must have one or more of the following: increased mood swings, irritability or anger, depressed mood, or anxiety/tension. Additionally, they must have one or more of the following to reach a total of five symptoms: anhedonia, difficulty concentrating, lethargy, changes in appetite, hypersomnia or insomnia, feelings of being overwhelmed or out of control, and/or experience breast tenderness or swelling. The symptoms lead to issues at work or school (i.e., decreased productivity and efficiency), within relationships

(i.e., discord in the intimate partner relationship or with children, friends, or other family members), and with usual social activities (i.e., avoidance of the activities).

You should have learned the following in this section:

- Mood disorder fall into one of two groups – depressive or bipolar disorders – with the key distinction between the two being episodes of mania/hypomania.
- Symptoms of depression fall into one of four categories – mood, behavioral, cognitive, and physical.
- Persistent Depressive Disorder shares symptoms with Major Depressive Disorder though they are usually not as severe and ebb and flow over a period of at least two years.
- Premenstrual dysphoric disorder presents as mood lability, irritability, dysphoria, and anxiety symptoms occurring often during the premenstrual phase of the cycle and remit around the beginning of menses or shortly thereafter.

Section 4.1 Review Questions

1. What are the different categories of mood disorder symptoms? Identify the symptoms within each category.
2. What are the key differences in a major depression and a persistent depressive disorder diagnosis?
3. What is premenstrual dysphoric disorder?

4.2. Clinical Presentation – Bipolar and Related Disorders

Section Learning Objectives

- Distinguish the forms bipolar disorder takes.
- Contrast a manic episode with a hypomanic episode.
- Define cyclothymic disorder.

4.2.1. Distinguishing Bipolar I and II Disorders

According to the DSM-5-TR (APA, 2022), there are two types of bipolar disorder—bipolar I and bipolar II. A diagnosis of bipolar I disorder is made when there is at least one manic episode. This manic episode can be preceded by and/or followed by a hypomanic or major depressive episode, however, diagnostic criteria for a manic episode is the *only* criteria that needs to be met for a bipolar I diagnosis. A diagnosis of bipolar II Disorder is made when there is a current or history of a hypomanic episode *and* a current or past major depressive episode.

Descriptions of both manic and hypomanic episodes follow below.

Making Sense of the Disorders

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

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

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

4.2.2. Manic and Hypomanic Episodes

4.2.2.1. Manic episode. The key feature of a **manic episode** is a specific period in which an individual reports abnormal, persistent, or expansive irritable mood for nearly all day, every day, for *at least one week* (APA, 2022). Additionally, the individual will display increased activity or energy during this same time. With regards to mood, an individual in a manic episode will appear excessively happy, often engaging haphazardly in sexual or interpersonal interactions. They also display rapid shifts in mood, also known as **mood lability**, ranging from happy, neutral, to irritable. At least three of the symptoms described below (four if the mood is only irritable) must be present and represent a noticeable change in the individual's typical behavior.

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

Despite the increased activity level, individuals experiencing a manic episode also require a decreased need for sleep (Criterion B2), sleeping as little as a few hours a night yet still feeling rested. Reduced need for sleep may also be a precursor to a manic episode, suggesting that a manic episode is to begin imminently. It is not uncommon for those experiencing a manic episode to be more talkative than usual. It can be difficult to follow their conversation due to the quick pace of their talking, as well as tangential storytelling. Additionally, they can be difficult to interrupt in conversation, often disregarding the reciprocal nature of communication (Criterion

B3). If the individual is more irritable than expansive, speech can become hostile and they engage in tirades, particularly if they are interrupted or not allowed to engage in an activity they are seeking out (APA, 2022).

Based on their speech pattern, it should not be a surprise that racing thoughts and flights of ideas (Criterion B4) also present during manic episodes. Because of these rapid thoughts, speech may become disorganized or incoherent. Finally, individuals experiencing a manic episode are distractable (Criterion B5).

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

Making Sense of the Disorders

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

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

4.2.3. Cyclothymic Disorder

Notably, there is a subclass of individuals who experience numerous periods with hypomanic symptoms that do not meet the criteria for a hypomanic episode and *mild* depressive symptoms (i.e., do not fully meet criteria for a major depressive episode). These individuals are diagnosed with **cyclothymic disorder** (APA, 2022). Presentation of these symptoms occur for two or more years and are typically interrupted by periods of normal mood not lasting more than two months at a time. The symptoms cause clinically significant distress or impairment in important areas of functioning, such as social and occupational. While only a small percentage of the population develops cyclothymic disorder, it can eventually progress into bipolar I or bipolar II disorder (Zeschel et al., 2015).

You should have learned the following in this section:

- An individual is diagnosed with bipolar I disorder if they have ever experienced a manic episode and are diagnosed with bipolar II disorder if the criteria has only been met for a hypomanic episode.
- A manic episode is characterized by a specific period in which an individual reports abnormal, persistent, or expansive irritable mood for nearly all day, every day, for at least one week.
- A hypomanic episode is characterized by abnormally and persistently elevated, expansive, or irritable mood and energy levels, though not as extreme as in mania, and must be present for at least four days. It is also not severe enough to cause impairments or hospitalization.
- Cyclothymic disorder includes periods of hypomanic and mild depressive symptoms without meeting the criteria for a depressive episode which lasts two or more years and is interrupted by periods of normal moods.

Section 4.2 Review Questions

1. What is the difference between bipolar I and II disorder?
2. What are the key diagnostic differences between a hypomanic and manic episode?
3. What is cyclothymic disorder?

4.3. Epidemiology

Section Learning Objectives

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

4.3.1. Depressive Disorders

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

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

4.3.2. Bipolar Disorders

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

4.3.3. Suicidality

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

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

You should have learned the following in this section:

- Major depressive disorder is experienced by about 7% of the population in the United States, afflicting young adults and women the most.
- Bipolar I disorder afflicts 1.5% and bipolar II disorder afflicts 0.8% of the U.S. population with bipolar II affecting women more than men and no gender difference being apparent for bipolar I.
- Individuals with a depressive disorder have a 17-fold increased risk for suicide while the lifetime risk of suicide for an individual with a bipolar disorder is estimated to be 20- to 30- fold greater than in the general population and 5-6% of individuals with bipolar disorder die by suicide.

Section 4.3 Review Questions

1. What are the prevalence rates of the mood disorders?
2. What gender differences exist in the rate of occurrence of mood disorders?
3. How do depressive and bipolar disorders compare in terms of suicidality (attempts and lethality)?

4.4. Comorbidity

Section Learning Objectives

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

4.4.1. Depressive Disorders

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

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

4.4.2. Bipolar Disorders

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

Bipolar II disorder is more often than not associated with one or more comorbid mental disorders, with anxiety disorders being the most common (38% with social anxiety, 36% with specific phobia, and 30% having generalized anxiety). As with bipolar I, substance use disorders are common with alcohol use (42%) leading the way, followed by cannabis use (20%). Premenstrual syndrome and premenstrual dysphoric disorder are common in women with bipolar II disorder especially (APA, 2022).

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

You should have learned the following in this section:

- Depressive disorders have a high comorbidity with substance use disorders, anxiety disorders, and some personality disorders.
- Bipolar disorders have a high comorbidity with anxiety disorders and substance abuse disorders while cyclothymic disorder is comorbid with substance-related disorders and sleep disorders.

Section 4.4 Review Questions

1. What are common comorbidities for the depressive disorders?
2. What are common comorbidities for bipolar disorders?

4.5. Etiology

Section Learning Objectives

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

4.5.1. Biological

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

4.5.1.1. Genetics. Like with any disorder, researchers often explore the prevalence rate of depressive disorders among family members to determine if there is some genetic component, whether it be a direct link or a predisposition. If there is a genetic predisposition to developing depressive disorders, one would expect a higher rate of depression within families than that of the general population. Research supports this with regards to depressive disorders as there is nearly a 30% increase in relatives diagnosed with depression compared to 10% of the general population (Levinson & Nichols, 2014). Similarly, there is an elevated prevalence among first-degree relatives for both Bipolar I and Bipolar II disorders as well.

Another way to study the genetic component of a disorder is via twin studies. One would expect identical twins to have a higher rate of the disorder as opposed to fraternal twins, as

identical twins share the same genetic make-up, whereas fraternal twins only share roughly 50%, similar to that of siblings. A large-scale study found that if one identical twin was diagnosed with depression, there was a 46% chance their identical twin was diagnosed with depression. In contrast, the rate of a depression diagnosis in fraternal twins was only 20%. Despite the fraternal twin rate still being higher than that of a first-degree relative, this study provided enough evidence that there is a strong genetic link in the development of depression (McGuffin et al., 1996).

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

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

4.5.1.2. Biochemical. As you will read in the treatment section, there is strong evidence of a biochemical deficit in depression and bipolar disorders. More specifically, low activity levels of norepinephrine and serotonin, have long been documented as contributing factors to

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

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

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

4.5.1.3. Endocrine system. As you may know, the endocrine system is a collection of glands responsible for regulating hormones, metabolism, growth and development, sleep, and mood, among other things. Some research has implicated hormones, particularly **cortisol**, a hormone released as a stress response, in the development of depression (Owens et al., 2014).

Additionally, **melatonin**, a hormone released when it is dark outside to assist with the transition to sleep, may also be related to depressive symptoms, particularly during the winter months.

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

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

4.5.2. Cognitive

The cognitive model, arguably the most conclusive model with regards to depressive disorders, focuses on the negative thoughts and perceptions of an individual. One theory often

equated with the cognitive model of depression is **learned helplessness**. Coined by Martin Seligman (1975), learned helplessness was developed based on his laboratory experiment involving dogs. In this study, Seligman restrained dogs in an apparatus and routinely shocked them regardless of their behavior. The following day, the dogs were placed in a similar apparatus; however, this time they were not restrained and there was a small barrier placed between the “shock” floor and the “safe” floor. What Seligman observed was that despite the opportunity to escape the shock, the dogs flurried for a bit, and then ultimately laid down and whimpered while being shocked.

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

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

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

Maladaptive attitudes, or negative attitudes about oneself, others, and the world around them are often present in those with depressive symptoms. These attitudes are inaccurate and often global. For example, “If I fail my exam, the world will know I’m stupid.” Will the entire world *really* know you failed your exam? Not likely. Because you fail the exam, are you stupid? No. Individuals with depressive symptoms often develop these maladaptive attitudes regarding everything in their life, indirectly isolating themselves from others. The **cognitive triad** also plays into the maladaptive attitudes in that the individual interprets these negative thoughts about their *experiences, themselves, and their futures*.

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

Finally, **automatic thoughts**, or the constant stream of negative thoughts, also leads to symptoms of depression as individuals begin to feel as though they are inadequate or helpless in

a given situation. While some cognitions are manipulated and interpreted negatively, Beck stated that there is another set of negative thoughts that occur automatically. Research studies have continually supported Beck's maladaptive thoughts, attitudes, and errors in thinking as fundamental issues in those with depressive disorders (Lai et al., 2014; Possel & Black, 2014). Furthermore, as you will see in the treatment section (Section 4.5), cognitive strategies are among the most effective forms of treatment for depressive disorders.

4.5.3. Behavioral

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

4.5.4. Sociocultural

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

4.5.4.1. Family-social perspective. Similar to that of the behavioral theory, the family-social perspective of depression suggests that depression is related to the unavailability of social support. This is often supported by research studies that show separated and divorced individuals are three times more likely to experience depressive symptoms than those that are married or even widowed (Schultz, 2007). While many factors lead a couple to separate or end their marriage, some relationships end due to a spouse's mental health issues, particularly depressive symptoms. Depressive symptoms have been positively related to increased interpersonal conflicts, reduced communication, and intimacy issues, all of which are often reported in causal factors leading to a divorce (Najman et al., 2014).

The family-social perspective can also be viewed oppositely, with stress and marital discord leading to increased rates of depression in one or both spouses (Nezlek et al., 2000). While some research indicates that having children provides a positive influence in one's life, it can also lead to stress both within the individual, as well as between partners due to division of work and discipline differences. Studies have shown that women who had three or more young children, and also lacked a close confidante and outside employment, were more likely than other mothers to become depressed (Brown, 2002).

4.5.4.2. Multi-cultural perspective. While depression is experienced across the entire world, one's cultural background may influence *what* symptoms of depression are presented. Common depressive symptoms such as feeling sad, lack of energy, anhedonia, difficulty concentrating, and thoughts of suicide are a hallmark in most societies; other symptoms may be more specific to one's nationality. More specifically, individuals from non-Western countries (China and other Asian countries) often focus on the physical symptoms of depression—tiredness, weakness, sleep issues—and less of an emphasis on the cognitive symptoms.

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

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

The first theory, *artifact theory*, suggests that the difference between genders is due to clinician or diagnostic systems being more sensitive to diagnosing women with depression than men. While women are often thought to be more “emotional,” easily expressing their feelings and more willing to discuss their symptoms with clinicians and physicians, men often withhold their symptoms or will present with more traditionally “masculine” symptoms of anger or aggression. While this theory is a possible explanation for the gender differences in the rate of depression, research has failed to support this theory, suggesting that men and women are

equally likely to seek out treatment and discuss their depressive symptoms (McSweeney, 2004; Rieker & Bird, 2005).

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

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

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

The final theory, *ruminant theory*, suggests that women are more likely than men to ruminate, or intently focus, on their depressive symptoms, thus making them more vulnerable to developing depression at a clinical level (Nolen-Hoeksema, 2012). Several studies have

supported this theory and shown that rumination of negative thoughts is positively related to an increase in depression symptoms (Hankin, 2009).

While many theories try to explain the gender discrepancy in depressive episodes, no single theory has produced enough evidence to fully explain why women experience depression more than men. Due to the lack of evidence, gender differences in depression remains one of the most researched topics within the subject of depression, while simultaneously being the least understood phenomena within clinical psychology.

You should have learned the following in this section:

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

Section 4.5 Review Questions

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

4.6. Treatment of Mood Disorders

Section Learning Objectives

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

4.6.1. Depressive Disorders

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

4.6.1.1. Psychopharmacology - Antidepressant medications. Antidepressants are often the most common first-line attempt at treatment for MDD for a few reasons. Oftentimes an individual will present with symptoms to their primary caregiver (a medical doctor) who will prescribe them some line of antidepressant medication. Medication is often seen as an “easier” treatment for depression as the individual can take the medication at their home, rather than

attending weekly therapy sessions; however, this also leaves room for adherence issues as a large percentage of individuals fail to take prescription medication as indicated by their physician.

Given the biological functions of neurotransmitters and their involvement in maintaining depressive symptoms, it makes sense that this is an effective type of treatment.

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

4.6.1.2. Psychopharmacology - Selective serotonin reuptake inhibitors (SSRIs).

SSRIs are among the most common medications used to treat depression due to their relatively benign side effects. Additionally, the required dose to reach therapeutic levels is low compared to the other medication options. Possible side effects from SSRIs include but are not limited to nausea, insomnia, and reduced sex drive.

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

4.6.1.3. Psychopharmacology - Tricyclic antidepressants. Although originally developed to treat schizophrenia, tricyclic antidepressants were adapted to treat depression after

failing to manage symptoms of schizophrenia (Kuhn, 1958). The term tricyclic came from the molecular shape of the structure: three rings.

Tricyclic antidepressants are like SSRIs in that they work by affecting brain chemistry, altering the number of neurotransmitters available for neurons. More specifically, they block the absorption or reuptake of serotonin and norepinephrine, thus increasing their availability for postsynaptic neurons. While effective, tricyclic antidepressants have been increasingly replaced by SSRIs due to their reduced side effects. However, tricyclic antidepressants have been shown to be more effective in treating depressive symptoms in individuals who have not been able to achieve symptom reduction via other pharmacological approaches.

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

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

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

While these drugs are effective, they come with serious side effects. In addition to the hypertensive episodes, they can also cause nausea, headaches, drowsiness, involuntary muscle jerks, reduced sexual desire, weight gain, etc. (APA, 2010). Despite these side effects, studies have shown that individuals prescribed MAOIs for depression have a treatment response rate of 50-70% (Krishnan, 2007). Overall, despite their effectiveness, MAOIs are likely the best treatment for late-stage, treatment-resistant depression patients who have exhausted other treatment options (Krishnan, 2007).

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

4.6.1.5. Psychotherapy - Cognitive behavioral therapy (CBT). CBT was founded by Aaron Beck in the 1960s and is a widely practiced therapeutic tool used to treat depression (and other disorders as well). The basics of CBT involve what Beck called the **cognitive triangle**—cognitions (thoughts), behaviors, and emotions. Beck believed that these three components are interconnected, and therefore, affect one another. It is believed that CBT can improve emotions in depressed patients by changing both cognitions (thoughts) and behaviors, which in return enhances mood. Common cognitive interventions with CBT include thought monitoring and recording, identifying cognitive errors, examining evidence supporting/negating cognitions, and

creating rational alternatives to maladaptive thought patterns. Behavioral interventions of CBT include activity planning, pleasant event scheduling, task assignments, and coping-skills training.

CBT generally follows four phases of treatment:

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

4.6.1.6. Psychotherapy - Behavioral activation (BA). BA is similar to the behavioral component of CBT in that the goal of treatment is to alleviate depression and prevent future relapse by changing an individual's behavior. Founded by Ferster (1973), as well as Lewinsohn

and colleagues (Lewinsohn, 1974; Lewinsohn, Biglan, & Zeiss, 1976), the goal of BA is to increase the frequency of behaviors so that individuals have opportunities to experience greater contact with sources of reward in their lives. To do this, the clinician assists the patient by developing a list of pleasurable activities that they can engage in outside of treatment (i.e., going for a walk, going shopping, having dinner with a friend). Additionally, the clinician assists the patient in identifying their negative behaviors—crying, sleeping in, avoiding friends—and monitoring them so that they do not impact the outcome of their pleasurable activities. Finally, the clinician works with the patient on effective social skills. By minimizing negative behaviors and maximizing pleasurable activities, the individual will receive more positive reward and reinforcement from others and their environment, thus improving their overall mood.

4.6.1.7. Psychotherapy - Interpersonal therapy (IPT). IPT was developed by Klerman, Weissman, and colleagues in the 1970s as a treatment arm for a pharmacotherapy study of depression (Weissman, 1995). The treatment was created based on data from post-World War II individuals who expressed a substantial impact on their psychosocial life events. Klerman and colleagues noticed a significant relationship between the development of depression and complicated bereavement, role disputes, role transitions, and interpersonal deficits in these individuals (Weissman, 1995). The idea behind IPT is that depressive episodes compromise interpersonal functioning, which makes it difficult to manage stressful life events. The basic mechanism of IPT is to establish effective strategies to manage interpersonal issues, which in return, will ameliorate depressive symptoms.

There are two main principles of IPT. First, depression is a common medical illness with a complex and multi-determined etiology. Since depression is a medical illness, it is also treatable and *not* the patient's fault. Second, depression is connected to a current or recent life

event. The goal of IPT is to identify the interpersonal problem that is related to the depressive symptoms and solve this crisis so the patient can improve their life situation while relieving depressive symptoms.

4.6.1.8. Multimodal treatment. While both pharmacological and psychological treatment alone is very effective in treating depression, a combination of the two treatments may offer additional benefits, particularly in the maintenance of wellness. Additionally, multimodal treatment options may be helpful for individuals who have not achieved wellness in a single modality.

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

4.6.2. Bipolar Disorder

4.6.2.1. Psychopharmacology. Unlike treatment for MDD, there is some controversy regarding effective treatment of bipolar disorder. One suggestion is to treat bipolar disorder aggressively with mood stabilizers such as Lithium or Depakote as these medications do not induce pharmacological mania/hypomania. These mood stabilizers are occasionally combined with antidepressants later in treatment *only* if absolutely necessary (Ghaemi, Hsu, Soldani & Goodwin, 2003). Research has shown that mood stabilizers are less potent in treating depressive

symptoms, and therefore, the combination approach is believed to help manage both the manic and depressive episodes (Nivoli et al., 2011).

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

4.6.2.2. Psychological treatment. Although psychopharmacology is the first and most widely used treatment for bipolar disorders, occasionally psychological interventions are also paired with medication as psychotherapy alone is not a sufficient treatment option. Majority of psychological interventions are aimed at medication adherence, as many bipolar patients stop taking their mood stabilizers when they “feel better” (Advokat et al., 2014). Social skills training and problem-solving skills are also helpful techniques to address in the therapeutic setting as individuals with bipolar disorder often struggle in this area.

4.6.3. Outcome of Treatment

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

Rates of relapse for major depressive disorder are often associated with individuals whose onset was at a younger age (particularly adolescents), those who have already experienced multiple major depressive episodes, and those with more severe symptomology, especially those presenting with severe suicidal ideation and psychotic features (APA, 2022).

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

You should have learned the following in this section:

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

Section 4.6 Review Questions

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

Module Recap

That concludes our discussion of mood disorders. You should now have a good understanding of the two major types of mood disorders – depressive and bipolar disorders. Be sure you are clear on what makes them different from one another in terms of their clinical presentation, epidemiology, comorbidity, and etiology. This will help you with understanding treatment options and their efficacy.

Part II. Mental Disorders – Block 1

Module 5: Trauma- and Stressor-Related Disorders

Module 5: Trauma- and Stressor-Related Disorders

Module Overview

In Module 5, we will discuss matters related to trauma- and stressor-related disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will consist of PTSD, acute stress disorder, adjustment disorder, and prolonged grief disorder. Prior to discussing these clinical disorders, we will explain what stressors are, as well as identify common stressors that may lead to a trauma- or stressor-related disorder. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of models to explain psychopathology (Module 2), and descriptions of various therapies (Module 3).

Module Outline

- 5.1. Stressors
- 5.2. Clinical Presentation
- 5.3. Epidemiology
- 5.4. Comorbidity
- 5.5. Etiology
- 5.6. Treatment

Module Learning Outcomes

- Define and identify common stressors.
- Describe how trauma- and stressor-related disorders present.
- Describe the epidemiology of trauma- and stressor-related disorders.

- Describe comorbidity in relation to trauma- and stressor-related disorders.
- Describe the etiology of trauma- and stressor-related disorders.
- Describe treatment options for trauma- and stressor-related disorders.

5.1. Stressors

Section Learning Objectives

- Define stressor.
- Identify and describe common stressors.

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

Among the most studied triggers for trauma-related disorders are combat and physical/sexual assault. Symptoms of combat-related trauma date back to World War I when soldiers would return home with “shell shock” (Figley, 1978). Unfortunately, it was not until

after the Vietnam War that significant progress was made in both identifying and treating war-related psychological difficulties (Roy-Byrne et al., 2004). With the more recent wars in Iraq and Afghanistan, attention was again focused on posttraumatic stress disorder (PTSD) symptoms due to the large number of service members returning from deployments and reporting significant trauma symptoms.

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

Now that we have discussed a little about some of the most commonly studied traumatic events, we will now examine the clinical presentation of posttraumatic stress disorder, acute stress disorder, adjustment disorder, and prolonged grief disorder.

You should have learned the following in this section:

- A stressor is any event that increases physical or psychological demands on an individual.
- It does not have to be personally experienced but can be witnessed or occur to a close family member or friend to have the same effect.
- Only a small percentage of people experience significant maladjustment due to these events.
- The most studied triggers for trauma-related disorders include physical/sexual assault and combat.

Section 5.1 Review Questions

1. Given an example of a stressor you have experienced in your own life.
2. Why are the triggers of physical/sexual assault and combat more likely to lead to a trauma-related disorder?

5.2. Clinical Presentation

Section Learning Objectives

- Describe how PTSD presents.
- Describe how acute stress disorder presents.
- Describe how adjustment disorder presents.
- Describe how prolonged grief disorder presents.

5.2.1. Posttraumatic Stress Disorder

Posttraumatic stress disorder, or more commonly known as PTSD, is identified by the development of physiological, psychological, and emotional symptoms following exposure to a traumatic event. Individuals must have been exposed to a situation where actual or threatened death, sexual violence, or serious injury occurred. Examples of these situations include but are not limited to witnessing a traumatic event as it occurred to someone else; learning about a traumatic event that occurred to a family member or close friend; directly experiencing a traumatic event; or being exposed to repeated events where one experiences an aversive event (e.g., victims of child abuse/neglect, ER physicians in trauma centers, etc.).

It is important to understand that while the presentation of these symptoms varies among individuals, to meet the criteria for a diagnosis of PTSD, individuals need to report symptoms among the four different categories of symptoms.

5.2.1.1. Category 1: Recurrent experiences. The first category involves *recurrent experiences* of the traumatic event, which can occur via dissociative reactions such as flashbacks; recurrent, involuntary, and intrusive distressing memories; or even recurrent distressing dreams

(APA, 2022, pgs. 301-2). These recurrent experiences must be specific to the traumatic event or the moments immediately following to meet the criteria for PTSD. Regardless of the method, the recurrent experiences can last several seconds or extend for several days. They are often initiated by physical sensations similar to those experienced during the traumatic events or environmental triggers such as a specific location. Because of these triggers, individuals with PTSD are known to avoid stimuli (i.e., activities, objects, people, etc.) associated with the traumatic event. One or more of the intrusion symptoms must be present.

5.2.1.2. Category 2: Avoidance of stimuli. The second category involves *avoidance* of *stimuli* related to the traumatic event and either one or both of the following must be present. First, individuals with PTSD may be observed trying to avoid the distressing thoughts, memories, and/or feelings related to the memories of the traumatic event. Second, they may prevent these memories from occurring by avoiding physical stimuli such as locations, individuals, activities, or even specific situations that trigger the memory of the traumatic event.

5.2.1.3. Category 3: Negative alterations in cognition or mood. The third category experienced by individuals with PTSD is *negative alterations in cognition or mood* and at least two of the symptoms described below must be present. This is often reported as difficulty remembering an important aspect of the traumatic event. It should be noted that this amnesia is not due to a head injury, loss of consciousness, or substances, but rather, due to the traumatic nature of the event. The impaired memory may also lead individuals to have false beliefs about the causes of the traumatic event, often blaming themselves or others. An overall persistent negative state, including a generalized negative belief about oneself or others is also reported by those with PTSD. Similar to those with depression, individuals with PTSD may report a reduced

interest in participating in previously enjoyable activities, as well as the desire to engage with others socially. They also report not being able to experience positive emotions.

5.2.1.4. Category 4: Alterations in arousal and reactivity. The fourth and final category is *alterations in arousal and reactivity* and at least two of the symptoms described below must be present. Because of the negative mood and increased irritability, individuals with PTSD may be quick-tempered and act out aggressively, both verbally and physically. While these aggressive responses may be provoked, they are also sometimes unprovoked. It is believed these behaviors occur due to the heightened sensitivity to potential threats, especially if the threat is similar to their traumatic event. More specifically, individuals with PTSD have a heightened startle response and easily jump or respond to unexpected noises just as a telephone ringing or a car backfiring. They also experience significant sleep disturbances, with difficulty falling asleep, as well as staying asleep due to nightmares; engage in reckless or self-destructive behavior, and have problems concentrating.

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

5.2.2. Acute Stress Disorder

Acute stress disorder is very similar to PTSD except for the fact that symptoms must be present from **3 days to 1 month** following exposure to one or more traumatic events. If the symptoms are present after one month, the individual would then meet the criteria for PTSD.

Additionally, if symptoms present immediately following the traumatic event but resolve by day 3, an individual would not meet the criteria for acute stress disorder.

Symptoms of acute stress disorder follow that of PTSD with a few exceptions. PTSD requires symptoms within each of the four categories discussed above; however, acute stress disorder requires that the individual experience nine symptoms across five different categories (intrusion symptoms, negative mood, dissociative symptoms, avoidance symptoms, and arousal symptoms; note that in total, there are 14 symptoms across these five categories). For example, an individual may experience several arousal and reactivity symptoms such as sleep issues, concentration issues, and hypervigilance, but does not experience issues regarding negative mood. Regardless of the category of the symptoms, so long as nine symptoms are present and the symptoms cause significant distress or impairment in social, occupational, and other functioning, an individual will meet the criteria for acute stress disorder.

Making Sense of the Disorders

In relation to trauma- and stressor-related disorders, note the following:

Diagnosis PTSD if symptoms have been experienced for at least one month

Diagnosis acute stress disorder ... if symptoms have been experienced for 3 days to one month

5.2.3. Adjustment Disorder

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

Unlike PTSD and acute stress disorder, adjustment disorder does not have a set of specific symptoms an individual must meet for diagnosis. Rather, whatever symptoms the individual is experiencing must be related to the stressor and must be significant enough to impair social, occupational, or other important areas of functioning and causes marked distress “...that is out of proportion to the severity or intensity of the stressor” (APA, 2022, pg. 319).

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

5.2.4. Prolonged Grief Disorder

The DSM-5 included a *condition for further study* called persistent complex bereavement disorder. In 2018, a proposal was submitted to include this category in the main text of the manual and after careful review of the literature and approval of the criteria, it was accepted in the second half of 2019 and added as a new diagnostic entity called prolonged grief disorder.

Prolonged grief disorder is defined as an intense yearning/longing and/or preoccupation with thoughts or memories of the deceased who died at least 12 months ago. The individual will present with at least three symptoms to include feeling as though part of oneself has died, disbelief about the death, emotional numbness, feeling that life is meaningless, intense loneliness, problems engaging with friends or pursuing interests, intense emotional pain, and avoiding reminders that the person has died.

Individuals with prolonged grief disorder often hold maladaptive cognitions about the self, feel guilt about the death, and hold negative views about life goals and expectancy. Harmful health behaviors due to decreased self-care and concern are also reported. They may also experience hallucinations about the deceased, feel bitter and angry, be restless, blame others for the death, and see a reduction in the quantity and quality of sleep (APA, 2022).

You should have learned the following in this section:

- In terms of stress disorders, symptoms lasting over 3 days but not exceeding one month, would be classified as acute stress disorder while those lasting over a month are typical of PTSD.
- If symptoms begin after a traumatic event but resolve themselves within three days, the individual does not meet the criteria for a stress disorder.
- Symptoms of PTSD fall into four different categories for which an individual must have at least one symptom in each category to receive a diagnosis. These categories include recurrent experiences, avoidance of stimuli, negative alterations in cognition or mood, and alterations in arousal and reactivity.
- To receive a diagnosis of acute stress disorder an individual must experience nine symptoms across five different categories (intrusion symptoms, negative mood, dissociative symptoms, avoidance symptoms, and arousal symptoms).
- Adjustment disorder is the least intense of the three disorders and does not have a specific set of symptoms of which an individual has to have some number. Whatever symptoms the person presents with, they must cause significant impairment in areas of functioning such as social or occupational, and several modifiers are associated with the disorder.
- Prolonged grief disorder is a new diagnostic entity in the DSM-5-TR and is defined as an intense yearning/longing and/or preoccupation with thoughts or memories of the deceased who died at least 12 months ago.

Section 5.2 Review Questions

1. What is the difference in diagnostic criteria for PTSD, Acute Stress Disorder, and Adjustment Disorder?
2. What are the four categories of symptoms for PTSD? How do these symptoms present in Acute Stress Disorder and Adjustment Disorder?
3. What is prolonged grief disorder?

5.3. Epidemiology

Section Learning Objectives

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

5.3.1. PTSD

The national lifetime prevalence rate for PTSD using DSM-IV criteria is 6.8% for U.S. adults and 5.0% to 8.1% for U.S. adolescents. There are currently no definitive, comprehensive population-based data using DSM-5 though studies are beginning to emerge (APA, 2022). It should not come as a surprise that the rates of PTSD are higher among veterans and others who work in fields with high traumatic experiences (i.e., firefighters, police, EMTs, emergency room providers). In fact, PTSD rates for combat veterans are estimated to be as high as 30% (NcNally, 2012). Between one-third and one-half of all PTSD cases consist of rape survivors, military combat and captivity, and ethnically or politically motivated genocide (APA, 2022).

Concerning gender, PTSD is more prevalent among females (8% to 11%) than males (4.1% to 5.4%), likely due to their higher occurrence of exposure to traumatic experiences such as childhood sexual abuse, rape, domestic abuse, and other forms of interpersonal violence. Women also experience PTSD for a longer duration. (APA, 2022). Gender differences are not found in populations where both males and females are exposed to significant stressors suggesting that both genders are equally predisposed to developing PTSD. Prevalence rates vary

slightly across cultural groups, which may reflect differences in exposure to traumatic events. More specifically, prevalence rates of PTSD are highest for African Americans, followed by Latinx Americans and European Americans, and lowest for Asian Americans (Hinton & Lewis-Fernandez, 2011). According to the DSM-5-TR, there are higher rates of PTSD among Latinx, African-Americans, and American Indians compared to whites, and likely due to exposure to past adversity and racism and discrimination (APA, 2022).

5.3.2. Acute Stress Disorder

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

As with PTSD, acute stress disorder is more common in females than males; however, unlike PTSD, there may be some neurobiological differences in the stress response, gender differences in the emotional and cognitive processing of trauma, and sociocultural factors that contribute to females developing acute stress disorder more often than males (APA, 2022). With that said, the increased exposure to traumatic events among females may also be a strong reason why women are more likely to develop acute stress disorder.

5.3.3. Adjustment Disorder

Adjustment disorders are relatively common as they describe individuals who are having difficulty adjusting to life after a significant stressor. In psychiatric hospitals in the U.S., Australia, Canada, and Israel, adjustment disorders accounted for roughly 50% of the admissions in the 1990s. It is estimated that anywhere from 5-20% of individuals in outpatient mental health treatment facilities have an adjustment disorder as their principal diagnosis. Adjustment disorder has been found to be higher in women than men (APA, 2022).

5.3.4. Prolonged Grief Disorder

As this is a new disorder, the prevalence of DSM-5 prolonged grief disorder is currently unknown. Using a different definition of the disorder a meta-analysis of studies across four continents suggests a pooled prevalence of 9.8%. It should be noted that these studies could only be loosely compared with one another making the reported prevalence rate questionable.

You should have learned the following in this section:

- Regarding PTSD, rates are highest among people who are likely to be exposed to high traumatic events, women, and minorities.
- As for acute stress disorder, prevalence rates are hard to determine since patients must seek medical treatment within 30 days, but females are more likely to develop the disorder.
- Adjustment disorders are relatively common since they occur in individuals having trouble adjusting to a significant stressor, though women tend to receive a diagnosis more than men.

Section 5.3 Review Questions

1. Compare and contrast the prevalence rates among the trauma and stress-related disorders.
2. What do we know about the prevalence rate for prolonged grief disorder and why?

5.4. Comorbidity

Section Learning Objectives

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

5.4.1. PTSD

Given the traumatic nature of the disorder, it should not be surprising that there is a high comorbidity rate between PTSD and other psychological disorders. Individuals with PTSD are more likely than those without PTSD to report clinically significant levels of depressive, bipolar, anxiety, or substance abuse-related symptoms (APA, 2022). There is also a strong relationship between PTSD and major neurocognitive disorders, which may be due to the overlapping symptoms between these disorders (Neurocognitive Disorders will be covered in Module 14).

5.4.2. Acute Stress Disorder

Because 30 days after the traumatic event, acute stress disorder becomes PTSD (or the symptoms remit), the comorbidity of acute stress disorder with other psychological disorders has not been studied. While acute stress disorder and PTSD cannot be comorbid disorders, several studies have explored the relationship between the disorders to identify individuals most at risk for developing PTSD. The literature indicates roughly 80% of motor vehicle accident survivors, as well as assault victims, who met the criteria for acute stress disorder went on to develop PTSD

(Brewin, Andrews, Rose, & Kirk, 1999; Bryant & Harvey, 1998; Harvey & Bryant, 1998). While some researchers indicated acute stress disorder is a good predictor of PTSD, others argue further research between the two and confounding variables should be explored to establish more consistent findings.

5.4.3. Adjustment Disorder

Unlike most of the disorders we have reviewed thus far, adjustment disorders have a high comorbidity rate with various other medical conditions (APA, 2022). Often following a critical or terminal medical diagnosis, an individual will meet the criteria for adjustment disorder as they process the news about their health and the impact their new medical diagnosis will have on their life. Other psychological disorders are also diagnosed with adjustment disorder; however, symptoms of adjustment disorder must be met independently of the other psychological condition. For example, an individual with adjustment disorder with depressive mood must not meet the criteria for a major depressive episode; otherwise, the diagnosis of MDD should be made over adjustment disorder. As the DSM-5-TR says, “adjustment disorders are common accompaniments of medical illness and may be the major psychological response to a medical condition” (APA, 2022).

5.4.4. Prolonged Grief Disorder

Prolonged grief disorder is commonly comorbid with MDD, PTSD if the death occurred in violent or accidental circumstances, substance use disorders, and separation anxiety disorder.

You should have learned the following in this section:

- PTSD has a high comorbidity rate with psychological and neurocognitive disorders while this rate is hard to establish with acute stress disorder since it becomes PTSD after 30 days.
- Adjustment disorder has a high comorbidity rate with other medical conditions as people process news about their health and what the impact of a new medical diagnosis will be on their life.
- Prolonged grief disorder has a high comorbidity with PTSD, MDD, separation anxiety disorder, and substance use disorders.

Section 5.4 Review Questions

1. What are the most common comorbidities among trauma and stress-related disorders?
2. Why is it hard to establish comorbidities for acute stress disorder?

5.5. Etiology

Section Learning Objectives

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

5.5.1. Biological

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

Researchers have studied the amygdala and HPA axis in individuals with PTSD, and have identified heightened amygdala reactivity in stressful situations, as well as excessive responsiveness to stimuli that is related to one’s specific traumatic event (Sherin & Nemeroff,

2011). Additionally, studies have indicated that individuals with PTSD also show a diminished fear extinction, suggesting an overall higher level of stress during non-stressful times. These findings may explain why individuals with PTSD experience an increased startle response and exaggerated sensitivity to stimuli associated with their trauma (Schmidt, Kaltwasser, & Wotjak, 2013).

5.5.2. Cognitive

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

5.5.3. Social

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

5.5.4. Sociocultural

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

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

You should have learned the following in this section:

- In terms of causes for trauma- and stressor-related disorders, an over-involvement of the hypothalamic-pituitary-adrenal (HPA) axis has been cited as a biological cause, with rumination and negative coping styles or maladjusted thoughts emerging as cognitive causes.
- Culture may lead to different interpretations of traumatic events thus causing higher rates among Hispanic Americans.
- Social and family support have been found to be protective factors for individuals most likely to develop PTSD.

Section 5.5 Review Questions

1. Discuss the four etiological models of the trauma- and stressor-related disorders. Which model best explains the maintenance of trauma/stress symptoms? Which identifies protective factors for the individual?

5.6. Treatment

Section Learning Objectives

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

5.6.1. Psychological Debriefing

One way to negate the potential development of PTSD symptoms is thorough **psychological debriefing**. Psychological debriefing is considered a type of crisis intervention that requires individuals who have recently experienced a traumatic event to discuss or process their thoughts and feelings related to the traumatic event, typically within 72 hours of the event (Kinchin, 2007). While there are a few different methods to a psychological debriefing, they all follow the same general format:

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

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

5.6.2. Exposure Therapy

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

In imaginal exposure, the individual mentally re-creates specific details of the traumatic event. The patient is then asked to repeatedly discuss the event in increasing detail, providing more information regarding their thoughts and feelings at each step of the event. During in vivo exposure, the individual is reminded of the traumatic event through the use of videos, images, or

other tangible objects related to the traumatic event that induces a heightened arousal response. While the patient is re-experiencing cognitions, emotions, and physiological symptoms related to the traumatic experience, they are encouraged to utilize positive coping strategies, such as relaxation techniques, to reduce their overall level of anxiety.

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

5.6.3. Cognitive Behavioral Therapy (CBT)

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

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

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

5.6.4. Eye Movement Desensitization and Reprocessing (EMDR)

In the late 1980s, psychologist Francine Shapiro found that by focusing her eyes on the waving leaves during her daily walk, her troubling thoughts resolved on their own. From this observation, she concluded that lateral eye movements facilitate the cognitive processing of traumatic thoughts (Shapiro, 1989).

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

1. **Patient History and Treatment Planning** - Identify trauma symptoms and potential barriers to treatment.
2. **Preparation** - Psychoeducation of trauma and treatment.
3. **Assessment** - Careful and detailed evaluation of the traumatic event. Patient identifies images, cognitions, and emotions related to the traumatic event, as well as trauma-related physiological symptoms.
4. **Desensitization and Reprocessing** - Holding the trauma image, cognition, and emotion in mind, while simultaneously assessing their physiological symptoms, the patient must track the clinician's finger movement for approximately 20 seconds. At this time, the patient must "blank it out" and let go of the memory.
5. **Installation of Positive Cognitions** - Once the negative image, cognition, and emotions are reduced, the patient must hold onto a positive image or thought while again tracking the clinician's finger movement for approximately 20 seconds.
6. **Body Scan** - Patient must identify any lingering bodily sensations while again tracking the clinician's fingers for a third time to discard any remaining trauma symptoms.

7. Closure - Patient is provided with positive coping strategies and relaxation techniques to assist with any recurrent cognitions or emotions related to the traumatic experience.
8. Reevaluation - Clinician assesses if treatment goals were met. If not, schedules another treatment session and identifies remaining symptoms.

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

The National Institute for Health and Care Excellence (NICE) says to consider EMDR for adults with a diagnosis of PTSD and who presented between 1 and 3 months after a non-combat related trauma if the person shows a preference for EMDR and to offer it to adults with a diagnosis of PTSD who have presented more than three months after a non-combat related trauma. They state that EMDR for adults should (cited directly from their website):

- be based on a validated manual
- typically be provided over 8 to 12 sessions, but more if clinically indicated, for example if they have experienced multiple traumas
- be delivered by trained practitioners with ongoing supervision
- be delivered in a phased manner and include psychoeducation about reactions to trauma; managing distressing memories and situations; identifying and treating target memories (often visual images); and promoting alternative positive beliefs about the self
- use repeated in-session bilateral stimulation (normally with eye movements but use other methods, including taps and tones, if preferred or more appropriate, such as for people who are visually impaired) for specific target memories until the memories are no longer distressing
- include the teaching of self-calming techniques and techniques for managing flashbacks, for use within and between sessions.

For more on NICE's PTSD guidance (2018) as it relates to EMDR, please see Sections 1.6.18 to

1.6.20: <https://www.nice.org.uk/guidance/ng116/chapter/Recommendations>

5.6.5. Psychopharmacological Treatment

While psychopharmacological interventions have been shown to provide some relief, particularly to veterans with PTSD, most clinicians agree that resolution of symptoms cannot be accomplished without implementing exposure and/or cognitive techniques that target the physiological and maladjusted thoughts maintaining the trauma symptoms. With that said,

clinicians agree that psychopharmacology interventions are an effective second line of treatment, particularly when psychotherapy alone does not produce relief from symptoms.

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

You should have learned the following in this section:

- Several treatment approaches are available to clinicians to alleviate the symptoms of trauma- and stressor-related disorders.
- The first approach, psychological debriefing, has individuals who have recently experienced a traumatic event discuss or process their thoughts related to the event and within 72 hours.
- Another approach is to expose the individual to a fear hierarchy and then have them use positive coping strategies such as relaxation techniques to reduce their anxiety or to toss the fear hierarchy out and have the person experience the most distressing memories or images at the beginning of treatment.
- The third approach is Cognitive Behavioral Therapy (CBT) and attempts to identify and challenge the negative cognitions surrounding the traumatic event and replace them with positive, more adaptive cognitions.
- The fourth approach, called EMDR, involves an 8-step approach and the tracking of a clinician's fingers which induces lateral eye movements and aids with the cognitive processing of traumatic thoughts.
- Finally, when psychotherapy does not produce relief from symptoms, psychopharmacology interventions are an effective second line of treatment and may include SSRIs, TCAs, and MAOIs.

Section 5.6 Review Questions

1. Identify the different treatment options for trauma and stress-related disorders. Which treatment options are most effective? Which are least effective?

Module Recap

In Module 5, we discussed trauma- and stressor-related disorders to include PTSD, acute stress disorder, adjustment disorder, and prolonged stress disorder. We defined what stressors were and then explained how these disorders present. In addition, we clarified the epidemiology, comorbidity, and etiology of each disorder. Finally, we discussed potential treatment options for trauma- and stressor-related disorders. Our discussion in Module 6 moves to dissociative disorders.

Part III. Mental Disorders – Block 1

Module 6: Dissociative Disorders

Module 6: Dissociative Disorders

Module Overview

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

Module Outline

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

Module Learning Outcomes

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

6.1. Clinical Presentation

Section Learning Objectives

- Describe dissociative disorders.
- Describe how dissociative identity disorder presents.
- Describe how dissociative amnesia presents.
- Describe how depersonalization/derealization presents.

Dissociative disorders are a group of disorders characterized by symptoms of disruption and/or discontinuity in consciousness, memory, identity, emotion, body representation, perception, motor control, and behavior (APA, 2022). These symptoms are likely to appear following a significant stressor or years of ongoing stress (i.e., abuse; Maldonado & Spiegel, 2014). Occasionally, one may experience temporary dissociative symptoms due to lack of sleep or ingestion of a substance; however, these would not qualify as a dissociative disorder due to the lack of impairment in functioning. Furthermore, individuals who suffer from acute stress disorder and PTSD often experience dissociative symptoms, such as amnesia, numbing, flashbacks, and depersonalization/derealization. However, because of the identifiable stressor (and lack of additional symptoms listed below), they meet diagnostic criteria for a stress disorder as opposed to a dissociative disorder.

There are three main types of dissociative disorders: dissociative identity disorder, dissociative amnesia, and depersonalization/derealization disorder.

6.1.1. Dissociative Identity Disorder (DID)

The key diagnostic criteria for **dissociative identity disorder** is the presence of two or more distinct personality states or an experience of possession (Criteria A). How overt or covert the personality states are depends on psychological motivation, stress level, cultural context, emotional resilience, and internal conflicts and dynamics (APA, 2022), and severe or prolonged stress may result in sustained periods of identify confusion/alteration. Those presenting as being possessed by spirits or demons and for a small proportion of non-possession-form cases, the alternate identifies are readily observable. Generally, though, the identities in non-possession-form dissociative identity disorder are not overtly displayed or only subtly displayed and when they are, it is just in a minority of individuals and manifests as different names, hairstyles, handwritings, wardrobes, accents, etc. If the alternate identities are not observable, their presence is identified through sudden alterations or discontinuities in the individual's sense of self and sense of agency, as well as recurrent dissociative amnesias (see the second criteria below; APA, 2022).

The second main diagnostic criteria (Criteria B) for dissociative identity disorder is that there must be a gap in the recall of events, information, or trauma due to the switching of personalities. These gaps are more excessive than typical forgetting one may experience due to a lack of attention. The dissociative amnesia presents as gaps in autobiographical memory, lapses in memory of well-learned skills or recent events, and discovering possessions for which there is no recollection of ever owning, and can involve everyday events and not just events that are stressful or traumatic.

It should be noted that most possession states occurring around the world are part of broadly accepted cultural or religious practice and should not be diagnosed as dissociative

identity disorder (Criteria D). The possession-form identities in dissociative identity disorder manifest most often as a spirit or supernatural being taking control and the individual speaking or acting in a distinctly different way. These identities present recurrently, are involuntary and unwanted, and cause significant distress or impairment (Criteria C). Impairment varies in adults from minimal (i.e., high functioning professionals) to profound. For those minimally affected, marital, family, relational, and parenting functions are more likely to be impaired by symptoms of dissociative identity disorder rather than their occupational and professional life.

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

The switching or shifting between personalities varies among individuals and can range from merely appearing to fall asleep, to very dramatic, involving excessive bodily movements, though for most, the change is subtle and may occur with only subtle changes in overt presentation. When sudden and unexpected, switching is generally precipitated by a significant stressor, as the subpersonality best equipped to handle the current stressor will present. The relationship between subpersonalities varies between individuals, with some individuals reporting knowledge of other subpersonalities while others have a one-way amnesic relationship with subpersonalities, meaning they are not aware of other personalities (Barlow & Chu, 2014). These individuals will experience episodes of “amnesia” when the primary personality is not present.

6.1.2. Dissociative Amnesia

Dissociative amnesia is identified by the inability to recall important autobiographical information, usually of a traumatic or stressful nature. It often consists of selective amnesia for a specific event or events or generalized amnesia for identity and life history. This type of amnesia is different from what one would consider permanent amnesia in that the information was successfully stored in memory but cannot be freely recollected. It is conceptualized as possibly being a reversible memory retrieval deficit. Additionally, individuals experiencing permanent amnesia often have a neurobiological cause, whereas dissociative amnesia does not (APA, 2022).

There are a few types of amnesia within dissociative amnesia. **Localized amnesia**, the most common type, is the inability to recall events during a specific period. The length of time within a localized amnesia episode can vary—it can be as short as the time immediately surrounding a traumatic event, to months or years, should the traumatic event occur that long (as commonly seen in abuse and combat situations). **Selective amnesia** is, in a sense, a component of localized amnesia in that the individual can recall some, but not all, of the details during a specific period. For example, a soldier may experience dissociative amnesia during the time they were deployed, yet still have some memories of positive experiences such as celebrating Thanksgiving or Christmas dinner with the members of their unit. **Systematized amnesia** occurs when an individual fails to recall a specific category of information such as not recalling a specific room in their childhood home.

Conversely, some individuals experience **generalized dissociative amnesia** in which they have a complete loss of memory for most or all of their life history, including their own identity, previous knowledge about the world, and/or well-learned skills. Individuals who experience this amnesia experience deficits in both *semantic* and *procedural* knowledge. This

means that individuals have no common knowledge of (i.e., cannot identify letters, colors, numbers) nor can they engage in learned skills (i.e., typing shoes, driving car). While generalized dissociative amnesia is extremely rare, it is also extremely frightening. The onset is acute, and the individual is often found wandering in a state of disorientation. Many times, these individuals are brought into emergency rooms by law enforcement following a dangerous situation such as an individual wandering on a busy road.

The distress and impairment suffered by those with dissociative amnesia resulting from childhood/adolescent traumatization varies. Some are chronically impaired in their ability to form and sustain satisfactory attachments while others are highly successful in their occupation due to compulsive overwork. And finally, a substantial subgroup of those afflicted by generalized dissociative amnesia develop a highly impairing, chronic autobiographical memory deficit that is not ameliorated by relearning their life history, resulting in poor overall functioning in most life domains (APA, 2022).

6.1.3. Depersonalization/Derealization Disorder

Depersonalization/derealization disorder is categorized by recurrent episodes of depersonalization and/or derealization. **Depersonalization** can be defined as a feeling of unreality or detachment from *oneself*. Individuals describe this feeling as an *out-of-body experience* where you are an observer of your thoughts, feelings, and physical being. Furthermore, some patients report feeling as though they lack speech or motor control, thus feeling at times like a robot. Distortions of one's physical body have also been reported, with various body parts appearing enlarged or shrunken. Emotionally, one may feel detached from their feelings, lacking the ability to *feel* emotions despite knowing they have them.

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

These episodes can last anywhere from a few hours to days, weeks, or even months. The onset is generally sudden, and like the other dissociative disorders, is often triggered by intense stress or trauma. Many individuals describe feeling like they are “crazy” or “going crazy” and fear they have irreversible brain damage. They experience an altered sense of time and may be obsessed about whether they really exist.

As one can imagine, depersonalization/derealization disorder can cause significant emotional distress, as well as impairment in one’s daily functioning. The disorder is associated with major morbidity and impairment occurs in both interpersonal and occupational spheres due to “...the hypoemotionality with others, subjective difficulty in focusing and retaining information, and a general sense of disconnectedness from life” (APA, 2022).

You should have learned the following in this section:

- Dissociative disorders are characterized by disruption in consciousness, memory, identity, emotion, perception, motor control, or behavior. They include dissociative identity disorder, dissociative amnesia, and depersonalization/derealization disorder.
- Dissociative identity disorder is the presence of two or more distinct personality states or an experience of possession.
- Dissociative amnesia is characterized by the inability to recall important autobiographical information, whether during a specific period (localized) or one's entire life (generalized).
- Depersonalization/derealization disorder includes a feeling of unreality or detachment from oneself (depersonalization) and feelings of unreality or detachment from the world (derealization).

Section 6.1 Review Questions

1. Identify the diagnostic criteria for each of the three dissociative disorders. How are they similar? How are they different?
2. What are the types of amnesia within dissociative amnesia?
3. What is the difference between depersonalization and derealization?

6.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of dissociative disorders.

