PSYCHOPATHOLOGY

Foundations for a Contemporary Understanding

FOURTH EDITION

Edited by

James E. Maddux and Barbara A. Winstead

ROUTLEDGE

PSYCHOPATHOLOGY

The fourth edition of *Psychopathology* is the most up-to-date text about the etiology and treatment of the most important psychological disorders. Intended for first-year graduate students in clinical psychology, counseling psychology, and related programs, this new edition, revised to be consistent with the DSM-5, continues to focus on research and empirically-supported information while also challenging students to think critically. The first part of the book covers the key issues, ideas, and concepts in psychopathology, providing students with a set of conceptual tools that will help them read more thoroughly and critically the second half of the book, which focuses on specific disorders. Each chapter in the second and third sections provides a definition, description, and brief history of the disorder it discusses, and outlines theory and research on etiology and empirically-supported treatments. This edition also features a companion website hosting lecture slides, a testbank, an instructor's manual, case studies and exercises, and more.

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Preface

We are pleased to offer the fourth edition of *Psychopathology: Foundations for a Contemporary Understanding*. This book was created—and revised—with students in mind. The length, organization, and level and style of writing reflect this intention. We had—and still have—two major goals in mind.

1. Providing up-to-date information about theory and research on the etiology and treatment of the most important psychological disorders. Toward this end, we chose well-known researchers who would not only be aware of the cutting edge research on their topics, but who were also contributing to it. This goal also demands frequent updating of information to reflect, as much as possible, the latest developments in the field.
2. Challenging students to think critically about psychopathology. We tried to accomplish this goal in two ways. First, we encouraged chapter authors to challenge traditional assumptions and theories concerning the topics about which they were writing. Second, and more important, we have included chapters that discuss, in depth, crucial and controversial issues facing the field of psychopathology, such as the definition of psychopathology, the influence of cultural and gender, the role of developmental processes, the validity of psychological testing, and the viability and utility of traditional psychiatric diagnosis. The first eight chapters in this book are devoted to such issues because we believe that a sophisticated understanding of psychopathology consists of much more than memorizing a list of disorders and their symptoms or memorizing the findings of numerous studies. It consists primarily of understanding ideas and concepts and understanding how to use those ideas and concepts to make sense of the research on specific disorders and the information found in formal diagnostic manuals.

Part I offers in depth discussions of a number of important ideas, concepts, and theories, which provide perspective on specific psychological disorders. The major reason for placing these general chapters in the first section before the disorders chapters is to give students a set of conceptual tools that will help them read more thoughtfully and critically the material on specific disorders.

Parts II and III deal with specific disorders of adulthood, childhood, and adolescence. We asked contributors to follow, as much as possible, a common format consisting of:

1. A definition and description of the disorder or disorders.
2. A brief history of the study of the disorder.
3. Theory and research on etiology.
4. Research on empirically supported interventions.

Editors must always make choices regarding what should be included in a textbook and what should not. A textbook that devoted a chapter to each and every disorder described in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) would be unwieldy and impossible to cover in a single semester. Our choices regarding what to include and what to exclude were guided primarily by our experiences regarding the kinds of psychological problems that clinical students typically encounter in their training and in their subsequent clinical careers. We also wanted to be generally consistent with the changes in nomenclature that appear in the fifth edition (DSM-5). For this reason, we have added new chapters on posttraumatic stress disorder, autism spectrum disorder, and dissociative disorders, and have separated the previous chapter on sexual dysfunctions and gender identity disorders into two chapters that provide expanded coverage of both topics. In addition, all of the chapters reflect—and many critique—the changes that were made in diagnostic criteria in the DSM-5. Finally, we have a new set of authors for the chapter on the biological bases of psychopathology.

For the first time, this book will be accompanied by a website that features links to additional resources and videos (including interviews with patients), and chapter summaries and (for instructors) PowerPoint lecture slides, a test bank, and an instructor's manual.

We continue to hope that instructors and students will find this approach to understanding psychopathology challenging and useful. We continue to learn much from our contributors in the process of editing their chapters, and we hope that students will learn as much as we have from reading what these outstanding contributors have produced.

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Part I

Thinking About Psychopathology

1

Conceptions of Psychopathology

A Social Constructionist Perspective

James E. Maddux, Jennifer T. Gosselin, and Barbara A. Winstead

A textbook about a topic should begin with a clear definition of the topic. Unfortunately, for a textbook on psychopathology, this is a difficult if not impossible task. The definitions or conceptions of *psychopathology*, and such related terms as *mental disorder*, have been the subject of heated debate throughout the history of psychology and psychiatry, and the debate is not over (e.g., Gorenstein, 1984; Horwitz, 2002; Widiger, Chapter 6 in this volume). Despite its many variations, this debate has centered on a single overriding question: Are *psychopathology* and related terms such as *mental disorder* and *mental illness* scientific terms that can be defined objectively and by scientific criteria, or are they *social constructions* (Gergen, 1985) that are defined largely or entirely by societal and cultural values? Addressing these perspectives in this opening chapter is important because the reader's view of everything in the rest of this book will be influenced by his or her view on this issue.

This chapter deals with *conceptions* of psychopathology. A conception of psychopathology is not a *theory* of psychopathology (Wakefield, 1992a). A conception of psychopathology attempts to define the term—to delineate which human experiences are considered psychopathological and which are not. A conception of psychopathology does not try to *explain* the psychological phenomena that are considered pathological, but instead tells us which psychological phenomena are considered pathological and thus need to be explained. A *theory* of psychopathology, however, is an attempt to explain those psychological phenomena and experiences that have been identified by the conception as pathological. Theories and explanations for what is currently considered to be psychopathological human experience can be found in a number of other chapters, including all of those in Part II.

Understanding various conceptions of psychopathology is important for a number of reasons. As explained by medical philosopher Lawrie Reznek (1987), "Concepts carry consequences—classifying things one way rather than another has important implications for the way we behave towards such things" (p. 1). In speaking of the importance of the conception of *disease*, Reznek wrote:

> The classification of a condition as a disease carries many important consequences. We inform medical scientists that they should try to discover a cure for the condition. We inform benefactors that they should support such research. We direct medical care towards the condition, making it appropriate to treat the condition by medical means such as drug therapy, surgery, and so on. We inform our courts that it is inappropriate to hold people responsible for the manifestations of the condition. We set up early warning detection services aimed at detecting the condition in its early stages when it is still amenable to successful treatment. We serve notice to health insurance companies and national health services that they are liable to pay for the treatment of such a condition. Classifying a condition as a disease is no idle matter.
>
> (Reznek, 1987, p. 1)

If we substitute *psychopathology* or *mental disorder* for the word *disease* in this paragraph, its message still holds true. How we conceive of psychopathology and related terms has wide-ranging implications for individuals, medical and mental health professionals, government agencies and programs, legal proceedings, and society at large.

Conceptions of Psychopathology

A variety of conceptions of psychopathology have been offered over the years. Each has its merits and its deficiencies, but none suffices as a truly scientific definition.

Psychopathology as Statistical Deviance A commonly used and "common sense" conception of psychopathology

is that pathological psychological phenomena are those that are *abnormal*—statistically deviant or infrequent. Abnormal literally means "away from the norm." The word "norm" refers to what is typical or average. Thus, this conception views psychopathology as deviation from statistical psychological normality.

One of the merits of this conception is its commonsense appeal. It makes sense to most people to use words such as *psychopathology* and *mental disorder* to refer only to behaviors or experiences that are infrequent (e.g., paranoid delusions, hearing voices) and not to those that are relatively common (e.g., shyness, a stressful day at work, grief following the death of a loved one).

A second merit to this conception is that it lends itself to accepted methods of measurement that give it at least a semblance of scientific respectability. The first step in employing this conception scientifically is to determine what is statistically normal (typical, average). The second step is to determine how far a particular psychological phenomenon or condition deviates from statistical normality. This is often done by developing an instrument or measure that attempts to quantify the phenomenon and then assigns numbers or scores to people's experiences or manifestations of the phenomenon. Once the measure is developed, *norms* are typically established so that an individual's score can be compared to the mean or average score of some group of people. Scores that are sufficiently far from average are considered to be indicative of "abnormal" or "pathological" psychological phenomena. This process describes most tests of intelligence and cognitive ability and many commonly used measures of personality and emotion (e.g., the Minnesota Multiphasic Personality Inventory).

Despite its commonsense appeal and its scientific merits, this conception presents problems. Perhaps the most obvious issue is that we generally consider only one "side" of the deviation to be problematic (see "Psychopathology as maladaptive," below). In other words, Intellectual Disability is pathological, intellectual genius is not. Major Depressive Disorder is pathological, unconstrained optimism is not. Another concern is that, despite its reliance on scientific and well-established psychometric methods for developing measures of psychological phenomena and developing norms, this approach still leaves room for subjectivity.

The first point at which subjectivity comes into play is in the *conceptual definition* of the construct for which a measure is developed. A measure of any psychological construct, such as intelligence, must begin with a conceptual definition. We have to ask ourselves "What is 'intelligence'?" Of course, different people (including different psychologists) will come up with different answers to this question. How then can we scientifically and objectively determine which definition or conception is "true" or "correct"? The answer is that we cannot. Although we have tried and true methods for developing a reliable and valid (i.e., it consistently predicts what we want to predict) measure of a psychological construct once we have agreed on its conception or definition, we cannot use these same methods to determine which conception or definition is true or correct. The bottom line is that there is not a "true" definition of intelligence and no objective, scientific way of determining one. Intelligence is not a thing that exists inside of people and makes them behave in certain ways and that awaits our discovery of its "true" nature. Instead, it is an abstract idea that is defined by people as they use the words "intelligence" and "intelligent" to describe certain kinds of human behavior and the covert mental processes that supposedly precede or are at least concurrent with the behavior.

We can usually observe and describe patterns in the way most people use the words *intelligence* and *intelligent* to describe the behavior of themselves and others. The descriptions of the patterns then comprise the definitions of the words. If we examine the patterns of the use of *intelligence* and *intelligent*, we find that, at the most basic level, they describe a variety of specific behaviors and abilities that society values and thus encourages; unintelligent behavior includes a variety of behaviors that society does not value and thus discourages. The fact that the definition of *intelligence* is grounded in societal values explains the recent expansion of the concept to include good interpersonal skills (e.g., social and emotional intelligence), self-regulatory skills, artistic and musical abilities, creativity, and other abilities not measured by traditional tests of intelligence. The meaning of *intelligence* has broadened because society has come to place increasing value on these other attributes and abilities, and this change in societal values has been the result of a dialogue or discourse among the people in society, both professionals and laypersons. One measure of intelligence may prove more reliable than another and more useful than another measure in predicting what we want to predict (e.g., academic achievement, income), but what we want to predict reflects what we value, and values are not derived scientifically.

Another point for the influence of subjectivity is in the determination of *how deviant* a psychological phenomenon must be from the norm to be considered abnormal or pathological. We can use objective, scientific methods to construct a measure such as an intelligence test and develop norms for the measure, but we are still left with the question of how far from normal an individual's score must be to be considered abnormal. This question cannot be answered by the science of psychometrics because the distance from the average that a person's score must be to be considered "abnormal" is a matter of debate, not a matter of fact. It is true that we often answer this question by relying on statistical conventions such as using one or two standard deviations from the average score as the line of division between normal and abnormal. Yet the decision to use that convention is itself subjective because a convention (from the Latin *convenire*, meaning "to come together"), is an agreement or contract made by people,

not a truth or fact about the world. Why should one standard deviation from the norm designate "abnormality"? Why not two standard deviations? Why not half a standard deviation? Why not use percentages? The lines between normal and abnormal can be drawn at many different points using many different strategies. Each line of demarcation may be more or less useful for certain purposes, such as determining the criteria for eligibility for limited services and resources. Where the line is set also determines the prevalence of "abnormality" or "mental disorder" among the general population (Kutchens & Kirk, 1997; Francis, 2013), so it has great practical significance. But no such line is more or less "true" than the others, even when those other are based on statistical conventions.

We cannot use the procedures and methods of science to draw a definitive line of demarcation between normal and abnormal psychological functioning, just as we cannot use them to draw definitive lines of demarcation between "short" and "tall" people or "hot" and "cold" on a thermometer. No such lines exist in nature awaiting our discovery.

Psychopathology as Maladaptive (Dysfunctional) Behavior Most of us think of psychopathology as behaviors and experiences that are not just statistically abnormal but also maladaptive (dysfunctional). Normal and abnormal are statistical terms, but *adaptive* and *maladaptive* refer not to statistical norms and deviations but to the effectiveness or ineffectiveness of a person's behavior. If a behavior "works" for the person—if the behavior helps the person deal with challenges, cope with stress, and accomplish his or her goals—then we say the behavior is more or less effective and adaptive. If the behavior does not "work" for the person in these ways, or if the behavior makes the problem or situation worse, we say it is more or less ineffective and maladaptive. The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) incorporates this notion in its definition of mental disorder by stating that mental disorders "are usually associated with significant distress or disability in social, occupational, or other important activities" (American Psychiatric Association, 2013, p. 20).

Like the statistical deviance conception, this conception has common sense appeal and is consistent with the way most laypersons use words such as *pathology*, *disorder*, and *illness*. As we noted above, most people would find it odd to use these words to describe statistically infrequent high levels of intelligence, happiness, or psychological wellbeing. To say that someone is "pathologically intelligent" or "pathologically well-adjusted" seems contradictory because it flies in the face of the commonsense use of these words.

The major problem with the conception of psychopathology as maladaptive behavior is its inherent subjectivity. Like the distinction between normal and abnormal, the distinction between adaptive and maladaptive is fuzzy and arbitrary. We have no objective, scientific way of making a clear distinction. Very few human behaviors are in and of themselves either adaptive or maladaptive; instead, their adaptiveness and maladaptiveness depend on the situations in which they are enacted and on the judgment and values of the actor and the observers. Even behaviors that are statistically rare and therefore abnormal will be more or less adaptive under different conditions and more or less adaptive in the opinion of different observers and relative to different cultural norms. The extent to which a behavior or behavior pattern is viewed as more or less adaptive or maladaptive depends on a number of factors, such as the goals the person is trying to accomplish and the social norms and expectations in a given situation. What works in one situation might not work in another. What appears adaptive to one person might not appear so to another. What is usually adaptive in one culture might not be so in another (see López & Guarnaccia, Chapter 4 in this volume). Even so-called "normal" personality involves a good deal of occasionally maladaptive behavior, which you can find evidence for in your own life and the lives of friends and relatives. In addition, people given official "personality disorder" diagnoses by clinical psychologists and psychiatrists often can manage their lives effectively and do not always behave in maladaptive ways.

Another problem with the "psychopathological = maladaptive" conception is that judgments of adaptiveness and maladaptiveness are logically unrelated to measures of statistical deviation. Of course, often we do find a strong relationship between the statistical abnormality of a behavior and its maladaptiveness. Many of the problems described in the DSM-5 and in this textbook are both maladaptive and statistically rare. There are, however, major exceptions to this relationship.

First, not all psychological phenomena that deviate from the norm or the average are maladaptive. In fact, sometimes deviation from the norm is adaptive and healthy. For example, IQ scores of 130 and 70 are equally deviant from norm, but abnormally high intelligence is more much adaptive than abnormally low intelligence. Likewise, people who consistently score abnormally low on measures of anxiety and depression are probably happier and better adjusted than people who consistently score equally abnormally high on such measures.

Second, not all maladaptive psychological phenomena are statistically infrequent and vice versa. For example, shyness is almost always maladaptive to some extent because it often interferes with a person's ability to accomplish what he or she wants to accomplish in life and relationships, but shyness is very common and therefore is statistically frequent. The same is true of many of the problems with sexual functioning that are included in the DSM as "mental disorders"—they are almost always maladaptive to some extent because they create distress and problems in relationships, but they are relatively common (see Gosselin, Chapter 13 in this volume).

Psychopathology as Distress and Disability Some conceptions of psychopathology invoke the notions of

subjective distress and *disability*. Subjective distress refers to unpleasant and unwanted feelings, such as anxiety, sadness, and anger. Disability refers to a restriction in ability (Ossorio, 1985). People who seek mental health treatment usually are not getting what they want out of life, and many feel that they are unable to do what they need to do to accomplish their valued goals. They may feel inhibited or restricted by their situation, their fears or emotional turmoil, or by physical or other limitations. Individuals may lack the necessary self-efficacy beliefs (beliefs about personal abilities), physiological or biological components, self-regulatory skills, and/or situational opportunities to make positive changes (Bergner, 1997).

As noted previously, the DSM incorporates the notions of distress and disability into its definition of mental disorder. In fact, subjective distress and disability are simply two different but related ways of thinking about adaptiveness and maladaptiveness rather than alternative conceptions of psychopathology. Although the notions of subjective distress and disability may help to refine our notion of maladaptiveness, they do nothing to resolve the subjectivity problem. Different people will define personal distress and personal disability in vastly different ways, as will different mental health professionals and different cultures. Likewise, people differ in their thresholds for how much distress or disability they can tolerate before seeking professional help. Thus, we are still left with the problem of how to determine normal and abnormal levels of distress and disability. As noted previously, the question "How much is too much?" cannot be answered using the objective methods of science.

Another problem is that some conditions or patterns of behavior (e.g., pedophilic disorder, antisocial personality disorder) that are considered psychopathological (at least officially, according to the *DSM*) are *not* characterized by subjective distress, other than the temporary distress that might result from social condemnation or conflicts with the law.

Psychopathology as Social Deviance Psychopathology has also been conceived as behavior that deviates from social or cultural norms. This conception is simply a variation of the conception of psychopathology as statistical abnormality, only in this case judgments about deviations from normality are made informally by people using social and cultural rules and conventions rather than formally by psychological tests or measures.

This conception also is consistent to some extent with common sense and common parlance. We tend to view psychopathological or mentally disordered people as thinking, feeling, and doing things that most other people do not do (or do not want to do) and that are inconsistent with socially accepted and culturally sanctioned ways of thinking, feeling, and behaving. Several examples can be found in DSM-5's category of paraphilic disorders.

The problem with this conception, as with the others, is its subjectivity. Norms for socially normal or acceptable behavior are not derived scientifically but instead are based on the values, beliefs, and historical practices of the culture, which determine who is accepted or rejected by a society or culture. Cultural values develop not through the implementation of scientific methods, but through numerous informal conversations and negotiations among the people and institutions of that culture. Social norms differ from one culture to another, and therefore what is psychologically abnormal in one culture may not be so in another (see López & Guarnaccia, Chapter 4 in this volume). Also, norms of a given culture change over time; therefore, conceptions of psychopathology will change over time, often very dramatically, as evidenced by American society's changes over the past several decades in attitudes toward sex, race, and gender. For example, psychiatrists in the 1800s classified masturbation, especially in children and women, as a disease, and it was treated in some cases by clitoridectomy (removal of the clitoris), which Western society today would consider barbaric (Reznek, 1987). Homosexuality was an official mental disorder in the DSM until 1973 (see also Gosselin, Chapter 13 in this volume).

In addition, the conception of psychopathology as social norm violations is at times in conflict with the conception of psychopathology as maladaptive behavior. Sometimes violating social norms is healthy and adaptive for the individual and beneficial to society. In the 19th century, women and African-Americans in the U.S. who sought the right to vote were trying to change well-established social norms. Their actions were uncommon and therefore "abnormal," but these people were far from psychologically unhealthy, at least not by today's standards. Earlier in the 19th century, slaves who desired to escape from their owners were said to have "drapetomania." Although still practiced in some parts of the world, slavery is almost universally viewed as socially deviant and pathological, and the desire to escape enslavement is considered to be as normal and healthy as the desire to live and breathe.