Dissociative disorders were once believed to be extremely rare; however, more recent research suggests that they may be more present in the general population than once thought. Estimates for the prevalence of dissociative identity disorder in U.S. adults is 1.5%, with women predominating in adult clinical settings. Symptom profiles, clinical history, and childhood trauma history show few gender differences though women have higher rates of somatization. Research shows that dissociative amnesia occurs in approximately 1.8% of the U.S. population. It is estimated that about one-half of all adults have experienced at least one episode of depersonalization/derealization during their life, however, symptomatology that meets full criteria for the disorder is markedly less common than these transient symptoms. A one-month prevalence of about 1-2% was reported in the United Kingdom (APA, 2022).

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

You should have learned the following in this section:

- Dissociative identity disorder has a prevalence of 1.5% and dissociative amnesia occurs in approximately 1.8% of the U.S. population.
- Estimates for depersonalization/derealization disorder are unknown, though it is believed that about half of all adults have experienced at least one episode during their life (i.e. transient symptoms and not full criteria).

Section 6.2 Review Questions

1. What are the prevalence rates for dissociative disorders? What are some identified barriers in determining prevalence rates of these disorders?

6.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of dissociative disorders.

Given that a traumatic experience often precipitates dissociative disorders, it should not be surprising that there is a high comorbidity between most dissociative disorders and PTSD (comorbidity of depersonalization/derealization disorder with PTSD is low). Similarly, depressive disorders are also commonly found in combination with dissociative disorders, likely due to the impact the disorders have on social and emotional functioning. In individuals with dissociative amnesia, a wide range of emotions related to their inability to recall memories

during the episode often present once the amnesia episode is in remission (APA, 2022). These emotions frequently contribute to the development of a depressive episode.

There has been some evidence of comorbid somatic symptom disorder and conversion disorder, particularly for those who experience dissociative amnesia. Furthermore, dependent, obsessive-compulsive, avoidant, and borderline personality traits/disorders are comorbid and for dissociative identity disorder and dissociative amnesia there is evidence of comorbid substance-related and feeding and eating disorders. Anxiety disorders are common for depersonalization/derealization disorder, and often individuals concurrently have unipolar depressive disorder.

You should have learned the following in this section:

- Many dissociative disorders have been found to have a high comorbidity with PTSD and depressive disorders.
- Somatic symptom and conversion disorders, as well as some personality disorders, have also been found to be comorbid.

Section 6.3 Review Questions

1. What are the common comorbid diagnoses for individuals with dissociative disorders?

6.4. Etiology

Section Learning Objectives

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

6.4.1. Biological

While studies on the involvement of genetic underpinnings need additional research, there is some suggestion that heritability rates for dissociation range from 50-60% (Pieper, Out, Bakermans-Kranenburg, Van Ijzendoorn, 2011). However, it is suggested that the combination of genetic and environmental factors may play a larger role in the development of dissociative disorders than genetics alone (Pieper, Out, Bakermans-Kranenburg, Van Ijzendoorn, 2011).

6.4.2. Cognitive

One proposed cognitive theory of dissociative disorders, particularly dissociative amnesia, is a memory retrieval deficit. More specifically, Kopelman (2000) theorizes that the combination of psychological stress and various other biopsychosocial predispositions affects the frontal lobes executive system's ability to retrieve autobiographical memories (Picard et al., 2013). Neuroimaging studies have supported this theory by showing deficits to several prefrontal regions, which is one area responsible for memory retrieval (Picard et al., 2013). Despite these

findings, there is still some debate over which specific brain regions within the executive system are responsible for the retrieval difficulties, as research studies have reported mixed findings.

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

6.4.3. Sociocultural

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

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

6.4.4. Psychodynamic

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

While a single incidence of repression may explain dissociative amnesia, psychodynamic theorists believe that dissociative identity disorder results from repeated exposure to traumatic experiences, such as childhood abuse, neglect, or abandonment (Dalenberg et al., 2012).

According to the psychodynamic perspective, children who experience repeated traumatic events such as physical abuse or parental neglect lack the support and resources to cope with these experiences. To escape from their current situation, children develop different personalities to essentially flee the dangerous situation they are in. While there is limited scientific evidence to support this theory, the nature of severe childhood psychological trauma is consistent with this theory, as individuals with dissociative identity disorder have the highest rate of childhood psychological trauma compared to all other psychiatric disorders (Sar, 2011).

You should have learned the following in this section:

- Though there is some evidence for a genetic component to dissociative disorders, a combination of genes and environment are thought to play a larger role.
- A cognitive explanation assumes a memory retrieval deficit, particularly related to dissociative amnesia, and differential hippocampus activation between subpersonalities in dissociative identity disorder.
- Mass media is also purported to have caused a rise in dissociative disorders due to the attention it gives these disorders in its publications and movies such as *Sybil*.
- Finally, repressed thoughts and feelings are thought to be the cause of dissociative disorders in the psychodynamic theory.

Section 6.4 Review Questions

1. How do the biological, cognitive, sociocultural, and psychodynamic perspectives differ in their explanation of the development of dissociative disorders?

6.5. Treatment

Section Learning Objectives

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

Treatment for dissociative disorders is limited for a few reasons. First, with respect to dissociative amnesia, many individuals recover on their own without any intervention. Occasionally treatment is sought out after recovery due to the traumatic nature of memory loss. Second, the rarity of these disorders has offered limited opportunities for research on both the development and effectiveness of treatment methods. Due to the differences between dissociative disorders, treatment options will be discussed specific to each disorder.

6.5.1. Dissociative Identity Disorder

The ultimate treatment goal for dissociative identity disorder is the **integration** of subpersonalities to the point of **final fusion** (Chu et al., 2011). Integration refers to the ongoing process of merging subpersonalities into one personality. Psychoeducation is paramount for integration, as the individual must understand their disorder, as well as acknowledge their subpersonalities. As mentioned above, many individuals have a one-way amnesic relationship with the subpersonalities, meaning they are not aware of one another. Therefore, the clinician

must first make the individual aware of the various subpersonalities that present across different situations.

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

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

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

Once an individual reaches final fusion, ongoing treatment is essential to maintain this status. In general, treatment focuses on social and positive coping skills. These skills are

particularly helpful for individuals with a history of traumatic events, as it can help them process these events, as well as help prevent future relapses.

6.5.2. Dissociative Amnesia

As previously mentioned, many individuals regain memory without the need for treatment; however, there is a small population that does require additional treatment. While there is no evidenced-based treatment for dissociative amnesia, both hypnosis and phasic therapy have been shown to produce some positive effects in patients with dissociative amnesia.

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

Another form of “hypnosis” is the use of barbiturates, also known as “truth serums,” to help relax the individual and free their inhibitions. Although not always effective, the theory is

that these drugs reduce the anxiety surrounding the unpleasant events enough to allow the individual to recall and process these memories in a safe environment (Ahern et al., 2000).

6.5.3. Depersonalization/Derealization Disorder

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

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

You should have learned the following in this section:

- Treatment for dissociative identity disorder involves the integration of subpersonalities to the point of final fusion and takes several steps to achieve.
- For some patients, this is not possible as they do not find final fusion to be a desirable outcome.
- Instead, the clinician will work to achieve resolution or sufficient integration and coordination of the subpersonalities to allow the person to function independently.
- For dissociative amnesia, hypnosis and phasic therapy are used, as well as barbiturates known as “truth serums.”
- Finally, diagnosis alone is sometimes enough to reduce the intensity of symptoms related to depersonalization/derealization disorder and due to the high comorbidity with anxiety and depression, alleviation of these secondary symptoms is often the goal of treatment.

Section 6.5 Review Questions

1. What is the treatment goal for dissociative identity disorder? How is it achieved?
2. What are the treatment options for dissociative amnesia and depersonalization/depersonalization disorder?

Module Recap

In this module, we discussed the dissociative disorders of Dissociative Identity Disorder, Dissociative Amnesia, and Depersonalization/Derealization Disorder in terms of their clinical presentation, epidemiology, comorbidity, etiology, and treatment approaches.

Part III. Mental Disorders – Block 2

Disorders Covered:

- 7. Anxiety Disorders
- 8. Somatic Symptom and Related Disorders
- 9. Obsessive-Compulsive and Related Disorders

Part III. Mental Disorders – Block 2

Module 7: Anxiety Disorders

Module 7: Anxiety Disorders

Module Overview

In Module 7, we will discuss matters related to anxiety disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include generalized anxiety disorder, specific phobia, agoraphobia, social anxiety disorder, and panic disorder. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the various therapies (Module 3).

Module Outline

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

Module Learning Outcomes

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

7.1. Clinical Presentation

Section Learning Objectives

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

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

7.1.1. Generalized Anxiety Disorder

Generalized anxiety disorder is characterized by an underlying excessive anxiety and worry related to a wide range of events or activities and lasting for more days than not for at least six months. While many individuals experience some degree of worry throughout the day,

individuals with generalized anxiety disorder experience worry of greater intensity and for longer periods than the average person (APA, 2022). Additionally, they are often unable to control their worry through various coping strategies, which directly interferes with their ability to engage in daily social and occupational tasks. To receive a diagnosis of generalized anxiety disorder, three or more of the following somatic symptoms must be present in adults as well: restlessness, fatigue, difficulty concentrating, irritability, muscle tension, and problems sleeping (APA, 2022; Gelenberg, 2000).

7.1.2. Specific Phobia

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

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

situational specific phobias while blood-injection-injury phobia is experienced by both men and women equally (APA, 2022).

7.1.3. Agoraphobia

Agoraphobia is defined as intense fear or anxiety triggered by two or more of the following: using public transportation such as planes, trains, ships, buses; being in large, open spaces such as parking lots or on bridges; being in enclosed spaces like stores or movie theaters; being in a crowd or standing in line; or being outside of the home alone. The individual fears or avoids these situations because they believe something terrible may occur and due to concern over not being able to escape or help not being available (APA, 2022). Active avoidance of the situations occurs and can be behavioral such as changing daily routines or using delivery to avoid entering a restaurant or cognitive such as using distraction to bear with an agoraphobic situation. The avoidance can result in the person being homebound. The fear or anxiety is out of proportion to the actual danger they pose and has been present for at least six months.

7.1.4. Social Anxiety Disorder

For **social anxiety disorder**, the anxiety or fear relates to social situations, particularly those in which an individual can be evaluated by others. More specifically, the individual is worried that they will be judged negatively and viewed as stupid, anxious, crazy, boring, or unlikeable, to name a few. Some individuals report feeling concerned that their anxiety symptoms will be obvious to others via blushing, stuttering, sweating, trembling, etc. These fears severely limit an individual's behavior in social settings and have occurred for six months or more.

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

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

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

7.1.5. Panic Disorder

Panic disorder consists of a series of recurrent, unexpected panic attacks coupled with the fear of future panic attacks. A panic attack is defined as a sudden or abrupt surge of fear or impending doom along with at least four physical or cognitive symptoms. Physical symptoms include heart palpitations, sweating, trembling or shaking, shortness of breath, feeling as though they are being choked, chest pain, nausea, dizziness, chills or heat sensations, and numbness/tingling. Cognitive symptoms may consist of feelings of derealization (feelings of unreality) or depersonalization (feelings of being detached from oneself), the fear of losing control or ‘going crazy,’ or the fear of dying (APA, 2022). While symptoms generally peak within a few minutes, it seems much longer for the individual experiencing the panic attack.

There are two key components to panic disorder—the attacks are *unexpected*, meaning there is nothing that triggers them, and they are *recurrent*, meaning they occur multiple times. Because these panic attacks occur frequently and are primarily “out of the blue,” they cause significant worry or anxiety in the individual as they are unsure of when the next attack will happen. In contrast to unexpected there are also expected panic attacks, or those that have an obvious trigger. The DSM-5-TR states that presence of expected panic attacks does not rule out the diagnosis of panic disorder as about half of individuals diagnosed with the disorder in the United States and Europe have both types of attacks (APA, 2022).

In some individuals, significant behavioral changes such as fear of leaving their home or attending large events occur as the individual is fearful an attack will happen in one of these situations, causing embarrassment. Additionally, individuals report worry that others will think they are “going crazy” or losing control if they were to observe an individual experiencing a

panic attack. Occasionally, an additional diagnosis of agoraphobia is given to an individual with panic disorder *if* their behaviors meet diagnostic criteria for this disorder as well.

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

You should have learned the following in this section:

- All anxiety disorders share the hallmark symptoms of excessive fear or worry related to behavioral disturbances.
- Generalized anxiety disorder is characterized by an underlying excessive worry related to a wide range of events or activities and an inability to control their worry through coping strategies.
- Specific phobia is characterized by fear or anxiety specific to an object or a situation and individuals can experience fear of more than one object.
- Agoraphobia is characterized by intense fear related to situations in which the individual is in public situations where escape may be difficult and help may not be able to come.
- Social anxiety disorder is characterized by fear or anxiety related to social situations, especially when evaluation by others is possible.
- Panic disorder is characterized by a series of recurrent, unexpected panic attacks coupled with the fear of future panic attacks.

Section 7.1 Review Questions

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

7.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of generalized anxiety disorder.
- Describe the epidemiology of specific phobia.
- Describe the epidemiology of agoraphobia.
- Describe the epidemiology of social anxiety disorder.
- Describe the epidemiology of panic disorder.

7.2.1. Generalized Anxiety Disorder

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

7.2.2. Specific Phobia

The prevalence rate for specific phobia is 8-12% in the United States and about 6% in European countries. There is a 2:1 ratio of females to males diagnosed with specific phobia. Prevalence rates are lower in older individuals and those from Asia, Africa, and Latin America.

7.2.3. Agoraphobia

The prevalence rate of agoraphobia worldwide for adolescents and adults is 1% to 1.7%. As with other anxiety disorders, women are twice as likely to be diagnosed with it. Older adults in the United States (aged 65 and up) have a 12-month prevalence of 0.4% and for older adults aged 55 and up in Europe and North America, the prevalence is 0.5%.

7.2.4. Social Anxiety Disorder

The overall prevalence rate of social anxiety disorder is significantly higher in the United States than in other countries, with an estimated 7% of the U.S. population diagnosed with social anxiety disorder, compared to 0.5% to 2.0% worldwide (median prevalence in Europe is 2.3%). A decrease in the diagnosis of social anxiety disorder among older individuals, aged 65 years and older, has been found. Regarding gender, there is a higher diagnosis rate in females than males. This gender discrepancy is greater among adolescents and young adults. Finally, non-Hispanic whites in the United States have a higher prevalence rate than Asian, Latinx, African American, and Caribbean Black descent (APA, 2022).

7.2.5. Panic Disorder

The 12-month prevalence for panic disorder in the general population is estimated at around 2-3% in adults and adolescents across the United States and several European countries. Higher rates of panic disorder are found in American Indians and non-Latinx whites. Females are more commonly diagnosed than males with a 2:1 diagnosis rate. Prevalence declines from about 1.2% in adults older than 55 to 0.7% in adults aged 64 and up.

You should have learned the following in this section:

- Prevalence rates for anxiety disorders range from 1.0% for agoraphobia up to 12% for specific phobia.
- For most anxiety disorders, females are twice as likely to be diagnosed.

Section 7.2 Review Questions

1. Create a table of the prevalence rates across the various anxiety related disorders. What are the differences between the disorders?
2. How do prevalence rates vary as a function of gender, race, nationality, and age?

7.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of generalized anxiety disorder.
- Describe the comorbidity of specific phobia.
- Describe the comorbidity of agoraphobia.
- Describe the comorbidity of social anxiety disorder.
- Describe the comorbidity of panic disorder.

7.3.1. Generalized anxiety disorder

There is a high comorbidity between generalized anxiety disorder and the other anxiety-related disorders, as well as unipolar depressive disorders. Comorbidity with substance use, neurodevelopmental, neurocognitive, psychotic, and conduct disorders is less common for those afflicted with generalized anxiety disorder. Generalized anxiety disorder is associated with

higher levels of suicidal ideation and behavior and psychological autopsy studies reveal it is the most frequent anxiety disorder diagnosed in suicides (APA, 2022).

7.3.2. Specific phobia

Other anxiety disorders, depressive and bipolar disorders, substance-related disorders, and somatic symptom disorder are typically comorbid with specific phobia. Additionally, personality disorders, in particular dependent personality disorder, are comorbid. Specific phobia is associated with the transition from suicidal ideation to attempt (APA, 2022).

7.3.3. Agoraphobia

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

7.3.4. Social Anxiety Disorder

Among the most common comorbid diagnoses with a social anxiety disorder are other anxiety-related disorders, major depressive disorder, and substance-related disorders. The high comorbidity rate among anxiety-related disorders and substance-related disorders is likely connected to the efforts of self-medicating to deal with social fears. For example, an individual with social anxiety disorder may consume more alcohol in social settings in efforts to alleviate the anxiety of the social situation. The comorbidity with major depressive disorder may be due to

the chronic social isolation associated with social anxiety disorder. Comorbidity has also been found with body dysmorphic disorder and avoidant personality disorder.

7.3.5. Panic disorder

Panic disorder rarely occurs in isolation, as 80% of individuals report symptoms of other anxiety disorders, major depressive disorder, bipolar I and bipolar II disorder, and possibly mild alcohol use disorder. Some individuals diagnosed with panic disorder also develop a substance-related disorder, likely as an attempt to treat their anxiety with alcohol or other substances. About 25% of patients report suicidal thoughts and the disorder may increase the risk for future suicidal behaviors but not deaths. (APA, 2022).

Unlike some of the other anxiety disorders, there is a high comorbidity with general medical symptoms. More specifically, individuals with panic disorder are more likely to report somatic symptoms such as dizziness, cardiac arrhythmias, COPD, asthma, irritable bowel syndrome, and hyperthyroidism (APA, 2022). The relationship between panic symptoms and these conditions is unclear.

You should have learned the following in this section:

- Many anxiety disorders are comorbid with one another.
- Other common comorbid disorders include depressive disorders and substance-related disorders.
- Agoraphobia has a high comorbidity with PTSD and panic disorder with general medical symptoms.
- Most anxiety disorders are associated with suicidal thoughts and behaviors, but not always deaths.

Section 7.3 Review Questions

1. What other disorders commonly occur with specific anxiety related disorders and why?
2. What anxiety-related disorder has a high comorbidity with medical symptoms?
3. What is the relationship of the disorders with suicidal ideation and attempts/behaviors? Be specific.

7.4. Etiology

Section Learning Objectives

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

7.4.1. Biological

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

7.4.1.2. Biological - Neurobiological structures. Researchers have identified several brain structures and pathways that are likely responsible for anxiety responses. Among those structures is the **amygdala**, the area of the brain that is responsible for storing memories related to emotional events (Gorman, Kent, Sullivan, & Coplan, 2000). When presented with a fearful situation, the amygdala initiates a reaction to ready the body for a response. First, the amygdala triggers the hypothalamic-pituitary-adrenal (HPA) axis to prepare for immediate action— either to fight or flight. The second pathway is activated by the feared stimulus itself, by sending a

sensory signal to the **hippocampus** and **prefrontal cortex**, to determine if the threat is real or imagined. If it is determined that no threat is present, the amygdala sends a calming response to the HPA axis, thus reducing the level of fear. If a threat is present, the amygdala is activated, producing a fear response.

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

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

pathway supports the theory that panic disorder is mediated by several neuroanatomical structures and their associated neurotransmitters.

7.4.2. Psychological

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

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

Social anxiety is also primarily explained by cognitive theorists. Individuals with social anxiety disorder tend to hold unattainable or extremely high social beliefs and expectations. Furthermore, they often engage in preconceived maladaptive assumptions that they will behave

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

7.4.2.2. Psychological – Behavioral. The behavioral explanation for the development of anxiety disorders is mainly reserved for phobias—both specific and social phobia. More precisely, behavioral theorists focus on **respondent conditioning** - when two events that occur close together become strongly associated with one another, despite their lack of causal relationship (see Module 2 for an explanation of respondent conditioning). Watson and Rayner's (1920) infamous Little Albert experiment is an example of how respondent conditioning can be used to induce fear through associations. In this study, Little Albert developed a fear of white rats by pairing a white rat with a loud sound. This experiment, although lacking ethical standards, was groundbreaking in the development of learned behaviors. Over time, researchers have been able to replicate these findings (in more ethically sound ways) to provide further evidence of the role of respondent conditioning in the development of phobias.

7.4.2.3. Psychological – Modeling is another behavioral explanation of the development of specific and social phobias. In modeling, an individual acquires a fear through observation and imitation (Bandura & Rosenthal, 1966). For example, when a young child observes their parent display irrational fear of an animal, the child may then begin to display similar behavior. Similarly, seeing another individual being ridiculed in a social setting may increase the chances

of developing social anxiety, as the individual may become fearful that they would experience a similar situation in the future. It is speculated that the maintenance of these phobias is due to the *avoidance* of the feared item or social setting, thus preventing the individual from learning that the object or situation is not something that should be feared.

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

7.4.3. Sociocultural

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

Gender has largely been researched within anxiety disorders due to the consistent discrepancy in the diagnosis rate between men and women. As previously discussed, women are routinely diagnosed with anxiety disorders more often than men, a trend that is observed throughout the entire lifespan. One potential explanation for this discrepancy is the influence of

social pressures on women. Women are more susceptible to experience traumatic experiences throughout their life, which may contribute to anxious appraisals of future events. Furthermore, women are more likely to use **emotion-focused coping**, which is less effective in reducing distress than **problem-focused coping** (McLean & Anderson, 2009). These factors may increase levels of stress hormones within women that leave them susceptible to develop symptoms of anxiety. Therefore, it appears a combination of genetic, environmental, and social factors may explain why women tend to be diagnosed more often with anxiety-related disorders.

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

You should have learned the following in this section:

- Biological causes of anxiety disorders include the serotonin transporter gene (5-HTTLPR); brain structures to include the amygdala, hippocampus, and prefrontal cortex; and the locus coeruleus and corticostriatal-thalamocortical (CSTC) circuit in relation to panic disorder.
- Psychological causes of anxiety disorders include maladaptive assumptions, the linking of events through respondent conditioning, modeling, and stimulus generalization as it relates to generalized anxiety disorder.
- Sociocultural causes of anxiety disorders include social pressures leading to a higher rate of diagnosis for women and discrimination and prejudice which affects ethnic minorities and other marginalized groups.

Section 7.4 Review Questions

1. Discuss the biological etiology of panic disorders. What brain structures and neurotransmitters are involved?
2. How does the cognitive model explain the development and maintenance of anxiety related disorders?
3. What is the difference between emotion-focused and problem-focused coping strategies? How do these two coping strategies explain differences in anxiety related disorders?
4. What are the effects of prejudice and discrimination on the development of anxiety disorders?

7.5. Treatment

Section Learning Objectives

- Describe treatment options for generalized anxiety disorder.
- Describe treatment options for specific phobia.
- Describe treatment options for agoraphobia.
- Describe treatment options for social anxiety disorder.
- Describe treatment options for panic disorder.

7.5.1. Generalized Anxiety Disorder

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

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

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

7.5.1.4. Biofeedback. Biofeedback provides a visual representation of a patient's physiological arousal. To achieve this feedback, a patient is connected to a computer that provides continuous information on their physiological states. There are several ways a patient can connect to the computer. Among the most common is electromyography (EMG). **EMG**

measures the amount of muscle activity currently experienced by the individual. An electrode is placed on a patient's skin just above a major muscle group, usually the forearm or the forehead. Other common areas of measurement are **electroencephalography** (EEG), which measures the neurofeedback or brain activity; **heart rate variability** (HRV), which measures autonomic activity such as heart rate or blood pressure; and **galvanic skin response** (GSR) which measures sweat.

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

7.5.2. Specific Phobias

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

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

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

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

Modeling is another common technique used to treat phobias (Kelly, Barker, Field, Wilson, & Reynolds, 2010). In this technique, the clinician approaches the feared object/subject

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

7.5.3. Agoraphobia

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

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

7.5.4. Social Anxiety Disorder

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

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

7.5.4.3. Cognitive restructuring. While exposure and social skills training are suitable treatment options, research routinely supports the need to incorporate cognitive restructuring as an additive component in treatment to provide substantial symptom reduction. Like cognitive restructuring previously discussed in the Mood Disorder module, the clinician will work with the therapist to identify negative, automatic thoughts that contribute to the distress in social

situations. The clinician can then help the patient establish new, positive thoughts to replace these negative thoughts. Research indicates that implementing cognitive restructuring techniques before, during, and after exposure sessions enhances the overall effects of treatment of social anxiety disorder (Heimberg & Becker, 2002).

7.5.5. Panic Disorder

7.5.5.1. Cognitive Behavioral Therapy (CBT). CBT is the most effective treatment option for individuals with panic disorder as the focus is on correcting misinterpretations of bodily sensations (Craske & Barlow, 2014). Nearly 80 percent of people with panic disorder report complete remission of symptoms after mastering the following five components of CBT for panic disorder (Craske & Barlow, 2014).

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

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

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

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

Cognitive restructuring. Cognitive restructuring, or the ability to recognize cognitive errors and replace them with alternate, more appropriate thoughts, is likely the most powerful part of CBT treatment for panic disorder, aside from the exposure part. As noted previously, cognitive restructuring involves identifying the role of thoughts in generating and maintaining emotions. The clinician encourages the patient to view these thoughts as “hypotheses” as opposed to fact, which allows the beliefs to be questioned and challenged. This is where the detailed recordings produced by self-monitoring are helpful. By discussing what the patient has recorded for the relationship between physiological arousal and thoughts/behaviors, the clinician

can help the patient restructure the maladaptive thought processes to more positive thought processes, which in return, helps to reduce fear and anxiety.

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

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

7.5.5.2. Pharmacological interventions. According to Craske & Barlow (2014), nearly half of patients with panic disorder present to psychotherapy already on medication, likely prescribed by their primary care physician. Some researchers argue that anti-anxiety medications

impede the progress of CBT treatment as the individual is not able to fully experience the physiological sensations during exposure sessions, thus limiting their ability to modify maladaptive thoughts and maintaining the panic symptoms. Results from large clinical trials suggest *no advantage* during or immediately after treatment of combining CBT and medication (Craske & Barlow, 2014). Additionally, when the medication was discontinued post-treatment, the CBT+ medication groups fared worse than the CBT treatment-only groups, thus supporting the theory that immersion in interoceptive exposure is limited due to the use of medication. Therefore, it is suggested that medications be reserved for those who do not respond to CBT therapy alone (Kampman, Keijers, Hoogduin & Hendriks, 2002).

You should have learned the following in this section:

- Treatment options for generalized anxiety disorder include benzodiazepines, rational-emotive therapy, CBT, and biofeedback.
- Treatment options for specific phobias include exposure treatments such as systematic desensitization, flooding, and modeling.
- Treatment options for agoraphobia include exposure and CBT techniques.
- Treatment options for social anxiety disorder include exposure treatment, social skills training, and cognitive restructuring.
- Treatment options for panic disorder include CBT, psychoeducation, self-monitoring, relaxation training, cognitive restructuring, exposure, and pharmacological interventions.

Section 7.5 Review Questions

1. Discuss the types of exposure treatments for individuals with anxiety disorders? Which are most effective? What have been some concerns with exposure treatment?
2. What is biofeedback? How is biofeedback used to treat anxiety related disorders?
3. What are the concerns with using pharmacological interventions in the treatment of anxiety disorders? Is there a time when it is helpful to use this treatment method?

Module Recap

Module 7, the first module of Unit 3, covered the topic of anxiety disorders. This discussion included generalized anxiety disorder, specific phobias, agoraphobia, social anxiety disorder, and panic disorder. As with other modules in this book, we discussed the clinical presentation, epidemiology, comorbidity, and etiology of the anxiety disorders. Treatment options included biological, psychological, and sociocultural options. In Module 8, we will discuss somatic symptom and related disorders.

Part III. Mental Disorders – Block 2

Module 8: Somatic Symptom and Related Disorders

Module 8: Somatic Symptom and Related Disorders

Module Overview

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

Module Outline

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

Module Learning Outcomes

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

8.1. Clinical Presentation

Section Learning Objectives

- Describe how somatic symptom disorder presents.
- Describe how illness anxiety disorder presents.
- Describe how functional neurological symptom (conversion) disorder presents.
- Describe how factitious disorder presents.

Psychological disorders that feature somatic symptoms are often challenging to diagnose due to the internalizing nature of the disorder, meaning there is no real way for a clinician to measure the somatic symptom. Furthermore, the somatic symptoms could take on many forms. For example, the individual may be *faking* the physical symptoms, *imagining* the symptoms, *exaggerating* the symptoms, or they could be real and triggered by external factors such as stress

or other psychological disorders. The symptoms also may be part of a real medical illness or disorder, and therefore, the symptoms should be treated medicinally.

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

8.1.1. Somatic Symptom Disorder

Individuals with **somatic symptom disorder** often present with multiple somatic symptoms at one time. These symptoms are significant enough to impact their daily functioning, such as preventing them from attending school, work, or family obligations. The symptoms can be localized (i.e., in one spot) or diffused (i.e., entire body), and can be specific or nonspecific (e.g., fatigue). Individuals with somatic symptom disorder often report excessive thoughts, feelings, or behaviors surrounding their somatic symptoms (APA, 2022). For example, individuals with somatic symptom disorder may spend an excessive amount of time or energy evaluating their symptoms, as well as the potential seriousness of their symptoms. A lack of medical explanation is not needed for a diagnosis of somatic symptom disorder, as it is assumed that the individual's suffering is authentic. Somatic symptom disorder is often diagnosed when another medical condition is present, as these two diagnoses are not mutually exclusive.

Somatic symptom disorder patients generally present with significant worry about their illness. Their interpretation of symptoms is often viewed as threatening, harmful, or troublesome (APA, 2022). Because of their negative appraisals, they fear that their medical status is more serious than it typically is, and high levels of distress are often reported. Oftentimes these patients will “shop” at different physician offices to confirm the seriousness of their symptoms.

8.1.2. Illness Anxiety Disorder

Illness anxiety disorder, previously known as hypochondriasis, involves an excessive preoccupation with having or acquiring a serious medical illness. The key distinction between illness anxiety disorder and somatic symptom disorder is that an individual with illness anxiety disorder does *not* typically present with any somatic symptoms. Occasionally an individual will present with a somatic symptom; however, the intensity of the symptom is mild and does not drive the anxiety. *Acquiring* a serious illness drives concerns and they will even avoid visiting a sick relative or friend for fear of jeopardizing their own health.

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

As one can imagine, the preoccupation and anxiety associated with attaining a medical illness severely impacts daily functioning. The individual will often spend copious amounts of time scanning and analyzing their body for “clues” of potential ailments. Additionally, an excessive amount of time is often spent on internet searches related to symptoms and rare illnesses. Illness becomes a central feature of the person’s identity and self-image. Although extreme, some cases of invalidism have been reported due to illness anxiety disorder (APA, 2022).

Making Sense of the Disorders

In relation to somatic symptom and related disorders, note the following:

For somatic symptom disorder the patient presents with multiple somatic symptoms at one time that are significant enough to impact their daily functioning

For illness anxiety disorder ... the patient does *not* typically present with any somatic symptoms but if they do, the symptoms are just mild in intensity

8.1.3. Functional Neurological Symptom Disorder (Conversion Disorder)

Functional neurological symptom (conversion) disorder occurs when an individual presents with one or more symptoms of altered voluntary motor or sensory function (APA, 2022). Common motor symptoms include weakness or paralysis, abnormal movements (e.g., tremors), and gait abnormalities (i.e., limping). Sensory symptoms include altered, reduced, or absent skin sensation, vision, or hearing. Less commonly seen are epileptic seizures and episodes of unresponsiveness resembling fainting or coma (Marshall et al., 2013). The disorder was called

“conversion disorder” in prior versions of the DSM and in the psychiatric literature. As noted, “The term “conversion” originated in psychoanalytic theory, which proposes that unconscious psychic conflict is “converted” into physical symptoms” (APA, 2022).

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

8.1.4. Factitious Disorder

Factitious disorder differs from the three previously discussed somatic disorders in that there is deliberate falsification of medical or psychological symptoms imposed on oneself or on another, with the overall intention of deception. While a medical condition may be present, the severity of impairment related to the medical condition is more excessive due to the individual’s need to deceive those around them. Even more alarming is that this disorder is not only observed in the individual leading the deception— it can also be present in another individual, often a child or an individual with a compromised mental status who is not aware of the deception behind their illness.

Some examples of factitious disorder behaviors include, but are not limited to, altering a urine or blood test, falsifying medical records, ingesting a substance that would indicate

abnormal laboratory results, physically injuring oneself, and inducing illness by injecting or ingesting a harmful substance. Although most individuals with factitious disorder seek treatment from health care professionals, some choose to mislead community members either in person or online about the illness or injury (APA, 2022). While it is unclear why an individual would want to fake their own (or someone else's) physical illness, there is some evidence suggesting that factors such as depression, lack of parental support during childhood, or an excessive need for social support may contribute to this disorder (McDermott, Leamon, Feldman, & Scott, 2012; Ozden & Canat, 1999; Feldman & Feldman, 1995).

Individuals with factitious disorder are at risk for experiencing psychological distress or functional impairment causing harm to themselves and others such as family, friends, health care professionals, and faith leaders. The DSM-5-TR states, "Whereas some aspects of factitious disorders might represent criminal behavior, such criminal behavior and mental illness are not mutually exclusive" (APA, 2022, pg. 368).

You should have learned the following in this section:

- Somatic symptom disorder is characterized by the presence of multiple somatic symptoms, whether localized or diffused and specific or nonspecific, at one time which impact daily functioning.
- Illness anxiety disorder is characterized by concern over having or acquiring a serious illness, and not the actual presence of somatic symptoms. Individuals spend a great deal of time scanning and analyzing their body for “clues” of potential ailments.
- Functional neurological symptom disorder is characterized by one or more symptoms of voluntary motor or sensory function.
- Factitious disorder is characterized by deliberate falsification of medical or psychological symptoms of oneself or another, with the overall intention of deception.

Section 8.1 Review Questions

1. What are some commonly shared features of somatic disorders?
2. Which somatic disorder usually accompanies a medical diagnosis?
3. What are the key distinctions between illness anxiety disorder and somatic symptom disorder?
4. What are the key differences between factitious disorder and the other somatic disorders?

8.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of somatic disorders.

The prevalence rates for somatic disorders are often difficult to determine; however, overall estimates of somatic symptom disorder are around 4-6%. There is a trend that females report more somatic symptoms than males; thus, more females are diagnosed with somatic symptom disorder than males (APA, 2022).

Seeing as illness anxiety disorder is a newer diagnosis (replacing hypochondriasis), prevalence rates are largely based on the previous disorder. Previous findings suggest that illness anxiety disorder occurs in 1.3% to 10% of the general population and is equal among males and females.

Prevalence rates of factitious disorder are largely unknown, likely due to the use of deception in individuals diagnosed with the disorder. Additionally, health care professionals infrequently record the diagnosis, even in recognized cases (APA, 2022).

And like the other somatic symptom disorders, the prevalence of functional neurological symptom disorder is unknown, even though transient functional neurological symptoms are common. In the United States and northern Europe, research shows that the incidence of individual persistent functional neurological symptoms to be around 4-12 of every 100,000 annually (APA, 2022).

You should have learned the following in this section:

- Though prevalence rates for somatic symptom disorders are hard to determine, it is believed that between 1 and 10% of the population suffer from one of these disorders.
- Females are more likely to be diagnosed with somatic symptom disorder and are as likely as males to be diagnosed with illness anxiety disorder.

Section 8.2 Review Questions

1. Create a table of the prevalence rates across the various somatic disorders. What are the differences between the disorders?
2. What gender differences are evident in the disorders, if any?

8.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of somatic disorders.

Given that half of psychiatric patients also have an additional medical disorder, 35% have an undiagnosed medical condition, and approximately 20% reported medical problems *caused* their mental condition, it should not come as a surprise that somatic disorders, in general, have high comorbidity with other psychological disorders (Felker, Yazel, & Short, 1996). More specifically, anxiety and depressive disorders are among the most commonly co-diagnosed disorders for somatic disorders. While there is not a lot of information regarding specific comorbidities among somatic symptom and related disorders, there is some evidence to suggest

that those with illness anxiety disorder are at risk of developing OCD and personality disorders. Similarly, personality disorders are more common in individuals with functional neurological symptom disorder than the general public. Somatic symptom disorder is also comorbid with PTSD and OCD. (APA, 2022). No comorbidity information is given for factitious disorder.

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

You should have learned the following in this section:

- Anxiety and depression have a high comorbidity with somatic symptom and related disorders.
- Functional neurological symptom disorder and illness anxiety disorder frequently occur with personality disorders.
- PTSD and OCD are comorbid with somatic symptom disorder.
- Central sensitivity syndrome also has high comorbidity with somatic disorders.

Section 8.3 Review Questions

1. In general, what other disorders often occur with somatic disorders?
2. Which disorder do we not know anything about?

8.4. Etiology

Section Learning Objectives

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

8.4.1. Psychodynamic

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

8.4.2. Cognitive

Cognitive theorists often believe that somatic disorders are a result of negative beliefs or exaggerated fears of physiological sensations. Individuals with somatic related disorders may

have a heightened sensitivity to bodily sensations. This sensitivity, combined with their maladaptive thought patterns, may lead individuals to overanalyze and interpret their physiological symptoms in a negative light.

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

8.4.3. Behavioral

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

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

8.4.4. Sociocultural

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

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

You should have learned the following in this section:

- Psychodynamic causes of somatic disorders include primary and secondary gains.
- Cognitive causes of somatic disorders include negative beliefs or exaggerated fears of physiological sensations.
- Behavioral causes of somatic disorders include reinforcers such as attention gained from others or receiving disability.
- Sociocultural causes of somatic disorders include familial influence and culture.

Section 8.4 Review Questions

1. How does catastrophizing contribute to the development and maintenance of somatic disorders?
2. How do somatic disorders develop according to behavioral theorists? Does this theory also explain how the symptoms are maintained? Explain.
3. What does the sociocultural model suggest regarding somatic disorders across cultures?

8.5. Treatment

Section Learning Objectives

- Describe treatment options for somatic disorders.

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

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

8.5.1. Psychotherapy

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

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

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

8.5.2. Psychopharmacology

Psychopharmacological interventions are rarely used due to possible side effects and unknown efficacy. Given that these individuals already have a heightened reaction to their physiological symptoms, there is a high likelihood that the side effects of medication would produce more harm than help. With that said, psychopharmacological interventions may be

helpful for those individuals who have comorbid psychological disorders such as depression or anxiety, which may negatively impact their ability to engage in psychotherapy (McGeary, Harzell, McGeary, & Gatchel, 2016).

You should have learned the following in this section:

- The biopsychosocial model of treatment is one of the most effective for somatic disorders as it considers the various biological, psychological, and social factors that influence the illness and presenting symptoms and includes a multidisciplinary approach.
- Psychotherapy options include interpersonal psychotherapy, CBT, and behavioral.
- Psychopharmacological interventions are rarely used for somatic disorders due to the side effects of the medication producing more harm than good. When used, they deal with comorbid disorders such as depression or anxiety.

Section 8.5 Review Questions

1. Discuss the difference between multidisciplinary and interdisciplinary approaches to treatment of somatic disorders.
2. What is the biopsychosocial model for treatment of somatic disorders? What are the three main components of this treatment?
3. Are there any treatments that are *not* effective in treating somatic disorders? If so, why?

8.6. Psychological Factors Affecting Other Medical Conditions

Section Learning Objectives

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

Although previously known as psychosomatic disorders, the DSM-5-TR has identified physical illnesses that are caused or exacerbated by biopsychosocial factors as *psychological factors affecting other medical conditions*. This disorder is different than all the previously mentioned somatic related disorders as the primary focus of the disorder is not the mental disorder, but rather the physical disorder. Psychological or behavioral factors adversely affect the medical condition by, "...influencing its course or treatment, by constituting an additional well-established health risk factor, or by influencing the underlying pathophysiology to precipitate or exacerbate symptoms or to necessitate medical attention" (APA, 2022, pg. 365). It is believed that a lack of positive coping strategies, psychological distress, or maladaptive health behaviors exacerbate these physical symptoms (McGeary, Harzell, McGeary, & Gatchel, 2016).

8.6.1. Psychophysiological Disorders

The most common types of psychophysiological disorders are headaches (migraines and tension), gastrointestinal (ulcer and irritable bowel), insomnia, and cardiovascular-related

disorders (coronary heart disease and hypertension). We will briefly review these disorders and discuss the associated psychological features believed to exacerbate symptoms.

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

Tension headaches are often described as a dull, constant ache localized to one part of the head or neck; however, it can co-occur in multiple places at one time. Unlike migraines, nausea, vomiting, and sensitivity to light do not often occur with tension headaches. Tension headaches, as well as migraines, are believed to be primarily caused by stress as they are in response to sustained muscle contraction that is often exhibited by those under extreme stress or emotion (Williamson, Barker, Veron-Guidry, 1994). In efforts to reduce the frequency and intensity of both migraines and tension headaches, individuals have found relief in relaxation techniques, as well as the use of biofeedback training to help encourage the relaxation of muscles.

8.6.1.2. Gastrointestinal. Among the two most common types of gastrointestinal psychophysiological disorders are **ulcers** and **irritable bowel syndrome (IBS)**. Ulcers, or painful sores in the stomach lining, occur when mucus from digestive juices are reduced, allowing digestive acids to burn a hole into the stomach lining. Among the most common type of ulcers are peptic ulcers, which are caused by the bacteria *H. pylori* (Sung, Kuipers, El-Serag,

2009). While there is evidence to support the involvement of stress in the development of dyspeptic symptoms, the evidence linking stress and peptic ulcers is slowly growing. (Purdy, 2013). Researchers believe that while *H. pylori* must be present for a peptic ulcer to develop, increased stress levels may impact the amount of digestive acid present in the stomach lining, thus increasing the frequency and intensity of symptoms (Sung, Kuipers, El-Serag, 2009).

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

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

8.6.1.3. Insomnia. Insomnia, the difficulty falling or staying asleep, occurs in more than one-third of the U.S. population, with approximately 10% of patients reporting chronic insomnia (Perlis & Gehrman, 2013). While exact pathways of chronic psychophysiological insomnia are unclear, there is evidence of some biopsychosocial factors that may predispose an individual to develop insomnia such as anxiety, depression, and overactive arousal systems (Trauer et al., 2015). Part of the difficulty with insomnia is the fact that these psychological symptoms can

impact one's ability to fall asleep; however, we also know that lack of adequate sleep also predisposes individuals to increased psychological distress. Due to this cyclic nature of psychological distress and insomnia, intervention for both sleep issues as well as psychological issues is vital to managing symptoms.

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

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

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

8.6.2. Treatments for Psychological Factors Affecting Other Medical Conditions

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

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

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

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

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

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

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

You should have learned the following in this section:

- Psychological factors affecting other medical conditions has as its primary focus the physical disorder, and not the mental disorder.
- The most common types of psychophysiological disorders include headaches to include migraines and tension, gastrointestinal to include ulcers and IBS, insomnia, coronary heart disease, and hypertension.
- Common treatments for these other medical conditions include relaxation training, biofeedback, hypnosis, traditional CBT treatments, and group therapy.

Section 8.6 Review Questions

1. What are the most common types of psychophysiological disorders?
2. Discuss the differences between the different types of headaches.
3. What is the difference between ulcers and irritable bowel syndrome?
4. What are the identified predictors to coronary heart disease and other cardiac events?
5. What are the most effective treatment options for psychophysiological disorders?

Module Recap

In Module 8, we discussed somatic disorders in terms of their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Somatic disorders included somatic symptom disorder, illness anxiety disorder, functional neurological symptom (conversion) disorder, and factitious disorder. We also discussed psychological factors affecting other medication conditions in relation to their clinical presentation, common types of psychophysiological disorders, and treatment.

Part III. Mental Disorders – Block 2

Module 9: Obsessive-Compulsive and Related Disorders

Module 9: Obsessive-Compulsive and Related Disorders

Module Overview

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

Module Outline

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

Module Learning Outcomes

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

9.1. Clinical Presentation

Section Learning Objectives

- Describe how obsessive compulsive disorder presents.
- Describe how body dysmorphic disorder presents.
- Describe how hoarding disorder presents.

9.1.1. Obsessive-Compulsive Disorder

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

Compulsions are time-consuming, repetitive behaviors or mental acts that an individual performs in response to an obsession. Common examples of compulsions are checking (e.g., repeatedly checking if the stove is turned off even though the first four-times they checked it was), counting (e.g., flicking the lights off and on exactly five times), hand washing, symmetry,

fears of harm to self or others, or repeating specific words (APA, 2022). These compulsive behaviors essentially alleviate the anxiety associated with the obsessive thoughts. For example, an individual may feel as though their hands are dirty after using utensils at a restaurant. They may obsess over this thought for some time, impacting their ability to interact with others or complete a specific task. This obsession will ultimately lead to the individual performing a compulsion where they will wash their hands with extremely hot water to rid all the germs, or even wash their hands a specified number of times if they also have a counting compulsion. At this point, the individual's anxiety should be temporarily relieved.

These obsessions and compulsions are more excessive than the typical "cleanliness" as they consume a large part of the individual's day. Additionally, they cause significant impairment in one's daily functioning. Given the example above, an individual with a fear of contamination may refuse to eat at restaurants, or they may bring their utensils from home. The frequency and severity of the obsessions and compulsions varies by patient, with some having mild to moderate symptoms and only spending 1-3 hours a day obsessing or engaging in compulsive behaviors, while other patients present with severe symptoms and have nearly constant intrusive thoughts or compulsions that can become incapacitating (APA, 2022).

9.1.2. Body Dysmorphic Disorder

Body dysmorphic disorder is another obsessive disorder; however, the focus of the obsessions is with perceived defects or flaws in one's physical appearance. A key feature of these obsessions is that they are *not* observable or appear slight to others. An individual who has a congenital facial defect or a burn victim who is concerned about their scars are *not* examples of an individual with body dysmorphic disorder. The obsessions related to one's appearance can run

the spectrum from feeling “unattractive” to “looking hideous.” While any part of the body can be a concern for an individual with body dysmorphic disorder, the most commonly reported areas are skin (acne, wrinkles, skin color), hair (particularly thinning or excessive body hair), and nose (size or shape; APA, 2022). Interestingly, the disorder can occur *by proxy* meaning the individual is not concerned with their own defects but those of another person, often a spouse or partner but at times, a parent, child, sibling, or stranger.

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

While most of us are guilty of engaging in some of these behaviors, to meet criteria for body dysmorphic disorder, one must spend a considerable amount of time preoccupied with their appearance (i.e., on average 3-8 hours a day), as well as display significant impairment in social, occupational, or other areas of functioning. Some individuals excessively tan, change their clothes repeatedly, or compulsively shop such as for beauty products. Camouflaging perceived defects is a common behavior and could involve applying makeup, adjusting a hat or one’s clothes, or covering the forehead or eyes with one’s hair, all to hide or cover the perceived defect or problem area (APA, 2022).

As the DSM-5-TR notes, body dysmorphic disorder has been associated with, “abnormalities in emotion recognition, attention, and executive function, as well as information-

processing biases and inaccuracies in interpretation of information and social situations” (APA, 2022, pg. 273). These individuals tend to express a bias for negative and threatening interpretations of facial expressions and situations that would be classified as ambiguous, for instance.

9.1.2.1. Muscle dysmorphia. While muscle dysmorphia is not a formal diagnosis, it is a common type of BDD, particularly within the male population. Muscle dysmorphia refers to the belief that one’s body is too small or lacks the appropriate amount of muscle definition (Ahmed, Cook, Genen & Schwartz, 2014). While the severity of BDD between individuals with and without muscle dysmorphia appears to be the same, some studies have found higher use of substance abuse (i.e., steroid use), poorer quality of life, and increased reports of suicide attempts in those with muscle dysmorphia (Pope, Pope, Menard, Fay Olivardia, & Philips, 2005). The DSM-5-TR instructs clinicians to specify if body dysmorphic disorder occurs with muscle dysmorphia.

9.1.2.2. Insight specifiers. Those diagnosed with body dysmorphic disorder vary in the degree of insight they have about the accuracy of their body dysmorphic disorder beliefs, ranging from good to absent/delusional. On average, insight is poor and at least one-third of those diagnosed with the disorder display absent/delusional insight. Mental health professionals would indicate the degree of insight regarding body dysmorphic disorder beliefs using *with good or fair insight*, *with poor insight*, or *with absent insight/delusional beliefs*. See page 272 of the DSM-5-TR for more information. Note that the insight specifier is used with OCD and hoarding disorders as well.

9.1.3. Hoarding Disorder

In hoarding disorder, the key feature is the *persistent* over-accumulation of possessions (APA, 2022). While we all obtain items throughout life, individuals with hoarding disorder continue to accumulate items without discarding possessions, regardless of their value or sentiment. This lack of discarding occurs over a long period and is not explained by a recent significant stressor (e.g., lost house in fire, so now keeps everything). For example, last week's newspaper would likely have no relevance to you or possibly any historical value, but those with hoarding disorder would keep this newspaper despite the lack of value or sentiment.

The most commonly hoarded items are newspapers, magazines, clothes, bags, books, mail, and paperwork (APA, 2022). While these items may be stored in attics and garages, individuals with a hoarding disorder also have these items cluttering their living space, sometimes to the extent that they are unable to utilize their furniture because it is covered in stuff. Cognitive factors contributing to the need to hold onto these non-sentimental items are fear of losing valuable information and fear of being wasteful. When asked to "clean out" their house or get rid of these items, individuals with hoarding disorder experience significant distress. Individuals with hoarding disorder display indecisiveness, avoidance, procrastination, perfectionism, difficulty planning and organizing tasks, and are easily distractible.

One's hoarding behaviors also impacts their daily functioning and causes impairment in social and occupational functioning. It can lead to low quality of life and in extreme cases, place the individual at risk for fire, falling, poor sanitation, and other health risks. Family relationships are often strained and conflict with neighbors and local authorities is common (APA, 2022).

You should have learned the following in this section:

- As part of OCD, obsessions are repetitive and persistent thoughts, urges, or images while compulsions are repetitive behaviors or mental acts that an individual performs in response to an obsession.
- Body dysmorphic disorder is characterized by obsessions over perceived defects or flaws in one's physical appearance.
- Muscle dysmorphia refers to the belief that one's body is too small or lacks the appropriate amount of muscle definition and is a type of body dysmorphic disorder common to men.
- Hoarding disorder is characterized by accumulating items without discarding possessions, regardless of their value or sentiment.

Section 9.1 Review Questions

1. Define obsessions and compulsions. Provide a list of examples of each thought/behavior.
2. What is body dysmorphic disorder? Give examples of characteristics that would *not* be consistent with a body dysmorphic disorder diagnosis.
3. Many of us save items throughout our lifetime that remind us of specific events. How is this different from hoarding?

9.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of OCD.
- Describe the epidemiology of body dysmorphic disorder.
- Describe the epidemiology of hoarding disorder.

9.2.1. OCD

The prevalence rate for OCD is approximately 1.2% both in the U.S. and worldwide (APA, 2022). Women are diagnosed with OCD more often than males; however, in childhood, boys are diagnosed more frequently than girls (APA, 2022). With respect to gender and symptoms, females are more likely to be diagnosed with cleaning related obsessions and compulsions. In contrast, males are more likely to display symptoms related to forbidden thoughts and symmetry (APA, 2022). The DSM-5-TR reports that the mean age of onset of OCD is 19.5 years with a quarter of cases starting by 14 years of age. Additionally, males have an earlier age of onset (5-15 yrs.) compared to females (20-24 yrs.; Rasmussen & Eisen, 1990).

9.2.2. Body Dysmorphic Disorder

The point prevalence rate for body dysmorphic disorder among U.S. adults is 2.4% while outside the U.S., the point prevalence is 1.7% to 2.9%. Gender-based prevalence rates indicate that women are more likely to be diagnosed with body dysmorphic disorder than men, though muscle dysmorphia is diagnosed more frequently in men. Additionally, women are more likely

to be preoccupied with weight, breasts, buttocks, legs, hips, and excessive body or facial hair while men have preoccupations with their genitals, body build, and thinning hair (APA, 2022).

9.2.3. Hoarding Disorder

While national studies on the prevalence rate of hoarding within the U.S. and internationally are not available, community surveys estimate clinically significant hoarding as occurring in 1.5% to 6.0% of the population (APA, 2022; Gilliam & Tolin, 2010). Clinical samples are more highly represented by females than males and older individuals (over the age of 65 years) are three times more likely to be diagnosed with hoarding disorder than younger adults.

You should have learned the following in this section:

- The prevalence rate for OCD is about 1.2% while body dysmorphic disorder is 2.4% and hoarding is estimated at 1.5% to 6%.
- In terms of gender, females are more likely to be diagnosed with the three disorders, though in terms of body dysmorphic disorder, males receive the muscle dysmorphia specifier more than females.
- Gender differences are also present for symptom presentation in OCD and the area of the body focused on in body dysmorphic disorder.

Section 9.2 Review Questions

1. What are the key gender differences related to OCD and body dysmorphic disorder?
2. How do the prevalence rate of the three disorders compare?

9.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of OCD.
- Describe the comorbidity of body dysmorphic disorder.
- Describe the comorbidity of hoarding disorder.

9.3.1. OCD

There is a high comorbidity between OCD and other anxiety disorders. Nearly 76% of individuals with OCD will be diagnosed with another anxiety disorder, most commonly panic disorder, social anxiety disorder, generalized anxiety disorder, or a specific phobia. Additionally, due to the nature of OCD and its symptoms, nearly 41% of those with OCD will also be diagnosed with a depressive or bipolar disorder (APA, 2022).

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

It should be noted that there are several disorders—schizophrenia, bipolar disorder, eating disorders, body dysmorphic disorder, and Tourette’s disorder – that OCD is much more common in. Therefore, clinicians who have a patient diagnosed with one of the disorders should also routinely assess patients for OCD (APA, 2022).

Finally, OCD has a mean rate of lifetime suicide attempts of 14.2%, a mean rate of lifetime suicidal ideation of 44.1%, and a mean rate of current suicidal ideation of 25.9%. Severity of OCD, the symptom dimension of unacceptable thoughts, a history of suicidality, and severity of comorbid depressive and anxiety symptoms are predictors of greater suicide risk (APA, 2022).

9.3.2. Body Dysmorphic Disorder

Major depressive disorder is the most common comorbid psychological disorder with body dysmorphic disorder and typically occurs after the onset of body dysmorphic disorder. Additionally, there are some reports of social anxiety disorder, OCD, and substance-related disorders (likely related to muscle enhancement; APA, 2022). Those with body dysmorphic disorder are four times more likely to have experienced suicidal thoughts and 2.6 times more likely to have made suicide attempts compared to healthy control subjects and those diagnosed with eating disorders, OCD, or any anxiety disorder.

9.3.3. Hoarding Disorder

Of those diagnosed with hoarding disorder, about 75% have a comorbid mood or anxiety disorder with major depressive disorder, social anxiety disorder, and generalized anxiety disorder being the most common comorbid conditions. Additionally, nearly 20% also meet the criteria for OCD (APA, 2022).

You should have learned the following in this section:

- OCD is shown to have a high comorbidity with anxiety and depressive disorders as well as tic disorder and ADHD in children.
- Body dysmorphic disorder has a high comorbidity with major depressive disorder.
- Hoarding disorder has a high comorbidity with mood and anxiety disorders.

Section 9.3 Review Questions

1. What are the most common comorbidities for OCD? Be specific.
2. This section discussed the OCD triad in children. What two other disorders complete this triad?
3. Which disorder is body dysmorphic disorder most comorbid with?
4. What can we say about comorbidities with hoarding disorder?

9.4. Etiology

Section Learning Objectives

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

9.4.1. Biological

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

9.4.1.1. Hereditary transmission. With regards to heritability studies, twin studies routinely support the role of genetics in the development of obsessive-compulsive behaviors, as monozygotic twins have a substantially greater concordance rate (80-87%) than dizygotic twins (47-50%; Carey & Gottesman, 1981; van Grootheest, Cath, Beekman, & Boomsma, 2005). Additionally, first degree relatives of patients diagnosed with OCD are at a 5-fold increase to develop OCD at some point throughout their lifespan (Nestadt, et al., 2000).

Interestingly, a study conducted by Nestadt and colleagues (2000) exploring the familial role in the development of obsessive-compulsive disorder found that family members of individuals with OCD had higher rates of both obsessions and compulsions than control families; however, the familial relationship with regards to obsessions were stronger than that of compulsions suggesting that there is a stronger heritability association for obsessions than compulsions.

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

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

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

9.4.1.3. Brain structures. Seeing as neurotransmitters have direct involvement in the development of obsessive-compulsive behaviors, it's only logical that brain structures that house these neurotransmitters also likely play a role in symptom development. Neuroimaging studies implicate the brain structures and circuits in the frontal lobe, more specifically, the orbitofrontal cortex, which is located just above each eye (Marsh et al., 2014). This brain region is responsible for mediating strong emotional responses and converts them into behavioral responses. Once the orbitofrontal cortex receives sensory/emotional information via sensory inputs, it transmits this information through impulses. These impulses are then passed on to the caudate nuclei, which filters through the many impulses received, passing along only the strongest impulses to the thalamus. Once the impulses reach the thalamus, the individual essentially reassesses the emotional response and decides whether to act (Beucke et al., 2013). It is believed that individuals with obsessive compulsive behaviors experience overactivity of the orbitofrontal cortex and a lack of filtering in the caudate nuclei, thus causing too many impulses to transfer to the thalamus (Endrass et al., 2011). Further support for this theory has been shown when individuals with OCD experience brain damage to the orbitofrontal cortex or caudate nuclei and experience remission of OCD symptoms (Hofer et al., 2013).

9.4.2. Cognitive

Cognitive theorists believe that OCD behaviors occur due to an individual's distorted thinking and negative cognitive biases. More specifically, individuals with OCD are more likely to overestimate the probability of harm, loss of control, or uncertainty in their life, thus leading them to over-interpret potential negative outcomes of events. Additionally, some research has indicated that those with OCD also experience disconfirmation bias, which causes the individual

to seek out evidence of their failure to perform the ritual or compensatory behavior correctly (Sue, Sue, Sue, & Sue, 2017). Finally, individuals with OCD often report the inability to trust themselves and their instincts, and therefore, feel the need to repeat the compulsive behavior multiple times to ensure it is done correctly. These cognitive biases are supported throughout research studies that repeatedly find individuals with OCD experience more intrusive thoughts than those without OCD (Jacob, Larson, & Storch, 2014).

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

9.4.3. Behavioral

The behavioral explanation of obsessive compulsive-related disorders focuses on compulsions rather than obsessions. Behaviorists believe that these compulsions begin with and are maintained through **operant conditioning**. How so? Well, an individual with OCD may experience negative thoughts or anxieties related to an unpleasant event (obsession; the event is a stimulus). These thoughts/anxieties cause significant distress to the individual, and therefore, they seek out some behavior (compulsion; the response) to alleviate these threats (i.e., escape behavior associated with negative reinforcement). This provides temporary relief to the individual, thus reinforcing the compulsive behaviors used to lessen the threat. Over time, the compulsive behaviors are reinforced due to the repeated exposure of the obsession and the temporary relief that comes with engaging in these compulsive behaviors (escape behavior).

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

You should have learned the following in this section:

- Biological causes of obsessive-compulsive disorders include hereditary transmission, neurotransmitter deficits particularly in relation to serotonin, and abnormal functioning in brain structures.
- Cognitive causes of obsessive-compulsive disorders include distorted thinking such as overestimating the probability of harm, loss of control, or uncertainty in their life, and negative cognitive biases such as disconfirmation bias.
- Behavioral causes of obsessive-compulsive disorders include operant conditioning.

Section 9.4 Review Questions

1. What are the biological implications regarding the etiology of OCD and related disorders? What brain structures have been linked to these disorders?
2. Discuss identified cognitive biases that are related to the development and maintenance of OCD and related disorders?
3. The behavioral model discusses how respondent conditioning may explain the development and maintenance of these disorders. What type of reinforcement is at work and how?

9.5. Treatment

Section Learning Objectives

- Describe treatment options for OCD.
- Describe treatment options for body dysmorphic disorder.
- Describe treatment options for hoarding disorder.

9.5.1. OCD

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

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

Within the hierarchy of obsessions, the individual is also gradually exposed to their obsession. For example, an individual obsessed with germs, may first watch a person sneeze on the computer in session. Once anxiety is managed and compulsions refrain at this level of exposure, the individual would move on to being present in the same room as a sick individual, to eventually shaking hands with someone obviously sick, each time preventing them from engaging in their compulsive behavior. Once this level of their hierarchy was managed, they would move on to the next obsession and so forth until the entire list was complete.

Treatment outcome for exposure and response prevention is very effective in treating individuals with OCD. In fact, some studies suggest up to an 86% response rate when treatment is completed (Foa et al., 2005). Combination treatments such as ERP with family counseling (utilizing CBT techniques) may increase this response rate even higher (Bolleau, 2011; Krebs & Heyman, 2015). Like most OCD related treatments, the largest barrier to treatment is getting patients to commit to treatment, as the repeated exposures and prevention of compulsive behaviors can be extremely distressing to patients.

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

9.5.2. Body Dysmorphic Disorder

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

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

9.5.2.1. Plastic surgery and medical treatments. Many individuals with body dysmorphic disorder seek out plastic surgery to attempt to correct their deficits. Phillips and colleagues (2001) evaluated treatments of patients with body dysmorphic disorder and found that 76.4% of the patients reported some form of plastic surgery or medical treatment, with dermatology treatment the most reported (45%) followed by plastic surgery (23%). The problem

with this type of treatment is that the individual is rarely satisfied with the outcome of the procedure, thus leading them to seek out additional surgeries on the same defect (Phillips et al., 2001). Therefore, it is important that medical professionals thoroughly screen patients for psychological distress before completing any medical treatment.

9.5.3. Hoarding Disorder

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

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

You should have learned the following in this section:

- Treatment options for OCD include exposure and response prevention, as well as SSRIs though the drug does not provide an added benefit in treatment.
- Treatment options for body dysmorphic disorder include exposure and response prevention and drugs clomipramine and SSRIs.
- Treatment options for hoarding disorder include exposure treatment, cognitive restructuring, and motivational interviewing.

Section 9.5 Review Questions

1. Discuss the various types of treatments for OCD. Which treatment option has the best outcome?
2. What are the different components of Exposure and Response Prevention? How do they work together to reduce OCD symptoms?
3. What are the most effective treatment approaches for body dysmorphic disorder?
4. According to Frost and Hartl (1996) what are the main components that contribute to the maintenance of hoarding disorder?

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 – obsessive compulsive and related disorders.

Part IV. Mental Disorders – Block 3

Disorders Covered:

- 10. Feeding and Eating Disorders
- 11. Substance-Related and Addictive Disorders

Part IV. Mental Disorders – Block 3

Module 10: Feeding and Eating Disorders

Module 10: Feeding and Eating Disorders

Module Overview

In Module 10, we will discuss matters related to feeding and eating disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will cover anorexia nervosa, bulimia nervosa, and binge eating disorder. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the therapies (Module 3).

Module Outline

- 10.1. Clinical Presentation
- 10.2. Epidemiology
- 10.3. Comorbidity
- 10.4. Etiology
- 10.5. Treatment

Module Learning Outcomes

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

10.1. Clinical Presentation

Section Learning Objectives

- Describe how anorexia nervosa presents.
- Describe how bulimia nervosa presents.
- Describe how binge-eating disorder (BED) presents.

Feeding and eating disorders are “...characterized by a persistent disturbance of eating or eating-related behavior that results in the altered consumption or absorption of food and that significantly impairs physical health or psychosocial functioning” (APA, 2022, pg. 371). They are very serious, yet relatively common mental health disorders, particularly in Western society, where there is a heavy emphasis on thinness and physical appearance. In fact, 13% of adolescents will be diagnosed with at least one eating disorder by their 20th birthday (Stice, Marti, & Rohde, 2013). Furthermore, a large number of adolescents will engage in significant disordered eating behaviors just below the clinical threshold (Culbert, Burt, McGue, Iacono & Klump, 2009). While there is no exact cause for eating disorders, the combination of biological, psychological, and sociocultural factors has been identified as major contributors in both the development and maintenance of eating disorders.

Within the DSM 5-TR (APA, 2022), six disorders are classified under the Feeding and Eating Disorders chapter: pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa, and binge-eating disorder. In this module, we will cover the latter three whose diagnostic criteria are **mutually exclusive**, meaning that only one of these diagnoses can be assigned at any given time due to substantial differences in their clinical course,

outcome, and treatment needs, despite a number of common psychological and behavioral features. For a discussion of the first three disorders, see Module 16 please.

For more on eating disorders in general, please visit the National Eating Disorders Association website below:

<https://www.nationaleatingdisorders.org/what-are-eating-disorders>

10.1.1. Anorexia Nervosa

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

Typical warning signs and symptoms are divided into two different categories: emotional/behavioral and physical. Some emotional and behavioral symptoms include dramatic weight loss; preoccupation with food, weight, calories, etc.; frequent comments about feeling "fat;" eating a restricted range of foods; making excuses to avoid mealtimes; and not eating in public. Physical changes may include dizziness, difficulty concentrating, feeling cold, sleep problems, thinning hair/hair loss, and muscle weakness, to name a few. When the individual loses weight, they view this as an impressive achievement and a sign of extraordinary discipline, while weight gain is seen as an unacceptable failure of self-control (APA, 2022).

The onset of the disorder typically begins with mild dietary restrictions such as eliminating carbs or specific fatty foods. As weight loss is achieved, the dietary restrictions progress to more severe, e.g., under 500 calories/day. Symptoms present in adolescence or

young adulthood and rarely before puberty or after age 40. The onset of the disorder typically is preceded by a stressful life event such as leaving home for college.

For more on anorexia nervosa, please visit the National Eating Disorders Association website below:

<https://www.nationaleatingdisorders.org/learn/by-eating-disorder/anorexia>

10.1.2. Bulimia Nervosa

Unlike anorexia nervosa where there is solely restriction of food, bulimia nervosa involves a pattern of recurrent binge eating behaviors. **Binge eating** can be defined as a discrete period of time where the amount of food consumed is significantly more than most people would eat during a similar time period. Individuals with bulimia nervosa often report a sense of lack of control over-eating during these binge-eating episodes. While not always the case, these binge-eating episodes are followed by a feeling of disgust with oneself, which leads to a **compensatory behavior** to rid the body of the excessive calories. These compensatory behaviors include vomiting, use of laxatives, fasting (or severe restriction), diuretics or other medications, or excessive exercise. This cycle of binge eating and compensatory behaviors occurs on average, at least once a week for three months (National Eating Disorder Association website; APA, 2022).

It is important to note that while there are periods of severe calorie restriction like anorexia, the two disorders cannot be diagnosed simultaneously. Therefore, it is important to determine the individual's weight when distinguishing between anorexia and bulimia. If an individual has a significantly low body weight and engages in binge/purging behaviors, the

diagnosis is anorexia; if the individual does *not* have a significantly low body weight and engages in binge/purging behaviors, the diagnosis is bulimia.

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

Making Sense of the Disorders

Though anorexia and bulimia share some common features, they differ as follows:

Diagnosis anorexia if **significantly low body weight with** severe calorie restriction

Diagnosis bulimia ... if body weight is within normal range but displays **calorie** restriction **AND** binge-eating episodes

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

Additionally, those with bulimia are often ashamed of their eating problems and attempt to hide the symptoms. The binge eating occurs in secrecy or as inconspicuously as possible. Common antecedents of binge eating include negative affect; interpersonal stressors; dietary restraint; boredom; and negative feelings linked to body weight, shape, and food.

For more on bulimia nervosa, please visit the National Eating Disorders Association website below:

<https://www.nationaleatingdisorders.org/learn/by-eating-disorder/bulimia>

10.1.3. Binge-Eating Disorder (BED)

Binge-eating disorder is similar to bulimia nervosa in that it involves recurrent binge eating episodes along with feelings of lack of control during the binge-eating episode. The binge-eating episodes are associated with at least three of the following: eating quicker than usual, eating until uncomfortably full, eating large amounts even if not hungry, eating alone, and feeling disgust with oneself or being depressed. Despite the feelings of shame and guilt post-binge, individuals with BED will not engage in vomiting, excessive exercise, or other compensatory behaviors. These binge eating episodes occur on average, at least once a week for 3 months.

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

Making Sense of the Disorders

Though bulimia and BED are similar, they differ as follows:

Diagnosis BED if binge eating occurs **WITHOUT** compensatory behaviors

Diagnosis bulimia ... if binge eating occurs **AND** there are compensatory behaviors to prevent weight gain

While much is still being researched about binge-eating disorder, current research indicates that the onset of BED is adolescence to early adulthood but can begin later in life. Those who seek treatment tend to be older than those with either bulimia or anorexia. Binge eating has been found to be common in adolescent and college-age samples and for all, is associated with social role adjustment issues, impaired health-related quality of life and life satisfaction, and increased medical morbidity and mortality (APA, 2022).

For more on binge eating disorder, please visit the National Eating Disorders Association website below:

<https://www.nationaleatingdisorders.org/learn/by-eating-disorder/bed>

You should have learned the following in this section:

- Anorexia nervosa involves the restriction of food, which leads to significantly low body weight relative to the individual's age, sex, and development, and an intense fear of gaining weight or becoming fat.
- Bulimia nervosa is characterized by a pattern of recurrent binge eating behaviors followed by compensatory behaviors.
- Binge-eating disorder is characterized by recurrent binge eating episodes along with a feeling of lack of control but no compensatory behavior to rid the body of the calories.

Section 10.1 Review Questions

1. What does mutually exclusive mean? What does it mean with respect to eating disorders?
2. What are the key differences in diagnostic criteria for anorexia, bulimia, and binge eating disorder?
3. Define compensatory behavior. What disorder is this found in?

10.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of anorexia nervosa.
- Describe the epidemiology of bulimia nervosa.
- Describe the epidemiology of binge eating disorder.

10.2.1. Anorexia Nervosa

According to the National Eating Disorder Alliance (NEDA) website, at any point in time more women (0.3-0.4%) than men (0.1%) will be diagnosed with anorexia. Anorexia nervosa is most prevalent in postindustrialized, high-income countries such as the United States, Australia, New Zealand, Japan, and many European countries. In the U.S., prevalence is lower among Latinx and non-Latinx Black Americans than non-Latinx Whites (APA, 2022).

10.2.2. Bulimia Nervosa

According to the NEDA website, at any point in time, 1.0% of women and 0.1% of men will meet the diagnostic criteria for bulimia nervosa. A study by Stice and Bohon (2012) found that between 1.1% and 4.6% of females and 0.1% to 0.5% of males will develop bulimia and that subthreshold bulimia occurs in 2.0% to 5.4% of adolescent females. The DSM reports that the 12-month prevalence ranges from 0.14% to 0.3% with higher rates in females and high-income countries. Rates are similar across ethnoracial groups across the U.S. (APA, 2022).

10.2.3. Binge Eating Disorder

Hudson et al. (2007) reports that BED is three times more common than anorexia and bulimia and is more common than breast cancer, HIV, and schizophrenia. It has also been found that between 0.2% and 3.5% of females and 0.9% and 2.0% of males will develop binge eating disorder with subthreshold binge eating disorder occurring in 1.6% of adolescent females (Stice & Bohon, 2012). The DSM reports a 12-month prevalence of 0.44% to 1.2% with rates 2-3 times higher in women, similar rates across ethnoracial groups in the United States and between most high-income industrialized countries (APA, 2022).

For more on statistics and research related to feeding and eating disorders, please visit the National Eating Disorders Association website below:

<https://www.nationaleatingdisorders.org/statistics-research-eating-disorders>

You should have learned the following in this section:

- BED is three times more common than anorexia and bulimia.
- All feeding and eating disorders are more common in women and high-income, industrialized countries.
- Only anorexia shows differences across ethnoracial groups in the United States.

Section 10.2 Review Questions

1. Which feeding and eating disorder is most common?
2. What gender differences occur with regards to the eating disorders?
3. Are there any other noteworthy similarities or differences in the prevalence rates of the three disorders?

10.3. Comorbidity

Section Learning Objectives

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

10.3.1. Anorexia Nervosa

Anorexia is rarely a single diagnosis. High rates of bipolar, depressive, and anxiety disorders are common among individuals with anorexia nervosa. Obsessive-compulsive disorder is more often seen in those with the restricting type of anorexia nervosa, whereas alcohol use disorder and other substance use disorders are more commonly seen in those with anorexia who engage in binge-eating/purging behaviors. Unfortunately, there is also a high rate of suicidality, with rates reported to be 18 times greater than in an age- and gender-matched comparison group. It is also estimated that between 9% and 25% of individuals with anorexia have attempted suicide (APA, 2022).

10.3.2. Bulimia Nervosa

The majority of individuals diagnosed with bulimia nervosa also present with at least one other mental disorder. Similar to anorexia nervosa, there is a high frequency of depressive symptoms (i.e., low self-esteem), as well as bipolar and depressive disorders. While some experience mood fluctuations because of their eating pattern (occurring at the same time or

following the development of bulimia), some individuals will identify mood symptoms prior to the onset of bulimia nervosa (APA, 2022).

Anxiety, particularly social anxiety, is often present in those with bulimia nervosa. However, most mood and anxiety symptoms resolve once an effective treatment of bulimia is established. Substance use disorder, and in particular alcohol use disorder, is also prevalent in those with bulimia, with about a 30% prevalence among those with bulimia. The substance abuse begins as a compensatory behavior (e.g., stimulant use is used to control appetite and weight) and over time, as the eating disorder progresses, so does the substance abuse. There is also a percentage of individuals with bulimia nervosa who display personality features that meet the criteria for at least one personality disorder, most often borderline personality disorder. Finally, about one-quarter to one-third of individuals with bulimia have had suicidal ideation and a comparable amount have attempted suicide.

10.3.3. BED

Research shows that BED shares similar comorbidities with anorexia nervosa and bulimia nervosa. Common comorbidities include major depressive disorder and alcohol use disorder. About 25% of those with BED have shown suicidal ideation (APA, 2022).

You should have learned the following in this section:

- Anorexia has a high comorbidity with bipolar, depressive, and anxiety disorders. OCD and alcohol use disorder are also comorbid but depend on the type of anorexia (restricting or binge-eating/purging).
- Bulimia has a high comorbidity with bipolar disorder, depressive symptoms and disorders, social anxiety, and substance use disorder.
- BED is highly comorbid with MDD and alcohol use disorder.
- There is a high rate of suicidal ideation with all three disorders.

Section 10.3 Review Questions

1. Discuss the comorbidity rates among the three main eating disorders.

10.4. Etiology

Section Learning Objectives

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

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

10.4.1. Biological

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

as strong for bulimia, identical twins still display a 23% concordance rate, compared to the 9% rate for fraternal twins.

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

10.4.2. 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 preferred treatment for eating disorders.

10.4.3. Sociocultural

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

10.4.3.1. Media. One commonly discussed contributor to eating disorders is the media. The idealization of thin models and actresses sends the message to young women (and adolescents) that to be popular and attractive, you must be thin. These images are not isolated to magazines, but are also seen in television shows, movies, commercials, and large advertisements on billboards and hanging in store windows. With the emergence of social media (e.g., Facebook, Snapchat, Instagram), exposure to media images and celebrities is even easier. Couple this with the ability to alter images to make individuals even thinner, it is no wonder many young people become dissatisfied with their body (Polivy & Herman, 2004).

10.4.3.2. Ethnicity. While eating disorders are not solely a “white woman” disorder, there are significant discrepancies when it comes to race, especially for anorexia nervosa. Why is this? Research indicates that black men prefer heavier women than do white men (Greenberg & Laporte, 1996). Given this preference, it should not be surprising that black women and children have larger ideal physiques than their white peers (Polivy & Herman, 2000). Since black women

are less driven to thinness, black women would appear to be less likely to develop anorexia; however, findings suggest this is not the case. Caldwell and colleagues (1997) found that high-income black women were equally as dissatisfied as high-income white women with their physique, suggesting body image issues may be more closely related to SES than that of race. The race discrepancies are also less significant in BED, where the prominent feature of the eating disorder is not thinness (Polivy & Herman, 2002).

10.4.3.3. Gender. Males account for only a small percentage of eating disorders. While it is unclear as to why there is such a discrepancy, it is likely somewhat related to cultural desires of women being “thin” and men being “muscular” or “strong.”

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

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

10.4.3.4. Family. Family influences are one of the strongest external contributors to maintaining eating disorders. Often family members are praised for their slenderness. Think about the last time you saw a family member or close friend- how often have you said, “You look great!” or commented on their appearance in some way? The odds are likely high. While the intent of the family member is not to maintain maladaptive eating behaviors by praising the

physical appearance of someone struggling with an eating disorder, they are indirectly perpetuating the disorder.

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

10.4.4. 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.

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

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

You should have learned the following in this section:

- Biological causes of eating disorders include a genetic predisposition and disruption in the neuroendocrine system.
- Cognitive causes of eating disorders include distorted thought patterns and an over-evaluation of body size.
- Sociocultural causes of eating disorders include the idealization of thin models and actresses by the media, SES, gender, and family involvement.
- The personality trait of perfectionism and low self-esteem are contributing factors to disorders related to eating, weight, and body shape.

Section 10.4 Review Questions

1. Define multidimensional disorders?
2. What evidence is there to suggest eating disorders are biologically driven?
3. According to the cognitive theory, eating disorders may be a variant of what other disorder?
4. Discuss the four sociocultural subgroups that explains development of eating disorders.
5. What are the two personality traits most commonly used to describe behaviors associated with eating disorders?

10.5. Treatment

Section Learning Objectives

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

10.5.1. Anorexia Nervosa

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

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

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

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

10.5.2. Bulimia Nervosa

Just as anorexia nervosa treatment initially focuses on weight gain, the first goal of bulimia nervosa treatment is to eliminate binge eating episodes and compensatory behaviors. The aim is to replace both negative behaviors with positive eating habits. One of the most effective ways to establish this is through Cognitive Behavioral Therapy (CBT).

10.5.2.1. CBT. Similar to anorexia nervosa, individuals with bulimia nervosa are expected to keep a journal of their eating habits; however, with bulimia nervosa, it is also important that the journal include changes in sensations of hunger and fullness, as well as other

feelings surrounding their eating patterns in efforts to identify triggers to their bingeing episodes (Agras, Fitzsimmons-Craft & Wilfley, 2017). Once these triggers are identified, psychologists will utilize specific behavioral or cognitive techniques to prevent the individual from engaging in binge episodes or compensatory behaviors.

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

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

10.5.2.2. Interpersonal Psychotherapy (IPT). IPT has also been established as an effective treatment for those with bulimia nervosa, particularly if an individual has not been successful with CBT treatment. The goal of IPT is to improve interpersonal functioning in those with eating disorders. Originally a treatment for depression, IPT-E was adapted to address the

social isolation and self-esteem problems that contribute to the maintenance of negative eating behaviors.

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

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

Phase Three is the final stage. The goals of this phase are to ensure that the changes made in Phase two are maintained. To achieve this, treatment sessions are spaced out, allowing patients more time to engage in their changed behavior. Additionally, relapse prevention (i.e., problem-solving ways *not* to relapse) is also discussed to ensure long term results. In doing this, the patient reviews the progress they have made throughout treatment, as well as identifying potential interpersonal issues that may arise, and how their treatment can be adapted to address those issues.

Support for IPT-E is limited; however, two extensive studies suggest that IPT-E is effective in treating bulimia nervosa, and possibly BED. While treatment is initially slower than

CBT, it is equally effective in long-term follow-up and maintenance of disorder (Fairburn, Marcus, & Wilson, 1993).

10.5.3. Binge Eating Disorder

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

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

10.5.4. Outcome of Treatment

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

Unfortunately, research has not identified any consistent predictors of positive outcomes for bulimia nervosa. However, there is some speculation that individuals with childhood obesity, low self-esteem, and those with a personality disorder have worse treatment outcomes. While treatment outcome for BED is still in its infancy, initial findings suggest that remission rates of BED are much higher than that for anorexia nervosa and bulimia nervosa.

You should have learned the following in this section:

- Treatment options for anorexia nervosa include CBT and FBT.
- Treatment options for bulimia nervosa include CBT, exposure and response prevention, and the three phases of interpersonal psychotherapy.
- Treatment options for BED include the taking of antidepressants to manage depressive symptoms, CBT, and interpersonal therapy.

Section 10.5 Review Questions

1. What is the initial (main) goal of treatment for anorexia?
2. What are the three phases of family-based treatment?
3. What is the goal for interpersonal psychotherapy? Discuss the three phases of IPT.
4. What is the overall treatment effectiveness of eating disorders?

Module Recap

Module 10 covered eating disorders in terms of their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. In Module 11, we will discuss substance-related and addictive disorders, which will conclude this part.

Part IV. Mental Disorders – Block 3

Module 11: Substance-Related and Addictive Disorders

Module 11: Substance-Related and Addictive Disorders

Module Overview

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

Module Outline

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

Module Learning Outcomes

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

11.1. Clinical Presentation

Section Learning Objectives

- Define substances and substance abuse.
- Describe properties of substance abuse.
- Describe how substance use disorder presents.
- Describe how substance intoxication presents.
- Describe how substance withdrawal presents.
- Define depressants and describe types.
- Define stimulants and describe types.
- Define hallucinogens/cannabis/combinations and describe types.
- Describe the effects of using drugs in combination.

11.1.1. Defining Terms and Adding Context

Substance-related and addictive disorders are among the most prevalent psychological disorders, with roughly 100 million people in the United States reporting the use of an illegal substance sometime throughout their life (SAMHSA, 2014). It is worth noting that the DSM-5 shifted terminology from *drug addiction* to *substance use disorder*, "...to describe the wide range of the disorder, from a mild form to a severe state of chronically relapsing, compulsive pattern of drug taking." The DSM-5 acknowledges that many clinicians will use the term *drug addiction* to describe more severe presentations, but it is omitted from the DSM-5 due to "...its uncertain definition and its potentially negative connotation" (APA, 2022, pg. 543).

What are substances? **Substances** are any ingested materials that cause temporary cognitive, behavioral, or physiological symptoms within the individual. The DSM uses 10 classes of substances: alcohol, caffeine, cannabis, hallucinogens, inhalants, opioids, sedatives, stimulants, tobacco, and other (or unknown).

Repeated use of these substances or frequent substance intoxication can develop into a long-term problem known as **substance abuse**. Abuse occurs when an individual consumes the substance for an extended period or must ingest large amounts of the substance to get the same effect a substance provided previously. The need to continually increase the amount of ingested substance is known as **tolerance**. As tolerance builds, additional physical and psychological symptoms present, often causing significant disturbances in an individual's personal and professional life. Individuals with substance abuse often spend a significant amount of time engaging in activities that revolve around their substance use, thus spending less time in recreational activities that once consumed their time.

Sometimes, there is a desire to reduce or abstain from substance use; however, cravings and **withdrawal** symptoms often prohibit this from occurring. Common withdrawal symptoms include, but are not limited to, cramps, anxiety attacks, sweating, nausea, tremors, and hallucinations. Depending on the substance and the tolerance level, most withdrawal symptoms last anywhere from a few days to a week. For those with extensive substance abuse or abuse of multiple substances, withdrawal should be closely monitored in a hospital setting to avoid severe consequences such as seizures, stroke, or even death.

According to the DSM-5-TR (APA, 2022), the substance-related disorders are divided into two groups: substance use disorders and substance-induced disorders which include substance intoxication and substance withdrawal. While there are some subtle differences in

symptoms, particularly psychological, physical, and behavioral symptoms, the general diagnostic criteria for substance use disorder, substance intoxication, and substance withdrawal remains the same across substances. These criteria are reviewed below, with more specific details of psychological, physical, and behavioral symptoms in the Section 11.1.5.: Types of Substances Abused.

11.1.2. Substance Use Disorder

The essential feature of **substance use disorder**, is a “...cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues using the substance despite significant substance-related problems” (APA, 2022, pg. 544) and can be diagnosed for all the substance classes except caffeine. Distress or impairment can be described as any of the following: inability to complete or lack of participation in work, school or home activities; increased time spent on activities obtaining, using, or recovering from substance use; impairment in social or interpersonal relationships; use of a substance in a potentially hazardous situation; psychological problems due to recurrent substance abuse; craving the substance; an increase in the amount of substance used over time (i.e., tolerance); difficulty reducing the amount of substance used despite a desire to reduce/stop using; and/or withdrawal symptoms (APA, 2022). While the number of these symptoms may vary among individuals, only two symptoms are required for a diagnosis of substance use disorder.

11.1.3. Substance Intoxication

For a diagnosis of **substance intoxication**, the individual must have recently ingested a substance. Immediately following the ingestion of this substance, significant behavioral and/or

psychological change is observed. In addition, physical and physiological symptoms present as a direct result of the substance ingested. As stated above, these behavioral, physical, and physiological symptoms are dependent on the type of substance that is ingested and, therefore, discussed in more detail within each substance category (i.e., depressants, stimulants, hallucinogens/cannabis/combo). This said, the most common changes involve disturbances of perception, wakefulness, attention, thinking, psychomotor behavior, interpersonal behavior, and judgment (APA, 2022).

11.1.4. Substance Withdrawal

Finally, **substance withdrawal** is diagnosed when there is cessation or reduction of a substance that has been used for a long period of time. Individuals undergoing substance withdrawal will experience physiological and psychological symptoms within a few hours after cessation/reduction. These symptoms cause significant distress or impairment in daily functioning (APA, 2022). As with substance intoxication, physiological and psychological symptoms during substance withdrawal are often specific to the substance abused and are discussed in more detail within each substance category later in the module.

11.1.5. Types of Substances Abused

For our purposes, the most abused substances will be divided into three categories based on how they impact one's physiological state: depressants, stimulants, and hallucinogens/cannabis/combo.

11.1.5.1. Depressants. Depressants include alcohol, sedative-hypnotic drugs, and opioids are known to have an inhibiting effect on one's central nervous system; therefore, they are often

used to alleviate tension and stress. Unfortunately, when used in large amounts, they can also impair an individual's judgment and motor activity.

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

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

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

The effect of ethyl alcohol in moderation allows for an individual to relax, engage more readily in conversation, and in general, produce a confident and happy personality. However, when consumption is increased or excessive, the central nervous system is unable to metabolize

the ethyl alcohol adequately, and adverse effects begin to present. Symptoms such as blurred vision, difficulty walking, slurred speech, slowed reaction time, and sometimes, aggressive behaviors are observed.

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

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

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

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

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

Opioids are unique in that they provide both euphoria and drowsiness. Tolerance to these drugs builds quickly, thus resulting in an increased need of the medication to produce desired effects. This rapid tolerance is also likely responsible for opioids’ highly addictive nature. Opioid

withdrawal symptoms can range from restlessness, muscle pain, fatigue, anxiety, and insomnia. Unfortunately, these withdrawal symptoms, as well as intense cravings for the drug, can persist for several months, with some reports up to years. Because of the intensity and longevity of these withdrawal symptoms, many individuals struggle to remain abstinent, and accidental overdoses are common (CDC, 2013).

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

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

Similar to opioids, **cocaine** is extracted from a South American plant—the coca plant—and produces feelings of energy and euphoria. It is the most potent natural stimulant known to date (Acosta et al., 2011). Low doses can produce feelings of excitement, talkativeness, and euphoria; however, as the amount of ingested cocaine increases, physiological changes such as rapid breathing, increased blood pressure, and excessive arousal can be observed. The

psychological and physiological effects of cocaine are due to an increase of *dopamine*, *norepinephrine*, and *serotonin* in various brain structures (Hart & Ksir, 2014; Haile, 2012).

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

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

Crack is a derivative of cocaine that is formed by combining cocaine with water and another substance (commonly baking soda) to create a solid structure that is then broken into smaller pieces. Because of this process, it requires very little cocaine to make crack, thus making it a more affordable drug. Coined for the crackling sound that is produced when it is smoked, it is also highly addictive, likely due to the fast-acting nature of the drug. While the effects of cocaine

peak in 20-30 minutes and last for about 1-2 hours, the effects of crack peak in 3-5 minutes and last only for up to 60 minutes (Addiction Centers of America).

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

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

While we are sure you are well aware of how **caffeine** is consumed, you may be surprised to learn that in addition to coffee, energy drinks, and soft drinks, caffeine can also be found in chocolate and tea. Because of the vast use of caffeine, it is the most widely consumed substance in the world, with approximately 90% of Americans consuming some form of caffeine each day (Fulgoni, Keast, & Lieberman, 2015). While caffeine is often consumed in moderate dosages, caffeine intoxication and withdrawal can occur. In fact, an increase in caffeine

intoxication and withdrawal have been observed with the simultaneous popularity of energy drinks. Common energy drinks such as Monster and Red Bull have nearly double the amount of caffeine of tea and coke (Bigard, 2010). While adults commonly consume these drinks, a startling 30% of middle and high schoolers also report regular consumption of energy drinks to assist with academic and athletic responsibilities (Terry-McElrath, O'Malley, & Johnston, 2014). The rapid increase in caffeinated beverages has led to a rise in ER visits due to the intoxication effects (SAMHSA, 2013).

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

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

individuals, as well as *within* the same individual. This means that the same amount of the same drug may produce a positive experience one time, but a negative experience the next time.

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

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

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

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

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

You should have learned the following in this section:

- An individual is diagnosed with substance use disorder, substance intoxication, or substance withdrawal specific to the substance or substances being ingested though the symptoms remain generally the same across substances.
- Substance use disorder occurs when a person experiences significant impairment or distress for 12 months due to the use of a substance.
- Substance intoxication occurs when a person has recently ingested a substance leading to significant behavioral and/or psychological changes.
- Substance withdrawal occurs when there is a cessation or reduction of a substance that has been used for a long period of time.
- Depressants include alcohol, sedative-hypnotic drugs, and opioids.
- Stimulants include cocaine and amphetamines, but caffeine as well.
- Hallucinogens come from natural sources and produce powerful changes in sensory perception.
- Cannabis is also derived from a natural plant and produces psychoactive effects.
- Many drugs are taken by users in combination which can have dangerous results depending on the interactions between the substances.

Section 11.1 Review Questions

1. What is a substance?
2. What is the difference between substance intoxication and substance abuse?
3. What is the difference between tolerance and withdrawal?
4. Create a table listing the three types of substances abused, as well as the specific substances within each category.
5. What are the common factors that affect alcohol absorption?
6. What are the effects of sedative-hypnotic drugs?
7. What receptors are responsible for increasing activity in alcohol and benzodiazepines?
8. What is responsible for the addictive nature of opioids?
9. Which neurotransmitters are implicated in cocaine use?
10. What are the different ways cocaine can be ingested?
11. List the common types of amphetamines.

11.2. Epidemiology

Section Learning Objectives

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

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

11.2.1. Depressants

Concerning depressant substances, men outnumber women in alcohol abuse 2 to 1 (Johnston et al., 2014). Ethnically, Native Americans have highest rate of alcoholism, followed by White, Hispanic, African, and Asian Americans. With regards to opioid use, roughly 1% of

the population has this disorder, with 80% of those being addicted to pain-reliever opioids such as oxycodone or morphine; the remaining 20% are heroin (SAMHSA, 2014).

11.2.2. Stimulants

Nearly 1.1% of all high school seniors have used cocaine within the past month (Johnston et al., 2014). Due to the high cost of cocaine, it is more commonly found in suburban neighborhoods where consumers have the financial means to purchase the drugs.

Methamphetamine is used by men and women equally. It is popular among biker gangs, rural America, and urban gay communities, as well as in clubs and all-night dance parties (aka raves; Hopfer, 2011).

A growing concern is the abuse of stimulant medication among college students as 17% of college students reported abusing stimulant medications. Greek organization membership, academic performance, and other substance use were the most highly correlated variables related to stimulant medication abuse.

11.2.3. Hallucinogens

Up to 14% of the general population have used LSD or another hallucinogen. Nearly 20 million adults and adolescents report current use of marijuana. Men report more than women. Sixty-five percent of individuals report their first drug of use was marijuana—labeling it as a gateway drug to other illicit substances (APA, 2022). Due to the increased research and positive effects of medicinal marijuana, the movement to legalize *recreational* marijuana has gained momentum, particularly in the Pacific Northwest of the United States.

You should have learned the following in this section:

- More men and Native Americans are addicted to depressants.
- Cocaine is more prevalent in suburban neighborhoods due to its cost and methamphetamine is used equally by men and women.
- Hallucinogens are used by up to 14% of the general population.

Section 11.2 Review Questions

1. Identify the gender and ethnicity differences of substance abuse across the three substance categories.
2. Are these substances abused by other unique groups of people?

11.3. Comorbidity

Section Learning Objectives

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

Substance abuse, in general, has a high comorbidity within itself (meaning abuse of multiple different substances), as well as with other mental health disorders. Researchers believe that substance abuse disorders are often secondary to another mental health disorder, as the substance abuse develops as a means to “self-medicate” the underlying psychological disorder. In fact, several large surveys identified alcohol and drug dependence to be twice as more likely in individuals with anxiety, affective, and psychotic disorders than the general public (Hartz et al., 2014). While it is difficult to identify exact estimates of the relationship between substance abuse and serious mental health disorders, the consensus among researchers is that there is a strong relationship between substance abuse and mood, anxiety, PTSD, and personality disorders (Grant et al., 2016).

You should have learned the following in this section:

- Substance abuse has a high comorbidity within itself and with mental health disorders such as mood, anxiety, PTSD, and personality disorders.

Section 11.3 Review Questions

1. With what other conditions are substance-related and addictive disorders highly comorbid?

11.4. Etiology

Section Learning Objectives

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

11.4.1. Biological

11.4.1.1. Genetics. Similar to other mental health disorders, substance abuse is genetically influenced. With that said, it is different than other mental health disorders in that if the individual is *not* exposed to the substance, they will not develop substance abuse.

Heritability of alcohol abuse is among the most well studied substances, likely because it is the only legal substance (except cannabis in some states). Twin studies have indicated a range of 50-60% heritability risk for alcohol disorder (Kendler et al., 1997). Studies exploring the heritability of other substance abuse, particularly drug use, suggests there may be a stronger heritability link than previously thought (Jang, Livesley, & Vernon, 1995). Twin studies indicate that the genetic component of drug abuse is stronger than drug use in general, meaning that genetic factors are more significant for abuse of a substance over nonproblematic use (Tsuang et al., 1996).

Merikangas and colleagues (1998) found an 8-fold increased risk for developing a substance abuse disorder across a wide range of substances.

Unique to substance abuse is the fact that both genetic and familial influence are both at play. What does this mean? Well, biologically, the individual may be genetically predisposed to

substance abuse; additionally, the individual may also be at risk due to their familial environment where their parents or siblings are also engaging in substance abuse. Individuals whose parents abuse substances may have a greater opportunity to ingest substances, thus promoting drug-seeking behaviors. Furthermore, families with a history of substance abuse may have a more accepting attitude of drug use than families with no history of substance abuse (Leventhal & Schmitz, 2006).

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

11.4.2. Cognitive

Cognitive theorists have focused on the beliefs regarding the anticipated effects of substance use. Defined as the *expectancy effect*, drug-seeking behavior is presumably motivated by the desire to attain a particular outcome by ingesting a substance. The expectancy effect can be defined in both positive and negative forms. Positive expectancies are thought to increase drug-seeking behavior, while negative expectancies would decrease substance use (Oei &

Morawska, 2004). Several studies have examined the expectancy effect on the use of alcohol. Those with alcohol abuse reported expectations of tension reduction, enhanced sexual experiences, and improved social pleasure (Brown, 1985). Additionally, observing positive experiences, both in person and through television or social media, also shapes our drug use expectancies.

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

11.4.3. Behavioral

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

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

Negative reinforcement, or the increase of a given behavior due to the removal of a negative effect, also plays a role in substance abuse in two different ways. First, many people

ingest a substance as an escape from their unpleasant life—whether it be physical pain, stress, or anxiety, to name a few. Therefore, the substance temporarily provides relief from a negative environment, thus reinforcing future substance abuse (Wise & Koob, 2013). Secondly, negative reinforcement is involved in symptoms of withdrawal. As previously mentioned, withdrawal from a substance often produces significant negative symptoms such as nausea, vomiting, uncontrollable shaking, etc. To eliminate these symptoms, an individual will consume more of the substance, thus again escaping the negative symptoms and enjoying the “highs” of the substance.

11.4.4. Sociocultural

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

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

families that promote religiosity may reduce substance use by promoting negative experiences (Galen & Rogers, 2004).

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

You should have learned the following in this section:

- Biological causes of substance-related and addictive disorders include the brain reward system and a genetic predisposition, though if the individual is not exposed to the substance they will not develop the substance abuse.
- Cognitive causes of substance-related and addictive disorders include the expectancy effect, and research provides stronger support for positive expectancy over negative expectancy.
- Behavioral causes of substance-related and addictive disorders include positive and negative reinforcement.
- Sociocultural causes of substance-related and addictive disorders include friends and the immediate environment.

Section 11.4 Review Questions

1. Discuss the *brain reward system*. What neurobiological regions are implicated within this system?
2. Define the *expectancy effect*. How does this explain the development and maintenance of substance abuse?
3. Discuss operant conditioning in the context of substance abuse. What are the reinforcers?
4. How does the sociocultural model explain substance abuse?

11.5. Treatment

Section Learning Objectives

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

Given the large number of the population 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).

11.5.1. Biological

11.5.1.1. Detoxification. Detoxification refers to the medical supervision of withdrawal from a specified drug. While most detoxification programs are inpatient for increased monitoring, some programs allow for outpatient detoxification, particularly if the addiction is not as severe. There are two main theories of detoxification—gradually decreasing the amount of the substance until the individual is off the drug completely, or eliminating the substance entirely while providing additional medications to manage withdrawal symptoms (Bisaga et al., 2015).

Unfortunately, relapse rates are high for those engaging in detoxification programs, particularly if they lack any follow-up psychological treatment.

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

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

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

11.5.2. Behavioral

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

11.5.2.2. Contingency management. Contingency management is a treatment approach that emphasizes *operant conditioning*—increasing sobriety and adherence to treatment programs through rewards. Originally developed to increase adherence to medication and reinforce opiate abstinence in methadone patients, contingency management has been adapted to increase abstinence in many different substance abuse treatment programs. In general, patients are “rewarded” with vouchers or prizes in exchange for abstinence from substance use (Hartzler, Lash, & Roll, 2012). These vouchers allow individuals to gain incentives specific to their

interests, thus increasing the chances of abstinence. Common vouchers include movie tickets, sports equipment, or even cash (Mignon, 2014).

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

11.5.3. Cognitive-Behavioral

11.5.3.1. Relapse prevention training. Relapse prevention training is essentially what it sounds like—identifying potentially high-risk situations for relapse and then learning behavioral skills and cognitive interventions to prevent the occurrence of a relapse. Early in treatment, the clinician guides the patient to identify any interpersonal, intrapersonal, environmental, and physiological risks for relapse. Once these triggers are identified, the clinician works with the patient on cognitive and behavioral strategies such as learning effective coping strategies, enhancing self-efficacy, and encouraging mastery of outcomes. Additionally, psychoeducation about how substance abuse is maintained, as well as identifying maladaptive thoughts and learning cognitive restructuring techniques, helps the patient make informed choices during high-risk situations. Finally, role-playing these high-risk situations in session allows patients to become comfortable engaging in these effective coping strategies that enhance their self-efficacy

and ultimately reducing the chances of a relapse. Research for relapse prevention training appears to be somewhat effective for individuals with substance-related disorders (Marlatt & Donovan, 2005).

11.5.4. Sociocultural

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

The overarching goal of AA is abstinence from alcohol. To achieve this, the participants are encouraged to “take one day at a time.” In using the 12 steps, participants are emboldened to admit that they have a disease, that they are powerless over this disease, and that their disease is more powerful than any person. Therefore, participants turn their addiction over to God and ask for help to right their wrongs and remove their negative character defects and shortcomings. The final steps include identifying and making amends to those who they have wronged during their alcohol abuse.

While studies examining the effectiveness of AA programs are inconclusive, AA’s membership indicates that 27% of its members have been sober less than one year, 24% have been sober 1-5 years, 13% have been sober 5-10 years, 14% have been sober 10-20 years, and

more than 22% have been sober over 20 years (Alcoholics Anonymous, 2014). Some argue that this type of treatment is most effective for those who are willing and able to abstain from alcohol as opposed to those who can control their drinking to moderate levels.

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

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

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

(Mignon, 2014). Essentially, the community around the patient reinforces the positive choices of abstaining from substance use.

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

You should have learned the following in this section:

- Biological treatment options for substance-related and addictive disorders include detoxification programs, agonist drugs, and antagonist drugs.
- Behavioral treatment options for substance-related and addictive disorders include aversion therapy and contingency management.
- Cognitive-behavioral treatment options for substance-related and addictive disorders include relapse prevention training.
- Sociocultural treatment options for substance-related and addictive disorders include Alcoholics Anonymous, residential treatment centers, and community reinforcement.

Section 11.5 Review Questions

1. Discuss the differences between agonist and antagonist drugs. Give examples of both.
2. What are the two behavioral treatments discussed in this module? Discuss their effectiveness.
3. What are the main components of the 12-step programs? How effective are they in substance abuse treatment?

Module Recap

And that concludes Part IV of the book and Block 3 of mental disorders. In this module, we discussed substance-related and addictive disorders to include substance use disorder, substance intoxication, and substance withdrawal. Substances include depressants, sedative-hypnotic drugs, opioids, stimulants, and hallucinogens. As in past modules, we discussed the clinical presentation, epidemiology, comorbidity, and etiology of the disorders. We then also discussed the biological, behavioral, cognitive-behavioral, and sociocultural treatment approaches.

Part V. Mental Disorders – Block 4

Disorders Covered:

- 12. Schizophrenia Spectrum and Other Psychotic Disorders
- 13. Personality Disorders

Part V. Mental Disorders – Block 4

Module 12: Schizophrenia Spectrum and Other Psychotic Disorders

Module 12: Schizophrenia Spectrum and Other Psychotic Disorders

Module Overview

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

Module Outline

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

Module Learning Outcomes

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

12.1. Clinical Presentation

Section Learning Objectives

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

12.1.1. The Clinical Presentation of Schizophrenia Spectrum Disorders

The schizophrenia spectrum and other psychotic disorders are defined by one of the following main symptoms: delusions, hallucinations, disorganized thinking (speech), disorganized or abnormal motor behavior, and negative symptoms. Individuals diagnosed with a schizophrenia spectrum disorder experience **psychosis**, which is defined as a loss of contact with reality. Psychosis episodes make it difficult for individuals to perceive and respond to environmental stimuli, causing a significant disturbance in everyday functioning. While there are a vast number of symptoms displayed in schizophrenia spectrum disorders, presentation of symptoms varies greatly among individuals, as there are rarely two cases similar in presentation, triggers, course, or responsiveness to treatment.

12.1.1.1. Delusions. **Delusions** are “fixed beliefs that are not amenable to change in light of conflicting evidence” (APA, 2022, pp. 101). This means that despite evidence contradicting one’s thoughts, the individual is unable to distinguish their thoughts from reality. The inability to

identify thoughts as delusional is likely likely due to a lack of insight. There are a wide range of delusions that are seen in the schizophrenia related disorders to include:

- ***Delusions of grandeur***- belief they have exceptional abilities, wealth, or fame; belief they are God or other religious saviors
- ***Delusions of control***- belief that others control their thoughts/feelings/actions
- ***Delusions of thought broadcasting***- belief that one's thoughts are transparent and everyone knows what they are thinking
- ***Delusions of persecution***- belief they are going to be harmed, harassed, plotted or discriminated against by either an individual or an institution; it is the most common delusion (Arango & Carpenter, 2010)
- ***Delusions of reference***- belief that specific gestures, comments, or even larger environmental cues are directed directly to them
- ***Delusions of thought withdrawal***- belief that one's thoughts have been removed by another source

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

12.1.1.2. Hallucinations. **Hallucinations** are “perception-like experiences that occur without an external stimulus” (APA, 2022, pg. 102). They can occur in any of the five senses: hearing (auditory hallucinations), seeing (visual hallucinations), smelling (olfactory hallucinations), touching (tactile hallucinations), and tasting (gustatory hallucinations).

Additionally, they can occur in a single modality or present across a combination of modalities

(e.g., having auditory and visual hallucinations). For the most part, individuals recognize that their hallucinations are not real and attempt to engage in normal behavior while simultaneously combating ongoing hallucinations.

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

12.1.1.3. Disorganized thinking (Speech). Among the most common cognitive impairments displayed in patients with schizophrenia are disorganized thoughts, communication, and speech. More specifically, thoughts and speech patterns may appear to be *circumstantial* or *tangential*. For example, patients may give unnecessary details in response to a question before they finally produce the desired response. While the question is eventually answered in circumstantial speech patterns, in tangential speech patterns the patient never reaches the point. Another common cognitive symptom is speech *incoherence* or word salad, where speech is “nearly incomprehensible and resembles receptive aphasia in its linguistic disorganization” (APA, 2022, pg. 102). *Derailment*, or the illogical connection in a chain of thoughts, is another common type of disorganized thinking. Although not always, derailment is often seen in *illogicality*, or the tendency to provide bizarre explanations for things.