Psychopathology as "Dyscontrol" or "Dysregulation" Some have argued that we should only consider as psychopathologies or mental disorders those maladaptive patterns of behaving, thinking, and feeling that are not within the person's ability to effectively control or self-regulate (Kirmayer & Young, 1999; Widiger & Sankis, 2000) . The basic notion here is that, if a person voluntarily behaves in maladaptive or self-destructive ways, then that person's behavior should not be viewed as in indication of or result of a mental disorder. Indeed, as does the notion of a physical or medical disorder, the term mental disorder seems to incorporate the notion that what is happening to the person is not within the person's control. The basic problem with this conception is that its draws an artificial line between "within control" (voluntary) and "out of control" (involuntary) that simply cannot be drawn. There are some behaviors that person might engage in that most of us would

agree are completely voluntary, deliberate, and intentional and some other behaviors that a person might engage in that most of us would agree are completely involuntary, non-deliberate, and unintentional. Such behaviors, however, are probably few and far between. The causes of human behavior are complex, to say the least, and environmental events can have such a powerful influence on any behavior that saying that anything that people do is completely or even mostly voluntary and intentional may be a stretch. In fact, considerable research suggests that most behaviors, most of the time, are automatic and therefore involuntary (Weinberger, Siefier, & Haggerty, 2010). Determining the degree to which a behavior is voluntary and within a person's control or involuntary and beyond a person's control is difficult, if not impossible. We also are left, once again, with the question of who gets to make this determination. The actor? The observer? The patient? The mental health professional?

Psychopathology as Harmful Dysfunction Wakefield's (1992a, 1992b, 1993, 1997, 1999, 2006) harmful dysfunction (HD) conception, presumably grounded in evolutionary psychology (e.g., Cosmides, Tooby, & Barkow, 1992), acknowledges that the conception of mental disorder is influenced strongly by social and cultural values. It also proposes, however, a supposedly scientific, factual, and objective core that is not dependent on social and cultural values (Wakefield, 2006). In Wakefield's words:

> a [mental] disorder is a harmful dysfunction wherein *harmful* is a value term based on social norms, and *dysfunction* is a scientific term referring to the failure of a mental mechanism to perform a natural function for which it was designed by evolution . . . a disorder exists when the failure of a person's internal mechanisms to perform their function as designed by nature impinges harmfully on the person's well-being as defined by social values and meanings.
>
> (Wakefield, 1992a, p. 373)

One of the merits of this conception is that it acknowledges that the conception of mental disorders must include a reference to social norms; however, this conception also tries to anchor the concept of mental disorder in a scientific theory—the theory of evolution.

Wakefield (2006) has reiterated this definition in writing that a mental disorder "satisfies two requirements: (1) it is negative or harmful according to cultural values; and (2) it is caused by a dysfunction (i.e., by a failure of some psychological mechanism to perform a natural function for which it was evolutionarily designed)" (p. 157). He and his colleagues also write, "Problematic mismatches between designed human nature and current social desirability are not disorders . . . [such as] adulterous longings, taste for fat and sugar, and male aggressiveness" (Wakefield, Horwitz, & Schmitz, 2006, p. 317).

However, the claim that identifying a failure of a "designed function" is a scientific judgment and not a value judgment is open to question. Wakefield's claim that dysfunction can be defined in "purely factual scientific" (Wakefield, 1992a, p. 383, 2010) terms rests on the assumption that the "designed functions" of human "mental mechanisms" have an objective and observable reality and, thus, that failure of the mechanism to execute its designed function can be objectively assessed. A basic problem with this notion is that although the physical inner workings of the body and brain can be observed and measured, "mental mechanisms" have no objective reality and thus cannot be observed directly—no more so than the "unconscious" forces that provide the foundation for Freudian psychoanalytic theory.

Evolutionary theory provides a basis for explaining human behavior in terms of its contribution to reproductive fitness. A behavior is considered more functional if it increases the survival of those who share your genes in the next generation and the next and less functional if it does not. Evolutionary psychology cannot, however, provide a catalogue of "mental mechanisms" and their natural functions. Wakefield states that "discovering what in fact is natural or dysfunctional may be extraordinarily difficult" (1992b, p. 236). The problem with this statement is that, when applied to human behavior, "natural" and "dysfunctional" are not properties that can be "discovered;" they are value judgments. The judgment that a behavior represents a dysfunction relies on the observation that the behavior is excessive and/or inappropriate under certain conditions. Arguing that these behaviors represent failures of evolutionarily designed "mental mechanisms" (itself an untestable hypothesis because of the occult nature of "mental mechanisms") does not absolve us of the need to make value judgments about what is excessive, inappropriate, or harmful and under what circumstances (Leising, Rogers, & Ostner, 2009). These are value judgments based on social norms, not scientific "facts," an issue that we will explore in greater detail later in this chapter (see also Widiger, Chapter 6 in this volume).

Another problem with the HD conception is that it is a moving target. For example, Wakefield modified his original HD conception by saying that it is concerned not with what a mental disorder *is* but only with what most scientists *think* it is. For example, he states that "My comments were intended to argue, not that PTSD [posttraumatic stress disorder] is a disorder, but that the HD analysis is capable of explaining why the symptom picture in PTSD is *commonly judged* to be a disorder" (1999, p. 390, emphasis added). Wakefield's original goal was to "define mental disorders *prescriptively*" (Sadler, 1999, p. 433, emphasis added) and to "help us decide whether someone is mentally disordered or not" (Sadler, 1999, p. 434). His more recent view, however, "avoids making any prescriptive claims, instead focusing on explaining the conventional clinical use of the disorder concept" (Sadler, 1999, p. 433). Wakefield "has abandoned his original task to be prescriptive and has now settled for being *descriptive*

only, for example, telling us why a disorder is judged to be one" (Sadler, 1999, p. 434, emphasis added).

Describing how people have agreed to define a concept is not the same as defining the concept in scientific terms, even if those people are scientists. Thus, Wakefield's HD conception simply offers a criterion that people (clinicians, scientists, and laypersons) might use to judge whether or not something is a "mental disorder." But consensus of opinion, even among scientists, is not scientific evidence. Therefore, no matter how accurately this criterion might describe how some or most people define "mental disorder," it no more or no less scientific than other conceptions that also are based on how some people agree to define "mental disorder." It is no more scientific than the conceptions involving statistical infrequency, maladaptiveness, or social norm violations (see also Widiger, Chapter 6 in this volume).

The DSM and International Classification of Diseases Definitions of Mental Disorder Any discussion of conceptions of psychopathology has to include a discussion of the most influential conception of all—that of the DSM. First published in 1952 and revised and expanded five times since, the DSM provides the organizational structure for virtually every textbook (including this one) on abnormal psychology and psychopathology, as well as almost every professional book on the assessment and treatment of psychological problems. (See Widiger, Chapter 6 in this volume, for a more detailed history of psychiatric classification, the DSM, and the International Classification of Diseases).

Just as a textbook on psychopathology should begin by defining its key term, so should a taxonomy of mental disorders. The difficulties inherent in attempting to define psychopathology and related terms are clearly illustrated by the definition of "mental disorder" found in the latest edition of the DSM, the DSM-5:

> A mental disorder is a syndrome characterized by 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. Mental disorders are usually associated with significant distress or disability in social, occupational, or other important activities. An expectable or culturally approved response to a common stressor or loss, such as the death of a loved one, is not a mental disorder. Socially deviant behavior (e.g., political, religious, or sexual) and conflicts that are primarily between the individual and society are not mental disorders unless the deviance or conflict results from a dysfunction in the individual, as described above.
>
> (American Psychiatric Association, 2013, p. 20)

All of the conceptions of psychopathology described previously can be found to some extent in this definition—statistical deviation (i.e., not "expectable"); maladaptiveness, including distress and disability; social norms violations; and some elements of the harmful dysfunction conception ("a dysfunction in the individual") although without the flavor of evolutionary theory. For this reason, it is a comprehensive, inclusive, and sophisticated conception and probably as good, if not better, than any proposed so far.

Nonetheless, it falls prey to the same problems with subjectivity as other conceptions. For example, what is the meaning of "clinically significant" and how should "clinical significance" be measured? Does clinical significance refer to statistical infrequency, maladaptiveness, or both? How much distress must a person experience or how much disability must a person exhibit before he/she is said to have a mental disorder? Who gets to judge the person's degree of distress or disability? How do we determine whether or not a particular response to an event is "expectable" or "culturally approved"? Who gets to determine this? How does one determine whether or not socially deviant behavior or conflicts "are primarily between the individual and society"? What exactly does this mean? What does it mean for a dysfunction to exist or occur "in the individual"? Certainly a biological dysfunction might be said to be literally "in the individual," but does it make sense to say the same of psychological and behavioral dysfunctions? Is it possible to say that a psychological or behavioral dysfunction can occur "in the individual" apart from the social, cultural, and interpersonal milieu in which the person is acting and being judged? Clearly, the DSM's conception of mental disorder raises as many questions as do the conceptions it was meant to supplant.

The World Health Organization's 10th edition of the *International Classification of Diseases and Related Health Problems* (ICD-10; World Health Organization, 1992) includes a *Classification of Mental and Behavioural Disorders* that is highly similar in format and content to the DSM-5. In fact, the two systems have evolved in tandem over the past several decades. In the ICD-10, the term "disorder" is used

> to imply the existence of a clinically recognizable set of symptoms or behavior associated in most cases with distress and with interference with personal functioning. Social deviance or conflict alone, without personal dysfunction, should not be included in mental disorder as defined here.
>
> (World Health Organization, 1992, p. 5)

Although less wordy than the DSM definition, the ICD definition contains the same basic ideas and the same interpretive problems. What is missing is the statement that a mental disorder exists "in an individual" although the term "personal dysfunction" can be interpreted as meaning the same thing.

Categories Versus Dimensions

The difficulty inherent in the DSM conception of psychopathology and other attempts to distinguish between normal

and abnormal or adaptive and maladaptive is that they are *categorical models* that attempt to describe guidelines for distinguishing between individuals who are normal or abnormal and for determining which specific abnormality or "disorder" a person has, to the exclusion of other disorders. In other words, people either "have" a given disorder or they do not. An alternative model, overwhelmingly supported by research, is the *dimensional model*. In the dimensional model, normality and abnormality, as well as effective and ineffective psychological functioning, lie along a continuum; so-called psychological disorders are simply extreme variants of normal psychological phenomena and ordinary problems in living (Keyes & López, 2002; Widiger, Chapter 6 in this volume). The dimensional model is concerned not with classifying people or disorders but with identifying and measuring individual differences in psychological phenomena, such as emotion, mood, intelligence, and personal styles (e.g., Lubinski, 2000; Williams, Chapter 9 in this volume.). Great differences among individuals on the dimensions of interest are expected, such as the differences we find on standardized tests of intelligence. As with intelligence, divisions between normality and abnormality may be demarcated for convenience or efficiency, but are not to be viewed as indicative of true discontinuity among "types" of phenomena or "types" of people. Also, statistical deviation is not viewed as necessarily pathological, although extreme variants on either end of a dimension (e.g., introversion-extraversion, neuroticism, intelligence) may be maladaptive if they lead to inflexibility in functioning.

This notion is not new. As early as 1860, Henry Maudsley commented that "there is no boundary line between sanity and insanity; and the slightly exaggerated feeling which renders a man 'peculiar' in the world differs only in degree from that which places hundreds in asylums" (as cited in Millon & Simonsen, 2010, p. 33).

Empirical evidence for the validity of a dimensional approach to psychological adjustment is strongest in the area of personality and personality disorders (Crego & Widiger, Chapter 12 in this volume; Skodol, 2010; Widiger & Trull, 2007). Factor analytic studies of personality problems among the general population and clinical populations with "personality disorders" demonstrate striking similarity between the two groups. In addition, these factor structures are not consistent with the DSM's system of classifying disorders of personality into categories and support a dimensional view rather than a categorical view. For example, most evidence strongly suggests that psychopathic personality (or antisocial personality), and other externalizing disorders of adulthood display a dimensional structure, not a categorical structure (Edens, Marcus, Lilienfeld, & Poythress, 2006; Krueger, Markon, Patrick, & Iacono, 2005; Larsson, Andershed, & Lichtenstein, 2006). The same is true of narcissism and narcissistic personality disorder (Brown, Budzek, & Tamorski, 2009). In addition, the recent emotional cascade model of borderline personality disorder, which highlights the interaction of emotional and behavioral dysregulation, although not presented explicitly as a dimensional model, is in almost every respect consistent with a dimension model (Selby & Joiner, 2009). The dimensional view of personality disorders is also supported by cross-cultural research (Alarcon et al., 1998).

Research on other problems supports the dimensional view. Studies of the varieties of normal emotional experiences (e.g., Carver, 2001; Oatley & Jenkins, 1992; Oatley, Keltner, & Jenkins, 2006) indicates that "clinical" emotional disorders are not discrete classes of emotional experience that are discontinuous from everyday emotional upsets and problems. Research on adult attachment patterns in relationships strongly suggests that dimensions are more accurate descriptions of such patterns than are categories (Fraley & Waller, 1998; Fossati, 2003; Hankin, Kassel, Abela, 2005). Research on self-defeating behaviors has shown that they are extremely common and are not by themselves signs of abnormality or symptoms of "disorders" (Baumeister & Scher, 1988). Research on children's reading problems indicates that "dyslexia" is not an all-or-none condition that children either have or do not have, but occurs in degrees without a natural break between "dyslexic" and "nondyslexic" children (Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992; Shaywitz, Morris, & Shaywitz, 2008; Snowling, 2006). Research indicates that attention-deficit/hyperactivity (Barkley, 2005), post-traumatic stress disorder (Rosen & Lilienfeld, 2008; Ruscio, Ruscio, & Keane, 2002), anxiety disorders (Eaton, Kessler, Wittchen, & Magee, 1994; Williams, Chapter 9 in this volume), depression (Costello, 1993a), somatoform disorders (Zvolensky, Eifert, & Garey, Chapter 15 in this volume), sexual dysfunctions and disorders (Gosselin, Chapter 13 in this volume) demonstrate this same dimensionality. Research on depression and schizophrenia indicates that these "disorders" are best viewed as loosely related clusters of dimensions of individual differences, not as disease-like syndromes (Claridge, 1995; Costello, 1993a, 1993b; Eisenberg et al., 2009; Flett, Vredenburg, & Krames, 1997). For example, a study on depressive symptoms among children and adolescents found a dimensional structure for all of the DSM-IV symptoms of major depression (Hankin, Fraley, Lahey, & Waldman, 2005).

The inventor of the term "schizophrenia," Eugene Bleuler, viewed so-called pathological conditions as continuous with so-called "normal" conditions and noted the occurrence of "schizophrenic" symptoms among normal individuals (Gilman, 1988). In fact, Bleuler referred to the major symptom of "schizophrenia" (thought disorder) as simply "*ungewonlich*," which in German means "unusual," not "bizarre," as it was translated in the first English version of Bleuler's classic monograph (Gilman, 1988). Essentially, the creation of "schizophrenia" was "an artifact of the ideologies implicit in nineteenth century European and American medical nosologies" (Gilman, 1988, p. 204). Indeed, research

indicates that the hallucinations and delusions exhibited by people diagnosed with a schizophrenic disorder are continuous with experiences and behaviors among the general population (Johns & van Os, 2001; van Os et al., 1999; see also Mittal et al., Chapter 17 in this volume). Recent research also suggests that dimensional measures of psychosis are better predictors of dysfunctional behavior, social adaptation, and occupational functioning than are categorical diagnoses (Rosenman, Korten, Medway, & Evans, 2003). Theory and research on neuroticism strongly suggests that is it provides the foundation for the development and anxiety and moods disorders and is best conceived as a dimension (Barlow, Sauer-Savala, Carl, Bullis, & Ellard, 2013). Finally, biological researchers continue to discover continuities between so-called normal and abnormal (or pathological) psychological conditions (Claridge, 1995; Livesley, Lang, & Vernon, 1998; Nettle, 2001).

Understanding the research supporting the dimensional approach is important because the vast majority of this research undermines the illness ideology's assumption that we can make clear, scientifically based distinctions between the psychologically well or healthy and the psychological ill or disordered. Inherent in the dimensional view is the assumption that these distinctions are not natural demarcations that can be "discovered;" instead, they are created or constructed "by accretion and practical necessity, not because they [meet] some independent set of abstract and operationalized definitional criteria" (Francis & Widiger, 2012, p. 111).

Dimensional approaches, of course, are not without their limitations, including the greater difficulties they present in communication among professionals compared to categories, and the greater complexity of dimensional strategies for clinical use (Simonsen, 2010). In addition, researchers and clinicians have not reached a consensus on which dimensions to use (Simonsen, 2010). Finally, dimensional approaches do not solve the "subjectivity problem" noted previously because the decision regarding how far from the mean a person's thoughts, feelings, or behavior must be to be considered "abnormal" remains a subjective one. Nonetheless, dimensional approaches have been gradually gaining great acceptance and will inevitably be integrated more and more into the traditional categorical schemes. [An extensive discussion of the pros and cons of categorical approaches are beyond the scope of this chapter. Detailed and informative discussions can be found in other recent sources (e.g., Grove & Vrieze, 2010; Simonsen, 2010).]

Dimensional conceptions of psychopathology did make some small inroads in the DSM-5, particularly in the new conception of "autism spectrum disorder" (which encompasses autistic disorder, Asperger's disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified) and an appendix that describes an "alternative DSM-5 model for personality disorders" based largely on dimensional research on personality. The rest of the document, however, remains a compendium of categories.

Social Constructionism and Conceptions of Psychopathology

If we cannot come up with an objective and scientific conception of psychopathology and mental disorder, then what way is left to us to understand these terms? How then are we to conceive of psychopathology? The solution to this problem is not to develop yet another definition of psychopathology. The solution, instead, is to accept the fact that the problem has no solution—at least not a solution at which we can arrive by scientific means. We have to give up the goal of developing a scientific definition and accept the idea that psychopathology and related terms are not the kind of terms that can be defined through the processes that we usually think of as scientific. We have to stop struggling to develop a scientific conception of psychopathology and attempt instead to try to understand the struggle itself—why it occurs and what it means. We need to better understand how people go about trying to conceive of and define psychopathology, what they are trying to accomplish when they do this, and how and why these conceptions are the topic of continual debate and undergo continual revision.

We start by accepting the idea that psychopathology and related concepts are abstract ideas that are not scientifically constructed but *socially* constructed. *Social constructionism* involves "elucidating the process by which people come to describe, explain, or otherwise account for the world in which they live" (Gergen, 1985, pp. 3–4). Social constructionism is concerned with "examining ways in which people understand the world, the social and political processes that influence how people define words and explain events, and the implications of these definitions and explanations—who benefits and who loses because of how we describe and understand the world" (Muehlenhard & Kimes, 1999, p. 234). From this point of view, words and concepts such as *psychopathology* and *mental disorder* "are products of particular historical and cultural understandings rather than . . . universal and immutable categories of human experience" (Bohan, 1996, p. xvi).

Universal or "true" definitions of concepts do not exist because these definitions depend primarily on who gets to do the defining. The people who define them are usually people with power, and so these definitions reflect and promote their interests and values (Muehlenhard & Kimes, 1999). Therefore, "When less powerful people attempt to challenge existing power relationships and to promote social change, an initial battleground is often the words used to discuss these problems" (Muehlenhard & Kimes, 1999, p. 234). Because the interests of people and institutions are based on their values, debates over the definition of concepts often become clashes between deeply and implicitly held beliefs about the way the world works

or should work and about the difference between right and wrong. Such clashes are evident in the debates over the definitions of *domestic violence* (Muehlenhard & Kimes, 1999), *child sexual abuse* (Holmes & Slapp, 1998; Rind, Tromovich, & Bauserman, 1998), and other such terms.

The social constructionist perspective can be contrasted with the *essentialist* perspective. Essentialism assumes that there are natural categories and that all members of a given category share important characteristics (Rosenblum & Travis, 1996). For example, the essentialist perspective views our categories of race, sexual orientation, and social class as objective categories that are independent of social or cultural processes. It views these categories as representing "empirically verifiable similarities among and differences between people" (Rosenblum & Travis, 1996, p. 2) and as "depict[ing] the inherent structure of the world in itself" (Zachar & Kendler, 2010, p. 128). In the social constructionist view, however, "reality cannot be separated from the way that a culture makes sense of it" (Rosenblum & Travis, 1996, p. 3). In social constructionism, such categories represent not what people *are* but rather the ways that people think about and attempt to make sense of differences among people. Social processes also determine what differences among people are more important than other differences (Rosenblum & Travis, 1996).

Thus, from the essentialist perspective, psychopathologies and mental disorders are natural entities whose true nature can be discovered and described. From the social constructionist perspective, however, they are abstract ideas that are defined by people and thus reflect their values—cultural, professional, and personal. The meanings of these and other concepts are not *revealed* by the methods of science but are *negotiated* among the people and institutions of society who have an interest in their definitions. In fact, we typically refer to psychological terms as "constructs" for this very reason—that their meanings are constructed and negotiated rather that discovered or revealed. The ways in which conceptions of so basic a psychological construct as the "self" (Baumeister, 1987) and "self-esteem" (Hewitt, 2002) have changed over time and the different ways they are conceived by different cultures (e.g., Cushman, 1995; Hewitt, 2002; Cross & Markus, 1999) provide an example of this process at work. Thus "all categories of disorder, even physical disorder categories convincingly explored scientifically, are the product of human beings constructing meaningful systems for understanding their world" (Raskin & Lewandowski, 2000, p. 21). In addition, because "what it means to be a person is determined by cultural ways of talking about and conceptualizing personhood . . . identity and disorder are socially constructed, and there are as many disorder constructions as there are cultures" (Neimeyer & Raskin, 2000, pp. 6–7; see also López & Guarnaccia, Chapter 4 in this volume). Finally, "if people cannot reach the objective truth about what disorder really is, then viable constructions of disorder must compete with one another on the basis of their use and meaningfulness in particular clinical situations" (Raskin & Lewandowski, 2000, p. 26). In other words, the debate about defining mental disorders continues because people continue to manufacture and modify the definitions they find most useful.

From the social constructionist perspective, sociocultural, political, professional, and economic forces influence professional and lay conceptions of psychopathology. Our conceptions of psychological normality and abnormality are not facts about people but abstract ideas that are constructed through the implicit and explicit collaborations of theorists, researchers, professionals, their clients, and the culture in which all are embedded and that represent a shared view of the world and human nature. For this reason, "mental disorders" and the numerous diagnostic categories of the DSM were not "discovered" in the same manner that an archeologist discovers a buried artifact or a medical researcher discovers a virus. Instead, they were invented (see Raskin & Lewandowski, 2000). By saying that mental disorders are invented, however, we do not mean that they are "myths" (Szasz, 1974) or that the distress of people who are labeled as mentally disordered is not real. Instead, we mean that these disorders do not "exist" and "have properties" in the same manner that artifacts and viruses do, even if they do have concomitant, complex biological processes. Therefore, a conception of psychopathology "does not simply describe and classify characteristics of groups of individuals, but . . . actively constructs a version of both normal and abnormal . . . which is then applied to individuals who end up being classified as normal or abnormal" (Parker, Georgaca, Harper, McLaughlin, & Stowell-Smith, 1995, p. 93).

Conceptions of psychopathology and the various categories of psychopathology are not mappings of psychological facts about people. Instead, they are social artifacts that serve the same sociocultural goals as do our conceptions of race, gender, social class, and sexual orientation—those of maintaining and expanding the power of certain individuals and institutions and maintaining social order, as defined by those in power (Beall, 1993; Parker et al., 1995; Rosenblum & Travis, 1996). As are these other social constructions, our concepts of psychological normality and abnormality are tied ultimately to social values—in particular, the values of society's most powerful individuals, groups, and institutions—and the contextual rules for behavior derived from these values (Becker, 1963; Kirmayer, 2005; Parker et al., 1995; Rosenblum & Travis, 1996). As McNamee and Gergen (1992) state: "The mental health profession is not politically, morally, or valuationally neutral. Their practices typically operate to sustain certain values, political arrangements, and hierarchies of privilege" (p. 2). Thus, the debate over the definition of psychopathology, the struggle over who gets to define it, and the continual revisions of the DSM are not aspects of a search for "truth." Rather, they are debates over the definition of socially constructed abstractions and struggles for the personal, political, and economic power that derives from the authority to define

these abstractions and thus to determine what and whom society views as normal and abnormal.

Millon (2010) has even suggested that the development of the DSM-IV was hampered by the reluctance of work groups to give up their rights over certain disorders once they were assigned them, even when it became clear that some disorders fit better with other work groups. In addition, over half of the members of the DSM-IV work groups (including every member of the work groups responsible for mood disorders and schizophrenia/psychotic disorders) had received financial support from pharmaceutical companies (Cosgrove, Krimsky, Vijayaragahavan, & Schneider, 2006).

As David Patrick (2005) concluded about a definition of mental disorder offered by the British government in a recent mental health bill, "The concept of mental disorder is of dubious scientific value but it has substantial political utility for several groups who are sane by mutual consent" (p. 435).

These debates and struggles are described in detail by Allan Horwitz in *Creating Mental Illness*. According to Horwitz,

> The emergence and persistence of an overly expansive disease model of mental illness was not accidental or arbitrary. The widespread creation of distinct mental diseases developed in specific historical circumstances and because of the interests of specific social groups . . . By the time the DSM-III was developed in 1980, thinking of mental illnesses as discrete disease entities . . . offered mental health professionals many social, economic, and political advantages. In addition, applying disease frameworks to a wide variety of behaviors and to a large number of people benefited a number of specific social groups including not only clinicians but also research scientists, advocacy groups, and pharmaceutical companies, among others. The disease entities of diagnostic psychiatry arose because they were useful for the social practices of various groups, not because they provided a more accurate way of viewing mental disorders.
>
> (Horwitz, 2002, p. 16)

Psychiatrist Mitchell Wilson (1993) has offered a similar position. He has argued that the dimensional/continuity view of psychological wellness and illness posed a basic problem for psychiatry because it "did not demarcate clearly the well from the sick" (p. 402) and that "if conceived of psychosocially, psychiatric illness is not the province of medicine, because psychiatric problems are not truly medical but social, political, and legal" (p. 402). The purpose of DSM-III, according to Wilson, was to allow psychiatry a means of marking out its professional territory. Kirk and Kutchins (1992) reached the same conclusion following their thorough review of the papers, letters, and memos of the various DSM working groups.

The social construction of psychopathology works something like this. Someone observes a pattern of behaving, thinking, feeling, or desiring that deviates from some social norm or ideal or identifies a human weakness or imperfection that, as expected, is displayed with greater frequency or severity by some people than others. A group with influence and power decides that control, prevention, or "treatment" of this problem is desirable or profitable. The pattern is then given a scientific-sounding name, preferably of Greek or Latin origin. The new scientific name is capitalized. Eventually, the new term may be reduced to an acronym, such as OCD (obsessive-compulsive disorder), ADHD (attention-deficit/hyperactivity disorder), and BDD (body dysmorphic disorder). Once a condition is referred to as a "disorder" in a diagnostic manual, it becomes *reified* and treated as if it were a natural entity existing apart from judgments and evaluations of human beings. The new disorder then takes on an existence all its own and becomes a disease-like entity. As news about "it" spreads, people begin thinking they have "it;" medical and mental health professionals begin diagnosing and treating "it;" and clinicians and clients begin demanding that health insurance policies cover the "treatment" of "it." Once the "disorder" has been socially constructed and defined, the methods of science can be employed to study it, but the construction itself is a social process, not a scientific one. In fact, the more "it" is studied, the more everyone becomes convinced that "it" really is "something."

Medical philosopher Lawrie Reznek (1987) has demonstrated that even our definition of physical disease is socially constructed. He writes:

> Judging that some condition is a disease is to judge that the person with that condition is less able to lead a good or worthwhile life. And since this latter judgment is a normative one, to judge that some condition is a disease is to make a normative judgment . . . This normative view of the concept of disease explains why cultures holding different values disagree over what are diseases . . . Whether some condition is a disease depends on where we choose to draw the line of normality, and this is not a line that we can discover . . . disease judgments, like moral judgments, are not factual ones.
>
> (Reznek, 1987, pp. 211–12)

Likewise, Sedgwick (1982) points out that human diseases are natural processes. They may harm humans, but they actually promote the "life" of other organisms. For example, a virus's reproductive strategy may include spreading from human to human. Sedgwick writes:

> There are no illnesses or diseases in nature. The fracture of a septuagenarian's femur has, within the world of nature, no more significance than the snapping of an autumn leaf from its twig; and the invasion of a human organism by cholera-germs carries with it no more the stamp of 'illness' than does the souring of milk by other forms of bacteria. Out of his anthropocentric self-interest, man has chosen to consider as "illnesses" or "diseases" those natural circumstances which precipitate death (or the failure to function according to certain values).
>
> (Sedgwick, 1982, p. 30)

If these statements are true of physical disease, they are certainly true of psychological "disease" or psychopathology. Like our conception of physical disease, our conceptions of psychopathology are social constructions that are grounded in sociocultural goals and values, particularly our assumptions about how people should live their lives and about what makes life worth living. This truth is illustrated clearly in the American Psychiatric Association's 1952 decision to include homosexuality in the first edition of the DSM and its 1973 decision to revoke its "disease" status (Kutchins & Kirk, 1997; Shorter, 1997). As stated by Wilson (1993), "The homosexuality controversy seemed to show that psychiatric diagnoses were clearly wrapped up in social constructions of deviance" (p. 404). This issue also was in the forefront of the debates over post-traumatic stress disorder, paraphilic rapism, and masochistic personality disorder (Kirk & Kutchins, 1997), as well as caffeine dependence, sexual compulsivity, low intensity orgasm, sibling rivalry, self-defeating personality, jet lag, pathological spending, and impaired sleep-related painful erections, all of which were proposed for inclusion in DSM-IV (Widiger & Trull, 1991). Others have argued convincingly that schizophrenia (Gilman, 1988), addiction (Peele, 1995), post-traumatic stress disorder (Herbert & Forman, 2010), personality disorder (Alarcon, Foulks, & Vakkur, 1998), dissociative identity disorder (formerly multiple personality disorder; Spanos, 1996), intellectual disability (Rapley, 2004) and both conduct disorder and oppositional defiant disorder (Mallet, 2007) also are socially constructed categories rather than disease entities.

With each revision, our most powerful professional conception of psychopathology, the DSM, has had more and more to say about how people should live their lives. The number of official mental disorders recognized by the American Psychiatric Association has increased from six in the mid-19th century to close to 300 in the DSM-5 (Francis & Widiger, 2012). Between 1952 and 2013, the number of pages in the DSM increased from 130 (mostly appendices) to over 900. As the scope of "mental disorder" has expanded with each DSM revision, life has become increasingly pathologized, and the sheer number of people with diagnosable mental disorders has continued to grow. Moreover, mental health professionals have not been content to label only obviously and blatantly dysfunctional patterns of behaving, thinking, and feeling as "mental disorders." Instead, we have defined the scope of psychopathology to include many common problems in living (Francis, 2013).

Consider some of the "mental disorders" found in the DSM-5. Cigarette smokers have tobacco-use disorder. If you try to quit, you are likely to develop the mental disorder tobacco withdrawal. If you drink large quantities of coffee, you may develop caffeine intoxication or caffeine-induced sleep disorder. What used to be known as simply "getting stoned" is the mental disorder cannabis intoxication—a mental disorder that afflicts millions of people every year—if not every day. If you have "a preoccupation with one or more perceived deflects or flaws in physical appearance that are not observable or appear slight to others" (American Psychiatric Association, 2013, p. 242) that causes you significant distress of dysfunction, you may have BDD. A child with "difficulties learning and using academic skills . . . that have persisted for at least 6 months, despite the provision of interventions that target those difficulties" (American Psychiatric Association, 2013, p. 66) may have the mental disorder specific learning disorder. (There is no mention of the possibility that the targeted interventions may have been the wrong interventions.) Toddlers who throw tantrums may have oppositional defiant disorder. Women who are irritable or emotionally labile before their menstrual period may have premenstrual dysphoric disorder. People who eat gum or ice may have pica. Adults who are not interested in sex may have female sexual interest/arousal disorder or male hypoactive sexual desire disorder. Women who have sex but do not have orgasms that are frequent enough or intense enough may have a female orgasmic disorder. For men, ejaculating too early and too late are both signs of a mental disorder.

Consider also some of the new disorders that were proposed for DSM-5: hypersexual disorder, temper dysregulation disorders of childhood, hoarding disorder, skin-picking disorder, psychosis risk syndrome, among others. Psychiatrist Allen Frances, the chair of the DSM-IV task force, has argued that these new "disorders" represent a further encroachment of the DSM into the realm of common problems in living (Frances, 2013). Nonetheless, hoarding disorder, disruptive mood dysregulation disorder (a renamed temper dysregulation disorder of childhood), and excoriation (skin-picking) disorder found their way into the DSM-5. Several other conditions (e.g. persistent complex bereavement disorder, internet gaming disorder) are listed as "conditions for further study" and therefore are likely to find there way into DSM-6.

In addition, "diagnostic fads" are sparked by each new edition. Francis notes four "epidemics" that were sparked by changes in from DSM-III to DSM-IV: autism, attention deficit/hyperactivity disorder, childhood bipolar disorder, and paraphilia not otherwise specified (Francis, 2013). He also warns that DSM-5 threatens to provoke new epidemics of at least four new disorders that emerged in DSM-5: Disruptive mood dysregulation disorder, binge-eating disorder, mild neurocognitive disorders, and "behavioral addictions" (Francis, 2013; see also Paris, 2013).

The past few years have witnessed media reports of epidemics of internet addiction, road rage, and "shopaholism." Discussions of these new disorders have turned up at scientific meetings and in courtrooms. They are likely to find a home in a future revision of the DSM if the media, mental health professions, and society at large continue to collaborate in their construction and if "treating" them and writing books about them become lucrative (Beato, 2010).

The social constructionist perspective does not deny that human beings experience behavioral and emotional difficulties—sometimes very serious ones. It insists,

however, that such experiences are not evidence for the existence of entities called "mental disorders" that can then be invoked as causes of those behavioral and emotional difficulties. The belief in the existence of these entities is the product of the all too human tendency to socially construct categories in an attempt to make sense of a confusing world.

The socially constructed illness ideology and associated traditional psychiatric diagnostics schemes, also socially constructed, have led to the proliferation of "mental illnesses" and to the pathologization of human existence (e.g., Francis, 2013). Given these precursors, it comes as no surprise that a highly negative clinical psychology evolved during the 20th century. The increasing heft and weight of the DSM, which has been accompanied by its increasing influence over clinical psychology, provides evidence for this. As the socially constructed boundaries of "mental disorder" have expanded with each DSM revision, more relatively mundane human behaviors have become pathologized; as a result, the number of people with diagnosable "mental disorders" has continued to grow. This growth has occurred largely because mental health professionals have not been content to label only the obviously and blatantly dysfunctional patterns of behaving, thinking, and feeling as "mental disorders." Instead, they (actually "we") have gradually pathologized almost every conceivable human problem in living. As a result of the growing dominance of the illness ideology among both professionals and the public, eventually everything that human beings think, feel, do, and desire that is not perfectly logical, adaptive, efficient or "creates trouble in human life" (Paris, 2013, p. 43) will become a "mental disorder" (Francis, 2013; Paris, 2013). This is not surprising given that Francis notes that in his more than two decades of working on three DSMs, "never once did he recall an expert make a suggestion that would reduce the boundary of his pet disorder" (Francis & Widiger, 2012, p. 118). DSM-5 has made normality "an endangered species" partly because we live in a society that is "perfectionistic in its expectations and intolerant of what were previously considered to be normal and expectable distress and individual differences" (Francis & Widiger, 2012, p. 116), but also partly because pharmaceutical companies are constantly trying to increase the market for their drugs by encouraging the loosening and expanding of the boundaries of mental disorders described in the DSM (Francis, 2013; Paris, 2013). Essentially, DSM-5 "just continues a long-term trend of expansion into the realm of normality" (Paris, 2013, p. 183). As it does, "with ever-widening criteria for diagnosis, more and more people will fall within its net [and] many will receive medications they do not need" (Paris, 2013, p. 38).

We acknowledge that DSM-5 is an improvement over DSM-IV in its greater attention to alternative dimensional models for conceptualizing psychological problems and its greater attention to the importance of cultural considerations in determining whether or not a problematic pattern should be viewed as a "mental disorder." Yet it remains steeped in the illness ideology for most of its 900 pages. For example, still included in the revised definition of *mental disorder* is the notion that a mental disorder is "a dysfunction in the individual" (p. 20)—an assumption that is inconsistent with almost every psychological and sociological conception of human functioning.

Summary and Conclusions

The debate over the conception or definition of *psychopathology* and related terms has been going on for decades, if not centuries, and will continue, just as we will always have debates over the definitions of truth, beauty, justice, and art. Our position is that *psychopathology* and *mental disorder* are not the kinds of terms whose "true" meanings can be discovered or defined objectively by employing the methods of science. They are *social constructions*—abstract ideas whose meanings are negotiated among the people and institutions of a culture and that reflect the values and power structure of that culture at a given time. Thus, the conception and definition of *psychopathology* always has been and always will be debated and always has been and always will be changing. It is not a static and concrete thing whose true nature can be discovered and described once and for all.

By saying that conceptions of psychopathology are socially constructed rather than scientifically derived, we are not proposing, however, that human psychological distress and suffering are not real or that the patterns of thinking, feeling, and behaving that society decides to label *psychopathology* cannot be studied objectively and scientifically. Instead, we are saying that it is time to acknowledge that science can no more determine the "proper" or "correct" conception of *psychopathology* and *mental disorder* than it can determine the "proper" and "correct" conception of other social constructions such as beauty, justice, race, and social class. We can nonetheless use science to study the phenomena that our culture refers to as psychopathological. We can use the methods of science to understand a culture's conception of mental or psychological health and disorder, how this conception has evolved, and how it affects individuals and society. We also can use the methods of science to understand the origins of the patterns of thinking, feeling, and behaving that a culture considers psychopathological and to develop and test ways of modifying those patterns.

Psychology and psychiatry will not be diminished by acknowledging that their basic concepts are socially and not scientifically constructed—no more than medicine is diminished by acknowledging that the notions of *health* and *illness* are socially constructed (Reznek, 1987), nor economics by acknowledging that the notions of *poverty* and *wealth* are socially constructed. Likewise, the recent controversy in astronomy over how to define the term *planet* (Zachar & Kendler, 2010) does not make astronomy any less scientific. Science cannot provide us with "purely

factual, scientific" definitions of these concepts. They are fluid and negotiated constructs, not fixed matters of fact.

As Lilienfeld and Marino have commented:

> Removing the imprimatur of science . . . would simply make the value judgments underlying these decisions more explicit and open to criticism . . . heated disputes would almost surely arise concerning which conditions are deserving of attention from mental health professionals. Such disputes, however, would at least be settled on the legitimate basis of social values and exigencies, rather than on the basis of ill-defined criteria of doubtful scientific status.
>
> (Lilienfeld and Marino, 1995, pp. 418–19)

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2

Developmental Psychopathology

Basic Principles

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Researchers and clinicians alike have exerted considerable effort to unravel the intricacies underlying disordered behavior in children and adults. In more recent decades, the foray into charting the precursors and developmental progression of childhood behavioral disturbance has emerged as its own unique subspecialty within clinical and developmental psychology. An exciting advance emanating from this burgeoning interest is the macroparadigm (Achenbach, 1990; Cicchetti, 2013) of developmental psychopathology. In their seminal article, Sroufe and Rutter (1984) defined developmental psychopathology as "the study of the origins and course of individual patterns of behavioral maladaptation, whatever the age of onset, whatever the causes, whatever the transformations in behavioral manifestation, and however complex the course of the developmental pattern may be" (p. 18). The primary focus of the developmental psychopathology perspective is to study the processes underlying continuity and change in patterns of both adaptive and maladaptive behavior from an interdisciplinary approach. A central tenet is the notion that no single theory can adequately explain all aspects of psychological maladjustment (Rutter, 2013). Instead, psychological functioning is best understood through reliance on and integration of multiple levels of analyses that arise from a variety of disciplines, each with unique theoretical views and methodological approaches. Accordingly, the developmental psychopathology approach draws on diverse scientific fields such as lifespan developmental psychology, clinical psychology, psychiatry, neuroscience, epidemiology, sociology, neuroendocrinology, genetics, among others, with the goal of providing a comprehensive knowledge base concerning the mutually influencing processes that underlie maladaptation as well as adaptation (Sroufe, 2013).