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

12.1.1.4. Disorganized/abnormal motor behavior. These symptoms manifest as childlike “silliness” to unpredictable agitation. *Catatonic behavior*, the decreased or complete lack of reactivity to the environment, is among the most commonly seen grossly disorganized motor behavior in schizophrenia. There runs a range of catatonic behaviors from *negativism* (resistance to instruction); *mutism* or *stupor* (complete lack of verbal and motor responses); *rigidity* (maintaining a rigid or upright posture while resisting efforts to be moved); or *posturing* (holding odd, awkward postures for long periods). There is one type of catatonic behavior, *catatonic excitement*, where the individual experiences hyperactivity of motor behavior, in a seemingly excited or delirious way. Other features include repeated stereotyped movements, staring, grimacing, and the echoing of speech (APA, 2022, pg. 102).

12.1.1.5. Negative symptoms. Up until this point, all the symptoms can be categorized as **positive symptoms**, or symptoms that are an over-exaggeration of normal brain processes; these symptoms are also new to the individual. The final diagnostic criterion is **negative symptoms**, which are defined as the inability or decreased ability to initiate actions, speech, express emotion, or feel pleasure (Barch, 2013). Negative symptoms often present before positive symptoms and remain once positive symptoms remit. Because of their prevalence through the course of the disorder, they are also more indicative of prognosis, with more negative symptoms suggesting a poorer prognosis. The poorer prognosis may be explained by the lack of effectiveness antipsychotic medications have in addressing negative symptoms (Kirkpatrick,

Fenton, Carpenter, & Marder, 2006). There are six main types of negative symptoms seen in patients with schizophrenia. Such symptoms include:

- **Diminished emotional expression** - Reduction in emotional expression; reduced display of emotional expression
- **Alogia** - Poverty of speech or speech content
- **Anhedonia** - Inability to experience pleasure
- **Asociality** - Lack of interest in social relationships
- **Avolition** - Lack of motivation for goal-directed behavior

12.1.2. Schizophrenia

As stated above, the hallmark symptoms of **schizophrenia** include the presentation of at least two of the following during a one month period: delusions, hallucinations, disorganized speech, disorganized/abnormal behavior, or negative symptoms. These symptoms create significant impairment in an individual's ability to engage in normal daily functioning such as work, school, relationships with others, or self-care, and continuous signs of the disturbance persist for at least 6 months. It should be noted that the presentation of schizophrenia varies significantly among individuals, as it is a heterogeneous clinical syndrome (APA, 2022).

While the presence of symptoms must persist for a minimum of 6 months to meet the criteria for a schizophrenia diagnosis, it is not uncommon to have **prodromal** symptoms that precede the active phase of the disorder and **residual** symptoms that follow it. These prodromal and residual symptoms are “subthreshold” forms of psychotic symptoms that do not cause significant impairment in functioning, with the exception of negative symptoms (Lieberman et al., 2001). Due to the severity of psychotic symptoms, mood disorder symptoms are also

common among individuals with schizophrenia; however, these mood symptoms are distinct from a mood disorder diagnosis in that psychotic features will exist beyond the remission of depressive symptoms.

12.1.3. Schizophreniform Disorder

Schizophreniform disorder is similar to schizophrenia, except for the length of presentation of symptoms. Schizophreniform disorder is considered an “intermediate” disorder between schizophrenia and brief psychotic disorder as the symptoms are present for at least one month but *not* longer than six months. Schizophrenia symptoms must be present for at least six months and a brief psychotic disorder is diagnosed when symptoms are present for *less* than one month. Approximately two-thirds of individuals who are initially diagnosed with schizophreniform disorder will have symptoms that last longer than six months, at which time their diagnosis is changed to schizophrenia (APA, 2022).

Another key distinguishing feature of schizophreniform disorder is the lack of criteria related to impaired functioning. While many individuals with schizophreniform disorder do display impaired functioning, it is not essential for diagnosis. Finally, any major mood episodes—either depressive or manic— that are present concurrently with the psychotic features must only be present for a short time, otherwise a diagnosis of schizoaffective disorder may be more appropriate (APA, 2022).

Making Sense of the Disorders

In relation to schizophrenia spectrum and other psychotic disorders, note the following:

Diagnosis brief psychotic disorder if symptoms have been present for *less* than one month

Diagnosis schizophreniform disorder if symptoms have been present for at least one month but *not* longer than six months

Diagnosis schizophrenia ... if the symptoms have been present for at least six months

12.1.4. Schizoaffective Disorder

Schizoaffective disorder is characterized by the psychotic symptoms included in schizophrenia *and* a concurrent uninterrupted period of a major mood episode—either a major depressive or manic episode. It should be noted that because the loss of interest in pleasurable activities is a common symptom of schizophrenia, to meet the criteria for a depressive episode within schizoaffective disorder, the individual must present with a pervasive depressed mood (APA, 2022). While schizophrenia and schizophreniform disorder do *not* have a significant mood component, schizoaffective disorder requires the presence of a depressive or manic episode for the majority, if not the total duration of the disorder. While psychotic symptoms are sometimes present in depressive episodes, they often remit once the depressive episode is resolved. For individuals with schizoaffective disorder, psychotic symptoms should continue for at least two weeks in the absence of a major mood disorder (APA, 2022). This is the key distinguishing feature between schizoaffective disorder and major depressive disorder with psychotic features.

12.1.5. Delusional Disorder

As suggestive of its title, **delusional disorder** requires the presence of at least one delusion that lasts for at least one month in duration. It is important to note that if an individual experiences hallucinations, disorganized speech, disorganized or catatonic behavior, or negative symptoms—in addition to delusions—they should *not* be diagnosed with delusional disorder as their symptoms are more aligned with a schizophrenia diagnosis. Unlike most other schizophrenia-related disorders, daily functioning is not overly impacted due to the delusions. Additionally, if symptoms of depressive or manic episodes present during delusions, they are typically brief compared to the duration of the delusions.

The DSM-V-TR (APA, 2022) has identified five main subtypes of delusional disorder to better categorize the symptoms of the individual's disorder. When making a diagnosis of delusional disorder, one of the following modifiers (in addition to mixed presentation) is included. **Erotomaniac delusion** occurs when an individual reports a delusion of *another person* being in love with them. Generally speaking, the individual whom the convictions are about is of higher status, such as a celebrity. **Grandiose delusion** involves the conviction of having great talent or insight. Occasionally, patients will report they have made an important discovery that benefits the general public. Grandiose delusions may also take on religious affiliation, as people believe they are prophets or even God. **Jealous delusion** revolves around the conviction that one's spouse or partner is/has been unfaithful. While many individuals may have this suspicion at some point in their relationship, a jealous delusion is much more extensive and generally based on incorrect inferences that lack evidence. **Persecutory delusion** involves the individual believing that they are being conspired against, spied on, followed, poisoned or drugged, maliciously maligned, harassed, or obstructed in pursuit of their long-term goals (APA, 2022).

Of all subtypes of delusional disorder, those experiencing persecutory delusions are the most at risk of becoming aggressive or hostile, likely due to the persecutory nature of their distorted beliefs. Finally, **somatic delusion** involves delusions regarding bodily functions or sensations. While these delusions can vary significantly, the most common beliefs are that the individual emits a foul odor despite attempts to rectify the smell; there is an infestation of insects on the skin; or that they have an internal parasite (APA, 2022). If no one delusion predominates, the **mixed type** specifier is used and if the dominant delusional belief cannot be clearly determined, use the **unspecified type** specifier. A separate specifier is used when the content of the delusions are deemed **bizarre** or implausible, not understandable, and not derived from ordinary life experience.

You should have learned the following in this section:

- Schizophrenia spectrum disorders are characterized by delusions, hallucinations, disorganized thinking (speech), disorganized or abnormal motor behavior, and negative symptoms.
- Delusions are beliefs that do not change even when conflicting evidence is presented and can be of grandeur, control, thought broadcasting, persecution, reference, and thought withdrawal.
- Hallucinations occur in any sense modality and most individuals recognize that they are not real.
- Disorganized thinking, abnormal motor behavior, catatonic behavior, and negative symptoms such as affective flattening, alogia, anhedonia, asociality, and avolition are also common to schizophrenia spectrum disorders.
- Schizophrenia is characterized by delusions, hallucinations, disorganized speech, disorganized/abnormal behavior, or negative symptoms lasting six months.
- Schizophreniform disorder is considered an “intermediate” disorder between schizophrenia and brief psychotic disorder as the symptoms are present for at least one month but not longer than six months.
- Schizoaffective disorder is characterized by the psychotic symptoms included in schizophrenia and a concurrent uninterrupted period of a major mood episode—either a depressive or manic episode.
- Delusional disorder requires the presence of at least one delusion that lasts for at least one month in duration to include erotomanic, grandiose, jealous, persecutory, and somatic.

Section 12.1 Review Questions

1. What are the four positive symptoms identified in a schizophrenia diagnosis? Define and identify their difference.
2. What is meant by negative symptoms? What are the negative symptoms observed in schizophrenia related disorders?
3. Identify diagnostic differences between schizophrenia, schizophreniform, schizoaffective, and delusional disorders.

12.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of schizophrenia spectrum disorders.

Schizophrenia occurs in approximately 0.3%-0.7% of the general population (APA, 2022). There is some discrepancy in rates of diagnosis between genders; these differences appear to be related to the emphasis of various symptoms. For example, men typically present with more negative symptoms, whereas women present with more affect-laden symptoms. Despite gender differences in the presentation of symptoms, there appears to be an equal risk for both genders to develop the disorder.

Schizophrenia typically occurs between late teens and mid-30s, with the onset of the disorder slightly earlier for males than females (APA, 2022). Earlier onset of the disorder is generally predictive of a worse overall prognosis. Onset of symptoms is typically gradual, with initial symptoms presenting similarly to depressive disorders; however, some individuals will present with an abrupt presentation of the disorder. Negative symptoms appear to be more predictive of prognosis than other symptoms. This may be due to negative symptoms being the most persistent, and therefore, most difficult to treat. Overall, an estimated 13.5% of individuals diagnosed with schizophrenia meet recovery criteria, according to one meta-analysis of 50 studies of individuals with broadly defined schizophrenia (APA, 2022).

Schizoaffective disorder, schizophreniform disorder, and delusional disorder prevalence rates are all significantly less than that of schizophrenia, occurring in 0.2% to 0.3% of the general population. While schizoaffective disorder is diagnosed more in females than males

(similar to schizophrenia but using the less stringent DSM-IV criteria), schizophreniform and delusional disorder appear to be diagnosed equally between genders (APA, 2022).

You should have learned the following in this section:

- Less than 1% of the general population is diagnosed with schizophrenia and 13.5% of these people fully recover from the disorder.
- Both genders have an equal risk of developing schizophrenia while men typically display more negative symptoms while women present with more affect-laden symptoms.
- Schizoaffective disorder, schizophreniform disorder, and delusional disorder have prevalence rates between 0.2 to 0.3%.

Section 12.2 Review Questions

1. Discuss the different prevalence rates across the schizophrenia related disorders. Are there differences among the disorders? Between genders?
2. Are there differences in prevalence rates depending on symptom presentations? If so, what?

12.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of schizophrenia spectrum disorders.

There is a high comorbidity between schizophrenia and substance abuse disorder and there is some evidence to suggest that the use of various substances (particularly marijuana) may place an individual at an increased risk of developing schizophrenia if the genetic predisposition is also present (see diathesis-stress model below; Corcoran et al., 2003). Additionally, there appears to be comorbidity with anxiety-related disorders, specifically panic disorder, and obsessive-compulsive disorder, among individuals with schizophrenia than compared to the general public. Schizotypal or paranoid personality disorder sometimes precede the onset of schizophrenia. About 5-6% of individuals diagnosed with schizophrenia die by suicide, about 20% have attempted suicide on at least one occasion, and many more have significant suicidal ideation.

It should also be noted that individuals diagnosed with a schizophrenia-related disorder are also at an increased risk for associated medical conditions such as weight gain, diabetes, metabolic syndrome, and cardiovascular and pulmonary disease (APA, 2022). This predisposition to various medical conditions is likely related to medications and poor lifestyle choices, and also place individuals at risk for a reduced life expectancy.

Schizoaffective disorder is comorbid with substance use disorders and anxiety disorders. Metabolic syndrome occurs at a higher rate than for the general population as well.

Comorbidity information is not given for delusional disorder or schizophreniform disorder.

You should have learned the following in this section:

- Schizophrenia has a high comorbidity with substance abuse disorders, anxiety-related disorders, OCD, and some medical conditions.
- Schizoaffective disorder is comorbid with substance use disorders, anxiety disorder, and metabolic syndrome.

Section 12.3 Review Questions

1. What comorbidities exist between schizophrenia spectrum and other psychotic disorders?

12.4. Etiology

Section Learning Objectives

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

12.4.1. Biological

12.4.1.1. Genetic/Family studies. Twin and family studies consistently support the biological theory. More specifically, if one identical twin develops schizophrenia, there is a 48% chance that the other will also develop the disorder within their lifetime (Coon & Mitter, 2007). This percentage drops to 17% in fraternal twins. Similarly, family studies have also found similarities in brain abnormalities among individuals with schizophrenia and their relatives; the more similarities, the higher the likelihood that the family member also developed schizophrenia (Scognamiglio & Houenou, 2014).

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

within the medial temporal lobe (Kurtz, 2015). Additional studies also indicate a reduction in the orbitofrontal regions of the brain, a part of the frontal lobe that is responsible for response inhibition (Kurtz, 2015).

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

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

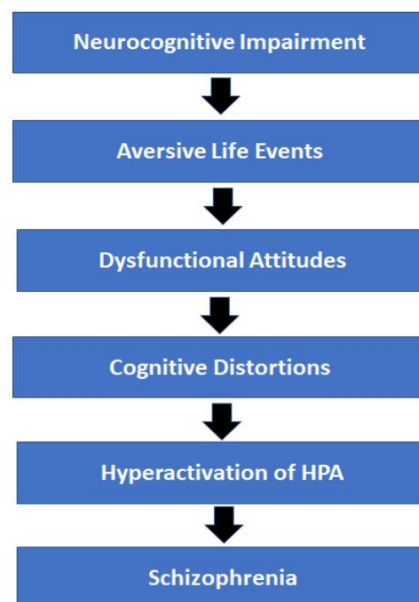
It is hypothesized that in combination with abnormal brain structures, persistently increased levels of glucocorticoids in brain structures may be the key to the onset of psychosis in prodromal patients (Corcoran et al., 2003). More specifically, stress exposure (and increased glucocorticoids) affects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

or exacerbation of symptoms is also a source of stress as they interfere with daily functioning (Walker & Diforio, 1997). This stress alone may be enough to initiate the onset of a relapse.

12.4.2. Psychological

12.4.2.1. Cognitive. The cognitive model utilizes some of the aspects of the diathesis-stress model in that it proposes that premorbid neurocognitive impairment places individuals at risk for aversive work/academic/interpersonal experiences. These experiences, in turn, lead to dysfunctional beliefs and cognitive appraisals, ultimately leading to maladaptive behaviors such as delusions/hallucinations (Beck & Rector, 2005). Beck proposed the following diathesis-stress model for how schizophrenia develops (See Figure 12.1).

Figure 12.1. Diathesis-Stress Model of the Development of Schizophrenia



Adapted from Beck & Rector, 2005, pg. 580

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

12.4.3. Sociocultural

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

12.4.3.2. Family dysfunction. Even for families with low levels of expressed emotion, there is often an increase in family stress due to the secondary effects of schizophrenia. Having a family member with schizophrenia increases the likelihood of a disruptive family environment due to managing the patient's symptoms and ensuring their safety while they are home (Friedrich

et al., 2015). Because of the severity of symptoms, families with a loved one diagnosed with schizophrenia often report more conflict in the home as well as more difficulty communicating with one another (Kurtz, 2015).

You should have learned the following in this section:

- Biological causes of schizophrenia spectrum and other psychotic disorders include genetics, several brain structures, and the HPA axis.
- Psychological causes of schizophrenia spectrum disorders include the diathesis-stress model.
- Sociocultural causes of schizophrenia spectrum disorders include families high in expressed emotion and family dysfunction.

Section 12.4 Review Questions

1. What evidence is there to support a biological model with respect to explaining the development and maintenance of the schizophrenia spectrum and other psychotic disorders?
2. Discuss the stress-vulnerability model with respect to schizophrenia spectrum and other psychotic disorders.
3. How does the sociocultural model explain the maintenance (and relapse) of schizophrenia related symptoms?

12.5. Treatment

Section Learning Objectives

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

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

12.5.1. Psychopharmacological

Among the first antipsychotic medications used for the treatment of schizophrenia was Thorazine. Developed as a derivative of antihistamines, Thorazine was the first line of treatment that produced a calming effect on even the most severely agitated patients and allowed for the organization of thoughts. Despite their effectiveness in managing psychotic symptoms, *conventional* antipsychotics (such as Thorazine and Chlorpromazine) also produced significant side effects similar to that of neurological disorders. Therefore, psychotic symptoms were

replaced with muscle tremors, involuntary movements, and muscle rigidity. Additionally, these conventional antipsychotics also produced **tardive dyskinesia** in patients, which included involuntary movements isolated to the tongue, mouth, and face (Tenback et al., 2006). While only 10% of patients reported the development of tardive dyskinesia, this percentage increased the longer patients were on the medication, as well as the higher the dose (Achalia, Chaturvedi, Desai, Rao, & Prakash, 2014). In efforts to avoid these symptoms, clinicians have been cognizant of not exceeding the clinically effective dose of conventional antipsychotic medications. If the management of psychotic symptoms cannot be resolved at this level, alternative medications are often added to produce a synergistic effect (Roh et al., 2014).

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

It should be noted that because of the harsh side effects of antipsychotic medications in general, many individuals, nearly one half to three-quarters of patients, discontinue the use of antipsychotic drugs (Leucht, Heres, Kissling, & Davis, 2011). Because of this, it is also important to incorporate psychological interventions along with psychopharmacological

treatment to both address medication adherence, as well as provide additional support for symptom management.

12.5.2. Psychological Interventions

12.5.2.1. Cognitive Behavioral Therapy (CBT). As discussed in previous chapters, the goal of treatment is to identify the negative biases and attributions that influence an individual's interpretations of events and the subsequent consequences of these thoughts and behaviors. For schizophrenia, CBT focuses on the maladaptive emotional and behavioral responses to psychotic experiences, which is directly related to distress and disability. Therefore, the goal of CBT is not on symptom reduction, but rather to improve the interpretations and understandings of these symptoms (and experiences) which will reduce associated distress (Kurtz, 2015). Common features of CBT for schizophrenia patients include psychoeducation about their disease and the course of their symptoms (i.e., ways to identify coming and going of delusions/hallucinations), challenging and replacing the negative thoughts/behaviors associated with their delusions/hallucinations to more positive thoughts/behaviors, and finally, learning positive coping strategies to deal with their unpleasant symptoms (Veiga-Martinez, Perez-Alvarez, & Garcia-Montes, 2008).

Findings from studies exploring CBT as a supportive treatment have been promising. One study conducted by Aaron Beck (the founder of CBT) and colleagues (Grant, Huh, Perivoliotis, Stolar, & Beck, 2011) found that recovery-oriented CBT produced a marked improvement in overall functioning as well as symptom reduction in patients diagnosed with schizophrenia. This study suggests that by focusing on targeted goals such as independent living, securing employment, and improving social relationships, patients were able to slowly move

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

12.5.3. Family Interventions

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

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

Psychoeducation is important for both the patient and family members as it is reported that more than half of those recovering from a psychotic episode reside with their family (Haddock & Spaulding, 2011). Therefore, educating families on the course of the illness, as well as ways to recognize onset of psychotic symptoms, is important to ensure optimal recovery.

Problem-solving is a crucial component in the family intervention model. Seeing as family conflict can increase stress within the home, which in return can lead to worsening of psychotic symptoms, family members benefit from learning effective methods of problem-solving to

address family conflicts. Additionally, teaching positive coping strategies for dealing with the symptoms of mental illness and its direct effect on the family environment may also alleviate some friction within the home

The third component, *CBT*, is similar to that described above. The goal of family-based CBT is to reduce negativity among family member interactions, as well as help family members adjust to living with someone with psychotic symptoms. These three components within the family intervention program have been shown to reduce re-hospitalization rates, as well as slow the worsening of schizophrenia-related symptoms (Pitschel-Walz, Leucht, Baumi, Kissling, & Engel, 2001).

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

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

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

You should have learned the following in this section:

- Psychopharmacological treatment options for schizophrenia spectrum disorders include antipsychotic drugs such as Thorazine, Chlorpromazine, Clozaril, Risperdal, and Abilify.
- Psychological treatment options for schizophrenia spectrum disorders include CBT, the goal of which is to improve the interpretations and understandings of symptoms (and experiences) which will reduce associated distress.
- Family interventions for schizophrenia spectrum disorders include psychoeducation, problem-solving skills, cognitive-behavioral therapy (CBT), social skills training, and inpatient/partial hospitalizations.

Section 12.5 Review Questions

1. Define tardive dyskinesia.
2. What pharmacological interventions have been effective in managing schizophrenia related disorder symptoms?
3. What is the main goal of family interventions? How is this achieved?

Module Recap

In our first module of Part V – Block 4, we discussed the schizophrenia spectrum and other psychotic disorders to include schizophrenia, schizophreniform disorder, schizoaffective disorder, and delusional disorder. We started by describing their common features, such as delusions, hallucinations, disorganized thinking, disorganized/abnormal motor behavior, and negative symptoms. This led to a discussion of the epidemiology, comorbidity, etiology, and treatment options for the disorders.

Part V. Mental Disorders – Block 4

Module 13: Personality Disorders

Module 13: Personality Disorders

Module Overview

In Module 13, we will cover matters related to personality disorders to include their clinical presentation, epidemiology, comorbidity, etiology, and treatment options. Our discussion will include Cluster A disorders of paranoid, schizoid, and schizotypal; Cluster B disorders of antisocial, borderline, histrionic, and narcissistic; and Cluster C personality disorders of avoidant, dependent, and obsessive-compulsive. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the therapies (Module 3).

Module Outline

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

Module Learning Outcomes

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

13.1. Clinical Presentation

Section Learning Objectives

- Define personality trait.
- Define personality disorder.
- List the defining features of personality disorders.
- Describe the three clusters.
- Describe how paranoid personality disorder presents.
- Describe how schizoid personality disorder presents.
- Describe how schizotypal personality disorder presents.
- Describe how antisocial personality disorder presents.
- Describe how borderline personality disorder presents.
- Describe how histrionic personality disorder presents.
- Describe how narcissistic personality disorder presents.
- Describe how avoidant personality disorder presents.
- Describe how dependent personality disorder presents.
- Describe how obsessive-compulsive personality disorder presents.

13.1.1. Overview of Personality Disorders

According to the DSM-5-TR, **personality traits** are “...enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personality contexts” while a **personality disorder** “...is an enduring pattern of inner experience and behavior that deviates markedly from the norms and expectations of the

individual's culture, is pervasive and inflexible, and has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment" (APA, 2022, pg. 733).

Personality disorders have four defining features, which include distorted thinking patterns, problematic emotional responses, over- or under-regulated impulse control, and interpersonal difficulties. While these four core features are universal among all ten personality disorders, the DSM-5-TR divides the personality disorders into three different clusters based on symptom similarities.

Cluster A is described as the odd or eccentric cluster and consists of paranoid, schizoid, and schizotypal personality disorders. The common feature between these three disorders is social awkwardness and social withdrawal. Often these behaviors are similar to those seen in schizophrenia; however, they tend to be not as extensive or impactful on daily functioning as seen in schizophrenia. In fact, there is a strong relationship between Cluster A personality disorders among individuals who have a relative diagnosed with schizophrenia (Chemerinksi & Siever, 2011).

Cluster B is the dramatic, emotional, or erratic cluster and consists of antisocial, borderline, histrionic, and narcissistic personality disorders. Individuals with these personality disorders often experience problems with impulse control and emotional regulation. Due to the dramatic, emotional, and erratic nature of these disorders, it is nearly impossible for individuals to establish healthy relationships with others.

And finally, **Cluster C** is the anxious or fearful cluster and consists of avoidant, dependent, and obsessive-compulsive personality disorders. As you read through the descriptions of the disorders, you will see an overlap with symptoms from the anxiety and depressive disorders. Cluster C disorders have the most treatment options of all the personality disorders,

likely because the overlapping anxiety and depressive disorders have well-established treatment options.

To meet the criteria for any personality disorder, the individual must display the pattern of behaviors in adulthood. Children cannot be diagnosed with a personality disorder. Some children may present with similar symptoms, such as poor peer relationships, odd or eccentric behaviors, or peculiar thoughts and language; however, a formal personality disorder diagnosis cannot be made until the age of 18. The DSM-5-TR reports that median prevalence across several countries is 3.6% for Cluster A disorders, 4.5% for Cluster B, 2.8% for Cluster C, and 10.5% for any personality disorder.

It is also noted that the clustering approach used in the DSM has not been consistently validated and has some serious limitations. As written, “An alternative to the categorical approach is the dimensional perspective that personality disorders represent maladaptive variants of personality traits that merge imperceptibly into normality and into one another” (APA, 2022, pg. 734). Interested readers should consult Section III of the DSM (beginning on page 881) for a full description of the dimensional model for personality disorders and an alternative model for personality disorders that utilizes a hybrid dimensional-categorical model approach.

13.1.2. Cluster A

13.1.2.1. Paranoid personality disorder. Paranoid personality disorder is characterized by a marked distrust or suspicion of others. Individuals interpret and believe that other’s motives and interactions are intended to harm them, and therefore, they are skeptical about establishing close relationships outside of family members—although, at times, even family members’ actions are also believed to be malevolent (APA, 2022). Individuals with

paranoid personality disorder often feel as though they have been deeply and irreversibly hurt by others even though they lack evidence to support that these others intended to or did hurt them. Because of these persistent suspicions, they will doubt relationships that show true loyalty or trustworthiness. Compliments are misinterpreted and they may view an offer of help as a criticism that they are not doing a good enough job on their own.

Individuals with paranoid personality disorder are also hesitant to share any personal information or confide in others as they fear the information will be used against them. Additionally, benign remarks or events are often viewed as demeaning or threatening. For example, if an individual with paranoid personality disorder was accidentally bumped into at the store, they would interpret this action as intentional, with the purpose of causing them injury. Because of this, individuals with paranoid personality disorder are quick to hold grudges and unwilling to forgive insults or injuries- whether intentional or not. They are known to quickly and angrily counterattack, either verbally or physically, in situations where they feel they were insulted (APA, 2022).

13.1.2.2. Schizoid personality disorder. Individuals with **schizoid personality disorder** display a persistent pattern of avoidance of social relationships, along with a limited range of emotional expression in interpersonal settings (APA, 2022). Similar to those with paranoid personality disorder, individuals with schizoid personality disorder do not have many close relationships; however, unlike paranoid personality disorder, this lack of connection is not due to suspicious feelings, but rather, the lack of desire to engage with others and the preference to engage in solitary behaviors. Individuals with schizoid personality disorder are often viewed as “loners” and prefer activities where they do not have to engage with others (APA, 2022). Established relationships rarely extend outside that of the family as they make no effort to start

or maintain friendships. This lack of establishing social relationships also extends to sexual behaviors, as these individuals report a lack of interest in engaging in sexual experiences with others.

Regarding the limited range of emotion, individuals with schizoid personality disorder are often indifferent to criticisms or praises of others and appear not to be affected by what others think of them. Individuals will rarely show any feelings or expressions of emotion and are often described as having a “bland” exterior (APA, 2022). In fact, individuals with schizoid personality disorder rarely reciprocate facial expressions or gestures typically displayed in normal conversations such as smiles or nods. Because of this lack of emotion, there is a limited need for attention or acceptance.

13.1.2.3. Schizotypal personality disorder. Schizotypal personality disorder is characterized by a range of impairment in social and interpersonal relationships due to discomfort in relationships, along with odd cognitive or perceptual distortions and eccentric behaviors (APA, 2022). Similar to those with schizoid personality disorder, individuals also seek isolation and have few, if any established relationships outside of family members.

One of the most prominent features of schizotypal personality disorder is **ideas of reference**, or the belief that unrelated events pertain to them in a particular and unusual way. Ideas of reference also lead to superstitious behaviors or preoccupation with paranormal activities that are not generally accepted in their culture (APA, 2022). The perception of special or magical powers, such as the ability to mind-read or control other’s thoughts, has also been documented in individuals with schizotypal personality disorder. Similar to schizophrenia, unusual perceptual experiences such as auditory hallucinations, as well as unusual speech patterns of derailment or incoherence, are also present.

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

13.1.3. Cluster B

13.1.3.1. Antisocial personality disorder. The essential feature of **antisocial personality disorder** is the persistent pattern of disregard for, and violation of, the rights of others. This pattern of behavior begins in late childhood or early adolescence and continues throughout adulthood. While this behavior presents before age 15, the individual cannot be diagnosed with antisocial personality disorder until the age of 18. Prior to age 18, the individual would be diagnosed with *conduct disorder*. Although not discussed in this book as it is a disorder of childhood, conduct disorder involves a repetitive and persistent pattern of behaviors that violate the rights of others or major age-appropriate norms. Common behaviors of individuals with conduct disorder that go on to develop antisocial personality disorder are aggression toward people or animals, destruction of property, deceitfulness or theft, or serious violation of rules (APA, 2022).

While commonly referred to as “psychopaths” or “sociopaths,” individuals with antisocial personality disorder fail to conform to social norms. This also includes legal rules as

individuals with antisocial personality disorder are often repeatedly arrested for property destruction, harassing/assaulting others, or stealing (APA, 2022). Deceitfulness is another hallmark symptom of antisocial personality disorder as individuals often lie repeatedly, generally to gain profit or pleasure. There is also a pattern of impulsivity—decisions made in the moment without forethought of personal consequences or consideration for others (Lang et al., 2015). This impulsivity also contributes to their inability to hold jobs as they are more likely to impulsively quit their jobs (Hengartner et al., 2014). Employment instability, along with impulsivity, also impacts their ability to manage finances; it is not uncommon to see individuals with antisocial personality disorder with large debts that they are unable to pay (Derefinko & Widiger, 2016).

While also likely related to impulsivity, individuals with antisocial personality disorder tend to be extremely irritable and aggressive, repeatedly getting into fights. The marked disregard for their safety, as well as the safety of others, is also observed in reckless behavior such as speeding, driving under the influence, and engaging in sexual and substance abuse behavior that may put themselves at risk (APA, 2022).

Of course, the most known and devastating symptom of antisocial personality disorder is the lack of remorse for the consequences of their actions, regardless of how severe they may be. Individuals often rationalize their actions as the fault of the victim, minimize the harmfulness of the consequences of their behaviors, or display indifference (APA, 2022). Overall, individuals with antisocial personality disorder have limited personal relationships due to their selfish desire and lack of moral conscience.

13.1.3.2. Borderline personality disorder. Individuals with **borderline personality disorder** display a pervasive pattern of instability in interpersonal relationships, self-image, and

affect (APA, 2022). The combination of these symptoms causes significant impairment in establishing and maintaining personal relationships. They will often go to great lengths to avoid real or imagined abandonment. Fears related to abandonment can lead to inappropriate anger as they often interpret the abandonment as a reflection of their own behavior. It is not uncommon to experience intense fluctuations in mood, often observed as volatile interactions with family and friends (Herpertz & Bertsch, 2014). Those with borderline personality disorder may be friendly one day and hostile the next.

To prevent abandonment, individuals with borderline personality disorder will often exhibit impulsive behaviors such as self-harm and suicidal behavior. In fact, individuals with borderline personality disorder engage in more suicide attempts, and completion of suicide is higher among these individuals than the general public (Linehan et al., 2015). Other impulsive behaviors, such as non-suicidal self-injury (cutting) and sexual promiscuity, are frequently seen within this population, typically occurring during high-stress periods (Sansone & Sansone, 2012). They often have chronic feelings of emptiness along with painful feelings of aloneness. Occasionally, hallucinations and delusions are present, particularly of a paranoid nature; however, these symptoms are often transient and recognized as unacceptable by the individual (Sieswerda & Arntz, 2007).

13.1.3.3. Histrionic personality disorder. **Histrionic personality disorder** is the first personality disorder that addresses pervasive and excessive emotionality and attention-seeking. These individuals are usually uncomfortable in social settings *unless* they are the center of attention. To help gain attention, the individual is often vivacious and dramatic, using physical gestures and mannerisms along with grandiose language. These behaviors are initially very charming to their audience; however, they begin to wear due to the constant need for attention to

be on them. If the theatrical nature does not gain the attention they desire, they may go to great lengths to draw attention, such as using a fictitious story or creating a dramatic scene.

To ensure they gain the attention they desire, individuals with histrionic personality disorder frequently dress and engage in sexually seductive or provocative ways. These sexually charged behaviors are not only directed at those in which they have a sexual or romantic interest but to the general public as well (APA, 2022). They often spend a significant amount of time on their physical appearance to gain the attention they desire.

Individuals with histrionic personality disorder are easily suggestible. Their opinions and feelings are influenced by not only their friends but also by current fads (APA, 2022). They also tend to exaggerate relationships, considering casual acquaintanceships as more intimate than they are.

13.1.3.4. Narcissistic personality disorder. Like histrionic personality disorder, **narcissistic personality disorder** also centers around the individual; however, with narcissistic personality disorder, individuals display a pattern of grandiosity along with a lack of empathy for others (APA, 2022). The grandiose sense of self leads to an overvaluation of their abilities and accomplishments. They often come across as boastful and pretentious, repeatedly proclaiming their superior achievements. These proclamations may also be fantasized to enhance their success or power. Oftentimes they identify themselves as “special” and will only interact with others of high status.

Given the grandiose sense of self, it is not surprising that individuals with narcissistic personality disorder need excessive admiration from others. While it appears that their self-esteem is hugely inflated, it is very fragile and dependent on how others perceive them (APA, 2022). Because of this, they may constantly seek out compliments and expect favorable

treatment from others. When this sense of entitlement is not upheld, they can become irritated or angry that their needs are not met.

A lack of empathy is also displayed in individuals with narcissistic personality disorder as they often struggle to (or choose not to) recognize the desires or needs of others. This lack of empathy also leads to exploitation of interpersonal relationships, as they are unable to understand other's feelings (Marcoux et al., 2014). They often become envious of others who achieve greater success or possessions than them. Conversely, they believe everyone should be envious of their achievements, regardless of how small they may be.

13.1.4. Cluster C

13.1.4.1. Avoidant personality disorder. Individuals with **avoidant personality disorder** display a pervasive pattern of social inhibition due to feelings of inadequacy and increased sensitivity to negative evaluations (APA, 2022). The fear of being rejected drives their reluctance to engage in social situations so that they may prevent others from evaluating them negatively. This fear extends so far that it prevents individuals from maintaining employment due to their intense fear of negative evaluation or rejection.

Socially, they have very few if any friends, despite their desire to establish social relationships. They actively avoid social situations in which they can develop new friendships out of the fear of being disliked or ridiculed. Similarly, they are cautious of new activities or relationships as they often exaggerate the potential negative consequences and embarrassment that may occur; this is likely a result of their ongoing preoccupation with being criticized or rejected by others. Within intimate relationships, their fear of being shamed or ridiculed leads to restraint, and they view themselves as socially inept (APA, 2022).

Making Sense of the Disorders

As you read the clinical description of avoidant personality disorder, did you think it sounded a lot like social anxiety disorder? You likely did as there is a great deal of overlap between the two disorders. So, how do they differ if they are to be regarded as separate diagnostic categories in the DSM? This difference is linked to self-concept. How so?

- In social anxiety disorder the negative self-concept is unstable and less pervasive and entrenched.
- In avoidant personality disorder, the negative self-concept is more stable as an enduring and pervasive pattern, typical of personality traits.

Additionally, avoidant personality disorder frequently occurs in the absence of social anxiety disorder and some separate risk factors have been identified for the two.

13.1.4.2. Dependent personality disorder. **Dependent personality disorder** is characterized by pervasive and excessive need to be taken care of by others (APA, 2022). This intense need leads to submissive and clinging behaviors as they fear they will be abandoned or separated from their parent, spouse, or another person with whom they are in a dependent relationship. They are so dependent on this other individual that they cannot make even the smallest decisions without first consulting with them and gaining their approval or reassurance. They often allow others to assume complete responsibility for their life, making decisions in nearly all aspects of their lives. Rarely will they challenge these decisions as their fear of losing this relationship greatly outweighs their desire to express their own opinion. Should the relationship end, the individual experiences significant feelings of helplessness and quickly seeks out another relationship to replace the old one (APA, 2022).

When they are on their own, individuals with dependent personality disorder express difficulty initiating and engaging in tasks on their own. They lack self-confidence and feel helpless when they are left to care for themselves or engage in tasks on their own. So that they do not have to engage in tasks alone, individuals will go to great lengths to seek out support of others, often volunteering for unpleasant tasks if it means they will get the reassurance they need (APA, 2022).

13.1.4.3. Obsessive-compulsive personality disorder (OCPD). OCPD is defined by an individual's preoccupation with orderliness, perfectionism, and ability to control situations that they lose flexibility, openness, and efficiency in everyday life (APA, 2022). One's preoccupation with details, rules, lists, order, organization, or schedules overshadows the larger picture of the task or activity. In fact, the need to complete the task or activity is significantly impacted by the individual's self-imposed high standards and need to complete the task perfectly, that the task often does not get completed. The desire to complete the task perfectly often causes the individual to spend an excessive amount of time on the task, occasionally repeating it until it is to their standard. Due to repetition and attention to fine detail, the individual often does not have time to engage in leisure activities or engage in social relationships. Despite the excessive amount of time spent on activities or tasks, individuals with OCPD will not seek help from others, as they are convinced that the others are incompetent and will not complete the task up to their standard.

Personally, individuals with OCD are rigid and stubborn, particularly with their morals, ethics, and values. Not only do they hold these standards for themselves, but they also expect others to have similarly high standards, thus causing significant disruption to their social interactions. The rigid and stubborn behaviors are also seen in their financial status, as they are

known to live significantly below their means to prepare financially for a potential catastrophe (APA, 2022). Similarly, they may have difficulty discarding worn-out or worthless items, despite their lack of sentimental value.

Though on the surface it may appear that OCPD and OCD are one and the same, there is a distinct difference as the personality disorder lacks definitive obsessions and compulsions (APA, 2022). In fact, most individuals with OCD do not have a pattern of behavior that meets criteria for this personality disorder.

You should have learned the following in this section:

- Personality disorders share the features of distorted thinking patterns, problematic emotional responses, over- or under-regulated impulse control, and interpersonal difficulties and divide into three clusters.
- Cluster A personality disorders are described as the odd/eccentric cluster and share as the common feature social awkwardness and social withdrawal. It consists of paranoid, schizoid, and schizotypal personality disorders.
- Cluster B personality disorders are described as the dramatic, emotional, or erratic cluster and consists of antisocial, borderline, histrionic, and narcissistic personality disorders.
- Cluster C is the anxious/fearful cluster and consists of avoidant, dependent, and obsessive-compulsive personality disorders.
- Paranoid personality disorder is characterized by a marked distrust or suspicion of others.
- Schizoid personality disorder is characterized by a persistent pattern of avoidance of social relationships, along with a limited range of emotion among social relationships.
- Schizotypal personality disorder is characterized by a range of impairment in social and interpersonal relationships due to discomfort in relationships, along with odd cognitive or perceptual distortions and eccentric behaviors.
- Antisocial personality disorder is characterized by a persistent pattern of disregard for, and violation of, the rights of others. They show no remorse for their behavior.

- Borderline personality disorder is characterized by a pervasive pattern of instability in interpersonal relationships, self-image, and affect.
- Histrionic personality disorder is characterized by pervasive and excessive emotionality and attention-seeking.
- Narcissistic personality disorder is characterized by a pattern of grandiosity along with a lack of empathy for others.
- Avoidant personality disorder is characterized by a pervasive pattern of social anxiety due to feelings of inadequacy and increased sensitivity to negative evaluations.
- Dependent personality disorder is characterized by pervasive and excessive need to be taken care of by others.
- OCPD is characterized by an individual's preoccupation with orderliness, perfectionism, and the ability to control situations that they lose flexibility, openness, and efficiency in everyday life.

Section 13.1 Review Questions

1. What are personality traits and how do they lead to personality disorders?
2. What are the three clusters? How are disorders grouped into these three clusters? Discuss the differences in symptom presentation between the three personality clusters.
3. Create a chart identifying each of the disorders among the three clusters. Be sure to include personality characteristics of each disorder. It is important to find characteristics unique to each personality disorder to aid in their identification.

13.2. Epidemiology

Section Learning Objectives

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

13.2.1. Cluster A

Disorders within Cluster A have a prevalence rate of around 2-5%. More specifically, according to Part II of the National Comorbidity Survey Replication, the estimated prevalence of paranoid personality disorder was 2.3%, schizoid personality disorder was 4.9%, and schizotypal personality disorder was 3.3%. Schizotypal personality disorder has been found to be more common in men while research on schizoid personality disorder leans to no gender difference in prevalence. As for paranoid personality disorder, it appears to be more common in men though the National Epidemiologic Survey on Alcohol and Related conditions found it to be more common in women (APA, 2022).

13.2.2. Cluster B

Using Part II of the National Comorbidity Survey Replication, it was found that for Cluster B personality disorders prevalence rates were: 0.6% for antisocial, 1.4% for borderline, 0.0% for histrionic, and 0.0% for narcissistic. It should be noted that the prevalence of histrionic personality disorder was 1.8% and narcissistic was 6.2% in the National Epidemiologic Survey on Alcohol and Related Conditions.

As for sex-and gender-related differences, antisocial personality disorder is three times more common in men and they present with irritability/aggression and reckless disregard for the safety of others more often than women. Borderline personality disorder is more common among women in clinical samples while community samples show no difference in prevalence, likely due to the tendency of women to seek help leading them to clinical settings. Histrionic personality disorder is more predominant in females in clinical settings, though some studies using structured assessments point to no difference in prevalence rates across the genders. Narcissistic personality disorder occurs more in men than women.

13.2.3. Cluster C

Using Part II of the National Comorbidity Survey Replication, it was found that for Cluster C personality disorders prevalence rates were: 5.2% for avoidant, 0.6% for dependent, and 2.4% for OCPD. Women are more likely to be diagnosed with avoidant and dependent personality disorders while OCPD appears to be equally prevalent in women and men.

For expanded information on the prevalence of the various personality disorders from the DSM-5-TR, please see Table 13. 1 below.

Table 13.1. Comparing Prevalence Rates by Data Source

Personality Disorder	Part II of NCSR ²	NESARC ³	International ⁴
Paranoid	2.3 ¹	4.4	3.2
Schizoid	4.9	3.1	1.3
Schizotypal	3.3	3.9	0.6
Antisocial	0.6	3.6	3.6
Borderline	1.4	5.9	2.7
Histrionic	0.0	1.8	0.9
Narcissistic	0.0	6.2	1.6
Avoidant	5.2	2.4	2.1
Dependent	0.6	0.5	0.4
Obsessive-compulsive	2.4	7.9	4.7

Notes:

- 1 – All numbers are reported as percentages
- 2 – Part II of the National Comorbidity Survey Replication
- 3 – NESARC – National Epidemiologic Survey on Alcohol and Related Conditions
- 4 – Values from reviews of epidemiological studies (5 to 7 studies with 3-6 data sets from the U.S.)
- Source – DSM-5-TR (APA, 2022)

You should have learned the following in this section:

- Prevalence rates of Cluster A personality disorders range from 2% to 5% with schizotypal being more common in men and there being no difference in schizoid and conflicting evidence for paranoid.
- Prevalence rates of Cluster B personality disorders range from 0.0% to 1.4% and antisocial and narcissistic are more common in men with borderline and histrionic being more common in women, in general.
- Prevalence rates of Cluster C personality disorders range from 0.6% to 5.2% with women being more likely to be diagnosed with avoidant and dependent personality disorders and OCPD appearing to be equally prevalent in women and men.

Section 13.2 Review Questions

1. What is the difference in prevalence rates across the three clusters? Are there any trends among gender?
2. Identify the most commonly occurring personality disorder. Which is the least common?

13.3. Comorbidity

Section Learning Objectives

- Describe the comorbidity of personality disorders.

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

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

narcissistic personality disorder were more likely to be diagnosed with generalized anxiety disorder and panic disorder; schizoid and avoidant personality disorders reported significant rates of generalized anxiety disorder; avoidant personality disorder had a higher diagnosis rate of social phobia. Substance use disorders occur less frequently across the ten personality disorders but are most common in individuals diagnosed with antisocial, borderline, and schizotypal personality disorders (Grant et al., 2015). Schizotypal personality disorder is also comorbid with brief psychotic disorder, schizophreniform disorder, delusional disorder, and schizophrenia while borderline is additionally comorbid with eating disorders, PTSD, and ADHD (APA, 2022).

You should have learned the following in this section:

- Mood disorders, anxiety disorders, and substance abuse disorders have a high comorbidity with personality disorders.
- Substance abuse disorders occur less frequently across the ten personality disorders but when they do, are comorbid with antisocial, borderline, and schizotypal personality disorders.

Section 13.3 Review Questions

1. With what other disorders are personality disorders comorbid?

13.4. Etiology

Section Learning Objectives

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

Research regarding the development of personality disorders is limited compared to that of other mental health disorders. The following is a general overview of contributing factors to personality disorders. While there is some research lending itself to specific causes of specific personality disorders, the overall contribution of biological, psychological, and social factors will be reviewed.

13.4.1. Biological

Research across the personality disorders suggests some underlying biological or genetic component; however, identification of specific mechanisms have not been identified in most disorders, except for those below. Because of this lack of concrete evidence, researchers argue that it is difficult to determine what role genetics plays into the development of these disorders compared to that of environmental influences. Therefore, while there is likely a biological predisposition to personality disorders, exact causes cannot be determined at this time.

Research on the development of *schizotypal personality disorder* has identified similar biological causes to that of schizophrenia—high activity of dopamine and enlarged brain ventricles (Lener

et al., 2015). Similar differences in neuroanatomy may explain the high similarity of behaviors in both schizophrenia and schizotypal personality disorder.

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

13.4.2. Psychological

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

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

abandonment throughout an individual's life (Newnham & Janca, 2014; Roepke & Varter, 2014; Caligor & Clarkin, 2010).

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

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

Cognitive distortions such as **dichotomous thinking**, also known as all-or-nothing thinking, are observed in several personality disorders. More specifically, dichotomous thinking

explains rigidity and perfectionism in OCPD, and the lack of self-sufficiency among individuals with dependent and borderline personality disorders (Weishaar & Beck, 2006). **Discounting the positive** also explains the underlying mechanisms for avoidant personality disorder (Weishaar & Beck, 2006). For example, individuals who have been routinely criticized or rejected during childhood may have difficulty accepting positive feedback from others, expecting only to receive rejection and harsh criticism. In fact, they may employ these misattributions to positive feedback to support their ongoing theory that they are constantly rejected and criticized by others.

13.4.2.3. Behavioral. Behavioral theorists apply three major theories to explain the development of personality disorders: modeling, reinforcement, and lack of social skills. In modeling, an individual learns maladaptive social patterns and behaviors by directly observing family members engaging in similar behaviors (Gaynor & Baird, 2007). While we cannot discredit the biological component of the familial influence, research does support an additive modeling or imitating component to the development of personality disorders, especially antisocial personality disorder (APA, 2022).

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

Finally, failure to develop normal social skills may explain the development of some personality disorders, such as avoidant personality disorder (Kantor, 2010).

13.4.3. Social

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

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

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

You should have learned the following in this section:

- Biological causes of personality disorders have not been identified in most disorders, the exception being schizotypal which has similar biological causes as schizophrenia and antisocial and borderline personality disorders which have similar neurological changes.
- Psychological causes of personality disorders include negative early childhood experiences; maladaptive thought patterns and cognitive distortions; and modeling, reinforcement, and lack of social skills.
- Social causes of personality disorders include high levels of psychological and social dysfunction within families and maltreatment.

Section 13.4 Review Questions

1. What personality disorders are most explained by the biological model?
2. How does the psychodynamic model explain the development of personality disorders?
3. What cognitive distortions are most discussed with respect to personality disorders?
4. What are the three behavioral theories used to explain the development of personality disorders?
5. Discuss the roll of attachment and how theorists have used it to explain the development of personality disorders.

13.5. Treatment

Section Learning Objectives

- Describe treatment options for personality disorders.

13.5.1. Cluster A

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

When patients are enrolled in treatment, cognitive-behavioral strategies are most commonly used with the primary intention of reducing anxiety-related symptoms. Additionally, attempts at cognitive restructuring—both identifying and changing maladaptive thought patterns—are also helpful in addressing the misinterpretations of other's words and actions, particularly for individuals with paranoid personality disorder (Kellett & Hardy, 2014). Schizoid personality disorder patients may engage in CBT techniques to help experience more positive emotions and more satisfying social experiences, whereas the goal of CBT for schizotypal personality disorder is to evaluate unusual thoughts or perceptions objectively and to ignore the

inappropriate thoughts (Beck & Weishaar, 2011). Finally, behavioral techniques such as social-skills training may also be implemented to address ongoing interpersonal problems displayed in the disorders.

13.5.2. Cluster B

13.5.2.1. Antisocial personality disorder. Treatment options for antisocial personality disorder are limited and generally not effective (Black, 2015). Like Cluster A disorders, many individuals are forced to participate in treatment, thus impacting their ability to engage in and continue with treatment. Cognitive therapists have attempted to address the lack of morality and encourage patients to think about the needs of others (Beck & Weishaar, 2011).

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

Within DBT, five main treatment components collectively help to reduce harmful behaviors (i.e., self-mutilation and suicidal behaviors) and replace them with practical, life-enhancing behaviors (Gonidakis, 2014). The first component is *skills training*. Generally performed in a group therapy setting, individuals engage in mindfulness, distress tolerance, interpersonal effectiveness, and emotion regulation. Second, individuals focus on *enhancing motivation* and applying skills learned in the previous component to specific challenges and events in their everyday life. The third, and often the most distinctive aspect of DBT, is the use

of *telephone* and *in vivo coaching* for DBT patients from the DBT clinical team. It is not uncommon for patients to have the cell phone number of their clinician for 24/7 availability of in-the-moment support. The fourth component, *case management*, consists of allowing the patient to become their own “case manager” and effectively use the learned DBT techniques to problem-solve ongoing issues. Within this component, the clinician will only intervene when absolutely necessary. Finally, the *consultation team*, is a service for the clinicians providing the DBT treatment. Due to the high demands of borderline personality disorder patients, the consultation team offers support to the providers in their work to ensure they remain motivated and competent in DBT principles to provide the best treatment possible.

Support for the effectiveness of DBT in borderline personality disorder patients has been implicated in several randomized control trials (Harned, Korslund, & Linehan, 2014; Neacsiu, Eberle, Kramer, Wisemeann, & Linehan, 2014). More specifically, DBT has shown to significantly reduce suicidality and self-harm behaviors in those with borderline personality disorders. Additionally, the drop-out rates for treatment are extremely low, suggesting that patients value the treatment components and find them useful in managing symptoms.

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

13.5.2.4. Narcissistic personality disorder. Of all the personality disorders, narcissistic personality disorder is among the most difficult to treat (with maybe the exception of antisocial personality disorder). Most individuals with narcissistic personality disorder only seek out treatment for those disorders secondary to their personality disorder, such as depression (APA, 2022). The focus of treatment is to address the grandiose, self-centered thinking, while also trying to teach patients how to empathize with others (Beck & Weishaar, 2014).

13.5.3. Cluster C

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

Unlike other personality disorders where individuals are skeptical of the clinician, individuals with dependent personality disorder try to place obligations of their treatment on the

clinician. Therefore, one of the main treatment goals for dependent personality disorder patients is to teach them to accept responsibility for themselves, both in and outside of treatment (Colli, Tanzilli, Dimaggio, & Lingardi, 2014). Cognitive strategies such as challenging and changing thoughts on helplessness and inability to care for oneself have been minimally effective in establishing independence. Additionally, behavioral techniques such as assertiveness training have also shown some promise in teaching individuals how to express themselves within a relationship. Some argue that family or couples therapy would be particularly helpful for those with dependent personality disorder due to the relationship between the patient and another person being the primary issue; however, research on this treatment method has not yielded consistently positive results (Nichols, 2013).

You should have learned the following in this section:

- Individuals with a Cluster A personality disorder do not often seek treatment and when they do, struggle to trust the clinician (paranoid and schizotypal) or are emotionally distant from the clinician (schizoid). When in treatment, cognitive restructuring and cognitive behavioral strategies are used.
- In terms of Cluster B, treatment options for antisocial are limited and generally not effective, borderline responds well to dialectical behavioral therapy (DBT), histrionic patients seek out help but are difficult to work with, and finally narcissistic are the most difficult to treat.
- For Cluster C, cognitive techniques aid with OCPD while gradual exposure to various social settings and social skills training help with avoidant. Clinicians use cognitive strategies to challenge thoughts on helplessness in patients with dependent personality disorder.

Section 13.5 Review Questions

1. What is the process in Dialectical Behavioral Therapy (DBT)? What does the treatment entail? What disorders are treated with DBT?
2. Given the difference in personality characteristics between the three clusters, how are the suggested treatment options different between cluster A, B, and C?

Module Recap

Module 13 covered three clusters of personality disorders: Cluster A, which includes paranoid, schizoid, and schizotypal; Cluster B, which includes antisocial, borderline, histrionic, and narcissistic; and Cluster C which includes avoidant, dependent, and obsessive-compulsive. We also covered the clinical description, epidemiology, comorbidity, etiology, and treatment of personality disorders.

Part VI. Mental Disorders – Block 5

Disorders Covered:

- 14. Neurocognitive Disorders
- 15. Contemporary Issues in Psychopathology

Part VI. Mental Disorders – Block 5

Module 14: Neurocognitive Disorders

Module 14: Neurocognitive Disorders

Module Overview

In Module 14, we will cover matters related to neurocognitive disorders (NCDs) to include their clinical presentation, epidemiology, etiology, and treatment options. Our discussion will include delirium, major neurocognitive disorder, and mild neurocognitive disorder. We also discuss nine subtypes to include: Alzheimer's disease, traumatic brain injury (TBI), vascular disorder, substance/medication induced, dementia with Lewy bodies, frontotemporal NCD, Parkinson's disease, Huntington's disease, and HIV infection. Be sure you refer Modules 1-3 for explanations of key terms (Module 1), an overview of the various models to explain psychopathology (Module 2), and descriptions of the therapies (Module 3).

Module Outline

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

Module Learning Outcomes

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

14.1. Clinical Presentation

Section Learning Objectives

- Describe how delirium presents.
- Describe how major neurocognitive disorder presents.
- Describe how mild neurocognitive disorder presents.

Unlike many of the disorders we have discussed thus far, neurocognitive disorders often result from disease processes or medical conditions. Therefore, it is important that individuals presenting with these symptoms complete a medical assessment to better determine the etiology behind the disorder.

There are three main categories of neurocognitive disorders—delirium, major neurocognitive disorder, and mild neurocognitive disorder. Within major and minor neurocognitive disorders are several subtypes due to the etiology of the disorder. For this book, we will review diagnostic criteria for both major and minor neurocognitive disorders, followed by a brief description of the various disease subtypes in the etiology section.

It is important to note as well that the criteria for the various NCDs are based on defined cognitive domains. These include the following, with a brief explanation of what it is:

1. Complex attention – Sustained, divided, or selective attention and processing speed
2. Executive function – planning, decision-making, overriding habits, mental flexibility, and responding to feedback/error correction

3. Learning and memory – includes cued recall, immediate or long-term memory, and implicit learning
4. Language – Includes expressive language and receptive language
5. Perceptual-motor – Includes any abilities related to visual perception, gnosis, perceptual-motor praxis, or visuo-constructional
6. Social cognition – Includes recognition of emotions and theory of mind

14.1.1. Delirium

Delirium is characterized by a notable disturbance in attention along with reduced awareness of the environment. The disturbance develops over a short period of time, representing a change from baseline attention and awareness, and fluctuates in severity during the day. There is also a disturbance in cognitive performance that is significantly altered from one's usual behavior. Disturbances in attention are often manifested as difficulty sustaining, shifting, or focusing attention. Additionally, an individual experiencing an episode of delirium will have a disruption in cognition, including confusion of where they are. Disorganized thinking, incoherent speech, and hallucinations and delusions may also be observed during periods of delirium.

Delirium is associated with increased functional decline and risk of being placed in an institution. That said, most people with delirium recover fully with or without treatment, especially if not elderly, but if undetected or the underlying cause is untreated, it may progress to stupor, coma, seizures, or death (APA, 2022).

14.1.2. Major Neurocognitive Disorder

Individuals with major neurocognitive disorder show a *significant* decline in both overall cognitive functioning (see the previously listed six domains) as well as the ability to independently meet the demands of daily living such as paying bills, taking medications, or caring for oneself (APA, 2022). While it is not necessary, it is helpful to have documentation of the cognitive decline via neuropsychological testing within a controlled, standardized testing environment. Information from close family members or caregivers is also important in documenting the decline and impairment in areas of functioning.

Within the umbrella of major neurocognitive disorder is **dementia**, a striking decline in cognition and self-help skills due to a neurocognitive disorder. The DSM-5-TR (APA, 2022) refrained from using this term in diagnostic categories as it is often used to describe the natural decline in degenerative dementias that affect older adults; whereas neurocognitive disorder is the preferred term used to describe conditions affecting younger individuals such as impairment due to traumatic brain injuries or other medical conditions. Therefore, while dementia is accurate in describing those experiencing major neurocognitive disorder due to age, it is not reflective of those experiencing neurocognitive issues secondary to an injury or illness.

14.1.3. Mild Neurocognitive Disorder

Individuals with mild neurocognitive disorder demonstrate a *modest* decline in one of the listed cognitive domains. The decline in functioning is not as extensive as that seen in major neurocognitive disorder, and the individual does *not* experience difficulty independently engaging in daily activities. However, they may require assistance or extra time to complete these tasks, particularly if the cognitive decline continues to progress.

It should be noted that the primary difference between major and mild neurocognitive disorder is the severity of the decline and independent functioning. Some argue that the two are earlier and later stages of the same disease process (Blaze, 2013). Conversely, individuals can go from major to mild neurocognitive disorder following recovery from a stroke or traumatic brain injury (Petersen, 2011). The DSM-5-TR describes major and mild NCD as existing on a spectrum of cognitive and functional impairment (APA, 2022, pg. 685).

You should have learned the following in this section:

- The criteria for the various NCDs are based on the cognitive domains of complex attention, executive function, learning and memory, language, perceptual-motor, and social cognition.
- Delirium is characterized by a notable disturbance in attention or awareness and cognitive performance that is significantly altered from one's usual behavior.
- Major neurocognitive disorder is characterized by a significant decline in both overall cognitive functioning as well as the ability to independently meet the demands of daily living.
- Mild neurocognitive disorder is characterized by a modest decline in one of the listed cognitive areas with no interference in one's ability to complete daily activities.

Section 14.1 Review Questions

1. What are the six cognitive domains the diagnostic criteria for NCDs are based on?
2. Define delirium. How does this differ from mild and major neurocognitive disorders?
3. What are the main differences between mild and major neurocognitive disorders?

14.2. Epidemiology

Section Learning Objectives

- Describe the epidemiology of neurocognitive disorders.

14.2.1. Delirium

The prevalence of delirium in the general community is relatively low at 1% to 2% based on data from the United States and Finland. For older individuals presenting to North American emergency departments, the rate is 8% to 17%. Prevalence rates for those admitted to the hospital range from 18% to 35%. For those in nursing homes or post-acute care settings prevalence is 20 to 22% and 88% for individuals with terminal illnesses at the end of life. Prevalence rates are lower for younger African Americans compared to White individuals of similar age.

14.2.2. Major and Mild NCD

Major and mild neurocognitive disorder prevalence rates vary widely depending on the etiological nature of the disorder and overall prevalence estimates are generally only available for older populations. Internationally, dementia occurs in 1-2% of individuals at age 65, and up to 30% of individuals by age 85. The female gender is associated with higher prevalence of dementia overall. Estimates for mild NCD among older individuals range from 2% to 10% at age 65 and 5% to 25% at age 85. In the U.S., incidence is highest in African Americans followed by American Indians/Alaska Natives, Latinx, Pacific Islanders, non-Latinx Whites, and Asian Americans.

14.2.3. Major and Mild NCD Subtypes

Alzheimer's disease, the most commonly diagnosed neurocognitive disorder, is observed in nearly 5.5 million Americans (Alzheimer's Association, 2017a), with 11% of those aged 65 and older and 32% older than 85 having dementia due to Alzheimer's disease. It should also be noted that somewhere between 60-90% of dementias are attributable to Alzheimer's disease, depending on the setting and diagnostic criteria. In terms of ethnoracial background in the U.S. the highest prevalence rates have been found among African Americans and Latinx of Caribbean origin (APA, 2022).

Over 2.87 million traumatic brain injuries (TBIs) happen each year within the United States, with men being 40% more likely to experience a TBI compared with women. The most common causes of TBI, in order of occurrence, are falls followed by collision with a moving or stationary object, automobile accidents, and assaults. It has also become increasingly recognized that concussion in sport causes mild TBI (APA, 2022).

You should have learned the following in this section:

- As individuals age, the rate of occurrence of delirium and dementia increases dramatically.
- Estimates for mild NCD among older individuals range from 2% to 10% at age 65 and 5% to 25% at age 85.
- As for Alzheimer's disease, prevalence rates are 11% of those aged 65 and older and 32% of those older than 85.
- Men are more likely to experience a TBI than women.

Section 14.2 Review Questions

1. What is the rate of occurrence of the neurocognitive disorders?

14.3. Etiology

Section Learning Objectives

- Define degenerative.
- Describe the symptoms and causes of Alzheimer's disease.
- Describe the symptoms and causes of traumatic brain injury (TBI).
- Describe the symptoms and causes of vascular disorders.
- Describe the symptoms and causes of substance/medication-induced major or mild NCD .
- Describe the symptoms and causes of dementia with Lewy bodies.
- Describe the symptoms and causes of frontotemporal NCD.
- Describe the symptoms and causes of Parkinson's disease.
- Describe the symptoms and causes of Huntington's disease.
- Describe the symptoms and causes of HIV infection.

Neurocognitive disorders occur due to a wide variety of medical conditions or injury to the brain. Therefore, this section will focus on a brief description of the nine different etiologies of neurocognitive disorders per the DSM-5-TR (APA, 2022). As you will see, most of these neurocognitive disorders are both **degenerative**, meaning the symptoms and cognitive deficits become worse over time, as well as related to a medical condition or disease.

Per the DSM-5-TR (APA, 2022), an individual will meet diagnostic criteria for *either* mild or major neurocognitive disorder as listed above. In order to specify the type of

neurocognitive disorder, additional diagnostic criteria specific to one of the following subtypes must be met.

14.3.1. Alzheimer's Disease

Alzheimer's disease is the most prevalent neurodegenerative disorder. While the primary symptom of Alzheimer's disease is the gradual progression of impairment in cognition, it is also important to identify concrete evidence of cognitive decline. This can be done in one of two ways: via genetic testing of the individual or a documented family history of the disease, or, through clear evidence of cognitive decline over time by repeated standardized neuropsychological evaluations (APA, 2022). It is crucial to identify these markers in making the diagnosis of Alzheimer's disease as some individuals present with memory impairment but eventually show a reversal of symptoms; this is not the case for individuals with Alzheimer's disease.

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

hippocampus, amygdala, and the cerebral cortex, areas associated with memory and cognition (Spires-Jones & Hyman, 2014).

Researchers have identified additional genetic and environmental influences in the development of Alzheimer's disorder. Genetically, the **apolipoprotein E (ApoE)** gene that helps to eliminate beta-amyloid by-products from the brain, has been implicated in the development of Alzheimer's disorder. One of the three variants of this gene, the e4 allele, appears to reduce the production of ApoE, thus increasing the number of beta-amyloid plaques within the brain. However, not all individuals with the e4 allele develop Alzheimer's disease; therefore, this explanation may better explain a vulnerability to Alzheimer's disease as opposed to the cause of the disease.

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

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

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

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

14.3.2. Traumatic Brain Injury (TBI)

TBIs occur when an individual experiences significant trauma or damage to the head. Neurocognitive disorder due to TBI is diagnosed when persistent cognitive impairment is observed immediately following the head injury, along with one or more of the following symptoms: loss of consciousness, posttraumatic amnesia, disorientation and confusion, or neurological impairment (APA, 2022).

The presentation of symptoms varies among individuals and depends largely on the location of the injury and the intensity of the trauma. Furthermore, the effects of a TBI can be temporary or permanent. Symptoms generally range from headaches, disorientation, confusion, irritability, fatigue, poor concentration, and emotional and behavioral changes. More severe

injuries can result in more significant neurological symptoms such as seizures, paralysis, and visual disturbances.

Major or mild NCD due to TBI may be comorbid with specified or unspecified depressive, anxiety, or personality disorders and PTSD. Rates of suicidal ideation are as high as 10% with rates of suicide attempt hovering around 0.8% to 1.7% (APA, 2022).

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

14.3.3. Vascular Disorders

Neurocognitive disorders due to vascular disorders can occur from a one-time event such as a stroke or ongoing subtle disruptions of blood flow within the brain (APA, 2022). The occurrence of these vascular disorders general begins with **atherosclerosis**, or the clogging of arteries due to a build-up of plaque. The **plaque** builds up over time, eventually causing the

artery to narrow, thus reducing the amount of blood able to pass through to other parts of the body. When these arteries within the brain become entirely obstructed, a **stroke** occurs. The lack of blood flow during a stroke results in the death of neurons and loss of brain function. There are two types of strokes—a **hemorrhagic stroke** that occurs when a blood vessel bursts within the brain and an **ischemic stroke**, which is when a blood clot blocks the blood flow in an artery within the brain (American Stroke Association, 2017).

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

After Alzheimer's disease, vascular disease is the second most common cause of NCD and population prevalence estimates are 0.98% for those between the ages of 71-79 years, 4.09% for individuals aged 80-89 years, and 6.19% for those aged 90 years and up. Within three months of a stroke, 20%-30% of people are diagnosed with dementia. Finally, stroke is more common in men up to age 65 and after that, it shifts to women. Vascular disease is frequently comorbid with major or mild NCD due to Alzheimer's disease and depression.

14.3.4. Substance/Medication-Induced Major or Mild NCD

Significant cognitive changes occur due to repetitive drug and alcohol abuse. Delirium can be observed in individuals with extreme substance intoxication, withdrawal, or even when multiple substances have been used within a close period (APA, 2022). While delirium symptoms are often transient during these states, mild neurocognitive impairment due to heavy substance abuse may remain until a significant period of abstinence is observed (Stavro, Pelletier, & Potvin, 2013).

14.3.5. Dementia with Lewy Bodies

Symptoms associated with neurocognitive disorder due to Lewy bodies include significant fluctuations in attention and alertness; recurrent visual hallucinations; impaired mobility; and sleep disturbances such as rapid eye movement sleep behavior disorder (APA, 2022). While the trajectory of the illness develops more rapidly than Alzheimer's disease, the survival period is similar in that most individuals do not survive longer than eight years post-diagnosis (Lewy Body Dementia Association, 2017).

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

14.3.6. Major or Mild Frontotemporal NCD

Frontotemporal NCD causes “progressive development of behavioral and personality change and/or language impairment” (APA, 2022, pg. 696). For the behavioral variant, individuals display at least three of the following: behavioral disinhibition, apathy or inertia, loss of sympathy or empathy, preservative or compulsive behavior, or hyperorality and dietary changes. For the language variant, they show prominent decline in language ability (i.e., speech production, word finding, object naming, grammar, or word comprehension). There is relative sparing of learning and memory and perceptual-motor functioning. Individuals with frontotemporal NCD commonly present in their 50s though the age of onset has a range of age 20 to 80 years. The median survival is 6-11 years after symptom onset and 3-4 years after diagnosis (APA, 2022).

14.3.7. Parkinson’s Disease

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

Onset of Parkinson’s disease is typically from age 50 to 89 years. Mild NCD develops early in the course of Parkinson’s disease while Major NCD does not occur until individuals are

much older. The prevalence of Parkinson's disease in the U.S. increases with age and is more common in men than women. The disease is comorbid with Alzheimer's disease and cerebrovascular disease. Depression, psychosis, REM sleep behavior disorder, apathy, and motor symptoms can make functional impairment worse (APA, 2022).

14.3.8. Huntington's Disease

Huntington's disease is a rare genetic disorder that involves involuntary movement, progressive dementia, and emotional instability. Due to the degenerative nature of the disorder, there is a shortened life-expectancy as death typically occurs 15-20 years post-onset of symptoms (Clabough, 2013). Although symptoms can present at any time, the average age of symptom presentation is during middle adulthood (between ages 35 and 45 years; APA, 2022). Symptoms generally begin with neurocognitive decline, particularly in executive function, along with changes in mood and personality. As symptoms progress, more physical symptoms present, such as facial grimaces, difficulty speaking, and repetitive movements. Because there is no treatment for Huntington's disease, the severity of the cognitive and physical impairments ultimately leads to complete dependency and the need for full-time care. Suicide is among the leading causes of death in Huntington's disease (APA, 2022).

14.3.9. HIV Infection

Not many people are aware that cognitive impairment is sometimes the first symptom of untreated HIV. While symptoms vary among individuals, slower mental processing, impaired executive function, problems with more demanding attentional tasks, and difficulty learning new information are among the most common early signs (APA, 2022). When HIV becomes active in

the brain, significant alterations of mental processes occur, thus leading to a diagnosis of *neurocognitive disorder due to HIV infection*. Significant impairment can also occur due to HIV-infection related inflammation throughout the central nervous system.

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

You should have learned the following in this section:

- Most neurocognitive disorders are degenerative meaning they become worse over time.
- Alzheimer's disease is characterized by the gradual progression of impairment in cognition as well as the presence of beta-amyloid plaques and neurofibrillary tangles.
- TBIs occur when an individual experiences significant trauma or damage to the head with the most common type being a concussion.
- Vascular disorders generally begin with atherosclerosis which leads to a stroke.
- Significant cognitive changes occur due to repetitive drug and alcohol abuse such as delirium.
- Dementia with Lewy bodies is characterized by significant fluctuations in attention and alertness; recurrent visual hallucinations; impaired mobility; and sleep disturbance.
- Frontotemporal NCD causes progressive declines in language or behavior due to the degeneration in the frontal and temporal lobes of the brain.
- Parkinson's disease is characterized by tremors of hands, arms, legs, or face; rigidity of the limbs and trunk; slowness in initiating movement; and drooping posture or impaired balance and coordination.
- Huntington's disease involves involuntary movement, progressive dementia, and emotional instability.
- HIV infection begins with slower mental processing, impaired executive function, problems with more demanding attentional tasks, and difficulty learning new information.

Section 14.3 Review Questions

1. Define degenerative. What disorders discussed in this module are considered degenerative?
2. Identify the biological causes of Alzheimer's disease.
3. What is a TBI?
4. How do vascular disorders occur?
5. What are Lewy bodies? How does dementia with Lewy bodies differ from Alzheimer's disease?
6. What are the main symptoms of Parkinson's disease? Huntington's disease?

14.4. Treatment

Section Learning Objectives

- Describe treatment options for neurocognitive disorders.

Treatment options for those with neurocognitive disorders are minimal at best, with most attempting to treat secondary symptoms as opposed to the neurocognitive disorder itself.

Furthermore, the degenerative nature of these disorders also makes it difficult to treat, as many diseases will progress regardless of the treatment options.

14.4.1. Pharmacological

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

Parkinson's disease has also found success in pharmacological treatment options. The medication *levodopa* increases dopamine availability, which provides relief of both physical and

cognitive symptoms. Unfortunately, there are also significant side effects such as hallucinations and psychotic symptoms; therefore, the medication is often only used when the benefits outweigh the negatives of the potential risks (Poletti & Bonuccelli, 2013).

14.4.2. Psychological

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

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

14.4.3. Support for Caregivers

Supporting caregivers is an important treatment option to include as the emotional and physical toll on caring for an individual with a neurocognitive disorder is often underestimated. According to the Alzheimer's Association (2017b), nearly 90% of all individuals with Alzheimer's disease are cared for by a relative. The emotional and physical demands on caring for a family member who continues to decline cognitively and physically can lead to increased

anger and depression in a caregiver (Kang et al. 2014). It is important that medical providers routinely assess caregivers' psychosocial functioning, and encourage caregivers to participate in caregiver support groups, or individual psychotherapy to address their own emotional needs.

You should have learned the following in this section:

- Pharmacological interventions for Alzheimer's disease target the neurotransmitters acetylcholine and glutamate and newer research is focused on the build-up of beta-amyloid and neurofibrillary tangles.
- Psychological treatments include cognitive and behavioral strategies such as playing board games, reading books, or social skills training.
- Caregivers need to join support groups to help them manage their own anger and depression, especially since 90% of such caregivers are relatives of the afflicted.

Section 14.4 Review Questions

1. Review the listed treatment options for neurocognitive disorders. What are the main goals of these treatments?

Module Recap

Our discussion in Module 14 turned to neurocognitive disorders to include the categories of delirium, major neurocognitive disorder, and Mild neurocognitive disorder. We also discussed the subtypes of Alzheimer's disease, traumatic brain injury (TBI), vascular disorder, substance/medication induced, dementia with Lewy bodies, frontotemporal NCD, Parkinson's disease, Huntington's disease, and HIV infection. The clinical description, epidemiology, etiology, and treatment options for neurocognitive disorders were discussed.

Part VI. Mental Disorders – Block 5

Module 15: Contemporary Issues in Psychopathology

Module 15: Contemporary Issues in Psychopathology

Module Overview

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

Module Outline

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

Module Learning Outcomes

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

15.1. Legal Issues Related to Mental Illness

Section Learning Objectives

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

15.1.1. Forensic Psychology/Psychiatry

According to the American Psychological Association, **forensic psychology/psychiatry** is when clinical psychology is applied to the legal arena in terms of assessment, treatment, and evaluation. Forensic psychology can also include the application of research from other subfields in psychology to include cognitive and social psychology. Training includes law and forensic psychology, and solid clinical skills are a must. According to APA, a forensic psychologist might “perform such tasks as threat assessment for schools, child custody evaluations, competency evaluations of criminal defendants and of the elderly, counseling services to victims of crime,

death notification procedures, screening and selection of law enforcement applicants, the assessment of post-traumatic stress disorder and the delivery and evaluation of intervention and treatment programs for juvenile and adult offenders.” A key issue investigated by forensic psychologists includes *mens rea* or the insanity plea. We will discuss this shortly.

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

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

15.1.2. Civil Commitment

15.1.2.1. What is civil commitment? When individuals with mental illness behave in erratic or potentially dangerous ways, to either themselves or others, then something must be done. The responsibility to act falls on the government through what is called *parens patriae* or “father of the country” or “country as parent.” Action, in this case, involves involuntary commitment in a hospital or mental health facility and is done to protect the individual and express concern over their well-being, much like a parent would do for their child. An individual can voluntarily admit themselves to a mental health facility, and upon doing so, staff will determine whether treatment and extended stay are needed.

15.1.2.2. Criteria for civil commitment. Though states vary in the criteria used to establish the need for civil commitment, some requirements are common across states. First, the individual must present a clear danger to either themselves or others. Second, the individual demonstrates that they are unable to care for themselves or make decisions about whether treatment

or hospitalization is necessary. Finally, the individual believes they are about to lose control, and so, needs treatment or care in a mental health facility.

15.1.2.3. Assessment of “dangerousness.” Dangerousness can best be defined as the person’s capacity or likelihood of harming themselves or others. Most people believe that those who are mentally ill are more dangerous than those free of mental illness, especially when espousing self-reported conservatism and RWA (Right-Wing Authoritarianism; Gonzales, Chan, and Yanos, 2017; DeLuca and Yanos, 2015) or after tragic events such as a mass shooting (Metzl & MacLeish, 2015). The media plays a role in this, and as McGinty et al. (2014) found, 70% of news coverage of serious mental illness (SMI) and gun violence over a 16-year period (1997 to 2012) focused on extreme events and described specific shootings by persons with SMI. The authors wrote, “Even in thematic news coverage focused on describing the general problem of SMI and gun violence, the majority of news stories did not mention that most people with SMI are not violent or that we lack tools capable of accurately identifying persons with SMI who are at heightened risk of committing future violence.” They concluded that media coverage of persons with SMI as violent might contribute to negative public attitudes.

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

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

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

Before we move on, consider for a minute that a person who is accused of a crime is innocent until proven guilty, has a trial, and if found guilty beyond a reasonable doubt (or almost

complete certainty) is only then incarcerated. This is not true for the mentally ill, who may be committed to a facility without ever having committed a crime or having a trial, but simply because they were judged as having the *potential* to do so (or was seen as dangerous). This *potential* means that there must be “clear and convincing” proof, which the U.S. Supreme Court defines as 75% certainty. The standard to commit is much different for those accused of criminal acts and those who are mentally ill.

15.1.3. Criminal Commitment

When people are accused of crimes but found to be mentally unstable, they are usually sent to a mental health institution for treatment. This is called **criminal commitment**.

Individuals may plead **not guilty by reason of insanity (NGRI)** or as it is also called, the **insanity plea**. When a defendant pleads NGRI they are acknowledging their guilt for the crime (*actus rea*) but wish to be seen as not guilty since they were mentally ill at the time (*mens rea*). The origins of the modern definition of insanity go back to Daniel M’Naghten in 1843 England. He murdered the secretary to British Prime Minister, Robert Peel, during an attempted assassination of the Prime Minister. He was found to be not guilty due to delusions of persecution, which outraged the public and led to calls for a more precise definition of insanity. The **M’Naghten rule** states that having a mental disorder at the time of a crime does not mean the person was insane. The individual also had to be unable to know right from wrong or comprehend the act as wrong. But how do you know what the person’s level of awareness was when the crime was committed?

Dissatisfaction with the M’Naghten rule led some state and federal courts in the U.S. to adopt instead the **irresistible impulse test** (1887), which focused on the inability of a person to

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

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

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

- significantly modified the standard for insanity previously applied in the federal courts

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

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

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

For more on the insanity plea, please visit:

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

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

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

You should have learned the following in this section:

- Forensic psychology is when clinical psychology is applied to the legal arena in terms of assessment, treatment, and evaluation, though it can include research from other subfields to include cognitive and social psychology.
- Civil commitment occurs when a person acts in potentially dangerous ways to themselves or others and can be initiated by the person or the government.
- Dangerousness is defined as the person's capacity of harming themselves or others and implies physical harm but not necessarily psychological abuse or the destruction of property.
- Criminal commitment occurs when a person is accused of a crime but found to be mentally unstable.
- Several rules or tests have been attempted to determine if a person is responsible for their actions at the time a crime was committed. These include the M'Naghten rule, irresistible impulse test, Durham test, and the American Law Institute standard.

Section 15.1 Review Questions

1. Describe the subfield of forensic psychology.
2. What is civil commitment and what criteria is used when establishing its need?
3. What does the concept of dangerousness mean?
4. What is criminal commitment?
5. Outline the various rules/tests used to determine if someone is responsible for their actions at the time of a crime.
6. Contrast the insanity plea with the concept of being competent to stand trial.

15.2. Patient's Rights

Section Learning Objectives

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

The following are several rights that patients with mental illness have. They include:

- **Right to Treatment** – In the 1966 case of *Rouse v. Cameron*, the D.C. District court said that the right to treatment is a constitutional right, and failure to provide resources cannot be justified due to insufficient resources. In the 1972 case of *Wyatt v. Stickney*, a federal court ruled that the state of Alabama was constitutionally obligated to provide all people who were committed to institutions with adequate treatment and had to offer more therapists, privacy, exercise, social interactions, and better living conditions for patients. In the case of *O'Connor v. Donaldson* (1975), the court ruled that patient's cases had to be reviewed periodically to see if they could be released. As well, if they are not a danger *and* are able to survive on their own or with help from family or friends, that they be released.
- **Right to Refuse Treatment** – As patients have the right to request treatment, they too have the right to refuse treatment such as biological treatment, psychotropic medications (*Riggins v. Nevada, 1992*), and electroconvulsive therapy.
- **Right to Less Restrictive Treatment** – In *Dixon v. Weinberger* (1975), a U.S. District Court ruled that individuals have a right to receive treatment in facilities less restrictive than mental institutions. The only patients who can be committed to hospitals are those unable to care for themselves.

- **Right to Live in a Community** – The 1974 U.S. District Court case, *Staff v. Miller*, ruled that state mental hospital patients had a right to live in adult homes in their communities.

You should have learned the following in this section:

- Patients with a mental illness have a right to treatment, to refuse treatment, to have less restrictive treatment, and to live in a community.

Section 15.2 Review Questions

1. What rights do patients with mental illness have and what court cases were pivotal to their establishment?

15.3. The Therapist-Client Relationship

Section Learning Objectives

- Describe three concerns related to the therapist-client relationship.

Three concerns are of paramount importance in terms of the therapist-client relationship. These include the following:

- **Confidentiality** – As you might have learned in your introductory psychology course, **confidentiality** guarantees that information about you is not disseminated without your consent. This applies to students participating in research studies as well as patients seeing a therapist.

- **Privileged communication** – Confidentiality is an ethical principle while **privileged communication** is a legal one, and states that confidential communications cannot be disseminated without the patient's permission. There are a few exceptions to this which include the client being younger than 16, when they are a dependent elderly person and a victim of a crime, or when the patient is a danger to him or herself or others, to name a few.
- **Duty to Warn** – In the 1976 *Tarasoff v. the Board of Regents of the University of California* ruling, the California Supreme Court said that a patient's right to confidentiality ends when there is a danger to the public, and that if a therapist determines that such a danger exists, they are obligated to warn the potential victim. Tatiana Tarasoff, a student at UC, was stabbed to death by graduate student, Prosenjit Poddar in 1969, when she rejected his romantic overtures, and despite warnings by Poddar's therapist that he was an imminent threat. The case highlights the fact that therapists have a legal and ethical obligation to their clients but, at the same time, a legal obligation to society. How exactly should they balance these competing obligations, especially when they are vague? The 1980 case of *Thompson v. County of Alameda* ruled that a therapist does not have a duty to warn if the threat is nonspecific.

You should have learned the following in this section:

- There are three concerns which are important where the therapist-client relationship is concerned – confidentiality, privileged communication, and the duty to warn.

Section 15.3 Review Questions

1. What are the three concerns related to the therapist-client relationship? Describe each and state any relevant court rulings relevant to them.

Check This Out

Can you play video games so much, that it becomes addictive? Does this mean that it is a diagnosable mental illness to be listed in the DSM 5-TR? Currently, the disorder is only listed in the DSM 5-TR as a *condition for further study* and is called **internet gaming disorder**. It is thought to include symptoms such as:

- Preoccupation with Internet games
- Withdrawal symptoms when not playing Internet games
- The person has tried to stop or curb playing Internet games, but has failed to do so
- The need to spend increasing amounts of time engage in Internet gaming
- A person has had continued overuse of Internet games even with the knowledge of how much they impact a person's life
- The person uses Internet games to relieve anxiety or guilt or to escape
- Loss of interests in previous hobbies and entertainment except for internet gaming

Interestingly, the DSM-5-TR says the mean prevalence of 12-month Internet gaming disorder is approximately 4.7% across multiple countries and is similar in Asian and Western countries. It is more common in males than females. It is comorbid with major depressive disorder, OCD, and ADHD.

And, the ICD now includes gaming disorder in its 11th edition.

For more on this “disorder,” check out the following articles:

- Psychology Today - <https://www.psychologytoday.com/us/blog/here-there-and-everywhere/201407/internet-gaming-disorder-in-dsm-5>
- The Cognitive Psychology of Internet Gaming Disorder (2014 article in Clinical Psychology Review) – <https://www.sciencedirect.com/science/article/pii/S0272735814000658>
- CNN – <https://www.cnn.com/2017/12/27/health/video-game-disorder-who/index.html>
- Huffington Post - https://www.huffingtonpost.com/christopher-j-ferguson/the-muddled-science-of-internet-gaming-disorder_b_9405478.html
- WHO - <http://www.who.int/features/qa/gaming-disorder/en/>

What do you think?

Module Recap

And that's it. Our final module explored some concepts that transcend any one mental disorder but affect people with mental illness in general. This included civil and criminal commitment and issues such as NGRI or the insanity plea, what makes someone dangerous and what we should do about it, and determining competency to stand trial. We then moved to patient rights, such as the right to treatment and, conversely, the right to refuse treatment. Finally, we ended by discussing the patient-therapist relationship and specifically, when the patient's right to confidentiality and privileged communication ends, and the therapist has a moral and legal obligation to warn. We hope you find these topics interesting and explore the issues further through the links that were provided and peer-reviewed articles that were cited.

Part VI. Mental Disorders – Block 5

Module 16: Disorders of Childhood Overview

Module 16: Disorders of Childhood Overview

Module Overview

In Module 16, we will provide an overview of disorders of childhood. Please note that a more comprehensive discussion can be found in our Behavioral Disorders of Childhood book (<https://opentext.wsu.edu/behavioral-disorders-childhood/>). Section 16.1 will cover the following disorders of infancy and early childhood to include disinhibited social engagement disorder, reactive attachment disorder, pica, rumination disorder, and avoidant/restrictive food intake disorder, enuresis, and encopresis. In Section 16.2 we will discuss developmental and motor-related disorders to include intellectual developmental disorder (intellectual disability), specific learning disorder, autism spectrum disorder, social (pragmatic) communication disorder, stereotypic movement, and tic disorders.

In Section 16.3 we cover behavior-related disorders to include ADHD, oppositional defiant disorder, conduct disorder, and intermittent explosive disorder. Finally, Section 16.4 will cover select disorders not previously presented in this book in previous sections. These disorders include the anxiety disorders of selective mutism and separation anxiety disorder and the obsessive-compulsive and related disorders of trichotillomania, and excoriation disorder.

For all disorders we will cover their clinical presentation, prevalence, comorbidity. We will also cover differential diagnosis when relevant. For a discussion of etiology, assessment, and treatment options please see the **Behavioral Disorders of Childhood** book from which this content is derived (see link above and throughout this book). As the two books are written by the same authors and follow the same format, the content of this module is consistent with The Fundamentals of Psychological Disorders book you have been reading through Module 15. Also

note that for disorders covered already in this book, the Behavioral Disorders of Childhood book will have more pertinent information related to childhood and adolescence. Our focus in this book has been on adulthood primarily.

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 therapies (Module 3).

Module Outline

- 16.1. Disorders of Infancy and Early Childhood
- 16.2. Developmental and Motor-related Disorders
- 16.3. Behavior-related Disorders
- 16.4. Other Disorders

Module Learning Outcomes

- Describe disorders of infancy and early childhood to include their clinical presentation, prevalence, and comorbidity.
- Describe developmental and motor-related disorders to include their clinical presentation, prevalence, and comorbidity.
- Describe behavior-related disorders to include their clinical presentation, prevalence, and comorbidity.
- Describe childhood-specific anxiety and obsessive-compulsive and related disorders to include their clinical presentation, prevalence, and comorbidity.

16.1. Disorders of Infancy and Early Childhood

Section Learning Objectives

- Describe the clinical presentation, prevalence, and comorbidity of disinhibited social engagement disorder.
- Describe the clinical presentation, prevalence, and comorbidity of reactive attachment disorder.
- Describe the clinical presentation, prevalence, and comorbidity of pica.
- Describe the clinical presentation, prevalence, and comorbidity of rumination disorder.
- Describe the clinical presentation, prevalence, and comorbidity of avoidant/restrictive food intake disorder.
- Describe the clinical presentation, prevalence, and comorbidity of enuresis.
- Describe the clinical presentation, prevalence, and comorbidity of encopresis.

16.1.1. Disinhibited Social Engagement Disorder

16.1.1.1. Clinical Presentation. We will explore disinhibited social engagement disorder in terms of: (1) how the child presents and (2) the history of the child. Let's start with how the child presents. These children, unlike children with reactive attachment disorder described below, tend to be overly social and interact with complete strangers. The child may walk up to someone in a store they have never met and hug them or even walk away with them. They may do this without ever hesitating when separating from their caregiver and might not even look to check back with their caregiver.

In terms of the history of the child, these children often experience *impaired caregiving*, which means that the caregiver does not sufficiently care for the child on a consistent basis. This

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

Disinhibited social engagement disorder may co-occur with developmental delays such as cognitive and language delays, stereotypies, and other signs of severe neglect. Signs of the disorder often persist even after these other signs of neglect are absent. As such, it is not uncommon for children with disinhibited social engagement disorder to present with no current signs of neglect. The DSM adds that the condition can present in children who show no signs of disordered attachment. “Thus, disinhibited social engagement disorder may be seen in children with a history of neglect who lack attachments or whose attachments to their caregivers range from disturbed to secure” (APA, 2022, pg. 299).

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

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

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

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

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

16.1.2. Reactive Attachment Disorder

16.1.2.1. Clinical Presentation. We will discuss reactive attachment disorder in much the same way as disinhibited social engagement disorder starting with how the child presents. Children with this disorder typically present as detached from others or emotionally withdrawn.

They do not seek comfort from caregivers or respond to physical touch when distressed. These children typically have low levels of expressed emotions, particularly positive emotions. They may experience unexplained irritability, sadness, and fearfulness.

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

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

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

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

16.1.2.4. Differential Diagnosis. Due to the significant deficit in emotional-social reciprocity, autism spectrum disorder must be differentiated from reactive attachment disorder.

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

For more on disinhibited social engagement disorder and reactive attachment disorder please visit: <https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-4-disinhibited-social-engagement-disorder-and-reactive-attachment/>

16.1.3. Pica

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

associated with specific biological abnormalities, though deficiencies in vitamins and minerals such as iron and zinc, have been reported.

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

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

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

16.1.4. Rumination Disorder

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

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

16.1.4.2. Prevalence. Limited European data suggests a prevalence of 1% to 2% of grade-school-age children.

16.1.4.3. Comorbidity. Rumination disorder also occurs more frequently in individuals with an intellectual disability (Olden, 2001). It can occur in the context of a concurrent medical condition or another mental disorder such as generalized anxiety disorder.

16.1.5. Avoidant/Restrictive Food Intake Disorder

16.1.5.1. Clinical Presentation. **Avoidant/restrictive food intake disorder** is a new disorder to the DSM 5 (APA, 2013) and replaces and extends the DSM-IV diagnosis of feeding disorder of infancy or early childhood (Norris & Katzman, 2015). It is an eating or feeding disturbance associated with at least one of the following: 1) significant weight loss, 2) significant nutritional deficiency, 3) dependence on enteral feeding or oral nutrition supplements, and 4)

marked interference with psychosocial functioning. The disturbance is not better explained due to a lack of available food or a culturally sanctioned practice.

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

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

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

For more on pica, rumination disorder, and avoidant/restrictive food intake disorder please visit: <https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-5-feeding-disorders/>

16.1.6. Enuresis

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

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

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

16.1.7. Encopresis

16.1.7.1. Clinical Presentation. Encopresis is defined as the repeated passage of feces into inappropriate places such as clothing or onto the floor, whether involuntary or intentional.

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

16.1.7.2. Prevalence. Most children older than four years of age diagnosed with encopresis have the subtype “with constipation and overflow incontinence.” The disorder affects 1% to 4% of children in high-income countries, while in some Asian countries a prevalence rate of 2% to 8% has been reported. It also affects children aged 4-6 years more than children aged 10-12 years. Encopresis is also higher among children who were low-income youth or were abused or neglected early. The gender ratio appears to be about equal in children younger than 5 years but is more common in boys than in girls among older children.

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

For more on enuresis and encopresis please visit: <https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-6-elimination-disorders/>

You should have learned the following in this section:

- A child with disinhibited social engagement disorder tends to be overly social and interact with complete strangers and have experienced *impaired caregiving*, which means that the caregiver does not sufficiently care for the child on a consistent basis.
- A child with reactive attachment disorder do not seek comfort from caregivers or respond to physical touch when distressed and has experienced a pattern of extremes of insufficient care.
- Pica is the act of eating items that are not food on a regular or recurring basis and over a period of at least one month.
- Rumination disorder is the frequent act of regurgitating food over a period of at least 1 month with no medical explanation such as gastroesophageal reflux and in the absence of a body-image/weight-related reason.
- Avoidant/restrictive food intake disorder is associated with at least one of the following: 1) significant weight loss, 2) significant nutritional deficiency, 3) dependence on enteral feeding or oral nutrition supplements, and 4) marked interference with psychosocial functioning.
- Enuresis is the repeated voiding of urine into bed or clothes and can be involuntary or intentional. It has occurred at least two times a week for the past three consecutive months and the child must be at least 5 years of age.
- Encopresis is defined as the repeated passage of feces into inappropriate places such as clothing or onto the floor, whether involuntary or intentional. There has been at least one such event each month for the past three months and the individual must be at least four years of age.

Section 16.1 Review Questions

1. In what ways are disinhibited social engagement disorder and reactive attachment disorder similar and different from one another?
2. What is pica?
3. What is rumination disorder?
4. What is avoidant/restrictive food intake disorder? How does food avoidance or restriction manifest?
5. What is enuresis?
6. What is encopresis?

16.2. Developmental and Motor-related Disorders

Section Learning Objectives

- Describe the clinical presentation, prevalence, and comorbidity of intellectual development disorder (intellectual disability).
- Describe the clinical presentation, prevalence, and comorbidity of specific learning disorder.
- Describe the clinical presentation, prevalence, and comorbidity of autism spectrum disorder (ASD).
- Describe the clinical presentation of social (pragmatic) communication disorder.
- Describe the clinical presentation, prevalence, and comorbidity of stereotypic movement disorder.
- Describe the clinical presentation, prevalence, and comorbidity of tic disorders.

16.2.1. Intellectual Developmental Disorder (Intellectual Disability)

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

When considering intellectual developmental disorder there are two primary areas of major deficits – intellectual functioning (Criterion A) and adaptive functioning (Criterion B; APA, 2022).

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

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

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

judgment, for example. Finally, the *practical domain* involves learning and self-management across life settings such as job responsibilities, personal care, and recreation.

16.2.1.1.3. Onset of intellectual developmental disorder (Criterion C). It

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

16.2.1.1.4. Severity specifiers. Rather than IQ scores, intellectual developmental disorder is assigned a severity specifier based on the level of delays related to adaptive functioning. Essentially, the more support someone needs, the more severe the intellectual disability. Severity ranges from Mild (least severe), to Moderate, to Severe, and Profound (most severe; APA, 2022). Severity is considered in relation to the three domains of conceptual, social, and practical. For instance, a specifier of severe would result the child having little understanding of written language or concepts involving numbers, quantity, and money (conceptual domain), speech and communication being focused on the here and now within everyday events (social

domain), and not being able to make responsible decisions regarding the well-being of self or others, necessitating supervision at all times (practical domain).

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

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

16.2.1.2. Prevalence. Intellectual development disorder occurs in approximately 1% of the overall general population while the global prevalence varies by country and level of development. Prevalence is 16 per 1,000 in middle-income countries but 9 per 1,000 in high-income countries (APA, 2022). Intellectual development disorder is more common in males than females, although sex ratios are inconsistent in the literature (APA, 2022; Einfeld & Emerson, 2008). It is hypothesized that the reason there is a higher occurrence of intellectual development disorder in males is due to general genetic vulnerability, often linked to X chromosome issues

that males experience (Harris, 2006). Prevalence is higher in youth than in adults and there are no significant differences between ethnoracial groups.

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

16.2.1.4. Onset. Onset of intellectual development disorder is in the developmental period, though etiology and severity of brain dysfunction affect exact age and characteristic features at onset. For individuals with more severe intellectual development disorder, delayed motor, language, and social milestones are typical within the first 2 years of life. For individuals with mild intellectual development disorder, impairments may not be identifiable until school age when problems with academic learning are evident.

16.2.2. Specific Learning Disorder

16.2.2.1. Clinical Presentation. A specific learning disorder is characterized by persistent difficulties learning critical academic skills during the years of formal schooling such as reading of single words accurately and fluently or arithmetic calculation; performance of the affected academic skills being well below expected for age; learning difficulties being apparent in the early school years for most individuals, and that the learning difficulties are considered “specific” for four reasons. First, the learning difficulties are not better explained by intellectual developmental disorder, global developmental delay, hearing or vision disorders, or neurological or motor disorders. Second, they cannot be attributed to more general external factors such as

economic or environmental disadvantage. Third, they cannot be attributed to neurological disorders such as a pediatric stroke, motor disorders, or to vision or hearing disorders. Fourth, a learning difficulty can be restricted to one academic skill or domain.

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

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

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

- **With impairment in reading** – The individual has trouble comprehending material, reading fluently and quickly, or reading words accurately.

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

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

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

Differences and Similarities between the Disorders

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

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

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

Specific learning disorder. To differentiate between a specific learning disorder and intellectual developmental disorder, it is useful to consider the discrepancy between what is expected of an individual (what they *can* do) and what they *are* doing. If an individual cannot perform averagely because their IQ is substantially below average, we could not expect them to perform at an average level on academic tasks. For example, if a person's IQ is 70 and they cannot function typically on cognitive tasks, we would not expect them to achieve an academic score of 100, a 30-point jump from what they can do to what they are doing. If an individual has an IQ of 70, we would expect their academic score to be around 70, which would not necessarily indicate a specific learning disorder, even though the score is low. In this case, they would be performing as expected, so the low score achievement score would reflect low cognitive abilities resulting from intellectual development disorder. However, if there is a large discrepancy between what a person can do and what they are doing, for example, someone with an IQ of 100 scoring only a 70 in an academic achievement task, this could indicate a specific learning disorder.

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

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

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

Specific learning disorder in the cognitively delayed and in the cognitively gifted.

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

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

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

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

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

For more on intellectual developmental and specific learning disorders please visit:
<https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-7-intellectual-disability-intellectual-developmental-disorder-ididd-learning-disorders/>

16.2.3. Autism Spectrum Disorder

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

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

16.2.3.1.2. Criterion B: Restricted and repetitive behaviors, interests, or activities. The child must present with at least two of the following. First, they engage in stereotyped movement, frequently called **stereotypy**, such as hand flapping, spinning, or any repetitive movement that does not have an obvious function. They might display **restricted or**

repetitive play such as lining up toys or fixating on a part of a toy or button. Individuals may have excessive and **restricted interests** such as being overly interested in history, dinosaurs, robotics, etc. Their interests may be so intense that, if discussing their interests, it is difficult to get them to move on to a new topic, or it may be the only thing about which they will engage in a conversation.

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

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

Although language delays are common in children with ASD, they are not necessarily required. Moreover, there is often a misconception that if a child has severe ASD, they are nonverbal, meaning they do not have language. This is not accurate. Children with ASD present very differently from each other. Regarding ASD, Dr. Stephen Shore said, “If you’ve met one

person with autism, you've met one person with autism." In other words, one child with ASD may have symptoms that present very differently from another child with ASD, but both children have ASD.

16.2.3.3. Specifiers for ASD. ASD is diagnosed within the context of *language development and intellectual development* given that these factors may indicate prognosis. As such, clinicians will assign specifiers that identify if there is any evidence of language impairment or intellectual impairment. For intellectual impairment, separate estimates of verbal and nonverbal skill are necessary. For language impairment, the clinician should consider receptive and expressive language skills separately since receptive may lag behind expressive language development in ASD.

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

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

16.2.3.5. Development and course. The behavioral features of ASD first become noticeable in early childhood as some children will present with a lack of interest in social interaction during their first year of life. For children in which skills have been lost, parents and

other caregivers may provide a history showing gradual or relatively rapid deterioration in social behaviors or language skills occurring between 12 and 24 months.

In terms of symptoms, the first symptoms of ASD typically involve delayed language development, often accompanied by lack of social interest or unusual social interactions, odd play patterns such as carrying a toy around but never playing with it, and unusual communication patterns such as knowing the alphabet but not responding when their name is said. During the second year, odd and repetitive behaviors, and the absence of typical play become more apparent (APA, 2022).

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

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

16.2.4. Social (Pragmatic) Communication Disorder

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

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

16.2.4.2. Development and course. Social (pragmatic) communication disorder is not typically diagnosed in children under four years of age. By age 4 or 5 years, most children have obtained adequate speech and language capabilities to allow for the identification of specific deficits in social communication, though milder forms of the disorder may go undetected until early adolescence, when language and social interactions become more complex.

Making Sense of the Disorders

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

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

For more on autism spectrum disorder and social (pragmatic) communication disorder please visit: <https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-8-autism-spectrum-disorder/#8.1>

16.2.5. Stereotypic Movement Disorder

16.2.5.1. Clinical Presentation. Stereotypic movement disorder involves “repetitive, seemingly driven, and apparently purposeless motor behavior” such as hand flapping, body rocking, or hitting one’s own body (APA, 2022, pg. 89). Whether efforts to stop such movements are successful is questionable. For children that are developing typically, directing attention to the movements, or distracting them from performing the movements, are successful. For children with neurodevelopmental disorders, such efforts are less successful. The behavior displayed varies, but each child has their own signature or individually patterned behavior. Duration of the

behavior is a few seconds to several minutes or longer, while the frequency can vary from many occurrences during a single day, to several weeks between episodes. The behaviors can occur during moments of boredom, excitement, stress, fatigue, or when the child is engrossed in other activities.

The stereotypic movements cause impairment in social, academic, or other activities and can result in self-injury. In fact, the mental health professional should specify if stereotypic movement disorder is with or without self-injurious behavior and whether the severity of the disorder is mild, moderate, or severe. A mild presentation is one in which the stereotypic movement is easily suppressed by a sensory stimulus or distraction. Severe presentation would be characterized by continuous movements that interfere with daily living. The onset of the disorder is during the early developmental period, typically within the first three years of life (APA, 2022).

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

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

16.2.5.4. Differential diagnosis: Autism spectrum disorder. Although the repetitive movements of stereotypic movement disorder may be reminiscent of behaviors seen in children

with autism and may develop in the same developmental timeframe (e.g., around or before age 3) as autism spectrum disorder, children with stereotypic movement disorder do not display social communication and reciprocity deficits. Moreover, they do not have circumscribed interests, difficulty with transitions, delayed speech, etc. The only displayed symptom related to autism spectrum disorder is the stereotypy related to behavior. As the DSM says, “When autism spectrum disorder is present, stereotypic movement disorder is diagnosed only when there is self-injury or when the stereotypic behaviors are sufficiently severe to become a focus of treatment” (APA, 2022, pg. 91).

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

16.2.5.6. Differential diagnosis: OCD. The absence of obsessions in stereotypic movement disorder distinguishes it from obsessive-compulsive disorder. As the DSM says, “In OCD the individual feels driven to perform repetitive behaviors in response to an obsession or according to rules that must be applied rigidly, whereas in stereotypic movement disorder the behaviors are seemingly driven but apparently purposeless” (APA, 2022, pg. 92).

OCD-related disorders of trichotillomania (e.g., pulling hair) and excoriation (e.g., picking at skin) may also seem difficult to differentiate from stereotypic movement disorder.

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.

Stereotypic movement disorder has an earlier onset than OCD-related disorders, which tends to be around puberty or later (APA, 2022).

16.2.6. Tic Disorders

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

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

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

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

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

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

Making Sense of the Disorders

In relation to motor disorders, note the following:

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

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

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

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

For more on stereotypic movement and tic disorders please visit:

<https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-9-motor-related-disorders/>

You should have learned the following in this section:

- Intellectual developmental disorder is characterized by intellectual functioning (Criterion A) and adaptive functioning (Criterion B) deficits and they must occur during the developmental period.
- A specific learning disorder is characterized by persistent difficulties learning critical academic skills during the years of formal schooling such as reading of single words accurately and fluently or arithmetic calculation; performance of the affected academic skills being well below expected for age; learning difficulties being apparent in the early school years for most individuals, and that the learning difficulties are considered “specific” (for four reasons).
- The essential features of ASD are twofold and include persistent impairment in reciprocal social communication and social interaction (Criterion A) and restricted, repetitive patterns of behavior, interests, or activities (Criterion B).
- Social (pragmatic) communication disorder is characterized by difficulty with pragmatics or the social use of language and communication.
- Stereotypic movement disorder involves repetitive, seemingly driven, and apparently purposeless motor behavior such as hand flapping, body rocking, or hitting one’s own body.
- According to the DSM, a tic is “a sudden, rapid, recurrent, nonrhythmic motor movement or vocalization.” Tics can be either *motor movements* (motor) or *vocalizations* (vocal) and can be simple or complex.

Section 16.2 Review Questions

1. How does intellectual development disorder present and what specifiers are used?
2. How does specific learning disorder present and what specifiers are used?
3. What distinguishes ASD and social (pragmatic) communication disorder?
4. What is stereotypic movement disorder? What specifiers are used with it?
5. What is a tic?
6. What are the three tic disorders and what makes them different from one another?

16.3. Behavior-related Disorders

Section Learning Objectives

- Describe the clinical presentation, prevalence, and comorbidity of ADHD.
- Describe the clinical presentation, prevalence, and comorbidity of oppositional defiant disorder.
- Describe the clinical presentation, prevalence, and comorbidity of conduct disorder.
- Describe the clinical presentation, prevalence, and comorbidity of intermittent explosive disorder.