Although on the surface the developmental psychopathology perspective may seem to be most similar to the sub-disciplines of child clinical and developmental psychology, its emphasis differs in important ways. Developmental psychopathology is interested in the processes that mediate or moderate the development of disordered behaviors, with a primary focus on the origins of the behaviors and how they manifest themselves in disorder or adaptation over development. Of equal interest are those precursors of maladaptation (e.g., presence of risk factors) that do not lead to disorder. In contrast to clinical psychology, the developmental psychopathology perspective does not focus on differential diagnosis, treatment, or prognosis but rather is interested in the pathways that lead toward and away from disorder. Further, developmental psychopathology researchers are interested in individual differences in patterns of adaptation rather than examining group differences in a particular aspect of a disorder. Relatedly, developmental psychopathology relies heavily on basic research emanating from lifespan developmental psychologists to help identify the complex links between specific normative developmental issues or tasks and the emergence of later disorder (Sroufe & Rutter, 1984). Examining and characterizing the effects of risk and protective factors (e.g., poverty, minority ethnic status, intelligence, socioeconomic status) on developmental processes as they relate to the emergence of disorder is a paradigmatic developmental psychopathology research agenda; it identifies the contextual influences that place children at risk for or buffers and protects them from maladaptation.

In sum, developmental psychopathology is a "conceptual approach that involves a set of research methods that capitalize on developmental and psychopathological variations to ask questions about mechanisms and processes" (Rutter, 2013, p. 1201). Psychopathology is viewed as developmental deviation in which specific aspects of the normative developmental trajectory have been derailed and,

consequently, maladaptive behaviors manifest (Sroufe & Rutter, 1984). The following sections of this chapter detail the core tenets of the developmental psychopathology perspective, with illustrative examples provided throughout.

Core Tenets

General Principles of Development An understanding of the developmental psychopathology perspective requires familiarity with the basic principles of development that underlie most developmental theories (Sroufe & Rutter, 1984). An essential starting point concerns the use of age as a developmental marker. Simply studying a sample of children or adolescents of a particular age does not necessarily constitute a developmental approach in and of itself, nor does it necessarily shed light on a developmental process. Rutter (1989) has suggested that to understand and study the processes that underlie age differences in a particular phenomenon, chronological age should be conceptualized as reflecting four types of influence, including cognitive and biological maturity as well as type and duration of environmental experiences. A developmental approach begins with a topic of interest (e.g., importance of language development in relational aggression) then, based on theory and empirical literature, hypotheses are offered about when differences in development processes may emerge. These hypotheses are then tested by selecting children of differing stages based on cognitive, biological, or experiential factors (e.g., toddler, preschool, early elementary age) or by selected abilities (e.g., expressive language abilities) that theoretically best illuminate important transitions or points of change in the topic of interest. When comparing atypically developing groups of children to a control or comparison group, it is necessary to match these children on constructs of importance to the research question (e.g., reading ability). The importance of specifying the underlying developmental process, as opposed to age, is exemplified in the literature examining the development of eating disorders. In particular, research has identified pubertal timing, in particular, as a risk factor for eating pathology (Baker, Thornton, Lichtenstein, & Bulik, 2012; Harden, Mendle, & Kretsche, 2012). Thus, although chronological age may sometimes be used as a marker of development, owing to its simplicity and convenience, the effect of its component parts (e.g., biological maturation) on the process under investigation must be considered.

Although one theory does not predominate in developmental psychology, there are a number of principles that characterize development (Santostefano, 1978). The principle of holism refers to the notion that development consists of a set of interrelated domains that exert transactional effects. Although researchers often refer to physical, social, cognitive, language, or emotional development as if they were separate, independent domains, development in one area influences development in the others. For example, worry is generally considered a normative developmental experience that is more common as children's cognitive abilities become more differentiated and complex (Muris, Merckelbach, Meetsers, & van den Brand, 2002). For children to experience complex worries (e.g., death concerns), they need the ability to engage in at least rudimentary abstract thought that involves anticipation of the future in which a possible array of potential negative outcomes is considered. Such cognitive skills are most reflective of later stages of cognitive development (i.e., concrete and formal operational periods; Piaget, 1972). Likewise, to manage the affective component of worry successfully, children must have developed emotion regulation skills. The absence, or delay, of these skills might contribute to chronic mismanagement of worry (i.e., avoidance, age-inappropriate clinginess to parents) and result in maladaptive functioning. Undoubtedly, children's emotional, cognitive, and social development are dynamically intertwined (Jacob, Suveg, & Whitehead, 2014; Tureck & Matson, 2012).

Directedness refers to the notion that children are active shapers of their environment and not passive recipients of experience (Scarr & McCartney, 1983). Thus, a child's unique developmental trajectory is the result of an interaction among genetic influences, a history of prior experiences, and a series of adaptations to environmental influences. Differentiation of modes and goals purports that, with development, children's behavior becomes more flexible with increased organization and differentiation. These developments, in turn, promote adaptation to the increasingly complex demands present in the environment. Individual differences in flexibility and behavioral organization then lead to different trajectories of psychological adjustment or deviation from developmental norms. Finally, the mobility of behavioral function principle states that earlier, more undifferentiated forms of behavior become hierarchically integrated into later forms of behavior. Interestingly, the earlier behavioral forms may lay dormant but can become activated under periods of stress, producing behaviors that appear to be regressed. For example, a child who has mastered toilet training may regress to earlier forms of behavior when stressed by the adjustment to the birth of a sibling. In this way, new development is based upon and builds on prior development attainment.

Overall, development is considered to be the interaction of genetic and environmental influences plus prior adaptation. The many dynamic transactions that occur among the various developing systems has a fanning out effect that cuts across different developmental systems and affects the course of development, a process known as developmental cascades (Masten & Cichetti, 2010). From within this model, the individual and their context is considered to be inseparable because of the mutual and continual interactions between them. The developmental psychopathology perspective is unique in its emphasis on prior experience when investigating the development of adaptation and disorder. That is, each developmental progression is considered to be a series of adaptations or

maladaptations that evolve over time to produce a specific outcome. Past experiences are critical in the unfolding of future behaviors because individuals interpret and respond to new situations based on their prior history. For example, as an outcome of poor parenting behaviors, a child who behaves aggressively in preschool with his peers begins to experience mild forms of peer rejection. Although this boy is placed into a new school environment for kindergarten, his history of unpleasant peer relations in preschool and his emergent hostile attribution bias (Dodge, 1980) contribute to his interpretation or social information processing of ambiguous overtures by peers as provocative and his subsequent selection of a confrontational response to these peers. Thus, this boy adopts an active role in creating his experiences (i.e., niche picking) and, in so doing, successive steps towards maladaptation are made. This cascade of effects is seen in the direct and indirect relations over time between peer rejection, social information processing, and, ultimately, aggressive behavior (Lansford, Malone, Dodge, Pettit, & Bates, 2010). The outcome of these behaviors is not immutable because change remains possible at all steps in development, but the interaction of genes, context, and prior adjustment will guide the direction of the outcome for a certain behavior (Sroufe, 1997).

Mutual Influence of Typical and Atypical Development

Another unique and defining feature of the developmental psychopathology perspective is its emphasis on the study of both typical and atypical development in concert, because they are mutually informing and provide a comprehensive understanding of development (Sroufe, 1990). From this perspective, psychopathology is defined as developmental deviation; the implication being that in order to understand what is considered atypical or abnormal, knowledge of what is normative is of utmost importance. (See also Chapter 1 in this volume.) Delineating the pathways to competent functioning when faced with conditions of adversity or other derailing environmental influence (e.g., risk research) is of key importance for constructing a framework to fully understand the complexities of development. Conversely, the study of atypical developmental processes helps to inform and clarify understanding of normative processes. In some instances, when studying normative behavior, the component processes involved in a developmental task are inextricably intertwined and integrated, making it difficult to distinguish each component and its role in the construction of the behavior under examination.

Consider the example of emotion regulation, a construct that has garnered considerable theoretical and empirical discussion (Cole, Martin, & Dennis, 2004). Emotion regulation is "the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one's goals" (Thompson, 1994, p. 27–8). There are numerous interwoven components that comprise emotion regulation, including emotional reactivity, coping strategies, and emotional understanding, to name just a few. For example, emotional reactivity is one's initial, unmodulated response to an emotion-provoking event, whereas emotion-coping strategies involve the modification of this reactivity through a variety of means (e.g., cognitive interpretation of the arousal, use of distraction, support seeking). To successfully regulate one's emotional experience in response to the demands of the social context, individuals must also employ emotion understanding skills to identify and label emotions and to understand the causes/consequences of emotional experiences. Thus, emotion regulation comprises numerous closely related, interacting processes, and studying each component in isolation from the other may create an incomplete picture of the phenomenon being studied.

Illumination of these component processes can sometimes be achieved through the investigation of atypical development in which functioning in one of the specific facets may have gone awry. For example, empirical research has found that anxious youth exhibit high emotional reactivity (Carthy, Horesh, Apter, & Gross, 2010), yet they do not exhibit deficits across all related emotion regulation processes. That is, anxious youth demonstrate less adaptive coping with emotional experiences, poorer understanding of how to dissemble or alter emotional expression but no differences from non-anxious youth in understanding of emotion cues and multiple emotions (Suveg et al., 2008; Suveg & Zeman, 2004). Taken together, anxious youth seem to have difficulty translating their knowledge of emotion cues into adaptive emotion regulation, suggesting that knowledge of emotion-related skills is a necessary but insufficient condition for adaptive emotion regulation. In sum, by neglecting to study the dynamic interplay between typical and atypical developmental processes, understanding of the pathways to both adaptation and disorder will be incomplete. Each lens or particular emphasis on a developmental process provides important insights into the strengths and vulnerabilities associated with different pathways or trajectories to adjustment or disorder.

The interpretation of a behavior as adaptive or maladaptive depends on the context in which the behavior occurs, and the outcome of the behavior. That is, the adaptive or maladaptive nature of specific behaviors can only be defined with respect to their ultimate end points or outcomes, and these outcomes may differ depending on an individual's unique contextual variables. For example, emotional competence reflects the flexible use of emotional displays that are sensitive to cues in the social context (Aldao, 2013). Children begin to learn these skills in early childhood, primarily through parental socialization of emotion (Zeman, Cassano, & Adrian, 2013). For children living in maltreating environments, however, the normative trajectory for learning these skills is altered. Research indicates that children who are physically maltreated have difficulties displaying their emotions in an

adaptive manner throughout childhood, as evidenced in infancy by less flexibility and sensitivity to environmental cues (Shields, Cicchetti, & Ryan, 1994). This pattern continues into middle childhood with emotional displays that are less situationally responsive in both family and peer contexts (Shipman & Zeman, 2001). Although functional in one context (i.e., the maltreating family), the same method of managing emotion may be maladaptive when utilized in another context (i.e., peers; Jenkins & Oatley, 1998).

In summary, "both positive adaptation and maladaptation can only be defined with respect to outcome, and developmental pathways are only fully defined by considering both the normal and abnormal outcomes in which they terminate and the strengths and liabilities of the patterns of adaptation and coping that mark their origins" (Sroufe, 1990, p. 336). The role of the developmental psychopathologist, then, is to delineate the component parts of the particular developmental process that promotes or inhibits optimal functioning by examining the mutual interplay between normative and atypical development.

Developmental Pathways Perspective When explicating the development of disorder or adaptation from a developmental psychopathology perspective, the concept of developmental pathways has been applied. To facilitate understanding of this construct, a commonly used metaphor is that of a tree in which adaptive and optimal development is represented by strong limbs emanating straight and upwards from the trunk (Sroufe, 1997; Waddington, 1957). Dysfunction or maladaptation is represented by successive growth on weaker branches leading away from the central, core of the tree. From this metaphor come four central propositions (Cicchetti & Rogosch, 2002; Sroufe, 1997; Sroufe & Rutter, 1984).

Disorder as Deviation From Normative Development First is the notion that disorder is considered to arise from a pattern of deviations from normative development that has evolved over time. Understanding what constitutes normative development is essential in order to determine what represents a deviation from the typical course. Certain pathways or branches represent adaptational failures that forecast the probability of later disorder. Repeated difficulty with mastering specific developmental tasks increases the likelihood of future maladjustment. From the tree metaphor, each adaptational failure adds an increment of growth on a branch leading away from the stabilizing strength of the core of the tree. One or two small failures are not likely to lead far from the core, but an abundance of these maladaptive developmental failures will further the distance from the core and increase the size of the wayward branch. Thus, disorder results when there is a repeated succession of deviations leading away from the blueprint of normative development.

Equifinality The second key proposition in the pathways framework, equifinality, purports that there are multiple pathways to a single outcome. That is, individuals may start on distinct points in their developmental origins and then experience varying influences at differing points in their developmental trajectory, yet have observably similar outcomes despite these differing developmental courses. For example, the pathways to later depression are quite varied. One adolescent could have a genetic predisposition for depression, whereas another adolescent may have experienced a maltreating home environment, and a third may have been raised by a depressed mother (Cicchetti & Rogosch, 2002). Yet the resultant outcome for all three individuals may converge on a depressive disorder, despite their unique preceding sets of biological and environmental influences. Thus, for researchers and clinicians, the principle of equifinality highlights the importance of determining the multitude of prior or predisposing factors that lead to outcomes of both adaptive and maladaptive functioning. Cicchetti and Sroufe (2000) comment that the research agenda with respect to equifinality has progressed from simply determining the antecedents of a behavior to addressing the more complex question, "What are the factors that initiate and maintain individuals on pathways probabilistically associated with X and a family of related outcomes?" (p. 257).

Multifinality The concept of multifinality refers to the notion that individuals may begin at a common starting point (e.g., the base of a branch), but the unfolding of the resultant pathways from that origin may diverge based on the interaction of prior experiences and biological factors that ultimately produce different patterns of adaptation or pathology. Even though the outcomes may appear to be quite different from a surface examination (phenotype), it is possible that their underlying causes and etiology (genotype) are more similar than dissimilar (Sroufe, 1997). These differing pathways are thought to arise from the dynamic interplay between risk and protective processes that are unique to each individual, and thus produce different pathways. For example, research has indicated that children from low-income, disadvantaged environments who have experienced at least one form of maltreatment exhibit a variety of different maladaptive outcomes. Moreover, a subset of the maltreated children exhibit remarkable resilience and appear to be protected by personal attributes of positive self-esteem, ego resilience, and ego control that are thought to be, in part, temperament based. Interestingly, the pathway to resilience for non-maltreated but low-income, disadvantaged children is reliant on relationship factors (i.e., maternal availability, relationship with camp counselor; Cicchetti & Rogosch, 1997). Thus, the interplay between the protective factors in this situation of adversity is crucial to understanding the divergence of pathways. A research agenda with this principle as a guide endeavors to answer the question, "What differentiates those progressing to

X from those progressing to Y and those being free from maladaptation or handicapping condition?" (Cicchetti & Sroufe, 2000, p. 257).

The Nature of Change Characterizing change and how it relates to the emergence of positive adaptation or disorder is of utmost importance to the developmental psychopathology perspective. Despite early adversity, change is thought to be possible at any juncture in development; pathology is not a stable entity that a child either has or does not have. Rather, the developmental psychopathology perspective asserts that the course of a maladaptive developmental trajectory can be modified in part, because individuals have a "self-righting" tendency to strive toward adaptive modes of functioning (Waddington, 1957). As described previously, this self-righting, self-organizational tendency has been documented in children who have experienced significant maltreatment within the context of other socioeconomic adversity (Cicchetti & Rogosch, 1997). From this framework, the role of the developmental psychopathology researcher is to investigate the factors that initiate and maintain the processes of self-righting that result in positive adaptation, and the underlying mechanisms that interfere with this self-organization process that steer individuals onto a path leading to maladaptive outcomes. Importantly, contextual factors can also moderate a maladaptive path. For instance, infants high in the temperament construct of behavioral inhibition (shy and withdrawn behaviors in response to novel situations) are at risk for internalizing problems across the developmental trajectory (Fox, Henderson, Marshall, Nichols, & Ghera, 2005). Yet, not all infants who display behavioral inhibition exhibit later maladaptation. Degnan, Henderson, Fox, and Rubin (2008) found that maternal negativity (neuroticism, depression) was related to social wariness in childhood for negatively reactive infants. In contrast, in the context of low levels of maternal negativity, there was no relation between infant negative reactivity and later social wariness.

Although change is possible at any point in the developmental trajectory, prior adaptation does place constraints on the possibility of future change. That is, the longer a child has been on a maladaptive or adaptive pathway, the more difficult or unlikely the possibility of change, particularly if development has crossed significant developmental milestones or stages (Sroufe, 1997). Using the tree metaphor, the farther the branch grows away from the trunk, the less support and nutrients it receives from the core of the tree. Thus, it becomes more difficult to redirect growth to rejoin the trajectory of positive adaptation. This construct is based on the notion that children are active shapers of their environment, in which they select particular experiences, interpret them according to their particular lens, and then exert an impact on the environment through their actions. All of these steps interact with each other. A particular type of maladaptation or psychopathology, then, is likely to become stronger over time, to the extent that the context facilitates the continuance of the behavior (Steinberg & Avenevoli, 2000). For example, for the boy who experienced peer rejection in preschool and continued this pattern in early elementary school, his interpretative frame or social information processing of social relationships (i.e., hostile attribution bias) will become internalized and solidified with additional experiences that may lead to an escalation of negative, aggressive peer relationships, and perhaps ultimately to significant antisocial behavior (Lansford et al., 2013). Research indicates that early intervention is critical to disrupting this dynamic chain of reinforcing behaviors and cognitions, and in essence, helping children rejoin the normative, adaptive path to social relationships. The more stable the path to antisocial and aggressive behavior, the more difficult positive adaptation at later time points becomes change (Conduct Problems Prevention Research, 2011).

In summary, the image of the branching tree provides a helpful metaphor to conceptualize the ways that pathways to both positive adaptation and maladaptation can occur. As with any metaphor, there are limitations to its applicability. Further, there are a finite number of possible pathways that exist, making the task of characterizing these trajectories a plausible goal rather than a hopeless task (Cicchetti & Rogosch, 2002). We now turn our attention to a few remaining constructs to be discussed from within the developmental psychopathology perspective.

Continuity One of the core issues of interest to developmental psychopathologists is determining whether the course of development is characterized by continuities or discontinuities across time and, if so, understanding their underlying mechanisms. A central research question concerns the prediction of adult psychopathology based on childhood behavior. That is, does depression at age 10 predict a stable pathway to depression in adulthood, barring any intervention efforts? Research over the past 30 years has made important strides in addressing this type of question (Rutter, 2013; Sroufe, 2013).

The course of typical and atypical development is considered to be lawful and coherent (Sroufe & Rutter, 1984) meaning that the way in which an individual develops in any given domain progresses in an orderly fashion that follows developmental principles of growth. This notion is not to be confused with behavioral stability or homotypic continuity, in which one would expect to see the same type of behavior exhibited across different developmental stages. This is rarely seen in development (Kagan, 1971). Rather, coherence (meaning congruity, consistency, logical connections) in development is expected, regardless of transformations in the observed behavior due to maturation. For example, in the development of locomotor skills in infancy and toddlerhood, there is coherence in development despite the appearance of dramatic transformations

in behavior that are exhibited in the progression of motor skills from sitting to crawling/scooting to walking to hopping. Coherence of development refers to the meaning of or the underlying processes involved in the behavior over time, rather than in the outward manifestation of the behavior. Thus, researchers look for continuity in processes that involve "persistence of the underlying organization and meaning of behavior despite changing behavioral manifestation" (Cicchetti & Rogosch, 2002, pp. 13–14).

Rutter (1981) has proposed that there are several different ways in which links are established between early development and later disorder. These linkages may be direct, in which an early experience: a) leads to or causes the disorder which then endures, b) leads to physical changes that then effect subsequent functioning, or c) results in a change in behavioral patterns that over time leads to maladaptive functioning or disorder. The linkages between early experience and later disorder may also operate in an indirect fashion in which: a) early experiences may change the dynamics and functioning of the family situation that then produces disordered behavior in the child over time, b) the experience of early stress affects the development of coping responses which can either result in increased sensitivity and compromised efforts to respond to stress or can buffer the child against the effects of stress and the development of disorder, c) through early experiences, the child experiences changes in self-concept which then influence his or her responses to future situations, and d) early experiences influence the individual's selection of subsequent environments. Thus, the way that issues and experiences at one developmental period are resolved sets the foundation for subsequent adaptations and issues at later stages. Children's development, then, is characterized by patterns of heterotypic continuities, discontinuities, and dramatic behavioral transformations, all of which make the study of the effect of early experience on later development extraordinarily challenging but also exciting in its potential for discovery.

Comorbidity The developmental psychopathology approach to understanding the nature of overlapping or co-existing diagnostic entities differs from the approach of traditional clinical child psychologists and psychiatrists. The term "comorbidity" has arisen from the medical model and implies the coexistence of two or more disorders from the current *Diagnostic and Statistical Manual of Mental Health* diagnostic system (American Psychiatric Association, 2013). Within this diagnostic system, comorbidity appears to be more typical than not (Caron & Rutter, 1991; Sroufe, 2013). From a developmental psychopathology perspective, however, comorbidity is viewed as a failure of the categorical system to characterize particular patterns of behavioral disturbance accurately. The focus of research, therefore, is concerned with developing classification systems based on patterns of adaptation and developmental outcomes using the pathways perspective. Adopting a developmental psychopathology perspective to classification systems may help to strengthen them. As such, it may be that instances of symptom overlap are due to many factors including: a) presence of shared risk factors, b) a comorbid association at the level of risk factors, c) the presence of a unique syndrome, and/or d) the occurrence of one disorder increasing the risk for the development and occurrence of another disorder (Caron & Rutter, 1991).

Research has attempted to explain common comorbidities in youth by examining variations in symptom patterns or underlying processes in particular developmental domains. For example, anxiety and depression commonly co-occur in children (Compas & Oppedisano, 2000) but research from a developmental psychopathology perspective has identified emotion-related variables that can differentiate the syndromes (Suveg, Hoffman, Zeman, & Thomassin, 2009). Specifically, poor emotion awareness, difficulties with emotion regulation, and high frequency of negative affect are emotion-related variables common to both anxiety and depression symptoms, but low frequency of positive affect is uniquely related to depression symptoms and frequency of emotion experience and somatic response to emotion activation are specific to anxiety symptoms of youth. Examining overlap in symptoms and syndromes is critical to better understand the underlying pathway(s) to the development of behavioral patterns and illuminates targets for prevention, a primary interest of developmental psychopathology researchers.

Franklin, Glenn, Jamieson, and Nock (2015) have articulated ways in which the developmental psychopathology approach can potentially advance issues of taxonomy and, in particular, the Research Domain Criteria Project (RDoC; Insel et al., 2010). The goal of the RDoC is to reclassify psychopathology based on underlying pathophysiology and behaviors. As discussed by Franklin et al. (2015), one potential weakness of this approach is biological reductionism, where there is an attempt to reduce subjective mental phenomena to objective physical phenomena. They caution that through reductionism, important information about the phenomenon of interest will be lost. To counter this problem, phenomena need to be examined at multiple levels of analysis that are then integrated. Although the types of information gained using such a complex approach are not likely to converge, it will provide important information that reflects the true depth of complexity of the process under examination with particular consideration given to the role of contextual variables. Of note, Franklin et al. (2015) articulate the many ways that developmental psychopathology principles can greatly enhance the further development of classification systems.

Risk and Resilience A typical developmental psychopathology research agenda is exemplified by risk and resilience research; namely, what biological and/or contextual

processes influence development either toward or away from adaptation. Risk research examines multiple levels of analysis and considers interactive (i.e., how variables influence one another) rather than main effects models. For example, differential susceptibility theories suggest that an individual's genetic composition can impact how sensitive a person is to environmental experiences (Belsky & Pleuss, 2009). In this way, a particular genetic variation in the context of positive environmental experiences may result in even better adaptation than if the variation was not present. Conversely, the same genetic variation in the context of poor or chronically stressful environmental experiences may result in the poorest outcomes. In one study, for example, Simons, Beach, Brody, Philibert, and Gibbons (2011) demonstrated that higher levels of aggression, anger, hostile view of relationships, and concern with "toughness" were found among African American children who exhibited genetic variations in the DRD4 and 5HTT alleles and who experienced adverse social conditions in comparison to youth with other genotypes. In contrast, youth with this genotype exhibited lower levels on the aggression and related variables in comparison to youth with other genotypes, when they experienced positive social circumstances. Other research has likewise examined complex gene-by-environment interactions (Brody et al., 2013; Lemery-Chalfant, Kao, Swann, & Goldsmith, 2013). Examining the interaction of multiple interacting factors that span genetic, physiological, behavioral, and environmental domains contributes to a better understanding of the processes underlying adaptation and maladaptation and mediators and moderators of the processes.

Although much debate surrounds the construct of resilience (Luthar, Cicchetti, & Becker, 2000), resilience is generally not viewed as a trait-like quality that the child simply "has" or is endowed with. Rather, resilience is thought to be a dynamic developmental process in which factors within the environment (e.g., secure attachment history) interact with characteristics of the child (e.g., intelligence) to produce positive outcomes or competence despite exposure to adverse conditions (e.g., living in a high-crime neighborhood; Luthar et al., 2000). Thus, simply being intelligent may not produce adaptation when faced with severe adversity, but a history of positive coping efforts in prior stressful situations and the presence of a secure attachment relationship with a primary caregiver may interact with a child's intelligence to yield a positive outcome (e.g., academic achievement) in a particular situation. Further, resilience is a multidimensional construct such that some children who are at high risk for maladaptation demonstrate competence in certain domains but not in others. Research has also revealed that although some individuals outwardly display resilience and competence in multiple domains, they experience internalizing symptoms (Luthar, 1993) or chronic health-related issues (Werner & Smith, 1992) indicating that resilience does not imply invulnerability.

Although this is an exciting and promising area of research and, in many ways, at the heart of the developmental psychopathology perspective, the current state of the field has been criticized for its definitional ambiguities, the heterogeneity of both the risk and competency factors, the instability of the resilience construct, and the overall utility of the concept of resilience (Luthar et al., 2000). Nevertheless, this line of inquiry has great potential for producing an increasingly in depth, complete understanding of adaptation (Panter-Brick & Leckman, 2013).

Cultural Issues Considering the emphasis in developmental psychopathology on contextual factors, the distinct role that culture plays in children's adaptation is receiving increasing attention. Research and practice must take into account the unique factors of children's cultural norms, socialization practices, and values when considering whether a particular behavior represents a maladaptive response to the dominant culture's demands (Abdullah & Brown, 2011). Given that the majority of developmental research has been conducted using Western, European-American, middle-class samples, it is important to recognize the dominant sociocultural perspective of this society in contrast to others. Mesquita and Markus (2004) have identified two distinct, prevalent cultural frameworks. The first model of agency termed "disjoint," is reflected in European American cultures with a focus on the self and the notion that the self should be independent, happy, and seek to control and influence the environment. The second model, termed "conjoint" is reflective of East Asian cultures in which the emphasis is on interdependence, belonging to social groups, and perception of the environment through the perspective of others. Thus, children developing within each of these cultures are likely to display different developmental trajectories and outcomes and the determination of whether an outcome is adaptive or maladaptive must be considered within the norms of the particular culture (see also Chapter 3 in this volume.)

In a cross-cultural comparison, Suveg et al. (2015) found that American children and their parents reported greater emotional expressiveness overall than did Chinese children and their parents. Further, family expression of positive emotion was positively related to emotion regulation for American children only, whereas family expression of negative emotion was positively related to under-controlled emotion regulation (externalizing types of regulation) for both American and Chinese children. A cultural context that has been rarely investigated is the emotional functioning of sub-Saharan African children. In a 2012 study, Morelen, Zeman, Perry-Parrish, and Anderson interviewed Ghanaian, Kenyan, and American children about their management of anger and sadness, and found an interesting pattern of both commonalities and differences across nationalities. Specifically, boys reported more control over sadness than girls with the opposite effect for anger regardless of country of origin.

Yet, American children reported more inhibition of sadness than the African youth with more anger expression by Ghanian than Kenyan and American youth. Collectively, such studies help to advance context-specific models of emotional development; an endeavor that exemplifies a developmental psychopathology approach.

From within a specific culture, researchers must also consider the role of subcultures that may place importance on values that potentially differ from those of the mainstream culture. For example, within the United States, social acceptance of boys in inner-city, high-crime areas is more likely to be based on aggressive behavior and low academic achievement rather than the typical profile of prosocial, competent behavior valued in middle-class America (Luthar & McMahon, 1996). Thus, examining the dynamic interplay of risk and protective factors in the development of disorder and positive adaptation must take into account the unique role of cultural factors when examining outcomes.

Conclusion

This chapter has examined the central tenets of the developmental psychopathology perspective and has highlighted its core principles with examples from research. The developmental psychopathology perspective is not a single theory, but rather an approach to the study of the intersection between adaptation and maladaptation that employs multiple levels of analyses to examine interacting and dynamic influences (i.e., genetic, physiological, environmental, contextual) on development. Not concerned with traditional diagnostic classification, the developmental psychopathology approach focuses on identifying processes that underlie pathways to adaptation and disorder and its related mediators and moderators. The role of cultural context in development is considered vitally important because it is essential for understanding the function, value, and appropriateness of a behavior. Further, by taking a process approach to understanding particular pathways to adaptation and disorder, specific targets can be identified for early prevention and intervention. Despite its clear contributions to our understanding of the implications of developmental deviations, challenges to the developmental psychopathology paradigm remain. For instance, because of its emphasis and interest in processes related to stability and change over time, expensive detailed longitudinal designs are needed to address these questions adequately. The statistical analyses of multiple interacting factors across different levels of analysis require large sample sizes, which can also be challenging to recruit and sustain over time, for a variety of reasons (e.g., resource availability). Further, multiple perspectives from varying fields (e.g., genetics, developmental psychology, sociology) provide the ideal approach to understanding psychosocial adaptation, yet the involvement, coordination and funding of a transdisciplinary teams and the integration of the resultant findings pose unique challenges. Nonetheless, the developmental psychopathology perspective offers a way of conceptualizing disorder based on developmental processes and pathways to adaptation with implications for prevention and intervention. This approach has appeal for a wide array of researchers and is likely to result in a more thorough, complete understanding of such phenomena than adopting simplistic approaches that focus primarily on observable behaviors.

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3

Psychopathology

A Neurobiological Perspective

Molly Nikolas, Kristian Markon, and Daniel Tranel

Introduction

Decades of work in psychological science and related disciplines have demonstrated that the brain is the foundation for human behavior. Additionally, the rise of behavioral and molecular genetic methodologies over the past 50 years has demonstrated that genetic factors play an important role in shaping brain development and, ultimately, personality and psychopathology. Advancements in non-invasive technologies have made the study of genes and brain functioning more accessible than ever, and treatments for psychopathology have been developed based upon this accumulating knowledge of neurobiological mechanisms. Understanding psychopathology must therefore undoubtedly include investigation of its biological bases, both genetic and neurobiological. This chapter introduces important concepts and issues regarding the biological bases of psychopathology, with a particular emphasis on genetic and neurobiological mechanisms and how discoveries in these areas hold great promise for the refinement of comprehensive etiologic models and treatment paradigms. We begin our chapter with an overview of fundamental concepts and topics in biological accounts of behavior, then discuss methods and methodological issues that are encountered in studying those biological substrates. Finally, we discuss the biological substrates of specific disorders and forms of psychopathology.

Neurobiological Foundations

An Introduction to the Human Brain and Neurotransmitters The human brain is extremely complex. It comprises something of the order of 50 to 100 billion neurons that each forms tens of thousands of connections to other neurons and exerts causal influences on behavior as well as changes in response to our experiences. We provide some basic information regarding brain structure and communication in Box 3.1 and Figure 3.1. This overview of brain structures and means of neuronal communication provides a primer of background knowledge of the biological bases of psychopathology. Needless to say, it is impossible to cover the basics of neuroanatomy, genetics, and biological psychology and findings specifically related to psychopathology in this chapter. We point interested readers to basic textbooks for more in-depth coverage of these concepts (Blumenfeld, 2010; Breedlove & Watson, 2013; Kandel, Schwartz, Jessell, Siegelbaum, & Hudspeth, 2012).

Box 3.1 Neurotransmitters and their Regulation

Coming in many forms, *neurotransmitters* are chemical messengers, mediating information transmission between neurons by passing from one neuron to act on *receptors* on another. Neurotransmitters are localized in different ways. Although many are found throughout the body, within the brain they may only be produced in specific regions; also, neurotransmitters often have different types of receptors, each of which is localized to specific brain regions. In this way, different neurotransmitters can have different patterns of behavioral associations, even as each serves multiple functions. Drugs often act through neurotransmitter pathways, such as by activating a receptor as an *agonist*, blocking receptors as an *antagonist*, inhibiting reuptake of the neurotransmitter into a neuron, affecting chemical synthesis or degradation, or some combination or variant of mechanisms.

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Acetylcholine is found throughout the brain and in muscle tissue. It mediates muscle contraction, and is involved in a number of cognitive processes, such as attention and memory. Acetylcholine acts on two different types of receptors, nicotinic acetylcholine receptors and muscarinic acetylcholine receptors. The nicotinic receptor is an *ionotropic* receptor; binding of acetylcholine to the receptor opens an ion channel that allows positive ions such as sodium and potassium to pass through. The muscarinic receptor is a *metabotropic* receptor; binding of acetylcholine to the receptor causes changes in associated proteins, which eventually cause other ion channels in the neural membrane to open and allow ions to pass through.

Dopamine is involved in numerous processes, especially reward and reinforcement, motor function, and cognition and attention. It is synthesized from the amino acid tyrosine, and is processed by enzymes (e.g., catechol-O-methyltransferase) to form other neurotransmitters in its family (i.e., the catecholamines). Many drugs of abuse directly or indirectly target dopamine pathways in various ways, and classic antipsychotic medications act as dopamine antagonists.

Epinephrine and **norepinephrine**, also known as adrenaline and noradrenaline, are examples of catecholamines formed from dopamine (norepinephrine from dopamine, and epinephrine from norepinephrine). They both have a wide variety of functions, and both are involved in fight-or-flight and fear responses, in part due to hypothalamopituitary axis sympathetic nervous system activity, as well as arousal and alertness. Norepinephrine pathways are also associated with decision making, and its reuptake is a mechanism of action of popular antidepressant and other psychotropic medications.

GABA, or gamma-aminobutyric acid, is the primary inhibitory neurotransmitter in the adult brain, in that it inhibits neural firing (note that inhibiting an inhibitory neuron could facilitate downstream neural responses). Very early in development, however, it has excitatory effects; it comes to act as an inhibitory neurotransmitter early during the postnatal period. Like acetylcholine, GABA binds to one of two types of receptors, an ionotropic and a metabotropic receptor. Many depressant substances (e.g., benzodiazepines and alcohol) act at least in part by facilitating GABA receptor activity.

Glutamate is the primary excitatory neurotransmitter in the adult brain, and the precursor to GABA. Like acetylcholine and GABA, there are ionotropic and metabotropic forms of glutamate receptors. Given its widespread distribution in the brain, glutamate functions in a wide variety of roles, and has been linked to a wide variety of mental disorders. Overexcitation of glutmate receptors, which sometimes occurs in traumatic brain injury or neurological disease, can lead to neurodegeneration or neuron death.

Serotonin is derived from the amino acid tryptophan, and also has a wide variety of roles in the brain and elsewhere. Serotonin has long been associated with behavioral and emotional regulation, with many antidepressants acting by preventing its elimination, either by inhibiting its reuptake, or by preventing its degradation. Many hallucinogenic substances also act via serotonin receptors, such as by acting as a serotonin agonist.

Neural Systems for Emotion and Feeling At the root of many forms of psychopathology are disturbances of *emotion*, including defects in perception, processing, expression, and regulation of emotion, as well as defects in the related capacity of *feelings* (the conscious processing and experience of emotions). Neurobiological systems for emotion have been elucidated by a long tradition of conventional neuropsychological studies and, more recently, by functional imaging approaches (especially functional magnetic resonance imaging, or fMRI). This work has pointed to a number of key structures and networks that are closely linked to emotions and feelings. Specifically, research has identified brainstem and hypothalamic nuclei, amygdala, insula cortex, anterior cingulate cortex, ventromedial prefrontal cortex, and somatosensory cortices, among others, as important for various aspects of emotion (Barrett, Mesquita, Ochsner, & Gross, 2007; Craig, 2002, 2008; Damasio, 1994; Damasio et al., 2000; Damasio, A. R., Damasio, H., & Tranel, 2013; Davidson & Irwin 1999; Lane, 2000; Lane et al., 1998; Tye et al., 2011; Wager, Phan, Liberzon, & Taylor, 2003). Some studies have pointed to focal brain regions associated with specific basic emotions (e.g., Vytal & Hamann, 2010), while others have reported a more distributed mapping between brain regions and emotions (e.g., Lindquist et al., 2012). Lesion and electrical stimulation studies have provided evidence for often quite specific associations between particular brain regions and particular emotions—e.g., specific brain regions for fear, or for sadness, or for disgust (Calder, Keane, Manes, Antoun, & Young, 2000; Dejjani et al., 1999; Feinstein, Adolphs, Damasio, & Tranel, 2011; Feinstein et al., 2013; Fried, Wilson, MacDonald, & Behnke, 1998). Also, for many of the bilateral cortical

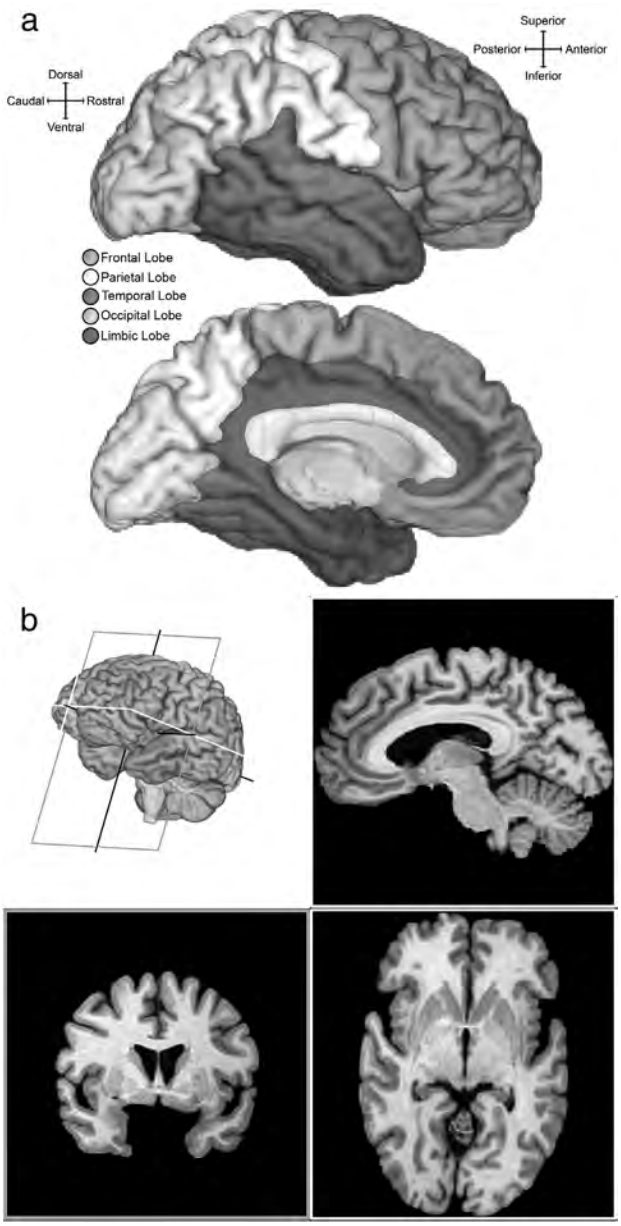

Figure 3.1 Human brain, with covering layers (skull, meninges) stripped to show cortical gyri and sulci of the cerebral hemispheres: (a) lateral (top) and medial (bottom) views of the cerebral hemispheres, colored according to key to show the five major lobes. Spatial axes labeled with conventional terms are shown to the left and right of the lateral view. (b) Upper left is a three-dimensional reconstruction of a normal brain (from a structural magnetic resonance scan), showing standard orthogonal planes of section in the sagittal (red box, upper right), coronal (blue box, lower left), and horizontal (or transverse, green box, lower right) dimensions. The idea for Figure 3.1b was suggested by Hanna Damasio (2000). This figure is reproduced in the color plate section.

structures, the right-hemisphere component has been shown to be particularly important (more so than the left-sided counterpart). We review below some of the principal emotion-related structures, along with examples of relevant empirical evidence and links to psychopathology.