16.3.1. Attention Deficit/Hyperactivity Disorder (ADHD)

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

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

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

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

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

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

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

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

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

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

16.3.1.5. Comorbidity. According to the CDC, approximately 60% of children with ADHD have another comorbid disorder (CDC, 2018a, September). About 50% of children with ADHD combined presentation, and about 25% of children with ADHD predominantly inattentive presentation, are also diagnosed with Oppositional Defiant Disorder (ODD). Conduct

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

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

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

16.3.1.6. Differential Diagnosis. ADHD should be distinguished from the following disorders:

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

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

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

16.3.1.6.3. 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).

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

For more on ADHD please visit: <https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-10-attention-deficit-hyperactivity-disorder/>

16.3.2. Oppositional Defiant Disorder

16.3.2.1. Clinical Presentation. **Oppositional defiant disorder** is characterized by a child that is defiant/argumentative, angry/irritable, and vindictive, and has shown this pattern of behavior for at least six months. Of the eight possible symptoms, the child must present with at least four of them. In terms of angry/irritable mood, they may lose their temper often, are easily annoyed or touchy, and are often angry and resentful. In terms of argumentative/defiant behavior the child argues with authority figures, actively defies or refuses to comply, deliberately annoys others, or blames others for their mistakes. Finally, they must have acted spiteful or vindictive at least twice within the past six months. Distress occurs in the child's immediate social context or affects social, occupational, educational, or other important areas of functioning. Functional consequences of these behaviors include frequent conflicts with parents, teachers, supervisors, peers, and romantic partners (APA, 2022). The disorder typically appears during the preschool years and rarely later than early adolescence.

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

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

16.3.2.4. Differential diagnosis. Oppositional defiant disorder should be distinguished from *conduct disorder*. Both disorders bring the individual in conflict with adults and authority figures, but the behaviors of oppositional defiant disorder are usually less severe than conduct

disorder and do not include aggression toward people or animals, destruction of property, or a pattern of theft or deceit. However, the impairment associated with oppositional defiant disorder may be equivalent or greater than that of conduct disorder. Finally, oppositional defiant disorder includes problems of emotional dysregulation which are absent from conduct disorder.

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

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

16.3.3. Conduct Disorder

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

15 symptoms of which the person must present with at least three in the past 12 months, with at least one criterion present in the past 6 months.

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

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

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

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

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

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

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

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

16.3.4. Intermittent Explosive Disorder

16.3.4.1. Clinical Presentation. Intermittent explosive disorder is characterized by recurrent behavioral outbursts which represent a failure to control aggressive impulses. It is manifested by one of the following: 1) verbal or physical aggression toward property, animals, or other individuals which occur twice a week on average, for up to three months; and 2) “...three behavioral outbursts involving damage or destruction of property and/or physical assault involving physical injury against animals or other individuals occurring within a 12-month period” (APA, 2022). The level of aggressiveness displayed by the individual is out of proportion with the experienced provocation or stressors and is not for the purpose of achieving a tangible objective such as money, power, or intimidation. The disorder should not be diagnosed in individuals younger than 6 years.

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

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

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

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

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

For more on oppositional defiant disorder, conduct disorder, and intermittent explosive disorder please visit: <https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-11-oppositional-and-conduct-disorder/>

You should have learned the following in this section:

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

Section 16.3 Review Questions

1. What symptoms are included in the inattention category?
2. What symptoms are included in the hyperactive and impulsive category?
3. When is ADHD most often identified and diagnosed and why?
4. Which of the disorders discussed in this section is the most severe?
5. Which of the disorders is associated with limited prosocial emotions?
6. Which of the disorders is not diagnosed in children under 6 years of age?
7. Which disorder is characterized by being irritable, argumentative, and vindictive?

16.4. Other Disorders

Section Learning Objectives

- Describe the clinical presentation, prevalence, and comorbidity of selective mutism.
- Describe the clinical presentation, prevalence, and comorbidity of separation anxiety disorder.
- Describe the clinical presentation, prevalence, and comorbidity of trichotillomania.
- Describe the clinical presentation, prevalence, and comorbidity of excoriation.

16.4.1. Anxiety Disorders – Selective Mutism

16.4.1.1. Clinical Presentation. Selective mutism is characterized by an absence of speech in particular social situations in which a person is expected to speak, such as at school or work, despite speaking in other situations. This lack of speech is not due to a communication disorder; does not occur exclusively during the course of autism spectrum disorder, schizophrenia, or another psychotic disorder; or due to a lack of knowledge of, or comfort with, the spoken language required in a specific social situation (APA, 2022). For example, a child with selective mutism may speak fluently and freely with a trusted caregiver in the privacy of their home (providing evidence that the child does not have a speech, language, or communication disorder) but produces no speech at school with peers or a teacher. The disturbance must have lasted for at least one month and is often marked by high social anxiety.

According to the DSM, associated features include, “excessive shyness, fear of social embarrassment, social isolation and withdrawal, clinging, compulsive traits, negativism, temper

tantrums, or mild oppositional behavior” (APA, 2022, pg. 222). The onset of the disorder is usually before 5 years but may not become apparent until entry into school.

16.4.1.2. Prevalence. Selective mutism is relatively rare, occurring in only 0.03% to 1.9% of the population and is more common in young children than adolescents and adults. There is conflicting evidence about gender, with some studies/samples showing equal distribution and others showing a higher prevalence in girls. Prevalence does not vary by race/ethnicity.

16.4.1.3. Comorbidity. Selective mutism is highly comorbid with other anxiety disorders, particularly social anxiety disorder followed by separation anxiety disorder and specific phobia. Communication disorders or delays also appear in some children with selective mutism (APA, 2022).

16.4.2. Anxiety Disorders – Separation Anxiety Disorder

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

attachment figure. This fear, anxiety, or avoidance is persistent and has lasted for at least four weeks in children and adolescents. For adults, it must last 6 months or more.

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

16.4.2.2. Prevalence. Separation anxiety disorder occurs in approximately 4% of children and decreases in prevalence from childhood through adolescence and adulthood. For instance, only 1.6% of adolescents in the United States have the disorder and for adults, 12-month prevalence ranges from 0.9% to 1.9%. In toddlers, separation anxiety disorder occurs about equally between girls and boys but in school-aged children, it is more prevalent for girls than boys.

16.4.2.3. Comorbidity. Separation anxiety disorder is highly comorbid with other anxiety disorders, such as generalized anxiety disorder and specific phobia (APA, 2022).

For more on separation anxiety disorder and selective mutism please visit:

<https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-13-anxiety-disorders/>

16.4.3. Obsessive Compulsive and Related Disorders - Trichotillomania

16.4.3.1. Clinical Presentation. Trichotillomania (Hair-Pulling) disorder is characterized by an individual recurrently pulling their hair out and results in hair loss. Despite attempts to cease the behavior, the individual continues with it. These behaviors cause distress and impairment and may also lead to other dermatological/medical conditions (APA, 2022).

Hair pulling may be accompanied by a range of behaviors or rituals such as searching for a particular kind of hair to pull such as those with a specific texture or color, pulling out hair in a specific way so that the root comes out intact, or visually examining or tactilely or orally manipulating the hair after it is pulled out such as pulling the strand between the teeth. Pain does not routinely accompany hair pulling.

It can be triggered by feelings of anxiety, boredom, and/or an increasing sense of tension, and may lead to gratification, pleasure, or a sense of relief once the hair is pulled out. It has been noted that for some, an “itch-like” or tingling sensation occurs and is alleviated once the hair has been pulled out. The act of hair pulling does not usually occur in the presence of other people with the exception of immediate family members. Some even have the urge to pull hair from other people or from pets, dolls, and other fibrous materials (APA, 2022).

Hair pulling can occur in infancy and will typically resolve during early development. Onset, though, most commonly coincides with, or follows, the onset of puberty. The usual course of trichotillomania is chronic with waxing and waning of symptoms being typical if untreated. The disorder manifests similarly across cultures and ethnic groups though there is not much data from non-Western regions.

16.4.3.2. Prevalence. Trichotillomania occurs in 1% to 2% of adults and adolescents. Females are more often diagnosed with this disorder than males, in adulthood. However, in childhood, males and females are equally impacted.

16.4.3.3. Comorbidity. The most common comorbid disorders are MDD and excoriation disorder. Other repetitive body-focused symptoms such as nail-biting occur in most individuals with trichotillomania and may warrant an additional diagnosis of other specified obsessive-compulsive and related disorder (APA, 2022).

16.4.4. Obsessive Compulsive and Related Disorders - Excoriation

16.4.4.1. Clinical Presentation. Excoriation (Skin-Picking) disorder is similar to trichotillomania, except it involves skin-picking which results in skin lesions. Despite attempts to cease the behavior, the individual continues with it. The skin picking causes impairment and may also lead to other medical consequences (APA, 2022).

Skin picking can be triggered by feelings of anxiety, boredom, and/or an increasing sense of tension, and may lead to gratification, pleasure, or a sense of relief once the skin or scab has been picked. It involves behaviors such as searching for a particular kind of scab to pull and examining, playing with, or swallowing the skin after it has been pulled. The act of skin picking usually does not occur in the presence of other people, except immediate family members, and some individuals report picking the skin of others. Pain is not typically associated with skin picking (APA, 2022).

Excoriation disorder usually begins during adolescence, commonly with or following the onset of puberty, and begins with a dermatological condition such as acne. The course is chronic

with some waxing and waning if untreated. It appears clinical features are similar across cultures, though there is limited data currently.

16.4.4.2. Prevalence. In terms of excoriation disorder, an online survey of over 10,000 adults between the ages of 18-69 and gender-matched to the U.S. population found that 2.1% self-identified as having current excoriation disorder while 3.1% reported lifetime excoriation disorder. Community samples show that 75% or more of individuals diagnosed with the disorder are women.

16.4.4.3. Comorbidity. OCD, trichotillomania, and MDD are comorbid with excoriation disorder, with depression comorbidity being more common in women. Other repetitive body-focused symptoms such as nail-biting occur in most individuals with excoriation disorder and may warrant an additional diagnosis of other specified obsessive-compulsive and related disorder (APA, 2022).

For more on trichotillomania and excoriation disorder please visit:

<https://opentext.wsu.edu/behavioral-disorders-childhood/chapter/module-14-obsessive-compulsive-and-related-disorders/>

You should have learned the following in this section:

- Separation anxiety disorder is characterized by excessive fear or anxiety concerning being separated from those to whom the individual is attached.
- Selective mutism is characterized by an absence of speech in particular social situations in which a person is expected to speak, such as at school or work, despite speaking in other situations.
- Trichotillomania (Hair-Pulling) disorder is characterized by an individual recurrently pulling their hair out and results in hair loss.
- Excoriation (Skin Picking) disorder involves skin picking which results in skin lesions.

Section 16.4 Review Questions

1. What is separation anxiety disorder?
2. What is selective mutism and how does it differ from social anxiety disorder?
3. What is the difference between trichotillomania disorder from excoriation disorder? How much pain do both involve for the afflicted?

Module Recap

The purpose of Module 16 was to provide an overview, not exhaustive coverage, of disorders present in childhood and adolescence. This included a discussion of the clinical presentation, prevalence, comorbidity, and differential diagnosis (when relevant) for these disorders. For a more comprehensive discussion, to include a discussion of etiology, assessment, and treatment options, please see our Behavioral Disorders of Childhood book (<https://opentext.wsu.edu/behavioral-disorders-childhood/>).

In this module, we covered:

- Disorders of infancy and early childhood to include disinhibited social engagement disorder, reactive attachment disorder, pica, rumination disorder, and avoidant/restrictive food intake disorder, enuresis, and encopresis.
- Developmental and motor-related disorders to include intellectual developmental disorder (intellectual disability), specific learning disorder, autism spectrum disorder, social (pragmatic) communication disorder, stereotypic movement, and tic disorders.
- Behavior-related disorders to include ADHD, oppositional defiant disorder, conduct disorder, and intermittent explosive disorder.
- Anxiety disorders of selective mutism and separation anxiety disorder
- Obsessive-compulsive and related disorders of trichotillomania, and excoriation disorder.

We hope this overview was helpful as we recognize many universities do not have a class specialized in mental disorders present during these critical developmental periods (i.e., childhood and adolescence).

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

Action potential – When the neuron depolarizes and fires

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

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

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

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

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

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

Alogia - Poverty of speech or speech content

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

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

Anhedonia - Inability to experience pleasure

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

Antecedents - The environmental events or stimuli that trigger a behavior

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

Apathy - General lack of interest

Asociality - Lack of interest in social relationships

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

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

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

Avolition - Lack of motivation of goal-directed behavior

Axon - Sends signals/information to neighboring neurons

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

B

Behavior modification - The process of changing behavior

Behavioral assessment - The measurement of a target behavior

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

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

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

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

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

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

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

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

C

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

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

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

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

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

Classification - The way in which we organize or categorize things

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

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

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

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

Clinical diagnosis - The process of using assessment data to determine if the pattern of symptoms the person presents with is consistent with the diagnostic criteria for a specific mental disorder set forth in an established classification system such as the DSM-5 or ICD-10

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

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

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

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

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

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

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

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

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

Cortisol - A hormone released as a stress response

Counterconditioning - The reversal of previous learning

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

Course – The particular pattern a disorder displays

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

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

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

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

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

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

Degenerative - Meaning the symptoms and cognitive deficits become worse overtime

Deinstitutionalization - The release of patients from mental health facilities

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

E

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

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

Emotional intelligence or **EI** – Is our ability to manage the emotions of others as well as ourselves and includes skills such as empathy, emotional awareness, managing emotions, and self-control

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

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

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

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

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

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

Etiology - The cause of the disorder

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 spirits were cast out through prayer, magic, flogging, starvation, having the person ingest horrible tasting drinks, or noise-making

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

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

F

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

Fixed Interval schedule (FI) – With a FI schedule, you will reinforce after some set amount of time

Fixed Ratio schedule (FR) – With this schedule, we reinforce some set number of responses

Flooding - Exposing the person to the maximum level of stimulus and as nothing aversive occurs, the link between CS and UCS producing the CR of fear should break, leaving the person unafraid

Forensic psychology/psychiatry - When clinical psychology is applied to legal arena in terms of assessment, treatment, and evaluation

Free association – In psychoanalytic theory, this technique involves the patient describing whatever comes to mind during the session

Frontal lobe – Part of the cerebrum that contains the motor cortex which issues orders to the muscles of the body that produce voluntary movement

Frontotemporal NCD - Causes progressive declines in language or behavior due to the degeneration in the frontal and temporal lobes of the brain; symptoms include significant changes in behavior and/or language

Fundamental attribution error - Occurs when we automatically assume a dispositional reason for another person's actions and ignore situational factor

G

GABA – Neurotransmitter responsible for blocking the signals of excitatory neurotransmitters responsible for anxiety and panic

Gaps - Holes in the literature of a given area

Generalizability – Begin able to apply your findings for the sample to the population

Generalized dissociative amnesia – A type of dissociative amnesia in which the person has a complete loss of memory of their entire life history, including their own identity

Generalized anxiety disorder - The most common anxiety disorder characterized by a global and persistent feeling of anxiety

Genital Stage – Beginning at puberty, sexual impulses reawaken and unfulfilled desires from infancy and childhood can be satisfied during lovemaking

Glial cells - The support cells in the nervous system that serve five main functions: as a glue and hold the neuron in place, form the myelin sheath, provide nourishment for the cell, remove waste products, and protect the neuron from harmful substances

Glutamate – Neurotransmitter associated with learning and memory

Grandiose delusion - Involves the conviction of having a great talent or insight

H

Habituation - When we simply stop responding to repetitive and harmless stimuli in our environment

Hippocampus - Our “gateway” to memory; Allows us to form spatial memories so that we can accurately navigate through our environment and helps us to form new memories about facts and events

Histrionic personality disorder - Addresses the pervasive and excessive need for emotion and attention from others; these individuals are often uncomfortable in social settings unless they are the center of attention

Hoarding – Focused on the persistent over-accumulation of possessions

Hypertension - -Chronically elevated blood pressure

Hypomanic episode - Persistently elevated, expansive, or irritable mood; May present as persistent increased activity or energy; Symptoms last at least 4 consecutive days and present most of the day, nearly every day; Includes at least three of the following: inflated self-esteem or grandiosity, decreased need for sleep, more talkative or pressured speech, flight of ideas, distractibility, increase in goal-directed activity or psychomotor agitation, or excessive involvement in activities that have a high potential for painful consequences

Hypothalamic-pituitary-adrenal (HPA) axis - Involved in the fear producing response and may be involved in the development of trauma symptoms

Hypothalamus – The part of the brain involved in drives associated with the survival of both the individual and the species; It regulates temperature by triggering sweating or shivering, and controls the complex operations of the autonomic nervous system

Hypothesis – A specific, testable prediction

Humanism - The worldview that emphasizes human welfare and the uniqueness of the individual

I

Id – According to Freud, is the impulsive part of personality that expresses our sexual and aggressive instincts

Ideas of reference - The belief that unrelated events pertain to them in a particular and unusual way

Identification – This is when we find someone who has found a socially acceptable way to satisfy their unconscious wishes and desires and we model that behavior

Illness anxiety disorder - Previously known as hypochondriasis, involves the excessive preoccupation with having or acquiring a serious medical illness

Incidence - The number of new cases in a population over a specific period of time

Independent variable (IV) – In an experiment, the variable that is manipulated

Inferential statistics – Statistics which allow for the analysis of two or more sets of numerical data

Insomnia - The difficult falling or staying asleep

Intellectualization- When we avoid emotion by focusing on intellectual aspects of a situation

Intelligence tests - Used to determine the patient's level of cognitive functioning and consists of a series of tasks asking the patient to use both verbal and nonverbal skills

Intermittent explosive disorder - Characterized by recurrent behavioral outbursts which represent a failure to control aggressive impulses

Ions - Charged particles found both inside and outside the neuron

Irritable bowel syndrome (IBS) - A chronic, functional disorder of the gastrointestinal tract including symptoms such as abdominal pain and extreme bowel habits (diarrhea and/or constipation)

J

Jealous delusion - Revolves around the conviction that one's spouse or partner is/has been unfaithful

K

L

Laboratory observation - A research method in which the scientist observes people or animals in a laboratory setting

Latency Stage – From 6-12 years of age, children lose interest in sexual behavior and boys play with boys and girls with girls

Latent content - The hidden or symbolic meaning of a dream

Law of effect (Thorndike, 1905) - The idea that if our behavior produces a favorable consequence, in the future when the same stimulus is present, we will be more likely to make the response again, expecting the same favorable consequence

Learning - Any relatively permanent change in behavior due to experience

Libido - The psychic energy that drives a person to pleasurable thoughts and behaviors

Lifetime prevalence - Indicates the proportion of a population that has had the characteristic at any time during their lives

Literature review - When we conduct a literature search through our university library or a search engine such as Google Scholar to see what questions have been investigated already and what answers have been found

Localized amnesia - The most common type of dissociative amnesia, is the inability to recall events during a specific period of time

M

Major Depressive Disorder – A mood disorder characterized by depressed mood most of the day or decreased interest or pleasure in all or most activities most of the day, along with insomnia or hypersomnia, fatigue, feelings of worthlessness, or difficulty concentrating to name a few symptoms; symptoms occur during a two week period

Major neurocognitive disorder – Individuals with the disorder show significant decline in both overall cognitive functioning as well as the ability to independently meet the demands of daily living such as paying bills, taking medications, or caring for oneself

Manic episode - Persistent elevated, expansive, or irritable mood. May present as persistent increased goal-directed activity or energy; Symptoms **last at least 1 week** and present most of the day, nearly

every day; includes three of the following: inflated self-esteem or grandiosity, decreased need for sleep, more talkative or pressured speech, flight of ideas, distractibility, increase in goal-directed activity or psychomotor agitation, or excessive involvement in activities that have a high potential for painful consequences

Manifest content - The person's actual retelling of the dream

Mass madness – or Group hysteria; When large numbers of people display similar symptoms and false beliefs; a term used during the Middle Ages

Medulla – The part of the brain that regulates breathing, heart rate, and blood pressure

Melatonin - A hormone released when it is dark outside to assist with the transition to sleep

Mental disorders - Characterized by psychological dysfunction which causes physical and/or psychological distress or impaired functioning and is not an expected behavior according to societal or cultural standards

Mental health epidemiology - Refers to the occurrence of mental disorders in a population

Mental hygiene movement - An idea arising in the late 18th century to the early 19th century with the fall of the moral treatment movement, it focused on the physical well-being of patients

Mental status examination - Used to organize the information collected during the clinical interview and systematically evaluates the patient through a series of questions assessing appearance and behavior to include grooming and body posture, thought processes and content to include disorganized speech or thought and false beliefs, mood and affect such that whether the person feels hopeless or elated, intellectual functioning to include speech and memory, and awareness of surroundings to include where the person is and what the day and time are

Migraine headaches - Headaches explained by a throbbing pain localized to one side of the head and often accompanied by nausea, vomiting, sensitivity to light, and vertigo

Model - A representation or imitation of an object

Modeling - Techniques used to change behavior by having subjects observe a model in a situation that usually causes them some anxiety

Moral treatment movement – An idea arising in Europe in the late 18th century and then in the United States in the early 19th century, it stressed affording the mentally ill respect, moral guidance, and humane treatment, all while considering their individual, social, and occupational needs

Myelin sheath - The white, fatty covering which: 1) provides insulation so that signals from adjacent neurons do not affect one another and, 2) increases the speed at which signals are transmitted

Multicultural psychology – The area of psychology which attempts to understand how the various groups, whether defined by race, culture, or gender, differ from one another

Multi-dimensional model – An explanation for mental illness that integrates multiple causes of psychopathology and affirms that each cause comes to affect other causes over time

N

Narcissistic personality disorder - Individuals display a pattern of grandiosity along with a lack of empathy for others

Naturalistic observation - A research method in which the scientist studies human or animal behavior in its natural environment which could include the home, school, or a forest

Negative Punishment (NP) – This is when something good is taken away or subtracted making a behavior less likely in the future

Negative Reinforcement (NR) – This is when something bad or aversive is taken away or subtracted due to your actions, making it that you will be more likely to make the same behavior in the future when the same stimuli presents itself

Negative symptoms – The inability or decreased ability to initiate actions, speech, expressed emotion, or to feel pleasure

Nerves - A group of axons bundled together like wires in an electrical cable

Neurological tests - Used to diagnose cognitive impairments caused by brain damage due to tumors, infections, or head injury; or changes in brain activity

Neuron - The fundamental unit of the nervous system

Neurotransmitter – When the actual code passes from one neuron to another in a chemical form

Nomenclature – A naming system

Norepinephrine – Neurotransmitter which increases the heart rate and blood pressure and regulates mood

Nucleus - The control center of the body

Q

Observation – Observing others either naturalistically or in a controlled environment

Observational learning - When we learn by observing the world around us

Obsessions - Repetitive and persistent thoughts, urges, or images

Obsessive compulsive disorder - More commonly known as OCD, the disorder requires the presence of both obsessions and compulsions

Obsessive-Compulsive personality disorder - Defined by an individual's preoccupation with orderliness, perfectionism, and ability to control situations that they lose flexibility, openness, and efficiency in everyday life

Operant conditioning - A type of associate learning which focuses on consequences that follow a response or behavior that we make (anything we do, say, or think/feel) and whether it makes a behavior more or less likely to occur

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

Paranoid personality disorder - Characterized by a marked distrust or suspicion of others

Parasympathetic nervous system – The part of the autonomic nervous system that calms the body after sympathetic nervous system arousal

Parietal lobe – The part of the cerebrum that contains the somatosensory cortex and receives information about pressure, pain, touch, and temperature from sense receptors in the skin, muscles, joints, internal organs, and taste buds

Peripheral nervous system - Consists of everything outside the brain and spinal cord; It handles the CNS's input and output and divides into the somatic and autonomic nervous systems

Period prevalence - Indicates the proportion of a population that has the characteristic at any point during a given period of time, typically the past year

Persecutory delusion - Involves the individual believing that they are being conspired against, spied on, followed, poisoned or drugged, maliciously maligned, harassed, or obstructed in pursuit of their long-term goals

Persistent (chronic) motor or vocal tic disorder - When either one or more motor tics or one or more vocal tic is present

Persistent Depressive Disorder – A mood disorder characterized by poor appetite or overeating, insomnia or hypersomnia, low self-esteem, low energy, and feelings of hopelessness lasting most of the day, for more days than not, for at least 2 years

Personality disorders - Have four defining features which include distorted thinking patterns, problematic emotional responses, over- or under- regulated impulse control, and interpersonal difficulties

Personality inventories - Ask clients to state whether each item in a long list of statements applies to them, and could ask about feelings, behaviors, or beliefs

Personality traits - Enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personality contexts

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

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 event

Positive psychology – The position in psychology that holds a more positive conception of human potential and nature

Positive Punishment (PP) – If something bad or aversive is given or added, then the behavior is less likely to occur in the future

Positive Reinforcement (PR) – If something good is given or added, then the behavior is more likely to occur in the future

Positive symptoms - Symptoms that are an over-exaggeration of normal brain processes

Preconscious – According to Freud, the level of personality that includes all of our sensations, thoughts, memories, and feelings

Presenting problem – The issue the person displays

Prevalence - The percentage of people in a population that has a mental disorder or can be viewed as the number of cases per some number of people

Prevention – When we identify the factors that cause specific mental health issues and implement interventions to stop them from happening, or at least minimize their deleterious effects

Prognosis - The anticipated course the mental disorder will take

Projection – When we attribute threatening desires or unacceptable motives to others

Projective tests – A psychological test which consists of simple ambiguous stimuli that can elicit an unlimited number of responses

Psychoanalysis - Psychoanalytic therapy used to understand the personality of a therapist's patient and to expose repressed material

Psychological debriefing - A type of crisis intervention that requires individuals who have recently experienced a traumatic event to discuss or process their thoughts and feelings related to the traumatic event, typically within 72 hours of the event

Psychological model – includes learning, personality, stress, cognition, self-efficacy, and early life experiences and how they affect mental illness

Psychological or psychogenic perspective - States that emotional or psychological factors are the cause of mental disorders and represented a challenge to the biological perspective

Psychological tests - Used to assess the client's personality, social skills, cognitive abilities, emotions, behavioral responses, or interests and can be administered either individually or to groups in paper or oral fashion

Psychopathology - The scientific study of psychological disorders

Psychosis - A loss of contact with reality

Public stigma – When members of a society endorse negative stereotypes of people with a mental disorder and discriminate against them

Punishment – Due to the consequence, a behavior/response is less likely to occur in the future

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

Reactive Attachment Disorder – A child with RAD presents as detached from others and like DSED, often experience impaired caregiving.

Reaction formation – When an impulse is repressed and then expressed by its opposite

Reactivity – When the observed changes behavior due to realizing they are being observed

Receptor sites – Locations where neurotransmitters bind to

Reinforcement – Due to the consequence, a behavior/response is more likely to occur in the future

Reinforcement schedule - The rule for determining when and how often we will reinforce a desired behavior

Relative refractory period - After a short period of time, the neuron can fire again, but needs greater than normal levels of stimulation to do so

Regression – When we move from a mature behavior to one that is infantile in nature

Reliable – When our assessment is consistent

Replication - Repeating a study to confirm its results

Repolarization – When the Na channels close and Potassium channels open; K has a positive charge and so the neuron becomes negative again on the inside and positive on the outside, or polarizes

Repression – When unacceptable ideas, wishes, desires, or memories are blocked from consciousness

Research design - Our plan of action of how we will go about testing the hypothesis

Resistance – According to psychoanalytic theory, is the point during free association that the patient cannot or will not proceed any further

Respondent conditioning (also called classical or Pavlovian conditioning) - Occurs when we link a previously neutral stimulus with a stimulus that is unlearned or inborn

Respondent Discrimination – When the CR is elicited by a single CS or a narrow range of CSs

Respondent Extinction – When the CS is no longer paired with the UCS

Respondent Generalization – When a number of similar CSs or a broad range of CSs elicit the same CR

Resting potential – When the neuron is waiting to fire

Reticular formation – The part of the brain responsible for alertness and attention

Reuptake reuptake - The process of the presynaptic neuron taking up excess neurotransmitters in the synaptic space for future use

Reversal or ABAB design – A study in which the control is followed by the treatment, and then a return to control and second administration of the treatment condition; builds replication in to the design

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

Schizoaffective disorder - Characterized by the psychotic symptoms included in criteria A of schizophrenia *and* a concurrent uninterrupted period of a major mood episode—either a depressive or manic episode

Schizoid personality disorder - Displays a persistent pattern of avoidance from social relationships along with a limited range of emotion among social relationships

Schizophrenia – A mental disorder that includes the presentation of at least two of the following for at least one month: delusions, hallucinations, disorganized speech, disorganized/abnormal behavior, or negative symptom

Schizophreniform Disorder – A mental disorder characterized by at least two of the following: delusions, hallucinations, disorganized speech, disorganized/abnormal behavior, and/or negative symptoms

Schizotypal personality disorder - Characterized by a range of impairment in social and interpersonal relationships due to discomfort in relationships, along with odd cognitive and/or perceptual distortions and eccentric behaviors

Scientific method - A systematic method for gathering knowledge about the world around us

Sedative-Hypnotic drugs - More commonly known as anxiolytic drugs, these drugs have a calming and relaxing effect on individuals

Selective amnesia - Is in a sense, a component of localized amnesia in that the individual can recall some, but not all, of the details during a specific time period

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 delusion - Involves delusions regarding bodily functions or sensations

Somatic nervous system - Allows for voluntary movement by controlling the skeletal muscles and carries sensory information to the CNS

Somatic Symptom Disorder – A somatic symptom or related disorder characterized by disproportionate and persistent thoughts of the seriousness of the symptom, high levels of anxiety about the symptom, and/or excessive time/energy spent focused on the symptom

Specific phobia - Observed when an individual experiences anxiety related to a specific object or subject

Spontaneous recovery – When the CS elicits the CR after extinction has occurred

Standardization – When we use clearly laid out rules, norms, and/or procedures in the process of assessing client's

Statistical significance - An indication of how confident we are that our results are due to our manipulation or design and not chance

Stereotypic Movement Disorder - A disorder in which an individual engages in repetitive movements and those movements have no clear functional purpose

Stigma - When negative stereotyping, labeling, rejection, and loss of status occur

Stressors - Any event- either witnessed firsthand, experienced personally or experienced by a close family member- that increases physical or psychological demands on an individual

Sublimation – When we find a socially acceptable way to express a desire

Substance abuse - Occurs when an individual consumes the substance for an extended period of time, or has to ingest large amounts of the substance to get the same effect a substance provided previously

Substance Intoxication – A substance use disorder characterized by recent ingestion of substance, significant behavioral or psychological changes immediately following the ingestion of substance, physical and physiological symptoms develop after ingestion of substance, and changes in behavior not attributable to a medical condition or other psychological disorder

Substance Use Disorder – A substance use disorder diagnosed when the individual presents with at least two criteria to include: substance is consumed in larger amounts over time, desire or inability to reduce quantity of substance use, cravings for substance use, use of the substance in potentially hazardous situations, tolerance of substance use, and withdrawal, to name a few (11 total criteria)

Substance Withdrawal - A substance use disorder characterized by cessation or reduction in substance that has been previously used for a long or heavy period of time, physiological and/or psychological symptoms within a few hours after cessation/reduction, physiological and/or psychological symptoms cause significant distress or impairment in functioning, and symptoms not attributable to a medical condition or other psychological disorder

Substances - Any ingested materials that cause temporary cognitive, behavioral, and/or physiological symptoms within the individual

Superego - According to Freud, the part of personality which represents society's expectations, moral standards, rules, and represents our conscience

Sympathetic nervous system - Involved when a person is intensely aroused; It provides the strength to fight back or to flee (fight-or-flight instinct)

Synapse - The point where the code passes from one neuron to another; Consists of three parts – the *axon* of the sending neuron; the *space* in between called the synaptic space, gap, or cleft; and the *dendrite* of the receiving neuron

Syndrome - Symptoms occurred regularly in clusters

Systematized amnesia - When an individual fails to recall a specific category of information

T

Target behavior - Whatever behavior we want to change and it can be in excess or needing to be reduced, or in a deficit state and needing to be increased

Tension headaches - Often described as a dull, constant ache that is localized to one part of the head/neck; however, it can co-occur in multiple places at one time

Thalamus – The major sensory relay center for all senses but smell

Thanatos - Our death instinct which is either directed inward as in the case of suicide and masochism or outward via hatred and aggression

Thematic Apperception Test – A projective test which asks the individual to write a complete story about each of 20 cards shown to them and give details about what led up to the scene depicted, what the characters are thinking, what they are doing, and what the outcome will be

Theory – A systematic explanation of a phenomenon

Threshold of excitation - -55mV or the amount of depolarization that must occur for a neuron to fire; It rises from -70mV to -55mV

Thyroid gland – The endocrine gland which regulates the body's rate of metabolism and so how energetic people are.

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 transferring to the therapist attitudes he/she held during childhood

Trauma-focused cognitive-behavioral therapy (TF-CBT) - An adaptation of CBT, that utilizes both CBT techniques, as well as trauma sensitive principles to address the trauma related symptoms

Treatment - Any procedure intended to modify abnormal behavior into normal behavior

Trephination - In which a stone instrument known as a *trephine* was used to remove part of the skull, creating an opening

Trial and error learning - Making a response repeatedly if it leads to success

Trichotillomania - Characterized by an individual recurrently pulling their hair out and results in hair loss

U

Ulcers - Or painful sores in the stomach lining, occur when mucus from digestive juices are reduced, thus allowing digestive acids to burn a hole into the stomach lining

Unconscious – According to Freud, the level of personality not available to us

Uni-dimensional model – A single factor explanation for mental illness

V

Validity – When the test measures what it says it measures

Variable Interval schedule (VI) – Reinforcing at some changing amount of time

Variable Ratio schedule (VR) – Reinforcing some varying number of responses

W

X

Y

Z

References

- Abass, A., Kisely, S., Town, J., Leichsenring, F., Driessen, E., DeMatt, S., ... Crowe, E. (2014). Short-term psychodynamic psychotherapies for common mental disorders. *Cochrane Database of Systematic Reviews*, 7, CD004687.
- Achalia, R., Chaturvedi, S., Desai, G., Rao, G., & Prakash, O. (2014). Prevalence and risk factors associated with tardive dyskinesia among Indian patients with schizophrenia. *Asian Journal of Psychiatry*, 9, 31-35.
- Acosta, M., Haller, D., & Schnoll, S. (2011). Cocaine and stimulants. In R.J. Frances, A.H. Mack, & S.I. Miller (Eds.), *Clinical textbook of addictive disorders* (3rd ed. pp. 183-218). New York, NY: Guilford Press.
- Addiction Centers of America – Differences between crack & Cocaine. Available at <https://americanaddictioncenters.org/cocaine-treatment/differences-with-crack/>
Accessed on November 20, 2017.
- Advokat, C., Comaty, J., Julien, R. (2014). *Julien's primer of drug reaction*. New York: Worth Publishers.
- Agras, W., Fitzsimmons-Craft, E., & Wilfley, D. (2017). Evolution of cognitive-behavioral therapy for eating disorders. *Behaviour Research and Therapy*, 88, 26-36.
- Agras, W., Lock, J., Brandt, H., Bryson, S., Dodge, E., Halmi, K., ... Woodside, B. (2014). Comparison of 2 family therapies for adolescent anorexia nervosa: a randomized parallel trial. *Journal of American Medical Association Psychiatry*, 71(11), 1279-1286.
- Ahern, G., Herring, A., Labiner, D., Weinand, M., & Hutzler, R. (2000). Affective self-report

- during the intracarotid sodium amobarbital test: Group differences. *Journal of the International Neuropsychological Society*, 6(6), 659-667.
- Ahmed, I., Cook, T., Genen, L., & Schwartz, R. (2014). Body dysmorphic disorder. Retrieved from <http://emedicine.medscape.com/article/291182overview0aw2aab6b2b3>.
- Alcoholics Anonymous 2014 Membership Survey*. (2014). AA World Services.
- Alla, S., Sullivan, S., & McCrory, P. (2012). Defining asymptomatic status following sports concussion: Fact or fallacy? *British Journal of Sports Medicine*, 46(8), 562-569.
- Allen, A. J., Leonard, H. L., & Swedo, S. E. (1995). Case study: a new infection-triggered, autoimmune subtype of pediatric OCD and Tourette's syndrome. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34(3), 307-311.
- Alter, C. (2014, March 10). Holder urges use of drug to combat heroin overdoses. *Time*.
- Alwin, N. (2006). The causes of personality disorder. In M. Sampson, R. McCubbin, & P. Tyrer (Eds.), *Personality disorder and community mental health teams: A practitioner's guide*. Sussex, England: John Wiley & Sons, Ltd.
- Alzheimer's Association. (2017a). Medications for memory loss. Retrieved from: https://www.alz.org/alzheimers_disease_standard_prescriptions.asp
- Alzheimer's Association. (2017b). Alzheimer's and dementia caregiver support groups. Retrieved from: <https://www.alz.org/care/alzheimers-early-mild-stage-caregiving.asp>
- American Academy of Child & Adolescent Psychiatry. (2013). *Marijuana and Teens*. Available at http://www.aacap.org/AACAP/Families_and_Youth/Facts_for_Families/FFF-Guide/Marijuana-and-Teens-106.aspx Accessed on November 20, 2017.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders*. (5th ed.). Arlington, VA: American Psychiatric Publishing.

- American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders*. (5th ed. Text Revision). Arlington, VA: American Psychiatric Publishing.
- American Stroke Association. (2017). Types of strokes. Retrieved from:
http://www.strokeassociation.org/STROKEORG/AboutStroke/TypesofStroke/Types-of-Stroke_UCM_308531_SubHomePage.jsp
- APA (American Psychiatric Association). (2000). *Diagnostic and statistical manual of mental disorders- Text revision* (4th ed.). Washington, DC: Author.
- Arango, C., & Carpenter, W. (2010). The schizophrenia construct: Symptomatic presentation. In D. Weinberger & P. Harrison (Eds), *Schizophrenia* (9-23). Oxford, UK: Wiley-Blackwell.
- Astbury, J. (2010). The social causes of women's depression: A question of rights violated? In D.C., Jack & A. Ali (Eds.), *Silencing the self across cultures: Depression and gender in the social world* (pp. 19-45). New York: Oxford University Press.
- Ayd, F. (1956). A clinical evaluation of Frenquel. *Journal of Nervous and Mental Disease*, 124, 507-509.
- Balakrishnan, S., & Alias, A. (2017). Usage of Social Stories in Encouraging Social Interaction of Children with Autism Spectrum Disorder. *Journal of ICSAR*, 1(2), 91-97.
- Bales, D., Timman, R., Andrea, H., Bussch-bach, J., Verheul, R., & Kamphuis, J. (2014). Effectiveness of day hospital mentalization-based treatment for patients with severe borderline personality disorder: A matched control study. *Clinical Psychology & Psychotherapy*, 22, 409-417.
- Barkley, R. A. (2015). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment*. (4th ed). Charleston, SC: Guilford Press
- Barlow., M. & Chu, J. (2014). Measuring fragmentation in dissociative identity disorder: The

- integration measure and relationship to switching and time in therapy. *European Journal of Psychotraumatology*, 5, DOI: [10.3402/ejpt.v5.22250](https://doi.org/10.3402/ejpt.v5.22250)
- Barnes, T. & Marder, S. (2011). Principles of pharmacological treatment in schizophrenia. In D.R. Weinberg & P. Harrison (Eds). *Schizophrenia* (pp. 515-524). Hoboken, NJ: Humana Press.
- Bauer, M. & Pfennig, A. (2005). Epidemiology of Bipolar Disorders. *Official Journal of the International League Against Epilepsy*, 46, 8-13.
- Baugh, C., Stamm, J., Riley, D., Gavett, B., Shenton, M., Lin, A., ... Stern, R. (2012). Chronic traumatic encephalopathy: Neurodegeneration following repetitive concussive and subconcussive brain trauma. *Brain Imaging and Behavior*, 6, 244-254.
- Bebbington, P. & Kuipers, E. (2011). Schizophrenia and psychosocial stresses. In D. Weinberger & P. Harrison (Eds). *Schizophrenia* (pp. 594-624). Hoboken, NJ: Humana Press.
- Beck, A. (1967). *Depression: Causes and treatment*. Philadelphia: University of Pennsylvania Press.
- Beck, A. (1991). Cognitive therapy: A 30-year retrospective, *American Psychologist*, 46(4), 368-375.
- Beck, A. (2002). Cognitive models of depression. In R. L. Leahy & E.T. Dowd (Eds). *Clinical advance sin cognitive psychotherapy: Theory and application* (pp. 29-61). New York: Springer.
- Beck, A. (2015). Theory of personality disorder. In A. Beck, D. Davis, & A. Freeman (Eds.), *Cognitive therapy of personality disorders*. (pp. 19-62). New York, NY: Guilford Press.
- Beck, A., & Rector, N. (2005). Cognitive approaches to schizophrenia: Theory and Therapy. *Annual Review of Clincial Psychology*, 1, 577-606.
- Beck, A. & Weishaar, M. (2011). Cognitive therapy. In R. Corsini & D. Wedding (Eds.),

- Current psychotherapies* (9th ed.). Belmont, CA: Brooks/Cole.
- Beck, A. & Weishaar, M. (2014). Cognitive therapy. In D. Wedding & R. Corsini (Eds.), *Current psychotherapies* (10th ed.). Independence, KY: Cengage Publishing.
- Belendiuk, K. & Riggs, P. (2014). Treatment of adolescent substance use disorders. *Current Treatment Options in Psychiatry, 1*, 175-188.
- Bellack, A. Morrison, R., Wixted, J. & Mueser, K. (1990). An analysis of social competence in schizophrenia. *British Journal of Psychiatry, 156*, 809-818.
- Bender, E. (2004). Data show wide variation in addiction treatment costs. *Psychiatric News, 39*, 11.
- Berkowitz, M. W., & Bier, M. C. (2007). What works in character education. *Journal of Research in Character Education, 5*(1), 29.
- Bernardy, N. & Friedman, M. (2015). Psychopharmacological strategies in the management of posttraumatic stress disorder (PTSD): What have we learned? *Current Psychiatry Report, 17*, 20-30.
- Bettis, A. H., Coiro, M. J., England, J., Murphy, L. K., Zelkowitz, R. L., Dejardins, L., ... & Compas, B. E. (2017). Comparison of two approaches to prevention of mental health problems in college students: enhancing coping and executive function skills. *Journal of American college health, 1*-10.
- Beucke, J., Sepulcre, J., Talukdar, T., Linnman, C., Zehenderliein, K., Endrass, T., . . . Kathmann, N. (2013). Abnormally high degree connectivity of the orbitofrontal cortex in obsessive-compulsive disorder. *American Journal of Psychiatry, 70*, 619-629.
- Bienvenu, O., Davydow, D., & Kendler, K. (2011). Psychiatric “diseases” vs. behavioral disorders and degree of genetic influence. *Psychological Medicine, 41*, 33-40.

- Bigard, A. (2010). Risks of energy drinks in youth. *Archives of Pediatrics*, *17*, 1625-1631.
- Bisaga, A., Sullivan, M., Glass, A., Mishlen, K., Pavlicova, M. Haney, M., ... Nunes, E. (2015). The effects of dronabinol during detoxification and the initiation of treatment with extended release naltrexone. *Drug and Alcohol Dependence*, *154*, 38-45.
- Bjornsson, A., Dyck, I., Moitra, E., Stout, R., Weisberg, R., Keller, M., & Phillips, K. (2011). The clinical course of body dysmorphic disorder in the Harvard/Brown Anxiety Research Project (HARP). *Journal of Nervous and Mental Disease*, *199*, 55-57.
- Black, D. (2015). The natural history of antisocial personality disorder. *Canadian Journal of Psychiatry*, *60*, 309-314.
- Black, M., Basile, K., Breiding, M., Smith, S., Walters, M., Merrick, M., . . . Stevens, M. (2011). *The National Intimate Partner and Sexual Violence Survey: 2010 summary report*. Retrieved from the Centers for Disease Control and Prevention, National Center for Injury Prevention and Control:
http://www.cdc.gov/ViolencePrevention/pdf/NISVS_Report2010-a.pdf
- Blaze, D. (2013). Neurocognitive disorders in DSM-5. *American Journal of Psychiatry*, *170*, 585-587.
- Boileau B. (2011). A review of obsessive-compulsive disorder in children and adolescents. *Dialogues in clinical neuroscience*, *13*(4), 401–411.
- Bokor, G. & Anderson, P. (2014). Obsessive compulsive disorder. *Journal of Pharmacy Practice*, *27*, 116-130.
- Borkovec, T. D., & Ruscio, A. M. (2001). Psychotherapy for generalized anxiety disorder. *The Journal of Clinical Psychiatry*, *62*, 37-42.
- Bourin, M., Malinge, M., & Guitton, B. (1995). Provocative agents in panic disorder. *Therapie*,

50(4), 301-306.

- Bradshaw, C. P. (2015). Translating research to practice in bullying prevention. *American Psychologist, 70*(4), 322.
- Brambrink, D. (2004). A comparative study for the treatment of anxiety in women using electromyographic biofeedback and progressive muscle relaxation and coping with stress: A manual for women. *Dissertation Abstracts International: Section B: The Science and Engineering, 65*, 3146.
- Brand, B., Lanius, R., Vermetten, E., Loewenstein, R. & Spiegel, D. (2012). Where are we going? An update on assessment, treatment, and neurobiological research in dissociative disorders as we move toward the DSM-5. *Journal of Trauma Dissociation, 13*(1), 9-13.
- Brewin, C., Andrews, B., Rose, S., & Kirk, M. (1999). Acute stress disorder and posttraumatic stress disorder in victims of violent crime. *American Journal of Psychiatry, 156*, 360-366.
- Brown, A. S., Begg, M. D., Gravenstein, S., Schaefer, C. A., Wyatt, R. J., Bresnahan, M., ... & Susser, E. S. (2004). Serologic evidence of prenatal influenza in the etiology of schizophrenia. *Archives of general psychiatry, 61*(8), 774-780.
- Brown, S. (1985). Reinforcement expectancies and alcoholism treatment outcome after a one-year follow-up. *Journal of Studies on Alcohol, 46*, 304-308,
- Brownstein, M. (1993). A brief history of opiates, opioid peptides, and opioid receptors. *Proceedings of the National Academy of Sciences, 90*, 5391-5393.
- Brugger, S. & Howes, O. (2017). Heterogeneity and homogeneity of regional brain structure in schizophrenia: A meta-analysis. *Journal of American Medical Association Psychiatry, 74*, 1104-1111.
- Bryant, R. (2010). The complexity of complex PTSD. *American Journal of*

- Psychiatry*, 167, 879-881.
- Bryant, R., Friedman, M., Spiegel, D., Ursano, R., & Strain, J. (2010). A review of acute stress disorder in DSM-5. *Depression and Anxiety*, 28, 802-817.
- Bryant, R. & Harvey, A. (1998). The relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *American Journal of Psychiatry*, 155, 625-629.
- Burkett, K., Morris, E., Manning-Courtney, P., Anthony, J., & Shambley-Ebron, D. (2015). African American families on autism diagnosis and treatment: The influence of culture. *Journal of autism and developmental disorders*, 45(10), 3244-3254.
- Cahill, S., Rothbaum, B., Resick, P., & Follette, V. (2009). Cognitive-behavioral therapy for adults. In Foa, E., Keane, T., Friedman, M., & Cohen, J. (Eds). *Effective Treatments of PTSD*, (pp. 139-222). New York, NY: Guilford Press.
- Caligor, E., & Clarkin, J. (2010). An object relations model fo personality and personality pathology. In J. Clarkin, P. Fonagy, & G. Gabbard (Eds.), *Psychodynamic psychotherapy for personality disorders: A clinical handbook* (pp. 3-36). Arlington, VA: American Psychiatric Publishing.
- Call, C., Walsh, T., & Attia, E. (2013). From DSM-IV to DSM-5: changes to eating disorder diagnoses. *Current Opinions on Psychiatry*, 26(6), 532-536.
- Campbell, K. & Peebles, R. (2014). Eating disorders in children and adolescents: state of the art review. *Pediatrics*, 134(3), 582-592.
- Capezzani, L., Ostacoli, L., Cavallo, M., Carletto, S., Fernandez, I., Solomon, R., . . . Cantelmi, T. (2013). EMDR and CBT for cancer patients: Comparative study of effects on PTSD, anxiety, and depression. *Journal of EMDR Practice and Research*, 7, 134-143.

- Carey, G. & Gottesman, I., Twin and family studies of anxiety, phobic, and obsessive compulsive disorders. In D. F. Klein & J. Rabkin (Eds.), *Anxiety: New research and changing concepts* (pp. 117-136). New York: Raven Press.
- Casey, E. History of Drug Use and Drug Users in the United States. Retrieved from www.druglibrary.org/schaffer/history/casey1.htm Accessed on November 4, 2017.
- Celani, P. (2014). A Fairbairnian structural analysis of the narcissistic personality disorder. *Psychoanalytic Review*, 101(3), 385-409.
- Center for Disease Control and Prevention Web-Based Injury Statistics Query and Reporting System. (2013). Leading causes of death reports. Retrieved from http://www.cdc.gov/injury/wisqars/fatal_injury_reports.htm Accessed on November 4, 2017.
- Centers for Disease Control and Prevention. (2018, October). *Tourette syndrome: Data & statistics*. Retrieved from <https://www.cdc.gov/ncbddd/tourette/data.html>
- Chemerinksi, E., & Siever, L. (2011). The schizophrenia spectrum personality disorders. In D. Weinberger & P. Harrison (Eds.). *Schizophrenia*, Hoboken, NJ: Wiley-Blackwell.
- Chen, E., Weissman, J., Zeffiro, T., Yiu, A., Eneva, K., Arlt, J., & Swantek, M. (2016). Family based therapy for young adults with anorexia nervosa restores weight. *International Journal of Eating Disorders*, 49(7), 701-707.
- Chen, L., Zhang, G., Hu, M., & Liang, X. (2013). Eye movement desensitization and reprocessing versus cognitive-behavioral therapy for adult posttraumatic stress disorder: Systematic review and meta-analysis. *Journal of Nervous & Mental Disease*, 203, 443-451.
- Chen, S., Zhou, R., Cui, H., & Chen, X. (2013). Deficits in cue detection underlie event-based

- prospective memory impairment in major depression: An eye tracking study. *Psychiatry Research*, 209(3), 453-458.
- Child Welfare Information Gateway. (2012). *Trauma-Focused Cognitive Behavioral Therapy for Children Affected by Sexual Abuse or Trauma*. Washington, DC: US Department of Health and Human Services, Children's Bureau.
- Chin-Chan, M., Navarro-Yepes, J., & Quintanilla-Vega, B. (2015). Environmental pollutants as risk factors for neurodegenerative disorders: Alzheimer and Parkinson diseases. *Frontiers in Cellular Neuroscience*, 9, 124.
- Choe, J. Y., Teplin, L. A., & Abram, K. M. (2008). Perpetration of violence, violent victimization, and severe mental illness: balancing public health concerns. *Psychiatric Services*, 59(2), 153-164.
- Chu, J., Dell, P., Van der Hart, O., Cardena, E., Barach, P., Somer, E., ... Twombly, J. (2011). Guidelines for treating dissociative identity disorder in adults, third revision, *Journal of Trauma & Dissociation*, 12, 115-187.
- Chung, T., Sealy, L., Abraham, M., Ruglovsy, C., Schall, J., & Maisto, S. (2014). Personal network characteristics of youth substance use treatment: Motivation for and perceived difficulty of positive network change. *Substance Abuse*, 36(3), 380-388.
- Cisler, J., Adams, T., Brady, R., Bridges, A., Lohr, J., & Olatunji, B. (2011). Unique affective and cognitive processes in contamination appraisals: Implications for contamination fear. *Journal of Anxiety Disorders*, 25, 28-35.
- Clabough, E. (2013). Huntington's disease: The past, present, and future search for disease modifiers. *Yale Journal of Biological Medicine*, 86, 217-233.
- Coffey, R. M., Mark, T., King, E., Harwood, H., McKusick, D., Genuardi, J., ... & Buck, J. A.