Right-Hemisphere Structures Right-hemisphere structures have long been considered important for emotions and feelings. Studies in behavioral neurology and neuropsychology, for example, have consistently supported the conclusion that for emotion processing, the right hemisphere leads the way, and the left hemisphere is less important (e.g., LaBar & LeDoux, 2003).[1] This may reflect some of the underlying hemispheric specializations associated with the right hemisphere, such as preferences for holistic, configural modes of processing that could facilitate handling of multidimensional and alogical stimuli that convey emotional tone. Facial expressions and prosody (the stress, intonation, and rhythm of speech that help convey emotional "coloring" in verbal utterances) are two illustrative examples of such stimuli (e.g., Borod, Haywood, & Koff, 1997). More generally and relevant to many forms of psychopathology, it has been proposed that the right hemisphere contains structures important for emotional processing which may have evolved to provide the neural machinery that operates aspects of social cognition (Bowers, Bauer, & Heilman, 1993; Gainotti, 2000; Heilman, Blonder, Bowers, & Valenstein, 2003).

Much of the specific evidence supporting these conclusions comes from neuropsychological studies. For example, it has been shown that lesions to right temporal and parietal cortices can impair emotional experience and arousal, as well as imagery for emotion (see Adolphs & Tranel, 2004). Neurological patients with right temporal and parietal damage often have difficulty discerning the emotional features of stimuli, with corresponding diminution in their emotional responsiveness, and this can occur for both visual and auditory stimuli (Borod et al., 1998; Van Lancker & Sidtis, 1992). Functional neuroimaging studies have also shown a preferential role of right-hemisphere structures in emotion recognition from facial expressions and from prosody (for a review, see Cabeza & Nyberg, 2000).

There have been two basic hypotheses regarding how and the extent to which the right hemisphere participates in emotion. The right-hemisphere hypothesis posits that the right hemisphere is specialized for processing all emotions. The valence hypothesis posits that the right hemisphere is specialized only for processing emotions of negative valence, whereas positive emotions are processed preferentially by the left hemisphere (Canli, 1999; Davidson, 1992, 2004). Both hypotheses have garnered some empirical support (e.g., Jansari, Tranel, & Adolphs, 2000), and resolution of this debate may require more precise specification of which components of emotion are under consideration. One distinction that seems to be important is the difference between recognition versus experience. Recognition of emotion (e.g., identifying emotions in external stimuli such as facial expressions and prosody) may accord more with the right-hemisphere hypothesis, while experience of emotion (e.g., arousal and feelings) may accord more with the valence hypothesis. For example, lesions to the right somatosensory cortex

have been associated with impaired visual recognition of emotional facial expressions, covering most primary emotions (e.g., sadness, fear, anger, surprise, disgust; however, visual recognition of happy facial expressions was not impaired by right somatosensory lesions), broadly consistent with the right-hemisphere hypothesis (Adolphs, H. Damasio, Tranel, Cooper, & A. Damasio, 2000). By contrast, studies of emotional experience have shown a lateralized pattern more consistent with the valence hypothesis. A well-established theory by Davidson (1992, 2004) posits an approach/withdrawal dimension, in which increased right-hemisphere activation correlates with increases in withdrawal behavior (including behaviors characteristic of emotions such as fear or sadness), and increased left-hemisphere activation correlates with increases in approach behaviors (including behaviors characteristic of emotions such as happiness and amusement).

Amygdala The amygdala is a key component in the so-called "limbic system" (Figure 3.2). The limbic system has long been connected to emotion-related functions, going back to the seminal formulations of Papez (1937) and MacLean (1952). The term has not been without controversy, and some scientists have underscored the fact that there is not even a consensus on exactly what structures do and do not belong to the limbic system (LeDoux, 2000). Nonetheless, the concept has remained useful, at least as a heuristic (see Feinstein et al., 2010), and the amygdala is very much at the center of the action insofar as emotional functions are concerned. In fact, the past couple of decades of work in cognitive neuroscience have propelled the amygdala to a place of notable importance in the neuroanatomy of emotion.

The amygdala is a bilateral structure composed of a collection of nuclei deep in the anterior temporal lobe. It

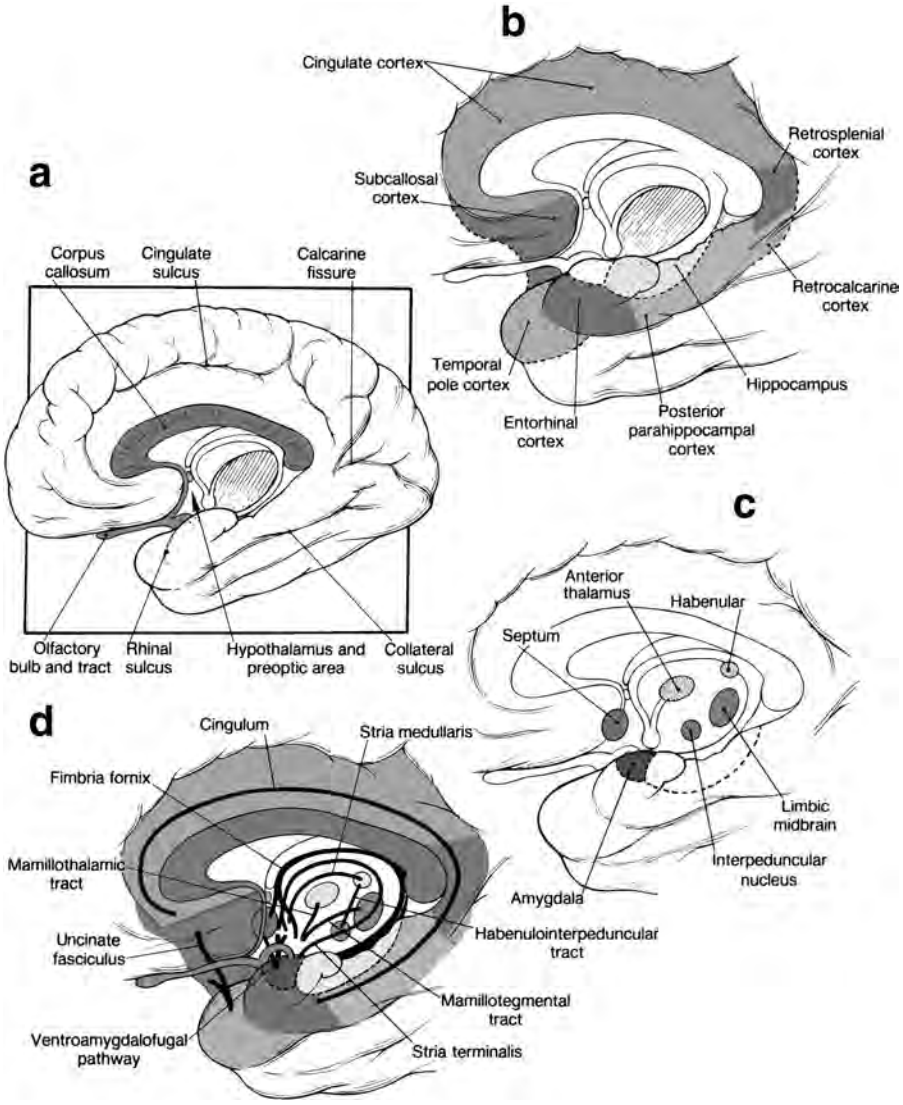

Figure 3.2 Limbic system, showing major structures, mostly on and near the midline, that comprise the interconnected cortical and subcortical components of the human limbic system: (a) Major landmarks; (b) major cortical components of the "limbic lobe"; (c) major subcortical components; (d) major pathways between limbic system components. The idea for this figure was suggested by Gary Van Hoesen (see Damasio, Van Hoesen, & Tranel, 1998). This figure is reproduced in the color plate section.

receives highly processed input from most sensory modalities, and in turn has extensive, reciprocal connections with many brain structures that are important for various aspects of emotion-related processing (Amaral, Price, Pitkanen, & Carmichael, 1992). Of particular importance, the amygdala has extensive bidirectional connections with the orbitofrontal cortices, known to be important for emotion and decision making (Bechara, H. Damasio, Tranel, & A. Damasio, 1997). The amygdala is also extensively and bidirectionally connected with the hippocampus, basal ganglia, and basal forebrain, key structures for memory and attention. The amygdala projects to the hypothalamus and other structures that are involved in controlling homeostasis, as well as visceral and neuroendocrine output. The amygdala is thus well-situated to link information about external stimuli conveyed from sensory cortices with modulation of decision making, memory, and attention, as well as somatic, visceral, and endocrine processes (see Adolphs and Tranel, 2004).

The amygdala makes critical contributions to a diverse array of emotional and social behaviors. Adolphs and Tranel (2004; see also Damasio, 1999) outlined three general principles:

1. The amygdala links perception of stimuli to an emotional response, using afferents from sensory cortices and efferents to emotion control structures such as hypothalamus, brainstem nuclei, and periaqueductal gray matter.[2]
2. The amygdala links perception of stimuli to modulation of cognition, based on bidirectional connections with structures involved in decision making, memory, and attention.
3. The amygdala links early perceptual processing of stimuli with modulation of such perception via direct feedback (e.g., Gallegos & Tranel, 2005). These various mechanisms allow the amygdala to contribute critically to emotion processing by modulating multiple processes simultaneously.

A number of psychopathological processes and psychiatric illnesses have been linked to pathology in the amygdala. For example, autism, post-traumatic stress disorder, generalized anxiety disorders, phobias, and schizophrenia have all been linked to amygdala pathology (e.g., Aggleton 2000). There is also evidence of amygdala dysfunction in mood disorders (Drevets, 2000; Davidson & Irwin, 1999). Histological (e.g., analyses of cell densities and neuronal arrangements) and volumetric (e.g., analyses of the volumes of gray and white matter structures) magnetic resonance imaging (MRI) studies have found abnormal amygdala cell density throughout development in individuals with autism and autism spectrum disorders, and fMRI work has shown abnormal amygdala activation in persons with autism, especially during emotion-related tasks (Baron-Cohen et al., 2000). Although surrounded by lively scientific debate, an "amygdala theory of autism" has remained a popular and largely valid heuristic for understanding the neurobiology of autism and related disorders in which emotional and social processing are notably disturbed.

Studies using neuropsychological and functional neuroimaging approaches have provided compelling evidence that the amygdala is involved in processing emotion via all principal sensory modalities, including visual, auditory, somatosensory, olfactory, and gustatory. Extensive work in animals has shown that the amygdala is important for fear conditioning (e.g., Davis, Walker, & Lee, 1997; LeDoux, 1996); and in humans, lesions to the amygdala impair the ability to acquire conditioned autonomic responses to stimuli that have been paired with unconditioned aversive stimuli (Bechara et al., 1995). In parallel, the acquisition of conditioned fear responses has been shown to activate the amygdala in functional imaging studies (Buechel, Morris, Dolan, & Friston, 1998).

Many neuropsychological investigations have pointed to a key role for the amygdala in the *recognition* of emotion from various types of stimuli (e.g., facial emotional expressions), as well as the *experience* of emotion triggered by emotional stimuli or emotional memories. For example, patients with focal, bilateral amygdala damage have been shown to be specifically and severely impaired in recognizing fear in facial expressions (Adolphs & Tranel, 2000). A similar impairment has been shown for anger and other highly arousing emotions similar to fear, which is consistent with the notion of a more general impairment in recognition of negative emotions in patients with bilateral amygdala damage (Adolphs et al., 1999). Functional neuroimaging studies have corroborated much of the lesion work, showing, for example, that the amygdala is strongly activated by tasks that require the recognition of signals of unpleasant and arousing emotions (Morris et al., 1996). In fact, visual, auditory, olfactory and gustatory stimuli have all been reported to activate the amygdala during processing of unpleasant and arousing emotions (Royet et al., 2000). Data such as these have led to the suggestion that the amygdala may play a role in recognizing highly arousing, unpleasant emotions (emotions that signal potential harm), and in the rapid triggering of appropriate physiological states related to these stimuli (Adolphs & Tranel, 2004). This mechanism may operate in a bottom-up, automatic fashion that can be below the level of conscious awareness. For example, Whalen et al. (1998) reported amygdala activation in participants who were shown facial expressions of fear that were presented so briefly the expressions could not be consciously recognized.

More generally, the amygdala appears to participate in the allocation of processing resources and the triggering of responses to stimuli that signal threat or are otherwise of particular and immediate importance or relevance to the organism (LeDoux, 1996). For example, Bechara (2004) has suggested that pleasant or aversive stimuli, such as

encountering a fear object (e.g., a snake), trigger quick, automatic, and obligatory affective/emotional responses via the amygdala. In addition, there is physiological evidence that responses triggered through the amygdala are short-lived and habituate quickly (Buchel et al., 1998). The amygdala provides a mechanism to link the features of external stimuli to emotional responses, which are evoked via visceral motor structures such as the hypothalamus and autonomic brainstem nuclei that produce changes in internal milieu and visceral structures, as well as behavior-related structures such as the striatum, periacqueductal gray (PAG), and other brainstem nuclei. The powerful and seemingly automatic nature of emotional responses triggered by the amygdala has important implications for psychopathology, including common psychiatric conditions such as post-traumatic stress disorder, phobias, and generalized anxiety disorders.

Ventromedial Prefrontal Cortex Given the extensive interconnections between the amygdala and orbitofrontal (or ventromedial prefrontal, more generally)[3] cortex, it is not surprising that lesions to the orbitofrontal cortex can produce emotion processing defects that are reminiscent of those associated with amygdala damage. Moreover, neuronal responses in the orbitofrontal cortex appear to be modulated by the emotional significance of stimuli (e.g., reward or punishment contingencies) in much the same way as such responses are modulated in the amygdala (e.g., Kawasaki et al., 2001; Rolls, 2000). Disconnecting the amygdala and orbitofrontal cortex can produce emotional impairments similar to those following lesions of either structure. In sum, there is considerable evidence that these two structures function as key components of a richly connected emotion processing network.

The medial part of the orbitofrontal cortex is part of a more extensive functional neuroanatomical system that has been termed the ventromedial prefrontal cortex (vmPFC; see footnote 1), and there is extensive evidence that the vmPFC plays a critical role in many aspects of higher-order emotional processing (Figure 3.3). For example, numerous neuropsychological and functional imaging investigations have documented that the vmPFC is crucial for moral reasoning, social conduct, and decision making, especially decision making that involves emotional and social situations (see Tranel, Bechara, & Damasio, 2000, for review). Damage to the vmPFC leads to poor judgment, bad decision making, and impaired emotions and feelings. Some of the most prominent defects are in the social realm, including social perception and social emotions (e.g., empathy, regret; see Damasio, Anderson, & Tranel, 2012). Patients with damage to the vmPFC cannot represent choice bias in the form of an emotional "hunch" (Bechara et al., 1997), and cannot trigger normal emotional responses to emotionally-charged and socially relevant stimuli (Damasio, A., Tranel, & Damasio, H., 1990).

Basal Ganglia The basal ganglia, especially on the right, also play an important role in emotion. It has been shown

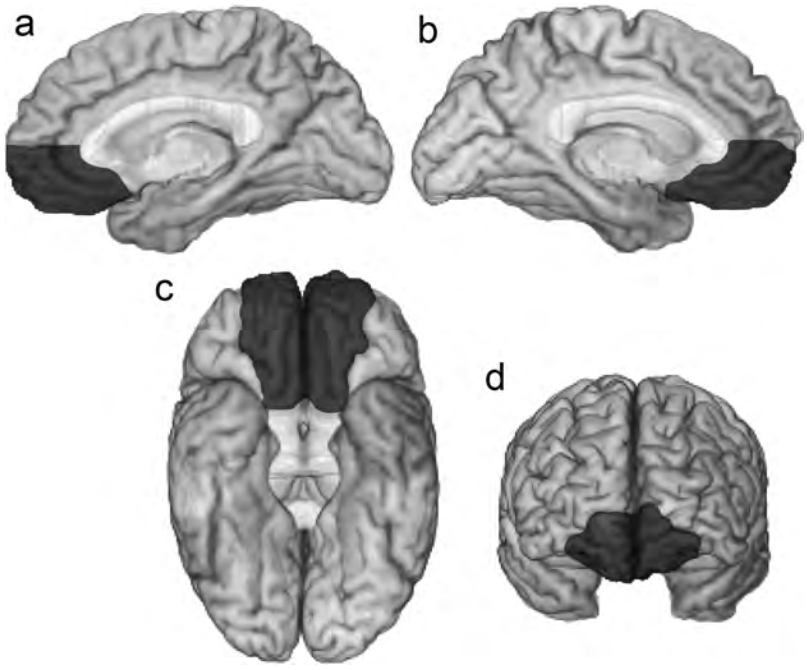

Figure 3.3 Ventromedial prefrontal cortex (vmPFC). The region of the prefrontal lobes (termed the "ventromedial prefrontal cortex") is marked in green on (a) mid-sagittal right hemisphere, (b) mid-sagittal left hemisphere, (c) ventral, and (d) frontal views. The vmPFC encompasses the medial part of the orbitofrontal cortex and the ventral sector of the mesial prefrontal cortex. This figure is reproduced in the color plate section.

that damage to the basal ganglia can produce impaired recognition of emotion from a variety of stimuli (Cancelliere & Kertesz, 1990). The right basal ganglia are activated by tasks requiring the processing of facial emotional expressions (Morris et al., 1996). Also, diseases that preferentially damage certain sectors of the basal ganglia, including obsessive-compulsive disorder, Parkinson's disease, and Huntington's disease, are marked by disturbances of emotions and feelings. For example, it has been shown that patients with obsessive-compulsive disorder have abnormal feelings of disgust and are impaired in the recognition of facial expressions of disgust (Sprengelmeyer et al., 1997). A hallmark sign in Parkinson's disease is impaired emotional expression, and patients with Parkinson's disease often have impaired feelings of emotions and impaired recognition of emotion. Studies have shown that patients with Huntington's disease have a selective impairment in recognizing disgust from facial expressions—that is, the patients have more difficulty recognizing disgust, compared with other facial emotions (Jacobs, Shuren, & Heilman, 1995; Sprengelmeyer et al., 1996).

Other Structures Several other structures are especially important to link stimulus perception to emotional response. The bed nucleus of the stria terminalis appears to have a role in anxiety, via the neuropeptide corticotropin-releasing factor. The substantia innominata and nuclei in the septum are important for emotional processing, and may mediate their effects through the neurotransmitter acetylcholine. A collection of nuclei in the brainstem (especially the locus ceruleus and Raphe nuclei) provide neuromodulatory functions that are critical for emotions and feelings. The ventral striatum is another important region. For example, the nucleus accumbens appears to be important for processing rewarding stimuli, and for causing behaviors that prompt an organism to seek stimuli that predict reward. The periaqueductal gray in the midbrain is also important, and stimulation in this area can produce panic-like behavioral and autonomic changes, as well as reports of panic-like feelings (Panksepp, 1998).

Genes and Behavior Enormous strides have been made over the past two decades regarding our understanding of the role of genetic processes in psychopathology. Reaction to reports regarding potential genetic impacts on complex behaviors (for example, findings of genetic influence on divorce reported by McGue & Lykken, 1992), however, have somewhat altered (or perhaps simplified) general understanding regarding the role of genes in the development of psychopathology. While there is clear evidence that genetic factors play important roles in the development of nearly every psychological disorder (Burt, 2009; Krueger et al., 2002), genes do *not* directly code for specific types of behavior—especially the complex behavior that is disordered in psychopathology (i.e., there is no "gene for schizophrenia"). Rather, genes encode proteins, and genetic variation likely impacts the development of psychopathology via its "upstream" consequences on protein formation and expression, which then have cascading effects on neurodevelopment and neurotransmission. Alterations or dysfunction in neurodevelopment may then be exhibited behaviorally via the manifestation of symptoms of psychopathology.

A Primer on Genetics What are genes and how might they impact psychopathology? At the most basic level, genes are long molecules of deoxyribonucleic acid (DNA), which are located at various positions within paired chromosomes. Humans have 23 paired chromosomes (46 total), which are contained within the cell nucleus; individuals inherit one pair of chromosomes from each of their parents. Chromosomes are numbered from 1 to 22 (with 1 being the longest and 22 being the shortest, referred to as autosomal); genes on these chromosomes encode proteins involved in the development of the body and brain. The 23rd pair of chromosomes determines an individual's sex; individuals with two copies of the X chromosome are genetically female; those with one X and one Y chromosome are genetically male.

Abnormalities in chromosomal division and inheritance have been linked to numerous disorders of health and development—perhaps the most notable example is Down syndrome. Also referred to as trisomy 22, Down syndrome occurs as the result of problematic division of the chromosomes during meiosis in the egg of the mother (termed chromosomal non-disjunction), such that an additional copy (or portion) of chromosome 22 is passed on to the child. The presence of this additional portion of the chromosome then results in changes in facial characteristics (e.g., flattened nasal bridge, small eyes) and intellectual impairments. Several chromosomal abnormalities have been linked to behavioral disorders; for example, William's syndrome is the result of deletion of approximately 26 genes in chromosome 7 and results in both physical changes (heart defects, flattened nasal bridge), as well as anxiety, attention problems, and intellectual disability (although individuals with William's syndrome may be highly verbal relative to their overall IQ, see Lashkari, Smith, & Graham, 1999). Similarly, 22q deletion syndrome, which involves a deletion of genes from the long arm of chromosome 22, has also been associated with numerous cognitive delays during childhood, as well as the development of psychosis during adolescence (Bassett & Chow, 2008). Importantly, however, the majority of psychological disorders are not the result of dysfunction or alteration of a single chromosome (or even a single gene). Rather, psychopathology likely results, in part, from numerous contributions from multiple genes. The term polygenic is used to describe most psychological disorders, which reflects the notion that many different genes likely each contribute a small portion to their etiology.

The total length of the human genome (all human DNA) is around three billion base pairs, which are estimated to contain approximately 20,000 different genes (U.S. Department of Energy Office of Science, 2009). An individual's DNA is contained with the cell nucleus of every cell in the human body. Importantly, however, not all base pairs are different across people; in fact, over 99% of base pairs do not vary across people at all, and likely code for proteins involved in the basic "construction" of a human being (i.e., proteins that create heart, skin, hair cells, or that provide information for cells to combine to create nose, hands, and feet, etc.). Only a small proportion of the DNA code is *polymorphic*; that is, only certain regions actually vary from person to person. This variation occurs because an individual inherits one set of code from their mother and the other set from their father, resulting in two *alleles* at each genetic locus. These alternate forms of the gene can be associated with phenotypic (or observable) differences. Classic Mendelian inheritance specified that each individual allele at a particular locus corresponded with a particular phenotype, or observable trait. Further, alleles could be dominant or recessive, such that dominant alleles would always be expressed over recessive alleles. Importantly, much of this theory was based on Mendel's experiments with peapod phenotypes, including pod color, shape, and height, among other physical characteristics, (see Fisher, 1936, for a discussion of Mendel's work), which may not apply to the inheritance of complex traits in humans. For decades, scientists applied the concepts of Mendelian inheritance to human phenotypes, such as eye color, positing that the allele for brown eyes was dominant over the alleles for blue eyes and green eyes. However, genetic investigations of eye color over the last several decades have indicated that it does not likely follow Mendelian patterns of inheritance and that approximately 16 different genes contribute to human eye color (White & Rabago-Smith, 2011).

While Mendelian models of inheritance are no doubt important for understanding genetic recombination and variation throughout the genome, it does not appear that psychopathology is inherited in this classical Mendelian sense. Rather, it appears that most psychological disorders are polygenic, meaning that many polymorphic variations at different regions across the genome each contribute a small portion to the etiology of psychopathology. The notion that behavioral traits and disorders have a genetic contribution has long been recognized and established by the field of behavior genetics. While work is currently underway to specify the exact nature of the genetic differences that may increase liability for psychopathology (see Methods of Gene Discovery, in this chapter, p. 41), the magnitude of genetic influence on psychological disorders has been consistently established via the use of twin and family studies, which we turn to next.

Twin and Family Studies The first evidence of a genetic contribution to psychopathology came largely from family studies. In these studies, the families of individuals with various types of disorders were studied to determine if the disorder or symptoms "ran in families." Family studies of psychopathology were conducted as early as the latter half of the 19th century (although some were motivated by eugenic ideology, which is *not* a current motivator of contemporary twin and family work). In general, family studies have shown that rates of psychological disorders (e.g., schizophrenia, mood disorders, attention-deficit hyperactivity disorder, ADHD) among family members of affected individuals tend to be higher than the prevalence rates in the general population (Biederman et al., 1992; Kendler, Karwoski-Shuman, & Walsh, 1996; Tienari, 1991). However, these family studies do not point specifically toward a genetic contribution to psychopathology. The advent of the classical twin design has since provided researchers with tools to quantify the magnitude of genetic influence on traits and behaviors.

The classical twin design uses samples of monozygotic (MZ) and dizygotic (DZ) twins to infer genetic influence, owing to some very basic differences regarding the degree of genetic relatedness among twins. MZ twins are the result of a single fertilized embryo that splits into two (for reasons still unknown); thus, MZ twins share 100% of their genes. In contrast, DZ twins are the result of two fertilized embryos and share, on average, 50% of their genes (as do all full biological siblings). Both MZ and DZ twins (unlike full biological siblings) also share a common prenatal environment. Similarities in a particular behavior, trait, or disorder among MZ twin pairs can then be compared with similarities among DZ twin pairs, in order to gauge whether or not there may be a potential role of genetic influences. This work began by examining concordance rates (described briefly below), but now uses biometric model fitting to quantify the proportion of variability due to genetic factors, (see Plomin, DeFries, McClearn, & McGuffin, 2008, for a review).

Initial twin studies quantified the genetic influence on psychological disorders via examination of twin concordance rates. That is, the relative difference in concordance rates between MZ and DZ twins is informative when inferring a genetic influence on a disorder or trait (e.g., depression). Higher concordance rates for MZ relative to DZ twins would suggest higher genetic influences, given that MZ twins share 100% of their genes, whereas DZ twins share 50% on average. Inherent in this framework is the equal environments assumption, which posits that twins reared together in the same family experience common *shared environmental factors* (i.e., influences that are shared among reared-together siblings and serve to increase sibling similarity) at 100%, regardless of twin zygosity. Furthermore, *non-shared or unique environmental factors* (those that are not common among twins in the same family and serve to decrease twin/sibling similarity) are by definition, shared at 0% for all twins, regardless of zygosity. Using this framework, twin concordance rates have been found to be substantially higher for MZ versus DZ twins for a variety of disorders,

including schizophrenia (Picchioni et al., 2010), mood disorders (Edvardsen et al., 2009; Kieseppa, Partonen, Haukka, Kaprio, & Lonngvist, 2004), ADHD (Sherman, McGue, & Iacono, 1997), anxiety disorders (Skre, Onstad, Torgersen, Lygren, & Kringlen, 1993), and autism spectrum disorders (Rosenberg et al., 2009).

However, these studies of overall twin concordance rates can be problematic, given that genes likely influence an underlying continuous liability for developing a psychological disorder and may vary dramatically depending on sample source (Walker, Downey, & Caspi, 1991). For example, concordance rates appeared to vary when examining twins drawn from clinical settings versus twins selected to represent the larger population, with the former considered to generate potential overestimates of genetic effects. Thus, twin methodology that can quantify the magnitude of genetic (and environmental) influences on psychopathology-relevant dimensions in the population can then provide more precise (and potentially less biased) estimates of the magnitude of genetic influence. This has been achieved via development of the biometric twin model (Plomin et al., 2008). Biometric twin models also make use of the difference in the proportion of genes shared between reared-together MZ and DZ twins. Using these differences, the variance within observed behaviors is partitioned into three components: additive genetic, shared environment, and non-shared environment plus measurement error. The additive genetic component (A) includes the effect of individual genes at different locations on the chromosome (or loci) summed together. The shared environment (C) is that part of the environment common to siblings that serves to increase sibling similarity regardless of the proportion of genes shared. The non-shared environment (E) encompasses environmental factors (and measurement error) differentiating twins within a pair (i.e., those effects that decrease twin correlations regardless of genetic relatedness). Non-additive genetic effects can also be estimated in biometric twin models. These effects represent the multiplicative effects of genes at different loci.

The proportion of the variability in psychological symptoms or traits that can be attributed to genetic variability is referred to as *heritability*. Ranging from 0 to 100%, this term provides an estimate of the magnitude of genetic influence on population-level individual differences in psychopathology. Multiple meta-analyses and reviews on heritability for psychological disorders have been conducted and the results are summarized in Figure 3.4. As can be seen there, the relative impact of genetic influences is high for several disorders and traits, including psychosis, mania, autism spectrum traits (i.e., social communication deficits), and inattention and hyperactivity–impulsivity (Cardno & Gottesman, 2000; Edvardsen et al., 2008; Nikolas & Burt, 2010; Ronald & Hoekstra, 2011). Moderate heritability (estimates of around 50%) have been found for several disorders (eating disorders,

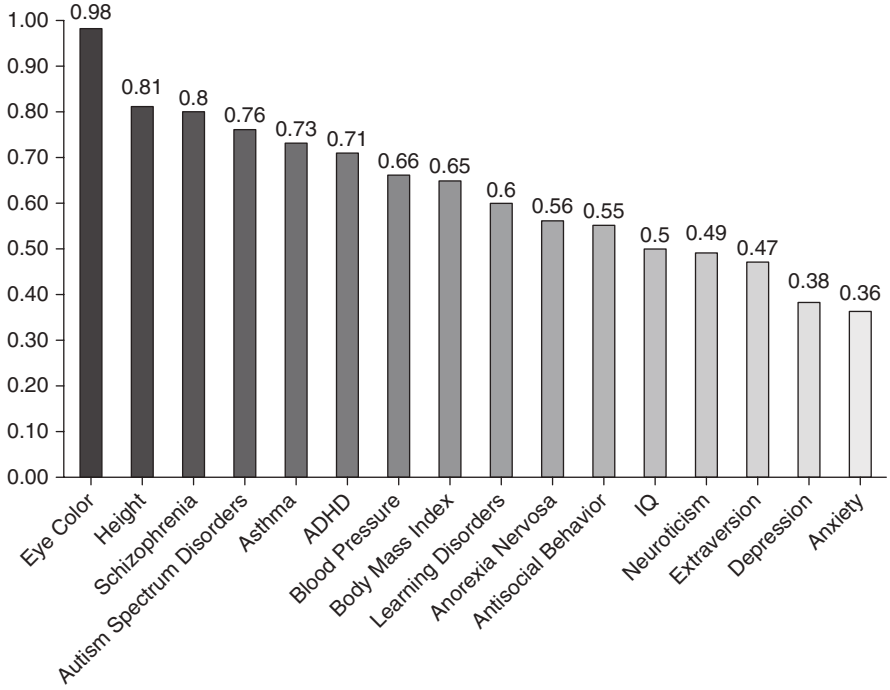

Figure 3.4 Heritability of physical traits and psychopathology. Heritability estimates for physical traits, dimensions of psychopathology, IQ, and personality traits are depicted, in descending order. These percentages refer to the proportion of variation in these traits in the population (e.g., height, eye color, IQ, schizophrenia) that is the result of genetic factors. Genetic influences are variable, but, importantly, are sizable and significant for nearly all forms of psychopathology and are equal to, and in some cases, greater than genetic contributions to common physical traits.

conduct disorders, substance-related disorders, personality disorders), as well as for measures of IQ and personality traits (Bouchard, 2013; Bulik et al., 2006; Gelhorn et al., 2005; Kendler, Maes, Sundquist, Ohlsson, & Sundquist, 2014; Pincombe, Luciano, Martin, & Wright, 2007; Wray, Birley, Sullivan, Visscher, & Martin, 2007). Somewhat lower, albeit sizable and significant, heritabilities have been reported for both anxiety and depression (Mosing et al., 2009).

Overall, twin studies have been critical in determining the overall magnitude of genetic influence on behavior. However, heritability is a population statistic that is sensitive to time, age, and context, and does not tell us anything about what specific genetic variants may be implicated in the causal processes underlying the development of psychopathology. Nonetheless, future twin work remains important for understanding factors that may cause alterations in genetic influence both within and across time. For example, Turkheimer and colleagues (2003) found that genetic influences on IQ appeared to change at different levels of socioeconomic status, such that genetic influences on IQ were *higher* for children in higher socioeconomic status groups. Thus, twin and family studies will likely remain integral to future work examining the complex interplay between genetic and environmental factors in the etiology of psychopathology.

Building from this background on neuroanatomy, neurotransmission, and genetics, we turn next to more specific discussion regarding the methods and approaches used to elucidate these biological contributors to psychopathology.

Methods and Approaches

Lesion Method

Background Neuropsychological approaches to the study of personality and psychopathology have long been a mainstay of the field. The logic is simple and straightforward, and entails observing systematic changes in personality that develop reliably after damage to specific brain structures. This approach—also called the "lesion method"—remains a fundamental and indispensable scientific approach to the study of brain–behavior relationships and psychopathology (e.g., Chatterjee, 2005; Fellows et al., 2005; Koenigs, Tranel, & Damasio, 2007; Rorden & Karnath, 2004). Historically, lesion studies provided the first sources of evidence for specific brain–behavior relationships regarding personality and psychopathology (e.g., the famous case of Phineas Gage; Harlow, 1868), later validated with converging evidence from other methods.

Using the lesion method, researchers can explore the association between focal damage to a particular brain region and impairment in a clearly defined psychological function. If damage to a particular brain region results in impairment to a particular psychological function, it is concluded that the brain region is *necessary* (albeit not necessarily *sufficient*) for that function (H. Damasio & A. Damasio, 2003). The lesion method is used in human participants who have incurred focal brain damage due to specific kinds of disease or injury (e.g., cerebrovascular disease, surgical treatment of epilepsy, tumor resection, focal trauma or infection). Such "naturally occurring" lesions do not affect all brain regions equally, as different types of brain insults tend to produce damage preferentially in certain areas of the brain. For example, herpes simplex encephalitis tends to affect limbic system structures; surgical treatment of epilepsy typically involves the anterior-mesial temporal lobes; and cerebrovascular accidents more commonly affect the perisylvian regions fed by the middle cerebral artery.

Limitations of the Lesion Method There are limitations to the lesion method, deriving from both issues of practicality as well as functional resolution. One practical limitation concerns access to relevant patients. The lesion method is frequently conducted within a medical center, because scientists need access to neurological patients, and this often requires collaborations with medical specialists including neurologists and neurosurgeons. Assembling reasonably sized groups of patients requires reliable referral pipelines of suitable patients, and scientists depend on medical specialists for such referrals. The lesion method also has limited functional resolution, owing, in turn, to limitations in both spatial and temporal resolution (see Huettel, Song, & McCarthy, 2004). Even relatively small, focal lesions may not affect a single functional unit of the brain—and in many instances, lesions affect brain tissue that is involved in multiple functions. Another challenge arises from the fact that higher-order functions typically depend on more than one brain area—and this is likely even more true of functions in the domain of personality. Accordingly, damage to a single brain area may not completely impair a function, because the patient can compensate using other brain structures in the functional system. Another limitation is that researchers obviously do not make lesions experimentally in humans (as is the case in animal research). Thus, researchers are dependent on "naturally occurring" lesions (stroke, medically indicated resections, etc., as outlined earlier). Such lesions do not occur in all areas of the brain with equal frequency, and they vary considerably in size and involve different proportions of gray and white matter (Figure 3.5).

Some of these limitations can be overcome with lesion overlap and subtraction approaches, or voxel-wise lesion symptom mapping approaches, which yield more precision in mapping lesion-deficit relationships (Rorden & Karnath, 2004; Rudrauf et al., 2008).

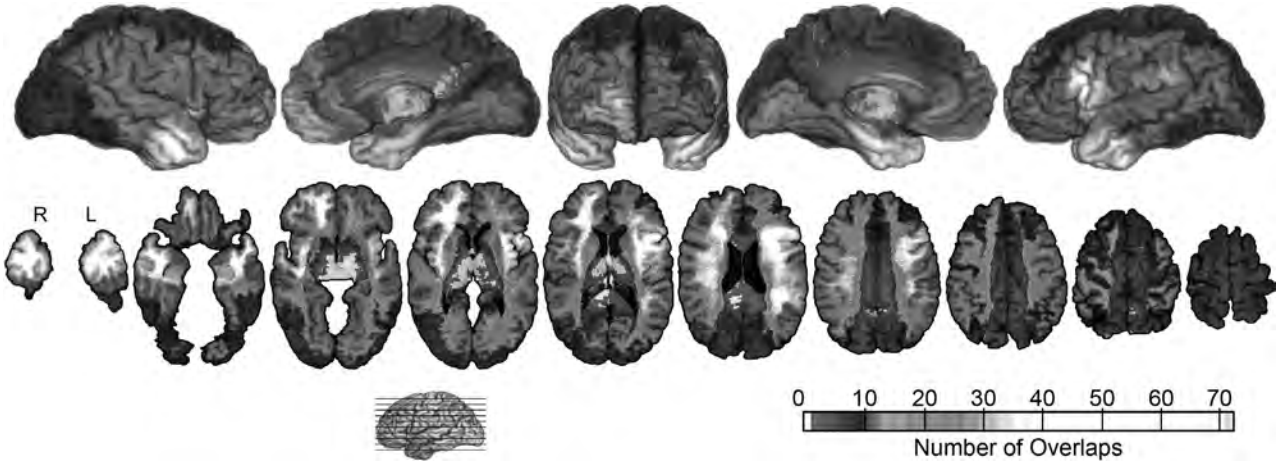

Figure 3.5 Lesion coverage map, showing overlaps of lesions that are represented in the Iowa Neurological Patient Registry. The color key denotes numbers of lesions overlapping in different brain regions. The map is derived from several hundred patients, and shows that certain areas of the brain (e.g., temporal poles, middle cerebral artery territory) are more highly sampled by lesions than are other areas (e.g., high midline structures, occipital cortices). This figure is reproduced in the color plate section.