- (2000). National Estimates of Expenditures for Mental Health and Substance Abuse Treatment, 1997. SAMHSA pub no SMA-00-3499. Rockville, Md. *Center for Substance Abuse Treatment and Center for Mental Health Services, Substance Abuse and Mental Health Services Administration.*
- Colli, A., Tanzilli, A., Dimaggio, G., & Lingiardi, V. (2014). Patient personality and therapist response: An empirical investigation. *American Journal of Psychiatry, 171*, 102-108.
- Conley, C. S., Shapiro, J. B., Kirsch, A. C., & Durlak, J. A. (2017). A meta-analysis of indicated mental health prevention programs for at-risk higher education students.
- Coon, D. & Mitter, J. (2007). *Introduction to psychology: Gateways to mind and behavior* (11th ed). Belmont, CA: Wadsworth.
- Corcoran, C., Walker, E., Huot, R., Mittal, V., Tessner, K., Kestler, L., & Malaspina, D. (2003). The stress cascade and schizophrenia: Etiology and onset. *Schizophrenia Bulletin, 29*, 671-692.
- Corrigan, P. W. (2016). Lessons learned from unintended consequences about erasing the stigma of mental illness. *World Psychiatry, 15*(1), 67-73.
- Corrigan, P. W., Bink, A. B., Schmidt, A., Jones, N., & Rüscher, N. (2016). What is the impact of self-stigma? Loss of self-respect and the “why try” effect. *Journal of Mental Health, 25*(1), 10-15.
- Corrigan, P. W., Larson, J. E., & Ruesch, N. (2009). Self-stigma and the “why try” effect: impact on life goals and evidence-based practices. *World psychiatry, 8*(2), 75-81.
- Cortina, L. & Kubiak, S. (2006). Gender and posttraumatic stress: Sexual violence as an explanation for women’s increased risk. *Journal of Abnormal Psychology, 115*, 753-759.
- Costa, P. T., & McCrae, R. R. (1992). Normal personality assessment in clinical practice: The

- NEO Personality Inventory. *Psychological assessment*, 4(1), 5.
- Craig, T. & Power, P. (2010). Inpatient provision in early psychosis. In P. Frech, J. Smith, D. Shiers, M. Reed & M. Rayne (Eds) *Promoting recovery in early psychosis: A practice manual* (pp. 17-26). Hoboken, NJ: Wiley-Blackwell.
- Craighhead, E., & Dunlop, B. (2014). Combination psychotherapy and antidepressant medication treatment for depression: For whom, when, and how. *Annual Review of Psychology*, 65, 267-300.
- Craske, M. & Barlow, D. (2014). Panic disorder and agoraphobia. In D. Barlow (Ed), *Clinical handbook of psychological disorders: A step-by-step treatment manual*. (pp. 1-61). New York, NY: Guilford Press.
- Culbert, K., Burt, S., McGue, M., Iacono, W., & Klump, K. (2009). Puberty and the genetic diathesis of disordered eating attitudes and behaviors. *Journal of Abnormal Psychology*, 118(4), 788-796.
- Culbert, K., Racine, E., & Klump, K. (2015). Research review: What we have learned about the causes of eating disorders- a synthesis of sociocultural, psychological, and biological research. *Journal of Child Psychology and Psychiatry*, 56, 1141-1164.
- Cummings, J. R., Lucas, S. M., & Druss, B. G. (2013). Addressing public stigma and disparities among persons with mental illness: The role of federal policy. *American journal of public health*, 103(5), 781-785.
- Czabała, C., Charzyńska, K., & Mroziak, B. (2011). Psychosocial interventions in workplace mental health promotion: an overview. *Health promotion international*, 26(suppl_1), i70-i84.
- Dalenberg, C., Brand, B., Gleaves, D., Dorahy, M., Loewenstein, R., Cardena, E., ... Spiegel, D.

- (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin*, 138(3), 550-588.
- de Bruin, E. J., Bögels, S. M., Oort, F. J., & Meijer, A. M. (2015). Efficacy of cognitive behavioral therapy for insomnia in adolescents: a randomized controlled trial with internet therapy, group therapy and a waiting list condition. *Sleep*, 38(12), 1913-1926.
- DeLeon, J., Cuesta, M., & Peralta, V. (1993). Delusions and hallucinations in schizophrenic patients. *Psychopathology*, 26, 286-291.
- DeLuca, J. S., & Yanos, P. T. (2016). Managing the terror of a dangerous world: Political attitudes as predictors of mental health stigma. *International journal of social psychiatry*, 62(1), 21-30.
- Dell, P. (2010). Involuntariness in hypnotic responding and dissociative symptoms. *Journal of Trauma & Dissociation*, 11, 1-18.
- Derefinko, K., & Widiger, T. (2016). Antisocial personality disorder. In S. Fatemi & P. Clayton (Eds.), *The medical basis of psychiatry*. New York, NY: Springer.
- Diclemente, C., Garay, M., & Gemmell, L. (2008). Motivational enhancement. In HD Kleber & M. Galanter (Eds). *The American Psychiatric Publishing textbook of substance abuse treatment* (4th ed., pp. 361-371). Arlington, VA: American Psychiatric Publishing.
- Ding, Y., Naganawa, M., Gallezot, J., Nabulsi, N., Lin, S., Ropchan, J., Weinzimmer, D., . . . Laurelle, M. (2014). Clinical doses of atomoxetine significantly occupy both norepinephrine and serotonin transports: Implications on treatment of depression and ADHD. *Neuroimage*, 86, 164-171.
- Donoghue, K., Doody, G., Murray, R., Jones, P., Morgan, C., Dazzan, P., . . . Maccabe, J. (2014).

- Cannabis use, gender and age of onset of schizophrenia: Data from the NESOP study. *Psychiatry Research*, 215, 528-532.
- Dougherty, D., Rauch, S., & Jenike, M. (2002). Pharmacological treatments for obsessive compulsive disorder. In P.E. Nathan & J.M. Gordon (Eds.), *A guide to treatments that work* (2nd ed., pp. 387-410). New York: Oxford University Press.
- Eagly, A. H., & Chaiken, S. (1993). *The psychology of attitudes*. Harcourt Brace Jovanovich College Publishers.
- Ebert, T., & Kotler, M. (2005). Prenatal exposure to influenza and the risk of subsequent development of schizophrenia. *IMAJ-RAMAT GAN*-, 7(1), 35.
- Edvardsen, J., Torgersen, S., Roysamb, E., Lygren, S., Skre, I., Onstad, S., & Oien, P. (2008). Heritability of bipolar spectrum disorders. Unity or heterogeneity. *Journal of Affective Disorders*, 106(3), 229-240.
- Egloff, N., Camara, R., von Kanel, R., Klinger, N., Marti, E., & Ferrari, M. (2014). Hypersensitivity and hyperalgesia in somatoform pain disorders. *General Hospital Psychiatry*, 36, 284-290.
- Eisenberg, D., Downs, M. F., Golberstein, E., & Zivin, K. (2009). Stigma and help seeking for mental health among college students. *Medical Care Research and Review*, 66(5), 522-541.
- El Nemer, F. M., Alian, D. M., Eldin, M. S., Khalil, H. E. M. (2014). Prevalence of pica among children attending pediatrics clinic at El-Menoufiya University Hospital. *American Journal of BioScience*, 2(4), 147-152.
- Ellis, A. (2014). Rational emotive behavior. In D. Wedding & R. Corsini (Eds.), *Current psychotherapies* (10th ed., pp. 151-192). Independence, KY: Cengage Publications.

- Endrass, T., Kloft, L., Kaufmann, C., & Kaufmann, N. (2011). Approach and avoidance learning in obsessive-compulsive disorder. *Depression & Anxiety, 28*, 166-172.
- Einfeld, S., & Emerson, E. (2008). Intellectual disability. *Rutter's child and adolescent psychiatry, 5*.
- Fairburn, C., Cooper, Z., & Shafran, R. (2003). Cognitive behavior therapy for eating disorders: A “transdiagnostic” theory. *Behavior Research and Therapy, 41*(5), 509-528.
- Fairburn, C., Jones, R., Peveler, R., Hope, R., & O’Connor, M. (1993). Psychotherapy and bulimia nervosa: The longer term effects of interpersonal psychotherapy, behaviour therapy, and cognitive behaviour therapy. *Archives of General Psychiatry, 50*, 419-428.
- Feldman, M. & Feldman, J. (1995). Tangled in the web: Countertransference in the therapy of factitious disorders. *International Journal of Psychiatry in Medicine, 25*, 389.
- Felker, B., Yazel, J., Short, D. Mortality and medical comorbidity among psychiatric patients: a review. *Psychiatric Services, 47*, 1356-1363.
- Ferster, C. (1973). A functional analysis of depression. *American Psychologist, 28*(10), 857-870.
- Fetissov, S. & Mequid, M. (2010). Serotonin delivery into the ventromedial nucleus of the hypothalamus affects differently feeding pattern and body weight in obese and lean Zucker rats. *Appetite, 54*(2), 346-353.
- Figley, C. (1978). Symptoms of delayed combat stress among a college sample of Vietnam veterans. *Military Medicine, 143*, 107-110.
- Filip, M., Frankowska, M., Sadakierska-Chudy, A., Suder, A., Szumiec, L., Mierzejewski, P., ..., Cryan, J. (2015). GABAB receptors as a therapeutic strategy in substance use disorders: Focus on positive allosteric modulators. *Neuropharmacology, 88*, 36-47.
- Fitch, W. L. (2007). AAPL Practice Guideline for the Forensic Psychiatric Evaluation of

- Competence to Stand Trial: An American legal perspective. *The journal of the American Academy of Psychiatry and the Law*, 35(4), 509-513.
- Foa, E., Hembree, E., Cahill, S., Rauch, S., Riggs, D., Feeny, N., & Yadin, E. (2005). Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: Outcome at academic and community clinics. *Journal of Consulting and Clinical Psychology*, 73, 953-964.
- Foa, E., Liebowitz, M., Kozak, M., Davies, S., Campeas, R., Franklin, M., . . . (2005). Treatment of obsessive compulsive disorder by exposure and ritual prevention, clomipramine, and their combination: A randomized, placebo-controlled trial. *American Journal of Psychiatry*, 162, 151-161.
- Foa, E., & McNally, R. (1996). Mechanisms of change in exposure therapy. In R. Rapee (Ed.), *Current controversies in the anxiety disorders* (pp. 329-343). New York, NY: Guilford Press.
- Foddy, M., Smithson, M., Schneider, S., and Hogg, M. (1999). *Resolving social dilemmas: Dynamics, structural, and intergroup aspects*. Philadelphia, PA: Psychology Press.
- Forbes, D., Creamer, M., Bisson, J., Cohen, J., Crow, B., Foa, E., . . . Ursano, R. (2010). A guide to guidelines for the treatment of PTSD and related conditions. *Journal of Trauma and Stress*, 23, 537-552.
- Forsman, A. K., Nordmyr, J., & Wahlbeck, K. (2011). Psychosocial interventions for the promotion of mental health and the prevention of depression among older adults. *Health promotion international*, 26(suppl_1), i85-i107.
- Frank, R. G. (2006). *Better but not well: Mental health policy in the United States since 1950*. JHU Press.

- Frank, R. G., Conti, R. M., & Goldman, H. H. (2005). Mental health policy and psychotropic drugs. *The Milbank Quarterly*, *83*(2), 271-298.
- Friborg, O., Martinsen, E., Martinussen, M., Kaiser, S. Overgard, K., & Rosenvinge, J. (2014). Comorbidity of personality disorders in mood disorders: A meta-analytic review of 122 studies from 1988-2010. *Journal of Affective Disorders*, *152-154*, 1-11.
- Friedrich, F., Gross, R., Wrobel, M., Klung, G., Unger, A., Fellingner, M., ... Wancata, J. (2014). Burden of mothers and fathers of persons with schizophrenia. *Psychiatrische Praxis*, *42*(4), 208-215.
- Frost, R., & Hartl, T. (1996). A cognitive-behavioral model of compulsive hoarding. *Behaviour Research and Therapy*, *34*, 341-350.
- Fulgoni, V., Keast, D., & Lieberman, H. (2015). Trends in intake and sources of caffeine in diets of the US adults: 2001-2010. *American Journal of Clinical Nutrition*, *101*, 1081-1087.
- Galanter, M. (2014). Alcoholics Anonymous and twelve-step recovery: A model based on social and cognitive neuroscience. *American Journal of Addictions*, *23*, 300-307.
- Galen, L. & Rogers, W. (2004). Religiosity, alcohol expectancies, drinking motives and their interaction in the prediction of drinking among college students. *Journal of Studies on Alcohol*, *65*, 469-476.
- Gallagher, M., Payne, L., White, K., Shear, K., Woods, S., Gorman, J., & Barlow, D. (2013). Mechanisms of change in cognitive behavioral therapy for panic disorder: The unique effects of self-efficacy and anxiety sensitivity. *Behaviour Research and Therapy*, *51*, 767-777.
- Gatchel, R., Peng, Y., Peters, M., Fuchs, P, & Turk, D. (2007). The biopsychosocial approach to

- chronic pain: Scientific advances and future directions. *Psychological Bulletin*, 133, 581-624.
- Garcia-Toro, M., Rubio, J., Gili, M., Roca, M., Jin, C., Liu, S., . . . Blanco, C. (2013). Persistence of chronic major depression: A national prospective study. *Journal of Affective Disorders*, 151, 306-312.
- Gaynor, S., & Baird, S. (2007). Personality disorders. In D. Woods & J. Kanter (Eds.), *Understanding behavior disorders: A contemporary behavioral perspective*. Reno, NV: Context Press.
- Ghaemi, S., Hsu, D., Soldani, F., Goodwin, F. (2003). Antidepressants in bipolar disorder: the case for caution. *Bipolar Disorders*, 5, 421-433.
- Gibbons, F., Kingsbury, J., Weng, C., Gerrard, M., Cutrona, C., Wills, T., & Stock, M. (2014). Effects of perceived racial discrimination on health status and health behavior: A differential mediation hypothesis. *Health Psychology*, 33(1), 11-19.
- Giedd, J. N., Rapoport, J. L., Garvey, M. A., Perlmutter, S., & Swedo, S. E. (2000). MRI assessment of children with obsessive-compulsive disorder or tics associated with streptococcal infection. *American Journal of Psychiatry*, 157(2), 281-283.
- Gijssman, H., Geddes, J., Riedell, J., Nolen, W., Goodwin, G. (2004). Antidepressants for bipolar depression: a systematic review of randomized, controlled trials. *American Journal of Psychiatry*, 161, 1537-1547.
- Gilliam, C., & Tolin, D. (2010). Compulsive hoarding. *Bulletin of the Menninger Clinic*, 74, 93-121.
- Gelenberg, A. (2000). Psychiatric and somatic markers of anxiety: Identification and pharmacologic treatment. *Primary Care Companion of the Journal of Clinical*

- Psychiatry*, 2, 49-54.
- Goff, D. & Simms, C. (1993). Has multiple personality disorder remained consistent over time? *Journal of Nervous and Mental Disease*, 181, 595-600.
- Gonidakis, F. (2014). Dialectical behavior therapy for borderline personality disorder. In C. Soldatos, P. Ruiz, D. Dikeos, M. Riba (Eds). *Pluralism in Psychiatry*. (pp. 145-148) Pianoro, Italy: Medimond.
- Gonzales, L., Chan, G., & Yanos, P. T. (2017). Individual and neighborhood predictors of mental illness stigma in New York state. *Stigma and health*, 2(3), 175.
- Gonzalez, H., Tarraf, W., Whitfield, K., & Vega, W. (2010). The epidemiology of major depression and ethnicity in the United States. *Journal of Psychiatric Research*, 44, 1043-1051.
- Gorman, J., Kent, J., Sullivan, G., & Coplan, J. (2000). Neuroanatomical hypothesis of panic disorder, revised. *American Journal of Psychiatry*, 157, 493-505.
- Gotlib, I. & Hammen, C. (2009). *Handbook of Depression*, 2nd ed. New York, NY: Guilford Press.
- Grant, B., Goldstein, R., Saha, T., Chou, P., Jung, J., Zhang, H., ...Hasin, D. (2015). Epidemiology of DSM-5 alcohol use disorder: Results from the national epidemiologic survey on alcohol and related conditions III. *Journal of American Medical Association Psychiatry*, 72(8), 757-766.
- Grant, P. M., Huh, G. A., Perivoliotis, D., Stolar, N. M., & Beck, A. T. (2012). Randomized trial to evaluate the efficacy of cognitive therapy for low functioning patients with schizophrenia. *Archives of General Psychiatry*, 69, 121-127.
- Grant, B., Saha, T., Ruan, J., Goldstein, R., Chou, P., ...Hasin, D. (2016). Epidemiology of

- DSM-5 Drug Use Disorder results from the national epidemiologic survey on alcohol and related conditions-III. *JAMA Psychiatry*, 73, 39-47.
- Greenberg, D. & Laporte, D. (1996). Racial differences in body type preferences of men for women. *International Journal of Eating Disorders*, 19, 275-78.
- Gruber, S., Sagar, A., Dahlgren, M., Racine, M., & Lukas, S. (2012). Age of onset of marijuana use and executive function. *Psychology of Addictive Behaviors*, 26, 496-506.
- Graziottin, A. & Serafini, A. (2009). Depression and the menopause: Why antidepressants are not enough? *Menopause International*, 15, 76-81.
- Guerdjikova, A., Mori, N., Casuto, L., & McElroy, S. (2017). Binge eating disorder. *Psychiatric Clinics of North America*, 40, 255-266.
- Gutman, D., Gorman, J., & Hirsch, J. (2004). Neuroanatomy of panic disorder: Implications of functional imaging in fear conditioning. In J. Gorman (Ed.), *Fear and anxiety: The benefits of translational research*. (pp. 119-134). Washington, DC: American Psychiatric Publishing.
- Haddock, G. & Spaulding, W. (2011). Psychological treatment of psychosis. In D. Weinberger & P. Harrison (Eds.). *Schizophrenia* (pp. 666-686). Hoboken, NJ: Humana Press.
- Haile, C. (2012). History use, and basic pharmacology of stimulants. In TR Kosten, TF Newton, R De L Garza, & C Haile (Eds.), *Cocaine and methamphetamine dependence: Advances in treatment* (pp. 13-84). Arlington, VA: American Psychiatric Publishing.
- Hakimian, M., & D'Souza, L. (2016). Effectiveness of Systematic Desensitization and Cognitive Behavior Therapy on Reduction of Obsessive Compulsive Disorder Symptoms: A Comparative Study. *European Online Journal of Natural and Social Sciences*, 5(1), pp-147.

- Hall, M., Levant, S., & DeFrances, C. (2012). Hospitalization for stroke in US hospitals, 1989-2009, *NCHS Data Brief, 95*, 1-8.
- Hankin, B. (2009). Development of sex differences in depressive and co-occurring anxious symptoms during adolescence: Descriptive trajectories and potential explanations in a multiwave prospective study. *Journal of Clinical Child and Adolescent Psychology, 38*, 460-472.
- Hanrahan, F., Field, A., Jones, F., & Davy, G. (2013). A meta-analysis of cognitive therapy for worry in generalized anxiety disorder. *Clinical Psychology Review, 33*, 120-132.
- Hansson, L., Jormfeldt, H., Svedberg, P., & Svensson, B. (2013). Mental health professionals' attitudes towards people with mental illness: Do they differ from attitudes held by people with mental illness?. *International Journal of Social Psychiatry, 59*(1), 48-54.
- Harned, M., Korslund, K., & Linehan, M. (2014). A pilot randomized controlled trial of Dialectical Behavior Therapy with and without the Dialectical Behavioral Therapy Prolonged Exposure protocol for suicidal and self-injuring women with borderline personality disorder and PTSD. *Behavioral Research Therapy, 55*, 7-17.
- Harris, J. C. (2006). *Intellectual disability: Understanding its development, causes, classification, evaluation, and treatment*. Oxford University Press.
- Harrison, A. J., Long, K. A., Tommet, D. C., & Jones, R. N. (2017). Examining the Role of Race, Ethnicity, and Gender on Social and Behavioral Ratings Within the Autism Diagnostic Observation Schedule. *Journal of Autism and Developmental Disorders, 1-13*.
- Hart, C. & Ksir, C. (2014). *Drugs, society, and human behavior* (15th ed.). East Windsor, NJ: McGraw-Hill Higher Education.
- Hartz, S., Pato, C., Medeiros, H., Cavazos-Rehg, P., Sobell, J., ...Pato, M. (2014). Comorbidity

- of severe psychotic disorders with measures of substance use. *JAMA Psychiatry*, 71, 248-254.
- Hartzler, B., Lash, S., & Roll, J. (2012). Contingency management in substance abuse treatment: A structured review of the evidence for its transportability. *Drug and Alcohol Dependence*, 122, 1-10.
- Harvey, A & Bryant, R. (1998). The relationship between acute stress disorder and posttraumatic stress disorder: A prospective evaluation of motor vehicle accident survivors. *Journal of Consulting and Clinical Psychology*, 66, 507-512.
- Hatzenbuehler, M. L., Phelan, J. C., & Link, B. G. (2013). Stigma as a fundamental cause of population health inequalities. *American journal of public health*, 103(5), 813-821.
- Hauer, P. (2010). Systematic affects of methamphetamine use. *South Dakota Medicine: Journal of the South Dakota State Medical Association*, 63, 285-297.
- Hedna, V., Bodhit, A., Ansari, S., Falchook, A., Stead, L., Heilman, K., & Waters, M. (2013). Hemispheric differences in ischemic stroke: Is left-hemisphere stroke more common? *Journal of Clinical Neurology*, 9(2), 97-102.
- Heimberg, R., & Becker, R. (2002). *Cognitive-behavioral group therapy for social phobia: Basic mechanisms and clinical strategies*. New York, NY: Guilford Press.
- Hengartner, M., Muller, M., Rodgers, S., Rossler, W., & Ajdacic-Gross, V. (2014). Occupational functioning and work impairment in association with personality disorder trait-scores. *Social Psychiatry and Psychiatric Epidemiology*, 49, 327-335.
- Herbert, J. (2007). Avoidant personality disorder. In W. O'Donohue, K. Fowler, & S. Lilienfeld (Eds.). *Personality Disorders: Toward the DSM-V*. Los Angeles, CA: Sage Publications.
- Herpertz, S., & Bertsch, K. (2014). The social-cognitive basis of personality disorders. *Current*

Opinion in Psychiatry, 27, 73-77.

- Hewitt, P., Flett, G., & Ediger, E. (1995). Perfectionism traits and perfectionistic self-presentation in eating disorder attitudes, characteristics, and symptoms. *International Journal of Eating Disorders*, 18, 317-326.
- Hiday, V. A., & Burns, P. J. (2010). Mental illness and the criminal justice system. *A handbook for the study of mental health: Social contexts, theories, and systems*, 478-498.
- Hurd, N., Varner, F., Caldwell, C., & Zimmerman, M. (2014). Does perceived racial discrimination predict changes in psychological distress and substance use over time? An examination among Black emerging adults. *Developmental Psychology*, 50(7), 1910-1918.
- Hinton, D. & Lewis-Fernandez, R. (2011). The cross-cultural validity of posttraumatic stress disorder: Implications for DSM-5. *Depression and Anxiety*, 28, 783-801.
- Hofer, H., Frigerio, S., Frischknecht, E., Gassmann, D., Gutbrod, K., & Muri, R. (2013). Diagnosis and treatment of an obsessive-compulsive disorder following traumatic brain injury: A single case and review of the literature. *Neurocase*, 19, 390-400.
- Horowitz, S. (2008). Shedding light on seasonal affective disorder. *Alternative and Complementary Therapies*, 14(6), 282-287.
- Houenou, J., Frommberger, J., Carde, S., Glasbrenner, M., Diener, C., Leboyer, M. & Wessa, M. (2011). Neuroimaging-based markers of bipolar disorder: Evidence from two meta-analyses. *Journal of Affective Disorders*, 132(3), 344-355.
- Hsu, J., Lee, W., Liao, Y., Lirng, J., Wang, S., & Fuh, J. (2015). Posterior atrophy and medial

- temporal atrophy scores are associated with different symptoms in patients with Alzheimer's disease and mild cognitive impairment. *PLOS ONE*, *10*(9), e0137121.
- Hudson, J.I., Hiripi, E., Pope, H.G. Jr., & Kessler, R.C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, *61*(3), 348-58. doi:10.1016/j.biopsych.2006.03.040.
- Hurd, N. M., Varner, F. A., Caldwell, C. H., & Zimmerman, M. A. (2014). Does perceived racial discrimination predict changes in psychological distress and substance use over time? An examination among Black emerging adults. *Developmental psychology*, *50*(7), 1910.
- Jacob, M., Larson, M., & Storch, E. (2014). Insight in adults with obsessive-compulsive disorder. *Comprehensive Psychiatry*, *55*, 896-903.
- Jacobson, N., Martell, C., & Dimidjian, S. (2001). Behavioral activation treatment for depression: Returning to contextual roots. *Clinical Psychology Science and Practice*, *8*(3), 255-270.
- Jang, K., Livesley, W., & Vernon, P. (1995). Alcohol and drug problems: a multivariate behavioral genetic analysis of co-morbidity. *Addiction*, *90*, 1213-1221.
- Jani, S., Johnson, R. S., Banu, S., & Shah, A. (2016). Cross-cultural bias in the diagnosis of borderline personality disorder. *Bulletin of the Menninger Clinic*, *80*(2), 146-165.
- Jansen, R., Penninx, B., Madar, V., Xia, K., Milaneschi, Y., Hottenga, J., . . . Sullivan, P. (2016). Gene expression in major depressive disorder. *Molecular Psychiatry*, *21*, 339-347.
- Jayawant, S. & Balkrishnana, R. (2005). The controversy surrounding OxyContin abuse: Issues and solutions. *Therapeutics and Clinical Risk Management*, *1*, 77-82.
- Jensen, M., McArthur, K., Barber, J., Hanley, M., Engel, J., . . . Patterson, D. (2006). Satisfaction

- with, and the beneficial side effects of hypnosis analgesia. *International Journal of Clinical and Experimental Hypnosis*, 54, 432-447.
- Jhanjee, S. (2014). Evidence based psychosocial interventions in substance use. *Indian Journal of Psychological Medicine*, 36, 112-118.
- Johnston, L., O'Malley, P., Miech, R., Bachman, J., & Schulenberg, J. (2014). *Monitoring the future national results on drug use, 1975-2013: Overview, key findings on adolescent drug use*. Ann Arbor, MI: Institute for Social Research, University of Michigan.
- Jones, B., Corbin, W., & Fromme, K. (2001). A review of expectancy theory and alcohol consumption. *Addiction*, 96, 57-72.
- Jones, H. L., Cross Jr, W. E., & DeFour, D. C. (2007). Race-related stress, racial identity attitudes, and mental health among Black women. *Journal of Black Psychology*, 33(2), 208-231.
- Jones, R., Harrison, C., & Ball, M. (2008). Secondary handicap & learning disability: A component analysis. *Mental Health and Learning Disabilities Research and Practice*, 5, 300-311.
- Kaczurkin, A., Asnaai, A., Hall-Clark, B., Peterson, A., Yarvis, J., Foa, E., & the STRONG STAR Consortium. (2016). Ethnic and racial differences in clinically relevant symptoms in active duty military personnel with posttraumatic stress disorder. *Journal of Anxiety Disorders*, 43, 90-98.
- Kalibatseva, Z., & Leong, F. T. (2014). A critical review of culturally sensitive treatments for depression: recommendations for intervention and research. *Psychological services*, 11(4), 433-450.
- Kampman, C., Keijers, G., Hoogduin, C., & Hendriks, G. (2002). A randomized, double-blind,

- placebo-controlled study of the effects of adjunctive paroxetine in panic disorder patients unsuccessfully treated with cognitive-behavioral therapy alone. *Journal of Clinical Psychiatry*, 63(9), 772-777.
- Kang, H., Myung, W., Na, D., Kim, S., Lee, J., Han, S., ... Kim, D. (2014). Factors associated with caregiver burden in patients with Alzheimer's disease. *Psychiatry Investigation*, 11(2), 152-159.
- Karnieli-Miller, O., Perlick, D. A., Nelson, A., Mattias, K., Corrigan, P., & Roe, D. (2013). Family members' of persons living with a serious mental illness: Experiences and efforts to cope with stigma. *Journal of Mental Health*, 22(3), 254-262.
- Kellett, S., & Hardy, G. (2014). Treatment of paranoid personality disorder with cognitive analytic therapy: A mixed methods single case experimental design. *Clinical Psychology & Psychotherapy*, 21(5), 452-464.
- Kelly, V., Barker, H., Field, A., Wilson, C. & Reynolds, S. (2010). Can Rachman's indirect pathways be used to un-learn fear? A prospective paradigm to test whether children's fears can be reduced using positive information and modeling a non-anxious response. *Behavior Research and Therapy*, 48, 164-170.
- Kessler, R., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K., . . . Wang, P. (2003). Results from the national comorbidity survey replication (NCS-R). *Journal of American Medical Association*, 289(23), 3095-3105.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of general psychiatry*, 62(6), 593-602.

- Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of general psychiatry*, *62*(6), 617-627.
- Kessler, R. C., Demler, O., Frank, R. G., Olfson, M., Pincus, H. A., Walters, E. E., ... & Zaslavsky, A. M. (2005). Prevalence and treatment of mental disorders, 1990 to 2003. *New England Journal of Medicine*, *352*(24), 2515-2523.
- Ketterer, M., Knysk, W., Khanal, S., & Hudson, M. (2006). Cardiovascular Disease. In M. Blumenfeld, J. Strain (Eds.). *Psychosomatic Medicine (109-119)*. Philadelphia, PA: Lippincott Williams & Williams.
- Kinchin, D. (2007). *A guide to psychological debriefing: Managing emotional decompression and post-traumatic stress disorder*. London, England: Jessica Kingsley Publishers.
- Kirkpatrick, B., Fenton, W., Carpenter, W., & Marder, S. (2006). The NIMH-MATRICES consensus statement on negative symptoms. *Schizophrenia Bulletin*, *32*, 214-219.
- Klerman, G., Weissman, M., Rounsaville, B., & Chevron, E. (1984). *Interpersonal psychotherapy of depression*. London: Jason Aronson Inc.
- Kopelman, M. (2000). Focal retrograde amnesia and the attribution of casualty: an exceptionally critical review. *Cognitive Neuropsychology*, *17*, 585-621.
- Kowal, S., Dall, T., Chakrabarti, R., Storm, M., & Jain, A. (2013). The current and projected economic burden of Parkinson's disease in the United States. *Movement Disorders*, *28*, 311-318.
- Krebs, G., & Heyman, I. (2015). Obsessive-compulsive disorder in children and adolescents. *Archives of disease in childhood*, *100*(5), 495–

499. doi: 10.1136/archdischild-2014-306934

- Krishan, K. (2007). Revisiting monamine oxidase inhibitors. *Journal of Clinical Psychiatry*, 68, 35-41.
- Kugelmass, H. (2016). "Sorry, I'm Not Accepting New Patients" An Audit Study of Access to Mental Health Care. *Journal of health and social behavior*, 57(2), 168-183.
- Kuhn, R. (1958). The treatment of depressive states with G-22355 (imipramine hydrochloride). *American Journal of Psychiatry*, 115, 459-464.
- Kumari, V., Uddin, S., Premkumar, P., Young, S., Gudjonsson, G., Raghuvanshi, S., ... Das, M. (2014). Lower anterior cingulate volume in seriously violent men with antisocial personality disorder or schizophrenia and a history of childhood abuse. *Australian and New Zealand Journal of Psychiatry*, 48(2), 153-161.
- Kurtz, M. (2015). *Schizophrenia and its treatment: Where is the progress?* New York, NY: Oxford University Press.
- Kuttler, S., Myles, B. S., & Carlson, J. K. (1998). The use of social stories to reduce precursors to tantrum behavior in a student with autism. *Focus on Autism and Other Developmental Disabilities*, 13(3), 176-182.
- Lahmann, C., Henningsen, P., & Noll-Hussong, M. (2012). Somatoform pain disorder-Overview. *Psuchatria Danubina*, 22, 453-458.
- Lai, C., Zauszniewski, J., Tang, T., Hou, S., Su, S., & Lai, P. (2014). Personal beliefs, learned resourcefulness, and adaptive functioning in depressed adults. *Journal of Psychiatric and Mental Health Nursing*, 21(3), 280-287.
- Lally, J., Conghaile, A., Quigley, S., Bainbridge, E., & McDonald, C. (2013). Stigma of mental illness and help-seeking intention in university students. *The Psychiatrist*, 37(8), 253-260.

- Lang, E., Benotsch, e., Frick, L., Lutgendorf, S., Berbaum, M., ...Spiegel, D. (2000). Adjunctive non-pharmacological analgesia for invasive medical procedures: A randomized trial. *Lancet*, 355, 1486-1490.
- Lang, F., Otte, S., Vasic, N., Jager, M. & Dudeck, M. (2015). Impulsiveness among short-term prisoners with antisocial personality disorder. *Psychiatrische Praxis*, 42(5). 274-277.
- Lazarov, O., Mattson, M., Perterson, D., Pimplika, S., & van Praag, H. (2010). When neurogenesis encounters aging and disease. *Trends in Neuroscience*, 33, 569-579.
- Le Grange, D., Lock, J., Accurso, E., Agras, W., Darcy, A., Forsberg, S., & Bryson, S. (2014). Relapse from remission at two- to four- year follow-up in two treatments for adolescent anorexia nervosa. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53(11), 1162-1167.
- Leichsenring, F., Salzer, S., Beutel, M. E., Herpertz, S., Hiller, W., Hoyer, J., ... & Ritter, V. (2014). Long-term outcome of psychodynamic therapy and cognitive-behavioral therapy in social anxiety disorder. *American Journal of Psychiatry*, 171(10), 1074-1082.
- Lener, M., Wong, E., Tang, C., Byne, W., Goldstein, K., Blair, N., ...Hazlett, E. (2015). White matter abnormalities in schizophrenia and schizotypal personality disorder. *Schizophrenia Bulletin*, 41(1). 300-310.
- Lenzenweger, M., Lane, M., Loranger, A., & Kessler, R. (2007). DSM-IV personality disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, 62, 553-564.
- Lervolino, A., Perroud, N., Fullana, M., Guipponi, M., Cherkas, L., Collier, D., & Mataix-Cols, D. (2009). Prevalence and heritability of compulsive hoarding: A twin study. *American Journal of Psychiatry*, 166, 1156-1161.
- Leucht, S., Heres, S., Kissling, W., & Davis, J. (2011). Evidence-base pharmacotherapy of

- schizophrenia. *International Journal of Neuropsychopharmacology*, 14, 269-284.
- Leventhal, A. & Schmitz, J. (2006). The role of drug use outcome expectancies in substance abuse risk: An interactional-transformational model. *Addictive Behaviors*, 31, 2038-2062.
- Levinson, D. & Nichols, W. (2014). *Major Depression and genetics*. Stanford, CA: Stanford, School of Medicine.
- Lewinsohn, P., Antonuccio, D., Steinmetz, J. & Teri, L. (1984). *The coping with depression course*. Eugene, OR: Castalia.
- Lewinsohn, P., Clarke, G., Hops, H., & Andrews, J. (1990). Cognitive-behavioral treatment for depressed adolescents. *Behavior Therapist*, 21, 385-401.
- Lewinsohn, P. (1974). A behavioral approach to depression. In R. M. Friedman & M. M. Katz (Eds.), *The psychology of depression: Contemporary theory and research* (pp. 157-185). New York: Wiley.
- Lewinsohn, P., Biglan, A., & Zeiss, A. (1976). Behavioral treatment of depression. In P. O. Davidson (Ed), *The behavioral management of anxiety, depression and pain* (pp. 91-146). New York: Brunner/Mazel.
- Lewis, M. & Petry, N. (2005). Contingency management treatments that reinforce completion of goal-related activities: Participation in family activities and its association with outcomes. *Drug and Alcohol Dependence*, 79, 267-271.
- Lewy Body Dementia Association. (2017). Lewy body dementia symptoms and diagnostic criteria. Retrieved from: <https://www.lbda.org/content/symptoms>
- Lieberman, J., Perkins, D., Belger, A., Chakos, M., Jarskog, F...Gilmore, J. (2001). The early stages of schizophrenia: speculations on pathogenesis, pathophysiology, and therapeutic approaches. *Journal of Biological Psychiatry*, 50(11), 884-897.

- Lilienfeld, S., Lynn, S., Kirsch, I., Chaves, J., Sarbin, T., Ganaway, G., & Powerll, R. (1999). Dissociative identity disorder and the sociocognitive model: Recalling the lessons of the past. *Psychological Bulletin*, *125*, 507- 523.
- Linehan, M., Armstrong, H., Suarez, A., Allmon, D. & Heard, H. (1991). Cognitive-behavioral treatment of chronically parasuicidal borderline patients. *Archives of General Psychiatry*, *48*, 1060-1064.
- Linehan, M., Korslund, K., Harned, M., Gallop, R., Lungu, A., Neacsiu, A., ...Murray-Gregory, A., (2015). Dialectical behavior therapy for high suicide risk individuals with borderline personality disorder: A randomized clinical trial and component analysis. *Journal of American Medical Association Psychiatry*, *72*(5), 475-482.
- Lo, C. C., & Cheng, T. C. (2014). Race, unemployment rate, and chronic mental illness: a 15-year trend analysis. *Social psychiatry and psychiatric epidemiology*, *49*(7), 1119-1128.
- Looper, K., Kirmayer, L. (2002). Behavioral medicine approaches to somatoform disorders. *Journal of Consulting and Clinical Psychology*, *70*, 810-827.
- Magugen, S., Luxton,D., Skopp, N., & Madden. E. (2012). Gender differences in traumatic experiences and mental health in active duty soldiers redeployed from Iraq and Afghanistan. *Journal of Psychiatric Research*, *46*, 311-316.
- Maldonado, J. & Spiegel, D. (2014). Dissociative Disorders. In R. Hales, S. Yudofsky, L., Weiss Roberts (Eds.), *The American Psychiatric Publishing Textbook of Psychiatry*, 6th ed.
- Mancebo, M., Eisen, J., Sibrava, N., Dyck, I., & Rasmussen, S. (2011). Patient utilization of cognitive-behavior therapy for OCD. *Behavior Therapy*, *42*, 399-412.
- March, J., Frances, A., Kahn, D., & Carpenter, D. (1997). The Expert Consensus Guideline series: Treatment of obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *58*,

1-72.

Marcoux, L., Michon, P., Lemelin, S., Voisin, J., Vachon-Preseau, E., & Jackson, P. (2014).

Feeling but not caring: Empatheic alteration in narcissitic men with high psychopathic traits. *Psychiatry Research*, 224(3), 341-348.

Marinova, Z., Chuang, D., & Fineberg, N. (2017). Glutamate-modulating drugs as a

potential therapeutic strategy in obsessive-compulsive disorder. *Current Neuropharmacology*, 15, 977-995.

Mark, T. L., Coffey, R. M., Vandivort-Warren, R., & Harwood, H. J. (2005). US spending for

mental health and substance abuse treatment, 1991-2001. *Health Affairs*, 24, W5.

Marlatt, G. & Donovan, D. (2005). *Relapse prevention: Maintenance strategies in the treatment*

of addictive behaviors. New York, NY: Guilford.

Marsh, R., Horga, G., Parashar, N., Wang, Z., Peterson, B., & Simpson, H. (2014). Altered

activation in fronto-striatal circuits during sequential processing of conflict in unmedicated adults with obsessive-compulsive disorder. *Biological Psychiatry*, 75, 615-622.

Marshall, S., Landau, M., Carroll, C., Schweiters, B., Llewellyn, A., Liskow, B., ...Bienenfeld,

D. (2013). Conversion disorders. Retrieved from

<http://emedicine.medscape.com/article/287464-overview>.

Mårtensson, G., Jacobsson, J. W., & Engström, M. (2014). Mental health nursing staff's attitudes

towards mental illness: an analysis of related factors. *Journal of psychiatric and mental health nursing*, 21(9), 782-788.

Mashiach-Eizenberg, M., Hasson-Ohayon, I., Yanos, P. T., Lysaker, P. H., & Roe, D. (2013).

Internalized stigma and quality of life among persons with severe mental illness: the mediating roles of self-esteem and hope. *Psychiatry research*, 208(1), 15-20.

Maslow, A. H. (1954). *Motivation and personality*. New York: Harper.

Masoumeh, H., & Lancy, D. S. (2016). Effectiveness of systematic desensitization and cognitive behaviour therapy in reduction of depression among obsessive compulsive disorder patients-A Comparative Study. *International Journal of Psychology and Psychiatry*, 4(1), 56-71.

McCabe, R., Wilsnack, S., West, B., & Boyd, C. (2010). Victimization and substance use disorders in a national sample of heterosexual and sexual minority women and men. *Addiction*, 105, 2130-2140.

McCallie, M., Blum, C., & Hood, C. (2006). Progressive muscle relaxation. *Journal of Human Behavior in the Social Environment*, 3, 51-66.

McDermott, B., Leamon, M., Feldman, M., & Scott, C. (2012). Factitious disorder and malingering. In JA Bourgeois, U Parthasarathi, & A Hategan (Eds.). *Psychiatry review and Canadian certification exam preparation guide* (pp. 267-276). Arlington, VA: American Psychiatric Publishing.

McGeary, D., Harzell, M., McGeary, C., & Gatchel, R. (2016). Somatic Disorders. In J.C. Norcross, G.R. VandenBos, and D.K. Freedheim (Eds). *APA Handbook of Clinical Psychology: Vol 4. Psychopathology and Health (209-223)*. Washington DC, US: American Psychological Association.

McGinty, E. E., Goldman, H. H., Pescosolido, B., & Barry, C. L. (2015). Portraying mental

- illness and drug addiction as treatable health conditions: effects of a randomized experiment on stigma and discrimination. *Social Science & Medicine*, 126, 73-85.
- McGinty, E. E., Webster, D. W., Jarlenski, M., & Barry, C. L. (2014). News media framing of serious mental illness and gun violence in the United States, 1997-2012. *American Journal of Public Health*, 104(3), 406-413.
- McGorry, P., Nelson, B., Goldstone, S., & Yung, A. (2010). Clinical staging: a heuristic and practical strategy for new research and better health and social outcomes for psychotic and related mood disorders. *Canadian Journal of Psychiatry*, 55, 486-496.
- McGrath, J., & Castle, D. (1995). Does influenza cause schizophrenia? A five year review. *Australian and New Zealand Journal of Psychiatry*, 29(1), 23-31.
- McGrath, J. J., Pemberton, M. R., Welham, J. L., & Murray, R. M. (1994). Schizophrenia and the influenza epidemics of 1954, 1957 and 1959: a southern hemisphere study. *Schizophrenia Research*, 14(1), 1-8.
- McGuffin, P., Katz, R., Watkins, S., & Rutherford, J. (1996). A hospital-based twin register of the heritability of DSM-IV unipolar depression. *Archives of General Psychiatry*, 53, 129-136.
- McKay, D., Sookman, D., Neziroglu, F., Wilhelm, S., Stein, D. J., Kyrios, M., ... & Veale, D. (2015). Efficacy of cognitive-behavioral therapy for obsessive-compulsive disorder. *Psychiatry research*, 225(3), 236-246.
- McKenna, K., Gallagher, K., Forbes, P., & Ibeziako, P. (2015). Ready, set, relax: Biofeedback-assisted relaxation training (BART) in a pediatric psychiatry consultation service. *Psychosomatics*, 56, 381-389.
- McKee, A., Stein, T., Nowinski, J., Stern, R., Daneshvar, D., Alvarez, V., ... & Cantu, R.

- (2013). The spectrum of disease in chronic traumatic encephalopathy. *Brain*, 136(1), 43-64.
- McLean, C. & Anderson, E. (2009). Brave men and timid women? A review of the gender differences in fear and anxiety. *Clinical Psychology Review*, 29, 496-505.
- McNally, R. (2003). *Remembering trauma*. Cambridge, MA: Belknap Press.
- McNally, R. (2004). Psychological debriefing does not prevent posttraumatic stress disorder. *Psychiatric Times*, p.71.
- McSweeney, S. (2004). Depression in women. In L. Cosgrove & P.J. Caplan (Eds.), *Bias in psychiatric diagnosis* (pp. 183-188). Northvale, NJ: Jason Aronson.
- Medford, N., Sierra, M., Baker, D., & David, A. (2005). Understanding and treating depersonalization disorder, *Advances in Psychiatric Treatment*, 11(2), 92-100.
- Mell, L. K., Davis, R. L., & Owens, D. (2005). Association between streptococcal infection and obsessive-compulsive disorder, Tourette's syndrome, and tic disorder. *Pediatrics*, 116(1), 56-60.
- Meloy, J. & Yakeley, J. (2010). Psychodynamic treatment of antisocial personality disorder: Psychodynamic psychotherapy for personality disorders: A clinical handbook. In J. Clarkin, P. Fonagy, & G. Gabbard (Eds.), *Psychodynamic psychotherapy for personality disorders: A clinical handbook*. (pp. 311-336). Arlington, VA: American Psychiatric Publishing.
- Melrose, S. (2015). Seasonal affective disorder: an overview of assessment and treatment approaches. *Depression research and treatment*, 2015.
- Melville, J. D., & Naimark, D. (2002). Punishing the insane: The verdict of guilty but mentally ill. *Journal of the American Academy of Psychiatry and the Law Online*, 30(4), 553-555.

- Merikangas, K., Stolar, M., Stevens, D., Goulet, J., Preisig, M., Fenton, B., ... Rounsaville, B. (1998). Familial Transmission of Substance Use Disorders. *Archives of General Psychiatry*, 55, 973-979.
- Metzl, J. M., & MacLeish, K. T. (2015). Mental illness, mass shootings, and the politics of American firearms. *American journal of public health*, 105(2), 240-249.
- Meyers, R. & Squires, D. (2001). *The community reinforcement approach: A guideline developed for the Behavioral Health Recovery Management project*. Albuquerque, NM: University of New Mexico Center on Alcoholism, Substance Abuse, and Addiction.
- Mignon, S. (2014). *Substance abuse treatment: Options, challenges, and effectiveness*. New York, NY: Springer Publishing.
- Millon, T. (2011). *Disorders of personality: Introducing a DSM/ICD spectrum from normal to abnormal* (3rd ed.). Hoboken, NJ: John Wiley Sons.
- Minsky, S., Vega, W., Miskimen, T., Gara, M., & Escobar, J. (2003). Diagnostic patterns in latino, african american, and european american psychiatric patients. *Archives of general psychiatry*, 60(6), 637-644.
- Moller, H.J., Grunze, H., & Broich, K. (2006). Do recent efficacy data on the drug treatment of acute bipolar depression support the position that drugs other than antidepressants are the treatment of choice?: A conceptual review. *European Archives of Psychiatry and Clinical Neuroscience*, 256, 1-16.
- Moscovitch, D., Rowa, K., Paulitzki, J., Ierullo, M., Chiang, B., Antony, M., & McCabe, R. (2013). Self-portrayal concerns and their relation to safety behaviors and negative affect in social anxiety disorder. *Behavior Research and Therapy*, 51, 476-486.
- Mossakowski, K. N. (2003). Coping with perceived discrimination: Does ethnic identity protect

- mental health?. *Journal of health and social behavior*, 318-331.
- Mossman, D., Noffsinger, S. G., Ash, P., Frierson, R. L., Gerbasi, J., Hackett, M., ... & Wall, B. W. (2007). AAPL practice guideline for the forensic psychiatric evaluation of competence to stand trial. *Journal of the American Academy of Psychiatry and the Law Online*, 35(Supplement 4), S3-S72.
- Munafò, M., Brown, S., & Hariri, A. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry*, 63(9), 852-857.
- Murphy, R., Straebl, S., Basden, S., Cooper, Z., & Fairbur, C. (2012). Interpersonal psychotherapy for eating disorders. *Clinical Psychology and Psychotherapy*, 19, 150-158.
- Murray, H. B., Thomas, J. J., Hinz, A., Munsch, S., & Hilbert, A. (2018). Prevalence in primary school youth of pica and rumination behavior: The understudied feeding disorders. *International Journal of Eating Disorders*.
- Naeem, F., Saeed, S., Irfan, M., Kiran, T., Mehmood, N., Gul, M., ... & Farooq, S. (2015). Brief culturally adapted CBT for psychosis (CaCBTp): a randomized controlled trial from a low income country. *Schizophrenia research*, 164(1), 143-148.
- Najman, J., Khatun, M., Mamun, A., Clavarino, A., Williams, G., Scott, J., . . . Alati, R. (2014). Does depression experienced by mothers lead to a decline in marital quality: A 21-year longitudinal study. *Social Psychiatry and Psychiatric Epidemiology*, 49, 121-132.
- National Center for Posttraumatic Stress Disorder. (n.d.) *Acute Stress Disorder*. Retrieved October 23, 2017, from <https://www.ptsd.va.gov/professional/treatment/early/acute-stress-disorder.asp>
- National Institute of Neurological Disorders and Stroke. (2017). NINDS Parkinson's disease

information page, Retrieved from <https://www.ninds.nih.gov/Disorders/All-Disorders/Parkinsons-Disease-Information-Page>

- Neacsiu, A., Eberle, J., Kramer, R., Wiesmann, T., & Linehan, M. (2014). Dialectical behavior therapy skills for transdiagnostic emotion dysregulation: A pilot randomized controlled trial. *Behaviour Research and Therapy*, *59*, 40-51.
- Neighbors, H. W., Trierweiler, S. J., Ford, B. C., & Muroff, J. R. (2003). Racial differences in DSM diagnosis using a semi-structured instrument: The importance of clinical judgment in the diagnosis of African Americans. *Journal of Health and Social Behavior*, *237-256*.
- Nestadt, G., Samuels, J., Riddle, M., Bienvenu, J., Liang, K., LaBuda, M., . . . Hoehn-Saric, R. (2000). A family study of obsessive-compulsive disorder, *Archives of General Psychiatry*, *57*, 358-363.
- Newnham, E. & Janca, A. (2014). Childhood adversity and borderline personality disorder: A focus on adolescence. *Current Opinion in Psychiatry*, *27*(1), 68-72.
- Nezlek, J., Hampton, C. & Shean, G. (2000). Clinical depression and day-to-day social interaction in a community sample. *Journal of Abnormal Psychology*, *109*, 11-19.
- Ng, E., Muntaner, C., Chung, H., & Eaton, W. W. (2014). Socioeconomic status and mental illness. *The Wiley Blackwell Encyclopedia of Health, Illness, Behavior, and Society*.
- NIAAA (National Institute on Alcohol Abuse and Alcoholism). (1997). *Alcohol Alert: Alcohol Metabolism*. No. 35, PH 371. Bethesda, MD: The Institute.
- Nichols, W. (2013). Integrative marital and family treatment of dependent personality disorders. In M. MacFarlane (Ed). *Family Treatment of Personality Disorders: Advances in Clinical Practice*. (pp. 173-204) New York, NY: Routledge.
- Nilni, Y., Rohan, K., & Zvolensky, M. (2012). The role of menstrual cycle phase and anxiety

- sensitivity in catastrophic misinterpretation of physical symptoms during a CO2 challenge. *Archives of Women's Mental Health*, 1-10.
- NIMH (National Institute of Mental Health; 2013). *Generalized anxiety disorder*. NIH Publications No. TR 10-4677. U.S. Department of Health and Human Services, National Institutes of Health.
- Nivoli, A., Colom, F., Murru, A., Pacchiarotti, I., Castro-Loli, P., Gonzalez-Pinto, A., ... Vieta, E. (2011). New treatment guidelines for acute bipolar depression: A systematic review. *Journal of Affective Disorders*, 129(1-3), 14-26.
- Nolen-Hoeksema, S. (2001). Gender differences in depression. *Current Directions in Psychological Research*, 10, 173-176.
- Nolen-Hoeksema, S. (2012). Emotion regulation and psychopathology: The role of gender. *Annual Review of Clinical Psychology*, 8, 161-187.
- Nolen-Hoeksema, S., Girgus, J., & Seligman, M. (1992). Predictors and consequences of childhood depressive symptoms: A 5-year longitudinal study. *Journal of Abnormal Psychology*, 101(3), 405-422.
- Norris, M. L., & Katzman, D. K. (2015). Change is never easy, but It is possible: reflections on avoidant/restrictive food intake disorder Two years after its introduction in the DSM-5. *Journal of Adolescent Health*, 57(1), 8-9.
- NSDUH (National Survey on Drug Use and Health). (2013). *Results from the 2012 National Survey on Drug Use and Health: Mental health findings*, NSDUH Series H-47, HHS Publication NO. (SMA) 13-4805. Rockville, MD: Substance Abuse and Mental Health Services Administration.
- O'Callaghan, E., Sham, P. C., Takei, N., Murray, R. M., & Glover, G. (1991). Schizophrenia

- after prenatal exposure to 1957 A2 influenza epidemic. *The Lancet*, 337(8752), 1248-1250.
- Oei, T. & Morawska, A. (2004). A cognitive model of binge drinking: The influence of alcohol expectancies and drinking refusal self-efficacy. *Addictive Behaviors*, 29, 159-179.
- Olatunji, B. O., Kauffman, B. Y., Meltzer, S., Davis, M. L., Smits, J. A., & Powers, M. B. (2014). Cognitive-behavioral therapy for hypochondriasis/health anxiety: A meta-analysis of treatment outcome and moderators. *Behaviour research and therapy*, 58, 65-74.
- Olden, K. W. (2001). Rumination. *Current Treatment Options in Gastroenterology*, 4(4), 351-358.
- Oliver, C., Petty, J., Ruddick, L., & Bacarese-Hamilton, M. (2012). The association between repetitive, self-injurious and aggressive behavior in children with severe intellectual disability. *Journal of autism and developmental disorders*, 42(6), 910-919.
- Owens, M., Herbert, J., Jones, P., Shakian, A., Wilkinson, P., Dunn, V., . . . Stuart, G. (2014). Elevated morning cortisol is a stratified population-level biomarker for major depression in boys only with high depressive symptoms. *Proceedings of the National Academy of Sciences of the United States of America*, 111(9), 3638-3643.
- Ozden, A. & Canat, S. (1999). Factitious hemoptysis. *Journal of American Child and Adolescent Psychiatry*, 38, 356-357.
- Papish, A., Kassam, A., Modgill, G., Vaz, G., Zanussi, L., & Patten, S. (2013). Reducing the stigma of mental illness in undergraduate medical education: a randomized controlled trial. *BMC medical education*, 13(1), 141.

- Paris, J. (1996). *Social factors in personality disorder: A biopsychosocial approach to etiology and treatment*. Cambridge: Cambridge University Press.
- Perlis, M. & Gehrman, P. (2013). Psychophysiological insomnia. *Encyclopedia of Sleep*, 203-204.
- Petersen, R. (2011). Mild cognitive impairment. *New England Journal of Medicine*, 364, 2227-2234.
- Phillips, K., Grant, J., Siniscalchi, J. & Albertini, R. (2001). Surgical and nonpsychiatric medical treatment of patients with body dysmorphic disorder. *Psychosomatics*, 42, 504-510.
- Phillips, K., Pagano, M., Menard, W., & Stout, R. (2006). A 12-month follow-up study of the course of body dysmorphic disorder. *American Journal of Psychiatry*, 163, 907-912.
- Piazza-Gardner, A. & Barry, G. (2013). The impact of alcohol on Alzheimer's disease: a systematic review. *Journal of Aging and Mental Health*, 17, 133-146.
- Picard, L., Mayor-Dubois, C., Maeder, P., XXX. (2013). Functional independence within the self-memory system: new insights from two cases of developmental amnesia. *Cortex*, 49, 1463-1481.
- Pieper, S., Out, D., Bakermans-Kranenburg, M., Van Ijzendoorn, M. (2011). Behavioral and molecular genetics of dissociation: the role of the serotonin transporter gene promoter polymorphism (5-HTTLPR). *Journal of Traumatic Stress*, 24, 373-380.
- Pitschel-Walz, G., Leucht, S., Bäumi, J., Kissling, W., & Engel, R. R. (2001). The effect of family interventions on relapse and rehospitalization in schizophrenia—A meta-analysis. *Schizophrenia Bulletin*, 27, 73–92.
- Poels, M., Ikram, M., van der Lugt, A., Horman, A., Niessen, W., Krestin, G., ... Vernooij, M.

- (2012). Cerebral microbleeds are associated with worse cognitive function. *Neurology*, 78, 326-333.
- Poletti, M., & Bonuccelli, U. (2013). Acute and chronic side effects of levodopa and dopamine agonists on patients with Parkinson's disease: A review. *Therapeutic Advances in Psychopharmacology*, 3, 101-113.
- Polivy, J. & Herman, C. (2002). Causes of eating disorders. *Annual Review of Psychology*, 53, 187-213.
- Polivy, J. & Herman, C. (2004). Sociocultural idealization of thin female body shapes: an introduction to the special issue on body image and eating disorders. *Journal of Social & Clinical Psychology*, 23, 1-6.
- Pope, C., Pope, H., Menard, W., Fay, C., Olivardia, R., & Philips, K. (2005). Clinical features of muscle dysmorphia among males with body dysmorphic disorder. *Body Image*, 2, 395-400.
- Possel, P. & Black, S. (2014). Testing three different sequential mediational interpretations of Beck's cognitive model of the development of depression. *Journal of Clinical Psychology*, 70(1), 72-94.
- Poulsen, S., Lunn, S., Daniel, S. I., Folke, S., Mathiesen, B. B., Katznelson, H., & Fairburn, C. G. (2014). A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. *American Journal of Psychiatry*, 171(1), 109-116.
- Prochaska, J., & Norcross, J. (2013). *Systems of psychotherapy: A transtheoretical analysis*. Nelson Education.
- Purdy, J. (2013). Chronic physical illness: A psychophysiological approach for chronic physical

- illness. *Yale Journal of Biological Medicine*, 86, 15-28.
- Raad, R., & Makari, G. (2010). Samuel Tuke's description of the retreat. *The American Journal of Psychiatry*, 167(8), 898.
- Raevouni, A., Keski-Rahkonen, & Hoek, H. (2014). A review of eating disorders in males. *Current Opinions on Psychiatry*, 27, 426-430.
- Raj, V., Rowe, A., Fleisch, S., Paranjape, S., Arain, A., & Nicolson, S. (2014). Psychogenic pseudosyncope: Diagnosis and management. *Autonomic Neuroscience: Basic and Clinical*, 184, 66-72.
- Rasmussen, S. & Eisen, J. (1990). Epidemiology of obsessive compulsive disorder. *The Journal of Clinical Psychiatry*, 51, 10-13.
- Reinhold, J., & Rickels, K. (2015). Pharmacological treatment for generalized anxiety disorder in adults: an update. *Expert Opinion in Pharmacotherapy*, 16, 1669-1681.
- Resick, P., Monson, C., & Rizvi, S. (2008). Posttraumatic stress disorder. In Barlow, D. (Ed), *Clinical Handbook of Psychological Disorders* (pp. 65-122). New York, NY: Guilford Press.
- Richardson, J., Roy, A., Shalat, S., von Stein, R., Hossain, M., Buckley, B., ...German, D. (2014). Elevated serum pesticide levels and risk for Alzheimer disease. *Journal of American Medical Association Neurology*, 71(3), 284-290.
- Richardson, L.F. (1998). Psychogenic dissociation in childhood: The role of the counseling psychologist. *Counseling Psychologist*, 26, 69-100.
- Rieker, P. & Bird, C. (2005). Rethinking gender differences in health: What we need to integrate social and biological perspectives. *Journal of Gerontology*, 60, S40-S47.

- Rihmer, Z., & Kiss, K. (2002). Bipolar disorders and suicidal behaviour. *Bipolar Disorders*, 4(s1), 21-25.
- Rodebaugh, T., Holaway, R., & Heimberg, R. (2004). The treatment of social anxiety disorder. *Clinical Psychology Review*, 24, 883-908.
- Roepke, S., & Varter, A. (2014). Narcissistic personality disorder: An integrative review of recent empirical data and current definitions. *Current Psychiatry Reports*, 16, 445.
- Roh, D., Chang, J., Kim, C., Cho, H., An, S., & Jung, Y. (2014). Antipsychotic polypharmacy and high-dose prescription in schizophrenia: A 5-year comparison. *Australian and New Zealand Journal of Psychiatry*, 48 (1), 52-60.
- Roose, S., & Spatz, E. (1999). Treating depression in patients with ischemic heart disease. *Drug Safety*, 20, 459-465.
- Roy-Byrne, P., Arguelles, L., Vitek, M., Goldberg, J., Keane, T., Ture, W., & Pitman, R. (2004). Persistence and change of PTSD symptomatology: A longitudinal co-twin control analysis of the Vietnam Era Twin Registry, *Social Psychiatry and Psychiatric Epidemiology*, 39, 681-685.
- Rozel, J. S., & Mulvey, E. P. (2017). The link between mental illness and firearm violence: implications for social policy and clinical practice. *Annual review of clinical psychology*, 13, 445-469.
- Rüsch, N., Nordt, C., Kawohl, W., Brantschen, E., Bärtzsch, B., Müller, M., ... & Rössler, W. (2014). Work-related discrimination and change in self-stigma among people with mental illness during supported employment. *Psychiatric Services*, 65(12), 1496-1498.
- Rüsch, N., Zlati, A., Black, G., & Thornicroft, G. (2014). Does the stigma of mental illness

contribute to suicidality?.

Rusyniak, D. (2011). Neurological manifestations of chronic methamphetamine abuse.

Neurologic Clinics, 29, 641-655.

Ryder, A. G., Sunohara, M., & Kirmayer, L. J. (2015). Culture and personality disorder: from a fragmented literature to a contextually grounded alternative. *Current opinion in psychiatry*, 28(1), 40-45.

Salkovskis, P., Thorpe, S., Wahl, K., Wroe, A., & Forrester, E. (2003). Neutralizing increases discomfort associated with obsessional thoughts: An experimental study with obsessional patients. *Journal of Abnormal Psychology*, 112, 709-715.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2013). *National survey on drug use and health, 2011 and 2012*. Washington, DC: Department of Health and Human Services.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2014). *Results from the 2013 National Survey on Drug Use and Health: Summary of national findings*, NSDUH Series H-48, HSS Publication No. (SMA) 14-4863. Rockville, MD: SAMHSA.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2015). *National Survey on Drug Use and Health (NSDUH)—Alcohol Use in Lifetime, Past Year, and Past Month among Persons Aged 12 or Older, by Demographic Characteristics: Percentages, 2014 and 2015*. Washington, DC: Department of Health and Human Services.

SAMHSA (Substance Abuse and Mental Health Services Administration). (2016). *Prescription*

- Drug Use and Misuse in the United States: Results from the 2015 National Survey on Drug Use and Health*. Washington, DC: Department of Health and Human Services.
- Sandler, R. (1990). Epidemiology of irritable bowel syndrome in the United States. *Gastroenterology*, *99*, 409-415.
- Sansone, R., & Sansone, L. (2012). Medically self-sabotaging behavior and its relationship with borderline personality. *Primary Care Reports*, *18*, 37-47.
- Sar, V. (2011). Epidemiology of dissociative disorders: An overview. *Epidemiology Research International*. Retrieved from <https://www.hindawi.com/archive/2011/404538/>
- Sar, V., Onder, C., Kilincaslan, A., Zoroglu, S., & Alyanak, B. (2014). Dissociative identity disorder among adolescents: Prevalence in a university psychiatric outpatient unit. *Journal of Trauma & Dissociation*, *15*(4), 402-419.
- Satcher, D. (2001). *Mental health: Culture, race, and ethnicity—A supplement to mental health: A report of the surgeon general*. US Department of Health and Human Services.
- Schmidt, U., Kalgwasser, S., & Wotjak, C. (2013). Biomarkers in posttraumatic stress disorder: Overview and implications for future research. *Disease Markers*, *35*, 43-54.
- Schmidt, N., Woolaway-Bickel, K., Trakowski, J., Santiago, H., Storey, J., Koselka, M., & Cook, J. (2000). Dismantling cognitive-behavioral treatment for panic disorder: Questioning the utility of breathing retraining. *Journal of Consulting and Clinical Psychology*, *68*(3), 417-424.
- Schomerus, G., Van der Auwera, S., Matschinger, H., Baumeister, S. E., & Angermeyer, M. C. (2015). Do attitudes towards persons with mental illness worsen during the course of life?

- An age-period-cohort analysis. *Acta Psychiatrica Scandinavica*, 132(5), 357-364.
- Schuch, J., Roest, A., Nolen, W., Penninx, B., & de Jonge, P. (2014). Gender difference in major depressive disorder: Results from the Netherlands study of depression and anxiety. *Journal of Affective Disorders*, 156, 156-163.
- Schulte, I., Petermann, F., & Noeker, M. (2010). Functional abdominal pain in childhood: From etiology to maladaptation. *Psychotherapy and Psychosomatics*, 79, 73-86.
- Schultz, G. (2007). Marital breakdown and divorce increases rates of depression, Stat-Can study finds. *LifeSiteNews.com*
- Schwartz, R. C., & Blankenship, D. M. (2014). Racial disparities in psychotic disorder diagnosis: A review of empirical literature. *World journal of psychiatry*, 4(4), 133.
- Schwartz, R., Brooner, R., Montoya, I., Currens, M., & Hayes, M. (2010). A 12-year follow-up of a methadone medical maintenance program. *The American Journal on Addictions*, 8, 293-299.
- Scognamiglio, C. & Houenou, J. (2014). A meta-analysis of fMRI studies in healthy relatives of patients with schizophrenia. *Australian and New Zealand Journal of Psychiatry*, 48(10), 907-916.
- Seidler, G. & Wagner, F. (2006). Comparing the efficacy of EMDR and trauma-focused cognitive-behavioral therapy in the treatment of PTSD: a meta-analytic study. *Psychological Medicine*, 36, 1515-1522.
- Seligman, M. (1975). *Helplessness*. San Francisco: Freeman.
- Selkoe, D. (1992). Alzheimer's disease: New insights into an emerging epidemic. *Journal of Geriatric Psychiatry*, 25(2), 211-227.

- Selkoe, D. (2011). Alzheimer's disease. *Cold Spring Harbor Perspectives in Biology*, 3(7).
- Selten, J. P., & Termorshuizen, F. (2017). The serological evidence for maternal influenza as risk factor for psychosis in offspring is insufficient: critical review and meta-analysis. *Schizophrenia research*, 183, 2-9.
- Shapiro, F. (1989). Eye movement desensitization: A new treatment for post-traumatic stress disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, 20, 211-217.
- Shapiro, F. & Maxfield, L. (2002). Eye movement desensitization and reprocessing (EMDR): Information processing in the treatment of trauma. *Journal of Clinical Psychology*, 58, 933-946.
- Sherin, J. & Nemeroff, C. (2011). Post-traumatic stress disorder: The neurobiological impact of psychological trauma. *Dialogues in Clinical Neuroscience*, 13, 263-278.
- Shoshani, A., & Steinmetz, S. (2014). Positive psychology at school: A school-based intervention to promote adolescents' mental health and well-being. *Journal of Happiness Studies*, 15(6), 1289-1311.
- Shulman, K., Herrmann, N., & Walker, S. (2013). Current place of monamine oxidase inhibitors in the treatment of depression. *CNS Drugs*, 27, 789-797.
- Sieswerda, S., & Arntz, A. (2007). Successful psychotherapy reduces hypervigilance in borderline personality disorder. *Behavioral and Cognitive Psychotherapy*, 35, 387-402.
- Sinopoli, V., Burton, C., Kronenberg, S., & Arnold, P. (2017). A review of the role of serotonin system genes in obsessive-compulsive disorder. *Neuroscience & Biobehavioral Reviews*, 80, 372-381.
- Skinner, B.F. (1990). Can psychology be a science of the mind? *American Psychologist*, 45, 1206-10.

- Skodol, A., Geier, T., Grant, B., & Hasin, D. (2014). Personality disorders and the persistence of anxiety disorders in a nationally representative sample. *Depression and Anxiety*, 1-8.
- Slovenko, R. (2009). *Psychiatry in law/law in psychiatry*. Taylor & Francis.
- Slovenko, R. (2011). The DSM in litigation and legislation. *The journal of the American Academy of Psychiatry and the Law*, 39(1), 6.
- Smith, B.L. (2012). Inappropriate prescribing. *Monitor on Psychology*, 43(6), 36.
- Sockol, L. E. (2015). A systematic review of the efficacy of cognitive behavioral therapy for treating and preventing perinatal depression. *Journal of Affective Disorders*, 177, 7-21.
- Soreff, S., & McInnes, L., (2014). Bipolar affective disorder. Retrieved from <http://emedicine.medscape.com/article/286342-overview>.
- Spires-Jones, T., & Hyman, B. (2014). The intersection of amyloid beta and tau at synapses in Alzheimer's disease. *Neuron*, 82(4), 756-771.
- Stahl, S. & Wise, D. (2008). The potential role of a corticotrophin-releasing factor receptor-1 antagonist in psychiatric disorders. *CNS Spectrums*, 13, 467-478.
- Staniloiu, A. & Markowitsch, H. (2010). Searching for the anatomy of dissociative amnesia. *Journal of Psychology*, 218, 96-108.
- Stavro, K., Pelletier, J., & Potvin, S. (2013). Widespread and sustained cognitive deficits in alcoholism: A meta-analysis. *Addiction Biology*, 18, 203-213.
- Steinert, C., Klein, S., Leweke, F., & Leichsenring, F. (2015). Do personality traits predict outcome of psychodynamically oriented psychosomatic inpatient treatment beyond initial symptoms? *British Journal of Clinical Psychology*, 54, 109-125.
- Stice, E. & Bohon, C. (2012). Eating Disorders. In *Child and Adolescent Psychopathology*, 2nd

- Edition*, Theodore Beauchaine & Stephen Linshaw, eds. New York: Wiley.
- Stice, E., Marti, C., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-Year prospective community study of young women. *Journal of Abnormal Psychology, 122*, 445-457.
- Stone, M. (2014). The spectrum of borderline personality disorder: A neurophysiological view. *Current Topics in Behavioral Neurosciences, 21*, 23-46.
- Street, A., Bell, M. & Ready, C. (2011). Sexual assault. In D.M. Benedek, & G.H. Wynn (Eds.) *Clinical manual for management of PTSD* (pp. 325-348) Arlington, VA: American Psychiatric Publishing.
- Strother, E., Lemberg, R., Stanford, S., & Turberville, D. (2012). Eating disorders in men: underdiagnosed, undertreated, and misunderstood. *Eating Disorders, 20*(5), 346-355.
- Sue, D., & Sue, D. (2016). *Counseling the culturally diverse: Theory and practice* (7th ed). Hoboken, NJ: Wiley.
- Sue, D., Sue, D., Sue, D., & Sue, S. (2017). *Essentials of understanding abnormal behavior*, 3rd Ed. Boston: MA, Cengage Learning.
- Sung, J., Kuipers, E., El-Serag, H. (2009). Systematic review: The global incidence and prevalence of peptic ulcer disease. *Aliment Pharmacological Theory, 29*, 938-946.
- Szalavits, M. (2013). How facebook improves memory. *Time*. Retrieved from Time website: <http://healthland.time.com/2013/03/01/how-facebook-improves-memory/>
- Szasz, T. S. (1960). The myth of mental illness. *American psychologist, 15*(2), 113.
- Sykes, M., Blanchard, E., Lackner, J., Keefer, L., & Krasner, S. (2003). Psychopathology in irritable bowel syndrome: Support for a psychophysiological model. *Journal of*

Behavioral Medicine, 26, 361-372.

- Tajfel, H. (1982). Social psychology of intergroup relations. *Annual Review of Psychology*, 33, 1–39.
- Tenback, D., van Harten, P., Sloof, C., & van Os, J. (2006). Evidence that early extrapyramidal symptoms predict later tardive dyskinesia: a prospective analysis of 10,000 patients in the European Schizophrenia Outpatient Health Outcomes (SOHO) study. *American Journal of Psychiatry*, 163, 1438-1440.
- Terry-McElrath, Y., O'Malley, P., & Johnston, L. (2014). Energy drinks, soft drinks, and substance use among United States secondary school students. *Journal of Addiction Medicine*, 8, 6-13.
- Thompson, D., Ramos, C., & Willett, J. (2014). Psychopathy: Clinical features, developmental basis and therapeutic challenges. *Journal of Clinical Pharmacy and Therapeutics*, 39(5), 485-495.
- Tomko, R. L., Trull, T. J., Wood, P. K., & Sher, K. J. (2014). Characteristics of borderline personality disorder in a community sample: comorbidity, treatment utilization, and general functioning. *Journal of personality disorders*, 28(5), 734-750.
- Trauer, J. Qian, M., Doyle, J., Rajaratnam, S., & Cunnington, D. (2015). Cognitive behavioral therapy for chronic insomnia: A systematic review and meta-analysis. *Annals of Internal Medicine*, 163, 191-204.
- Tsai, G., Condie, D., Wu, M., & Chang, I. (1999). Functional magnetic resonance imaging of personality switches in a woman with dissociative identity disorder. *Harvard Review of Psychiatry*, 7, 119-122.

- Tsuang, M., Lyons, M., Eisen, S., Goldberg, J., True, W., Nang, L., ... Eaves, L. (1996). Genetic influences on abuse of illicit drugs. *American Journal of Medical Genetics*, 5, 473-477.
- Tuckey, M. & Scott, J. (2014). Group critical incident stress debriefing with emergency services personnel: A randomized controlled trial. *Anxiety, Stress, and Coping*, 27, 38-54.
- Turner, J. C. (1987). *Rediscovering the social group: A self-categorization theory*. Oxford, England: Basil Blackwell.
- Unger, J., Schwartz, S., Huh, J., Soto, D., & Baezconde-Garbanati, L. (2014). Acculturation and perceived discrimination: Predictors of substance use trajectories from adolescence to emerging adulthood among Hispanics. *Addictive Behaviors*, 39(9), 1293-1296.
- van Grootheest, D., Cath, D., Beekman, A., & Boomsma, D. (2005). Twin studies on obsessive-compulsive disorder: A review. *Twin Research and Human Genetics*, 8, 450-458.
- Vassallo, M., Durant, J., Biscay, V., Lebrun-Frenay, C., Dunais, B., Laffon, M., ... Dellamonica, P. (2014). Can high central nervous system penetrating antiretroviral regimens protect against the onset of HIV-associated neurocognitive disorders? *AIDS*, 28, 493-501.
- Veale, D., Boocock, A., Goumay, K., Dryden, W., Shah, F., Wilson, R., Walburn, J. (1996). Body dysmorphic disorder. A survey of fifty cases. *The British Journal of Psychiatry*, 169, 196-201.
- Veiga-Martinez, C., Perez-Alvares, M., Garcia-Montes, G. (2008). Acceptance and commitment therapy applied to treatment of auditory hallucinations. *Clinical Case Studies*, 7, 118-135.
- Wagner, K., Ritt-Olson, A., Chou, C., Pokhrel, P., Duan, L., Baezconde-Garbanati, L., ... Unger, J. (2012). Associations between parental family structure, family functioning, and substance use among Hispanic/Latino adolescents. *Psychology of Addictive Behaviors*,

24, 98-108.

Wahlbeck, K. (2015). Public mental health: the time is ripe for translation of evidence into practice. *World Psychiatry, 14*(1), 36-42.

Walker, E. & Diforio, D. (1997). Schizophrenia: A neural diathesis-stress model. *Psychological Review, 104*, 667-685.

Wang, P. S., Berglund, P., Olfson, M., Pincus, H. A., Wells, K. B., & Kessler, R. C. (2005). Failure and delay in initial treatment contact after first onset of mental disorders in the National Comorbidity Survey Replication. *Archives of general psychiatry, 62*(6), 603-613.

Wang, P. S., Lane, M., Olfson, M., Pincus, H. A., Wells, K. B., & Kessler, R. C. (2005). Twelve-month use of mental health services in the United States: results from the National Comorbidity Survey Replication. *Archives of general psychiatry, 62*(6), 629-640.

Watson, J. & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology, 3*, 1-4.

Watt, M., O'Connor, R., Stewart, S., Moon, E., & Terry, L. (2008). Specificity of childhood learning experiences in relation to anxiety sensitivity and illness/injury sensitivity: Implications for health anxiety and pain. *Journal of Cognitive Psychotherapy: An International Quarterly, 22*, 128-143.

Weare, K., & Nind, M. (2011). Mental health promotion and problem prevention in schools: what does the evidence say?. *Health promotion international, 26*(suppl_1), i29-i69.

Weaver, M. & Schnoll, S. (2008). Hallucinogens and club drugs. In H.D. Kleber & M Galanter (Eds), *The American Psychiatric Publishing textbook of substance abuse treatment* (4th ed. pp. 191-200). Arlington, VA: American Psychiatric Publishing.

- Weishaar, M. & Beck, A. (2006). Cognitive theory of personality and personality disorders. In S. Strack (Ed). *Differentiating normal and abnormal personality* (2nd ed. pp. 113-135). New York: Springer Publishing Co.
- Whelan, E. & Cooper, P. (2000). The association between childhood feeding problems and maternal eating disorder: a community study. *Psychological Medicine*, 30, 69-77.
- Whiffen, V. & Demidenko, N. (2006). Mood disturbance across the life span. In J. Worell & C. D. Goodheart (Eds.), *Handbook of girls' and women's psychological health* (pp. 51-59). New York: Oxford University Press.
- Whiteford, H. A., Degenhardt, L., Rehm, J., Baxter, A. J., Ferrari, A. J., Erskine, H. E., ... & Burstein, R. (2013). Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. *The Lancet*, 382(9904), 1575-1586.
- Wilens, T., Yule, A., Martelon, M., Zulauf, C., & Faraone, S. (2014). Parental history of substance use disorders (SUD) and SUD in offspring: A controlled family study of bipolar disorder. *American Journal of Addictions*, 23(5), 440-446.
- Wilhelm, S., Otto, M., Lohr, B., & Deckersbach, T. (1999). Cognitive behavior group therapy for body dysmorphic disorder: a case series. *Behaviour Research and Therapy*, 37, 71-75.
- Williamson, D. (1981). Behavioral treatment of migraine and muscle-contraction headaches: Outcome and theoretical explanations. In M. Hersen, R. Eisler, & P. Miller (Eds.), *Progress in behavior modification*, New York, NY: Academic Press.
- Williamson, D. Barker, S., & Veron-Guidry, S (1994). Psychophysiological Disorders. In: V. Van Hasselt & M. Hersen (Ed.) *Advanced Abnormal Psychology* (373). Boston, MA: Springer.

- Williams, M. & Viscusi, J. (2015). Hoarding disorder and a systematic review of treatment with cognitive behavioral therapy. *Cognitive Behaviour Therapy*, 45, 93-110.
- Wilson, R., Segawa, E., Boyle, P., & Bennett, D. (2012). Influence of late-life cognitive activity on cognitive health. *Neurology*, 78(15), 1123-1129.
- Wise, R., & Koob, G. (2013). The development and maintenance of drug addiction. *Neuropsychopharmacology*, 39, 254-262.
- Witthoft, M. & Hiller, W. (2010). Psychological approaches to origins and treatments of somatoform disorders. *Annual Review of Clinical Psychology*, 6, 257-283.
- Wolpe, J. (1997). *From psychoanalytic to behavioral methods in anxiety disorders: A continuing evolution*. Brunner/Mazel.
- World Health Organization. (2013). *Guidelines for the management of conditions that are specifically related to stress*. Geneva, Switzerland: Author.
- Wu, L., Ringwalt., C., Weiss, R., & Blazer, D. (2009). Hallucinogen-related disorders in a national sample of adolescents: the influence of ecstasy/MDMA use. *Drug and Alcohol Dependence*, 104, 156-166.
- Yadin, E. & Foa, E. (2009). How to reduce distress and repetitive behaviors in patients with OCD. *Current Psychiatry*, 8, 19-23.
- Yanos, P. T., Roe, D., & Lysaker, P. H. (2011). Narrative enhancement and cognitive therapy: a new group-based treatment for internalized stigma among persons with severe mental illness. *International journal of group psychotherapy*, 61(4), 576-595.
- Yes, Bayer Promoted Heroin – Here are the ads that prove it. Business Insider

- Website. <http://www.businessinsider.com/yes-bayer-promoted-heroin-for-children-here-are-the-ads-that-prove-it-2011-11?op=1>. Published on November 17, 2011. Accessed on November 4, 2017.
- Zafra, J. (2016). The Use of Structural Family Therapy With a Latino Family: A Case Study. *Journal of Systemic Therapies*, 35(4), 11-21.
- Zeanah, C. H., Chesher, T., Boris, N. W., and the American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI). (2016). Practice parameter for the assessment and treatment of children and adolescents with reactive attachment disorder and disinhibited social engagement disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 55(11), 990-1003.
- Zerbe, K. (2008). *Integrated treatment of eating disorders: Beyond the body betrayed*. New York, NY: W.W. Norton & Co.
- Zeschel, E., Bingmann, T., Bechdolf, A., Kruger-Oezguerdal, S., Correll, C., Leopold, K., . . . Juckel, G. (2015). Temperament and prodromal symptoms prior to first manic/hypomanic episodes: Results from a pilot study. *Journal of Affective Disorders*, 173, 339-344.

Index

#

20th to 21st Centuries, and views of mental illness – Section 1.4.6

A

Abnormal behavior – Section 1.1.1

Abnormal psychology – Section 1.1.3

Absolute refractory period – Section 2.2.1.4

Acceptance techniques – Section 2.3.3.5

Action potential – Section 2.2.1.4

Acute Stress Disorder – Section 5.2.2

 Epidemiology of – Section 5.3.2

 Comorbidity of – Section 5.4.2

Adjustment Disorder – Section 5.2.3

 Epidemiology of – Section 5.3.3

 Comorbidity of – Section 5.4.3

Affect, types of – Section 2.1

Affective flattening – Section 12.1.1.6

Affective states – Section 2.1

Agoraphobia – Section 7.1.3

 Comorbidity – Section 7.3.3

 Epidemiology of – Section 7.2.3

 Treatment of – Section 7.5.3

All-or-nothing principle Section 2.2.1.4

Alogia – Section 12.1.1.6

Alzheimer’s disease – Section 14.3.1

American Law Institute standard – Section 15.1.3

Amygdala – Section 2.2.1.6

Anal Stage – Section 2.3.1.2

Anhedonia – Section 12.1.1.6

Anorexia nervosa – Section 10.1.1

 and EDNOS – Section 10.2.3

 Comorbidity – Section 10.4.1

 Treatment of – Section 10.6.1

Antecedents – Section 3.1.3.6

Antisocial personality disorder – Section 13.1.3.1

 Treatment of – Section 13.5.2.1

Apathy – Section 12.1.1.6

Asociality – Section 12.1.1.6

Attention deficit/hyperactivity disorder – Section 16.3.1

Attributions and cognitive errors – Section 2.3.3.3

Autism spectrum disorder – Section 16.2.3

Avoidant personality disorder – Section 13.1.4.1

Avoidant/restrictive food intake disorder – Section 16.1.5

Avolition – Section 12.1.1.6

Axon – Section 2.2.1.3

Axon terminals – Section 2.2.1.3

B

Bandura, A. – Section 2.3.2.4

Behavioral assessment – Section 3.1.3.6

Behavioral model – Section 2.3.2

Evaluation of – Section 2.3.2.5

Related to mood disorders – Section 4.5.3

Related to anxiety disorders – Section 7.4.2.2

Related to somatic disorders – Section 8.4.3

Related to obsessive compulsive disorders – Section 9.4.3

Related to substance use and addictive disorders – Section 11.4.3; 11.5.2

Related to personality disorders – Section 13.4.2.3

Behavior modification – Section 2.3.2.5

Behaviors – Section 3.1.3.6

Binge eating disorder (BED) – Section 10.1.3

and EDNOS – Section 10.2.2

Comorbidity – Section 10.4.3

Treatment of – Section 10.6.3

Biological model – Section 2.2

Related to mood disorders – Section 4.5.1

Related to trauma and stressor related disorders – Section 5.5.1

Related to dissociative disorders – Section 6.4.1

Related to anxiety disorders – Section 7.4.1

Related to obsessive compulsive disorders – Section 9.4.1

Related to eating disorders – Section 10.5.1

- Related to substance use and addictive disorders – Section 11.4.1; 11.5.1
- Related to schizophrenic disorders – Section 12.4.1
- Related to personality disorders – Section 13.4.1
- Biological or somatogenic perspective – Section 1.4.6.1
- Bipolar Disorder I and II – Section 4.2
 - Epidemiology of – Section 4.3.2
 - Comorbidity of – Section 4.4.2
 - Treatment of – Section 4.6.2
- Bobo Doll experiment – Section 2.3.2.4
- Body Dysmorphic Disorder – Section 9.1.2
 - Epidemiology of – Section 9.2.2
 - Comorbidity of – Section 9.3.2
 - Treatment of – Section 9.5.2
- Borderline personality disorder – Section 13.1.3.2
 - Treatment of – Section 13.5.2.2
- Brain structure and chemistry – Section 2.2.1
- Bulimia nervosa – Section 10.1.2
 - and EDNOS – Section 10.2.4
 - Comorbidity – Section 10.4.2
 - Treatment of – Section 10.6.2

C

- Cardiovascular – Section 8.6.1.4
- Catatonic behavior – Section 12.1.1.5
- Cathartic method – Section 1.4.6.2
- Cerebellum – Section 2.2.1.6
- Cerebrum, lobes – Section 2.2.1.6
- Chronic traumatic encephalopathy (CTE) – Section 14.3.2
- Civil commitment – Section 15.1.2
- Classification – Section 1.2
- Classification systems – Section 3.2.1
- Client-centered therapy – Section 2.3.4.1
- Client-therapist relationship – Section 3.3.1.4; Section 15.3
- Clinical assessment – Section 3.1.1
- Clinical description – Section 1.2
- Clinical diagnosis – Section 3.2.1
- Clinical interview – Section 3.1.3.2
- Cognitive behavioral therapy – Section 2.3.3.5; Section 5.6.3
 - In relation to schizophrenic disorders – Section 12.5.2.1
- Cognitive coping skills training – Section 2.3.3.5
- Cognitive model – Section 2.3.3
 - Evaluation of – Section 2.3.3.6
 - Related to mood disorders – Section 4.5.2
 - Related to trauma and stressor related disorders – Section 5.5.1
 - Related to dissociative disorders – Section 6.4.2

- Related to anxiety disorders – Section 7.4.2.1
- Related to somatic disorders – Section 8.4.2
- Related to obsessive compulsive disorders – Section 9.4.2
- Related to eating disorders – Section 10.5.2
- Related to substance use and addictive disorders – Section 11.4.2; 11.5.3
- Related to schizophrenic disorders – Section 12.4.2.1
- Related to personality disorders – Section 13.4.2.2
- Cognitive restructuring – Section 2.3.3.5
- Cognitive therapies – Section 2.3.3.5
- Comorbidity – Section 1.2
 - Of mood disorders – Section 4.4
 - Of trauma and stressor related disorders – Section 5.4
 - Of dissociative disorders – Section 6.3
 - Of somatic disorders – Section 8.3
 - Of obsessive compulsive disorders – Section 9.3
 - Of eating disorders – Section 10.4
 - Of substance-related and addictive disorders – Section 11.3
 - Of schizophrenic disorders – Section 12.3
 - Of personality disorders – Section 13.3
- Competent to stand trial – Section 15.1.3
- Computed tomography (CT scan) – Section 3.1.3.4
- Concussion – Section 14.3.2
- Conditioning – Section 2.3.2.1
- Conduct disorder – Section 16.3.3

Confidentiality – Section 15.3
Consequences – Section 3.1.3.6
Contingencies – Section 2.3.2.3
Conversion disorder – Section 8.1.3
Counterconditioning – Section 2.3.2.2
Course – Section 1.2
Cost of mental illness – Section 1.1.2
Criminal commitment – Section 15.1.3
Cultural-sensitive therapies – Section 2.4.4
Culture – Section 1.1.2
Current views/trends, in mental illness – Section 1.4.7

D

Dangerousness – Section 1.1.2; 15.1.2.3
Deinstitutionalization – Section 1.4.7.2
Delirium – Section 14.1.1
Delusional disorder – Section 12.1.5
Delusions – Section 12.1.1.1
Dementia – Section 14.1.2
 With Lewy bodies – Section 14.3.5
Dendrites – Section 2.2.1.3
Denial – Section 2.3.1.3
Dependent personality disorder – Section 13.1.4.2
Depersonalization/Derealization Disorder – Section 6.1.3

Treatment of – Section 6.5.3

Depolarized – Section 2.2.1.4

Depressants – Section 11.1.2.1

Epidemiology of – 11.2.1

Depressive disorders

Epidemiology of – Section 4.3.1

Comorbidity of – Section 4.4.1

Treatment of – Section 4.6.1

Deviance – Section 1.1.2

Diagnosis, elements of – Section 3.2.2.2

Disinhibited social engagement disorder – Section 16.1.1

Disorganized thinking – Section 12.1.1.3

Displacement – Section 2.3.1.3

Dissociative disorders – Section 6.1

Dissociative amnesia disorder – Section 6.1.2

Treatment of – Section 6.5.2

Dissociative identity disorder – Section 6.1.1

Treatment of – Section 6.5.1

Distress – Section 1.1.2

Dopamine – Section 2.2.1.5

Dream Analysis – Section 2.3.1.4

DSM – Section 3.2.2

Disorder categories – Section 3.2.2.3

Durham test (products test) – Section 15.1.3

Duty to Warn – Section 15.3

Dysfunction – Section 1.1.2

E

Eating Disorder Not Otherwise Specified (EDNOS) – Section 10.2

Ego defense mechanisms – Section 2.3.1.3

Electroconvulsive therapy – Section 2.2.3.2

Emotion

 Defined – Section 2.1

 Characteristics of – Section 2.2

Enactive learning – Section 2.3.2.4

Environmental factors, and mental illness – Section 2.4.3

Endorphins – Section 2.2.1.5

Enuresis – Section 16.1.6

Encopresis – Section 16.1.7

Enzymatic degradation – Section 2.2.1.4

Epidemiological study – Section 1.5.2.4

Epidemiology – Section 1.2

 Of mood disorders – Section 4.3

 Of trauma and stressor related disorders – Section 5.3

 Of dissociative disorders – Section 6.2

 Of anxiety disorders – Section 5.2

 Of somatic disorders – Section 8.2

 Of obsessive-compulsive disorders – Section 9.2

- Of eating disorders – Section 10.3
- Of substance-related and addictive disorders – Section 11.2
- Of schizophrenic disorders – Section 12.2
- Of personality disorders – Section 13.2
- Of neurocognitive disorders – Section 14.2
- Eros – Section 2.3.1.1
- Erotomanic delusion – Section 12.1.5
- Etiology – Section 1.2
 - of mood disorders – Section 4.5
 - of trauma and stressor related disorders – Section 5.5
 - Of dissociative disorders – Section 6.4
 - Of anxiety disorders – Section 7.4
 - Of somatic disorders – Section 8.4
 - Of obsessive compulsive disorders – Section 9.4
 - Of eating disorders – Section 10.5
 - Of substance-related and addictive disorders – Section 11.4
 - Of schizophrenic disorders – Section 12.4
 - Of personality disorders – Section 13.4
 - Of neurocognitive disorders – Section 14.3
- Excoriation (Skin-Picking) disorder - Section 16.4.4
- Existential perspective – Section 2.3.4.2
 - Evaluation of – Section 2.3.4.3
- Exorcism – Section 1.4.1
- Experiments – Section 1.5.2.5

Exposure therapy – Section 5.6.2

Extinction, operant conditioning – Section 2.3.2.3

Eye Movement Desensitization and Reprocessing (EMDR) – Section 5.6.4

F

Factitious disorder – Section 8.1.4

Family interventions

 In relation to schizophrenic disorders – Section 12.5.3

Federal Insanity Defense Reform ACT (IDRA) – Section 15.1.3

Fire – Section 2.2.1.4

Fixated – Section 2.3.1.2

Flooding – Section 2.3.2.2

Forensic/legal psychology – Section 15.1.1

Free Association – Section 2.3.1.4

Frontotemporal NCD – Section 14.3.6

Fundamental attribution error – Section 2.3.3.3

G

GABA – Section 2.2.1.5

Gastrointestinal – Section 8.6.1.1

Gender factors, and mental illness – Section 2.4.2

Generalized Anxiety Disorder – Section 7.1.1

 Comorbidity – Section 7.3.1

 Epidemiology of – Section 7.2.1

Treatment of – Section 7.5.1

Genetic explanations for mental illness – Section 2.2.2.1

Genital Stage – Section 2.3.1.2

Glial Cells – Section 2.2.1.3

Glutamate – Section 2.2.1.5

Grandiose delusion – Section 12.1.5

Greco-Roman Thought – Section 1.4.2

Guilty but mentally ill (GBMI) – Section 15.1.3

H

Habituation – Section 2.3.2.1

Hallucinations – Section 12.1.1.1

Hallucinogens/Cannabis/Combination – Section 11.1.2.5

Epidemiology of – 11.2.3

Headaches – Section 8.6.1.1

Heuristics – Section 1.3

Hippocampus – Section 2.2.1.6

History, of mental illness – Section 1.4

Histrionic personality disorder – Section 13.1.3.3

Treatment of – Section 13.5.2.3

HIV Infection – Section 14.3.9

Hoarding – Section 9.1.3

Epidemiology of – Section 9.2.3

Comorbidity of – Section 9.3.3

Treatment of – Section 9.5.3

Hormonal imbalances – Section 2.2.2.2

Huntington’s disease – Section 14.3.8

Hypomanic Episode – Section 4.2

Humanistic perspective – Section 2.3.4.1

Evaluation of – Section 2.3.4.3

Hypertension – Section 8.6.1.1

Hypnosis – Section 8.6.2.3

Hypothalamus – Section 2.2.1.6

I

ICD – Section 3.2.3

Ideas of reference – Section 13.1.2.3

Identification – Section 2.3.1.3

Illness anxiety disorder – Section 8.1.2

Incidence – Section 1.2

Insomnia – Section 8.6.1.3

Intellectual Developmental Disorder (Intellectual Disability) – Section 16.2.1

Intellectualization – Section 2.3.1.3

Intelligence tests – Section 3.1.3.7

Intensity, and emotion – Section 2.2.3

Intermittent explosive disorder – Section 16.3.4

Ions – Section 2.2.1.4

Irresistible impulse test – Section 15.1.3

J

Jealous delusion – Section 12.1.5

K

L

Latency Stage – Section 2.3.1.2

Latent content, of dreams – Section 2.3.1.4

Law of effect – Section 2.3.2.3

Learning – Section 2.3.2.1

Levels of personality (conscious, preconscious, unconscious) – Section 2.3.1.1

Libido – Section 2.3.1.1

M

Magnetic Resonance Imaging (MRI) – Section 3.1.3.4

Major Depressive Disorder – Section 4.1

Major Depressive Episode – Section 4.1

Major Neurocognitive Disorder – Section 14.1.2

Maladaptive cognitions – Section 2.3.3.4

Managed health care – Section 1.4.7.3

Manic Episode – Section 4.2

Manifest content, of dreams – Section 2.3.1.4

Medulla – Section 2.2.1.6

Mental disorders – Section 1.1.3

Mental health, professionals – Section 1.6.1

Mental hygiene movement – Section 1.4.5

Mental status examination – Section 3.1.3.2

Mesmerism – Section 1.4.6.2

Middle Ages, and views of mental illness – Section 1.4.3

Mild Neurocognitive Disorder – Section 14.1.3

M’Naghten rule – Section 15.1.3

Model – Section 2.1.2

Modeling – Section 2.3.2.4

Mood – Section 2.1

Mood disorders – Module 4

 Symptoms of – Module 4.1

 Types of – Module 4.1

Moral treatment movement – Section 1.4.5

Multicultural factors, and mental illness – Section 2.4.4

Multicultural psychology – Section 1.4.7.4

Multidimensional Models – Section 2.1.2

Multimethod study – Section 1.5.2.6

Myelin Sheath – Section 2.2.1.3

N

Narcissistic personality disorder – Section 13.1.3.4

 Treatment of – Section 13.5.2.4

Negative symptoms – Section 12.1.16

Nerves – Section 2.2.1.3

Nervous system, parts of – Section 2.2.1.2

 Communication in – Section 2.2.1.1

Neural Transmission – Section 2.2.1.4

Neurological tests – Section 3.1.3.4

Neurotransmitters – Section 2.2.1.4

Neuron – Section 2.2.1.3

NGRI – Section 15.1.3

Nomenclature – Section 1.2

Norepinephrine – Section 2.2.1.5

Norms (social) – Section 1.1.2

Nucleus – Section 2.2.1.3

O

Observational learning – Section 2.3.2.1; 2.3.2.4

OCD (Obsessive Compulsive Disorder) – Section 9.1.1

 Epidemiology of – Section 9.2.1

 Comorbidity of – Section 9.3.1

 Treatment of – Section 9.5.1

Obsessive-compulsive personality disorder (OCPD) – Section 13.1.4.2

Operant conditioning – Section 2.3.2.3

Opioids – Section 11.1.2.3

Oppositional defiant disorder – Section 16.3.2

Oral Stage – Section 2.3.1.2

P

Panic disorder – Section 7.1.5

 Comorbidity – Section 7.3.5

 Epidemiology of – Section 7.2.5

 Treatment of – Section 7.5.5

Paranoid personality disorder – Section 13.1.2.1

Parkinson's disease – Section 14.3.7

Parts of personality (id, ego, superego) – Section 2.3.1.1

Patient's rights – Section 15.2

Pavlov, Ivan Petrovich – Section 2.3.2.2

Perceptual set – Section 2.1

Persecutory delusion – Section 12.1.5

Persistent Depressive Disorder – Section 4.1

Personality

 Related to eating disorders – Section 10.5.4

Personality, development of – Section 2.3.1.2

Personality, structure of – Section 2.3.1.1

Personality disorders – Section 13.1.1

Personality inventories – Section 3.1.3.3

Psychological tests – Section 3.1.3.3

Phallic Stage – Section 2.3.1.2

Pharmacological – Section 14.4.1

Physical examination – Section 3.1.3.5

Pica – Section 16.1.3

Polarized – Section 2.2.1.4

Pons – Section 2.2.1.6

Positive psychology – Section 1.1.1

Positive symptoms – Section 12.1.16

Positron Emission Tomography (PET) – Section 3.1.3.4

Prevention science – Section 1.4.7.6

Prescription rights for psychologists – Section 1.4.7.5

Presenting problem – Section 1.2

Prevalence – Section 1.2

Primary reinforcers/punishers – Section 2.3.2.3

Privileged communication – Section 15.3

Professional journals – Section 1.6.2.2

Professional societies – Section 1.6.2.1

Prognosis – Section 1.2

Projection – Section 2.3.1.3

Projective tests – Section 3.1.3.3

Psychiatric drugs – Section 1.4.7.2

Psychoanalysis – Section 2.3.1.4

Psychodynamic theory – Section 2.3.1

Evaluation of – Section 2.3.1.5

Related to dissociative disorders – Section 6.4.4

Related to somatic disorders – Section 8.4.1

Related to personality disorders – Section 13.4.2.1

Psychological or psychogenic perspective – Section 1.4.6.2

Psychological debriefing – Section 5.6.1

Psychomotor symptoms – Section 12.1.1.4

Psychopathology – Section 1.1.3

Psychopharmacology – Section 2.2.3.1

 In relation to trauma and stressor related disorders – Section 5.6.5

 In relation to somatic disorders – Section 8.5.2

 In relation to schizophrenic disorders – Section 12.5.1

Psychophysiological Disorders – Section 8.6.1

Psychosis – Section 12.1.1

Psychosurgery – Section 2.2.3.3

Psychotherapy – Section 3.3.1.3

 Related to somatic disorders – Section 8.5.1

Psychotropic drugs – Section 2.2.3.1

PTSD – Section 5.2.1

 Epidemiology of – Section 5.3.1

 Comorbidity of – Section 5.4.1

Punishment – Section 2.3.2.3

Q

R

Rape – Section 5.1

Rationalization – Section 2.3.1.3

Reaction formation – Section 2.3.1.3

Reactive attachment disorder – Section 16.1.2

Reactivity – Section 3.1.3.1

Reappraisal – Section 2.1

Receptor sites – Section 2.2.1.4

Reform movement, and views of mental illness – Section 1.4.5

Regression – Section 2.3.1.3

Reinforcement – Section 2.3.2.3

Reinforcement schedules – Section 2.3.2.3

Relative refractory period – Section 2.2.1.4

Reliability – Section 3.1.2

Renaissance, and views of mental illness – Section 1.4.4

Repolarized – Section 2.2.1.4

Repression – Section 2.3.1.3

Research Method – Section 1.5.2

Resistance – Section 2.3.1.4

Respondent conditioning – Section 2.3.2.2

Respondent discrimination – Section 2.3.2.2

Respondent extinction – Section 2.3.2.2

Respondent generalization – Section 2.3.2.2
Resting potential – Section 2.2.1.4
Reticular formation – Section 2.2.1.6
Reuptake – Section 2.2.1.4
Reversal design (ABAB design) – Section 1.5.2.5
Rogers, C. – Section 2.3.4.1
Rumination disorder – Section 16.1.4

S

Schemas and cognitive errors – Section 2.3.3.2
Schizoaffective disorder – Section 12.1.4
Schizoid personality disorder – Section 13.1.2.2
Schizophrenia – Section 12.1.2
Schizophreniform disorder – Section 12.1.3
Schizotypal personality disorder – Section 13.1.2.3
Scientific method – Section 1.5.1
Secondary reinforcers/punishers – Section 2.3.2.3
Sedative-Hypnotic drugs – Section 11.1.2.2
Selective mutism – Section 16.4.1
Self-monitoring – Section 3.1.3.6
Self-serving bias – Section 2.3.3.3
Sensitization – Section 2.3.2.1
Separation anxiety disorder – Section 16.4.2
Serotonin – Section 2.2.1.5

Social anxiety disorder – Section 7.1.4

Comorbidity – Section 7.3.4

Epidemiology of – Section 7.2.4

Treatment of – Section 7.5.4

Social cognition – Section 1.3; 2.3.3.2

Social identity theory – Section 1.3

Social (pragmatic) communication disorder – Section 16.2.4

Sociocultural Model – Section 2.4

Evaluation of – Section 2.4.5

Related to mood disorders – Section 4.5.4

Related to trauma and stressor related disorders – Section 5.5.4

Related to dissociative disorders – Section 6.4.3

Related to anxiety disorders – Section 7.4.3

Related to somatic disorders – Section 8.4.4

Related to eating disorders – Section 10.5.3

Related to substance use and addictive disorders – Section 11.4.4; 11.5.4

Related to schizophrenic disorders – Section 12.4.3

Related to personality disorders – Section 13.4.3

Socioeconomic factors, and mental illness – Section 2.4.1

Soma – Section 2.2.1.3

Somatic delusion – Section 12.1.5

Somatic symptom and related disorders – Section 8.1

Somatic symptom disorder – Section 8.1.1

Specific learning disorder – Section 16.2.2

Specific Phobia – Section 7.1.1

- Comorbidity – Section 7.3.2
- Epidemiology of – Section 7.2.2
- Treatment of – Section 7.5.2

Spontaneous recovery – Section 2.3.2.2

Stages of personality development – Section 2.3.1.2

Standardization – Section 3.1.2

Stereotypic movement disorder – Section 16.2.5

Stigma – Section 1.3

Stimulants – Section 11.1.2.4

- Epidemiology of – 11.2.2

Stressors – Section 5.1

Sublimation – Section 2.3.1.3

Substances – Section 11.1.1

- Types of – Section 11.1.2

Substantia nigra – Section 2.2.1.6

Suicidality – Section 4.3.3

Synapse – Section 2.2.1.4

Synaptic gap/cleft/space – Section 2.2.1.4

Syndrome – Section 3.2.1

T

Target behavior – Section 3.1.3.6

Thalamus – Section 2.2.1.6

Thanatos – Section 2.3.1.1

Thematic Apperception Test – Section 3.1.3.3

Thorndike – Section 2.3.2.3

Tic disorders – Section 16.2.6

Transference – Section 2.3.1.4

Trauma-focused Cognitive behavioral therapy – Section 5.6.3

Threshold of excitation – Section 2.2.1.4

Tolerance – Section 11.1.1

Traumatic Brain Injury (TBI) – Section 14.3.2

Treatment – Section 1.2; Section 3.3.1

 Related to mood disorders – Section 4.6

 Related to trauma and stressor related disorders – Section 5.6

 Related to dissociative disorders – Section 6.5

 Related to anxiety disorders – Section 7.5

 Related to somatic disorders – Section 8.5

 Related to Psychological Factors Affecting Other Medical Conditions – Section 8.6.2

 Related to obsessive compulsive disorders – Section 9.5

 Related to eating disorders – Section 10.6

 Related to substance-related and addictive disorders – Section 11.5

 Related to schizophrenic disorders – Section 12.5

 Related to personality disorders – Section 13.5

Related to neurocognitive disorders – Section 14.4

Trial and error learning – Section 2.3.2.3

Trichotillomania – Section 16.4.3

Trephination – Section 1.4.1

U

Uni-Dimensional Model – Section 2.1.1

V

Validity – Section 3.1.2

Cross-sectional – Section 3.1.3.1

Vascular disorders – Section 14.3.3

Viral infections – Section 2.2.2.1

W

Watson, J.B. – Section 1.3.2.1; Section 2.1

Withdrawal – Section 1.1.1.1

X

Y

Z