Connectivity among brain areas creates additional challenges. Higher functions, perhaps especially functions such as "personality," depend on brain networks and not just isolated brain regions, and the networks include both gray matter and the white matter fiber tracts that connect various cortical regions (Friston, 2000). Specific brain regions reside in a larger, highly interconnected context, and coordinated processing among distributed networks of brain regions is required for higher functions such as cognition and personality. This appears to be particularly true as the target functions become more complex, such as personality, problem solving, and so on—for example, "moving your thumb" can be performed with a small bit of motor cortex, whereas "feeling happy" requires a more complex network of structures. Within the last few years, investigators have arrived at broadly similar descriptions of the spatial layout of functional systems of brain regions across the entire human cortex (Figure 3.6) using correlations of resting-state fMRI blood oxygen level dependent (BOLD) signal (Power, Schlaggar, Lessov-Schlaggar, & Petersen, 2013; Warren et al., 2014).

Despite these limitations, the lesion method has remained the "gold standard" approach to understanding brain–behavior relationships, especially in regard to complex functions such as personality. The relationship of behavior and cognition to brain function has been informed by 150 years of neuropsychological studies describing specific deficits following focal brain lesions (Damasio, 1989; McCarthy & Warrington, 1990; Shallice, 1988). This "localization of function" approach has been immensely productive and strongly influences clinical practice in neurology, psychiatry, neurosurgery, and neuropsychology, especially regarding diagnosis, prognosis, and rehabilitation of brain-injured patients (Lezak, Howieson, Bigler, & Tranel, 2012).

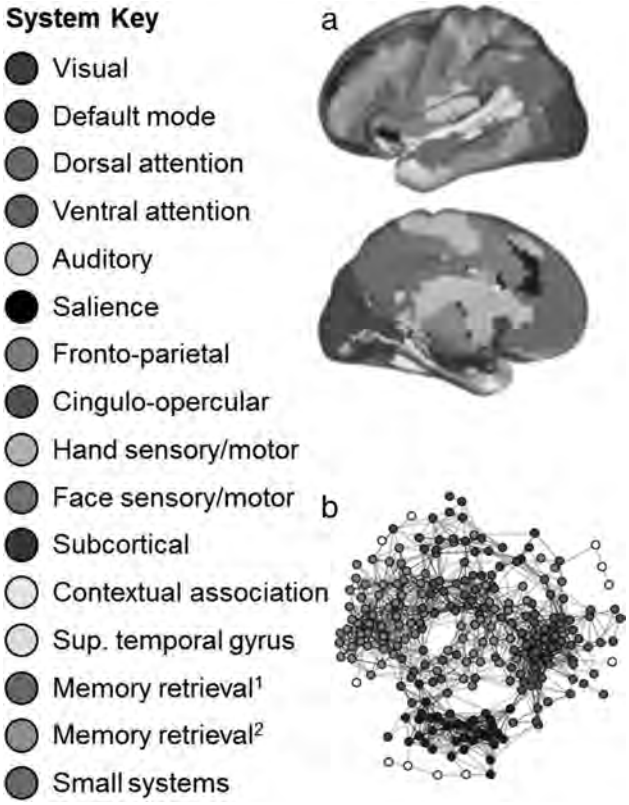

Figure 3.6 "Various brain "networks" that have been derived from resting state functional magnetic resonance imaging studies. The color key denotes 15 various networks, named for known or presumed functional and/or structural correlates, and also "small systems." (a) shows the networks plotted on lateral (upper) and medial (lower) hemispheric views; (b) shows the nature of interconnectivity of the networks. The idea for this figure was inspired by Warren et al. (2014). This figure is reproduced in the color plate section.

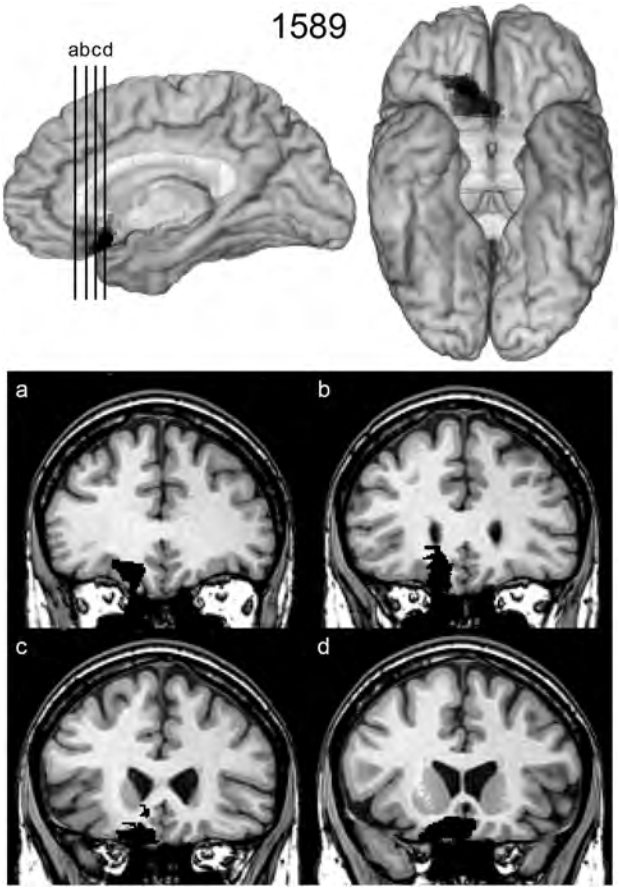

Figure 3.7 Patient 1589 has a small right ventromedial prefrontal lesion, shown (in red) in medial (upper left) and ventral (upper right) hemispheric views and in coronal cuts (a–d). By radiologic convention, the left hemisphere is on the right in the coronal views, and the right hemisphere is on the left (this perspective also applies to all subsequent patient examples, Figures 3.8 to 3.11). This figure is reproduced in the color plate section.

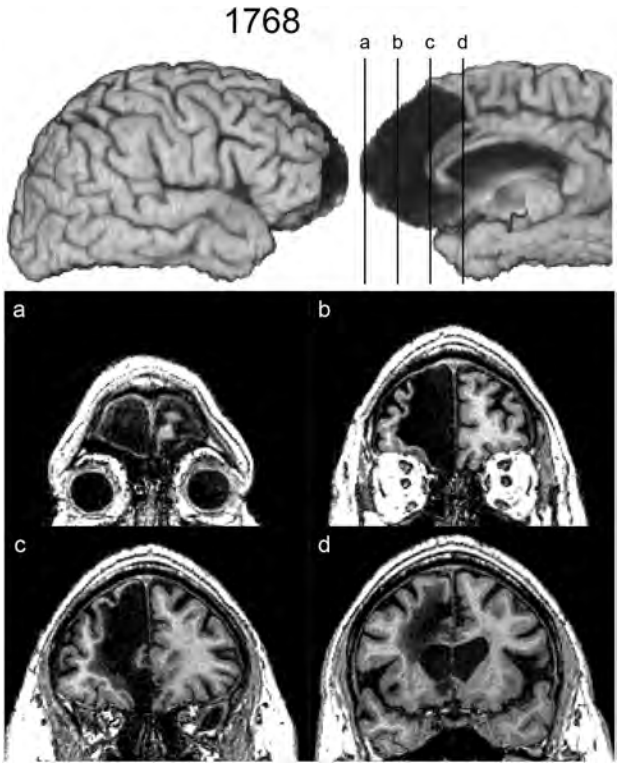

Figure 3.8 Patient 1768 has a large right ventromedial prefrontal lesion that extends into superior mesial and anterior lateral sectors. The lesion is shown in lateral (upper left) and medial (upper right) hemispheric views and in coronal cuts (a–d).

Case Examples

Patient 1589 (Figure 3.7) worked as a minister and counselor until suffering subarachnoid hemorrhage associated with rupture of an anterior communicating artery aneurysm, and resultant right orbitofrontal lesion, at the age of 32. Prior to the injury, he had obtained graduate degrees in ministry and counseling, and had worked very successfully in these professions. Subsequently, he has been unable to maintain gainful employment. He was often terminated for failing to keep appointments, and failing to complete necessary paperwork. He left the ministry because he had developed interpersonal problems at each church he was assigned to. His wife of 24 years eventually left him, noting significant changes in personality and social conduct. He demonstrates severe and chronic impairments in behavioral organization, judgment, planning, and decision making. He is unable to manage basic exigencies of life; e.g., even when he has sufficient funds he often failed to pay bills, and he is driving on expired license plates. He has gained a substantial amount of weight, eating unhealthy foods in large quantities, noting that he feels "less restrained" and goes for "instant gratification." His cognitive profile is largely normal with respect to intelligence, attention, perception, language, and standardized tests of executive function.

Patient 1768 (Figure 3.8), a widowed diesel mechanic, suffered a right frontal lobe infarction at the age of 54, resulting in a right ventromedial prefrontal lesion. Cognitive testing revealed stable, but relatively mild, impairments in anterograde memory and aspects of executive function. Otherwise, his cognitive profile is normal with respect to intelligence, attention, language, and perception. Behaviorally, he demonstrated significant personality changes, including abulia, blunted affect, reduced motivation, minimal goal-oriented actions, anosognosia, verbal perseverations, distractibility, and an inability to complete tasks. He became a widower after his stroke and became dependent on his daughter for all meals (she manages a local restaurant) and financial management.

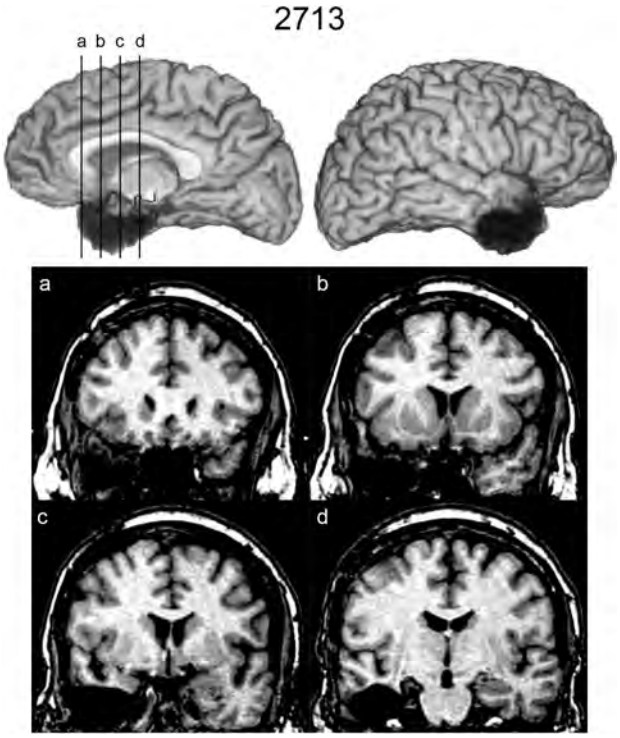

Figure 3.9 Patient 2713 has a right anterior temporal lesion, shown in medial (upper left) and lateral (upper right) hemispheric views and in coronal cuts (a–d). The lesion encompasses the right amygdala.

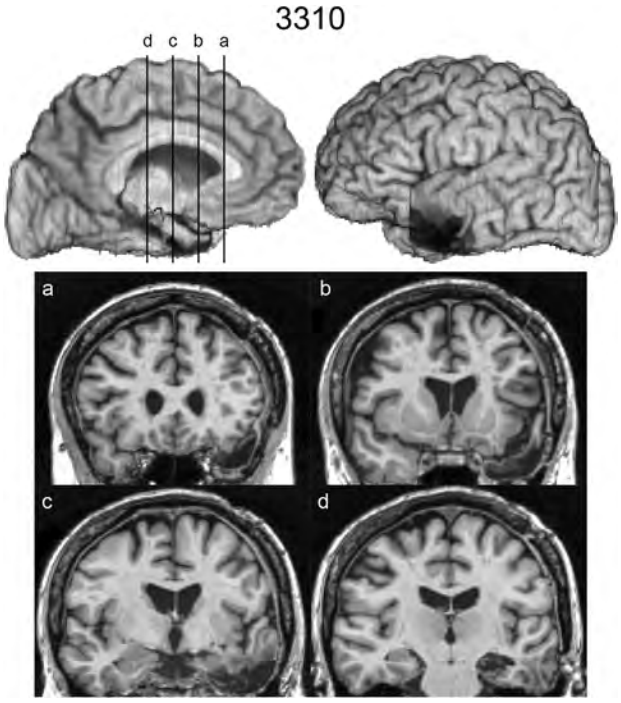

Figure 3.10 Patient 3310 has a left anterior temporal lesion, shown in medial (upper left) and lateral (upper right) hemispheric views and in coronal cuts (a–d). The lesion encompasses the left amygdala.

He noted "I can sit and watch TV all day," and will only take breaks for meals. He also noted difficulties with intentional movements with his left hand (e.g., his left hand will continue to hold onto a towel when he tries to set it down). He is no longer working, although he believes he is still fully capable.

Patient 2713 (Figure 3.9) is a 52-year-old right-handed man who was in the army for several years (first as a drill sergeant, then in administration), and later employed as an independent contractor. He and his wife raised five children, and also brought several foster children into their home. He underwent a right anterior temporal lobectomy (including amygdalohippocampectomy), at age 46, for pharmacoresistant epilepsy, and has remained seizure free. Neuropsychological testing indicates generally normal cognitive abilities with respect to intelligence, attention, memory, language, perception. He had impaired performances on standardized tests of executive functioning, due to impulsivity and distractibility. He demonstrated impaired performances on a laboratory test of complex decision making (the Iowa Gambling Task), failing to develop anticipatory skin conductance responses to disadvantageous choices. Behaviorally, he has significant changes in personality and emotional processing. His wife noted that he is more labile and irritable, and would overreact to minor events to the point of remaining upset for several days. He was unable to return to employment as an independent contractor, owing to his emotional lability and frequent outbursts. He and his wife discontinued caring for foster children.

Patient 3310 (Figure 3.10) is a 51-year-old, right-handed married woman, who was employed as a secretary for a local manufacturer. She underwent a left anterior temporal lobectomy (including amygdalohippocampectomy), at age 49. Her seizure frequency was reduced, but not eliminated. Neuropsychological testing revealed defects in verbal memory, and some aspects of language (naming, and auditory comprehension). Deficits were also evident in her performances on standardized tests of executive function, primarily due to impulsivity and perseveration. She had impairments on laboratory tests of complex decision making, chiefly due to an inability to develop anticipatory skin conductance responses to disadvantageous choices. Behaviorally, she developed numerous changes in personality and emotional processing, marked especially by emotional lability, irritability, depression, and anxiety. She demonstrated disinhibition (e.g., frequent use of profanity and inappropriate humor), and dramatic changes in affect. She was irritable at times, and then would suddenly become inexplicably euthymic. Because of her constellation of cognitive and behavior impairments she has been awarded disability.

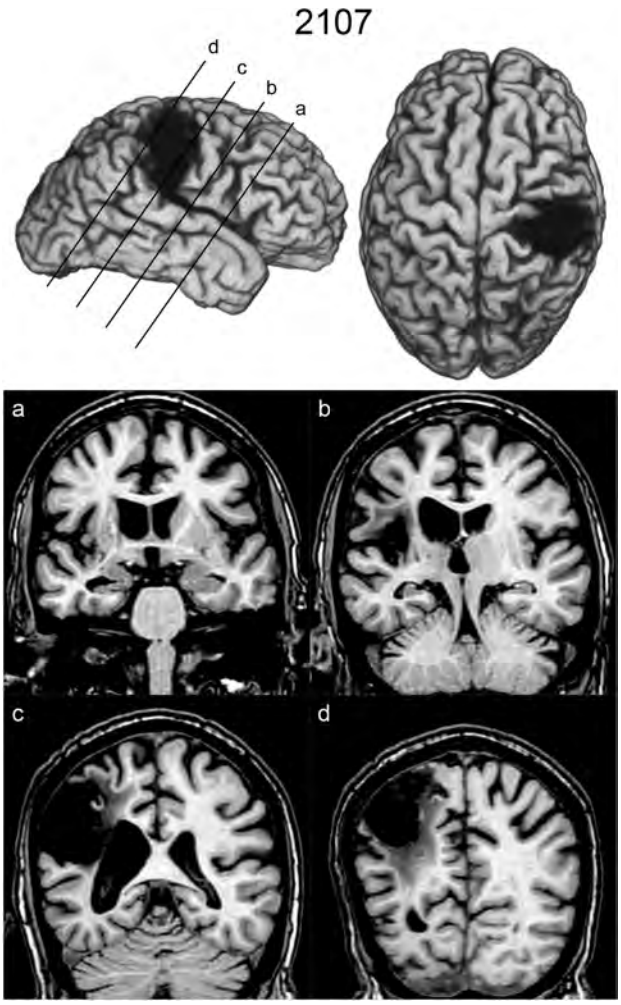

Figure 3.11 Patient 2107 has a right parietal lesion, shown in lateral (upper left) and superior (upper right) hemispheric views and in oblique coronal cuts (a–d). The lesion encompasses the somatosensory cortices and includes dorsolateral, supramarginal gyrus, and insula regions on the right.

Patient 2107 (Figure 3.11), a 63-year-old, right-handed, married man, suffered an infarction of the right middle cerebral artery, at the age of 55, which resulted in mild left hemiparesis. Neuropsychological testing has shown consistent, mild impairments in aspects of visual processing (including left visual inattention), but otherwise normal cognitive abilities with respect to intelligence, attention, memory, language, and standardized tests of executive function. Behaviorally, he has anosognosia for his impairments and numerous changes in personality. He has lability (e.g., cries easily), anxiety, diminished motivation, and poor organizational skills. He has become much more dependent on his wife for everyday activities, owing to problems with indecisiveness. He also demonstrates deficits in emotional and social judgments, including difficulties recognizing affect from static facial expressions, and limited recognition of an emotional/social setting from a video depiction of inanimate objects. Despite repeatedly being told that he should not drive, he frequently has asked about driving privileges. Even though he has been told this numerous times, he has never expressed any anger or outward disappointment, generally acknowledging it with a statement like "okay, maybe next time, right?" He was previously employed as a custodian, but as a result of his cognitive and behavioral deficits he took early retirement.

Functional Neuroimaging Functional neuroimaging approaches have become another commonly used tool for investigating neurobiological substrates of personality and psychopathology. Multiple functional neuroimaging methods exist, most of which involve using some measure of blood flow or metabolism or a closely related parameter to index variations in neural activity across different brain regions. Some designs experimentally change psychological or behavioral state to make inferences about underlying changes in neural activity; other designs are observational in their approach to understanding neural activity.

In positive emission tomography (PET), for example, radioactively labeled substances are introduced into the bloodstream, where they are taken up into the brain. These substances, which become sequestered during neural activity, emit radiation that is imaged across the brain (e.g., radioactively labeled glucose analogues are used to supply energy to neurons; radioactive serotonin or dopamine antagonists bind to receptors). By mapping the location of this radiation, one can make inferences about neural activity involving those radioactively labeled substances (e.g., where a dopamine antagonist is being taken up in the brain, or differences between individuals in how it is binding to dopamine receptors).

In fMRI, similarly, changes in magnetic field properties due to oxygenated versus deoxygenated blood hemoglobin are measured across the brain to determine where blood flow, and thus, brain activity, is occurring. When neural activity increases in a given area, the fraction of oxygenated blood will increase in that area (i.e., the BOLD response). Oxygenated and deoxygenated blood have different magnetic properties, which can be detected from an electromagnetic "echo" when probed with radio pulses. By observing changes in the properties of this echo as a function of changes in experimental conditions (e.g., observing neutral versus negative images), one can make inferences about the patterns of brain activity involved in a psychological process. Increasingly, fMRI is also used to identify how BOLD response, and therefore neural activity, is correlated across different regions of the brain, either as it occurs in the absence of experimental manipulation (e.g., resting-state designs) or across conditions of an experiment. These latter sorts of designs help us to understand not only which regions are involved in a particular neuropsychological process, but also how their activity is networked and coordinated.

Although functional imaging techniques have become an indispensable tool in modern neurobiological research, like any other methods, they also have important pitfalls.

For instance, different functional imaging techniques vary in their spatial versus temporal resolution; fMRI, for example, provides relatively detailed information about where neural activity occurs, but can be limited in providing information about when it occurs; in contrast, electroencephalography (EEG; which involves measuring changes in scalp electrical currents as a function of changes in brain activity) provides relatively detailed information about when neural activity occurs, but not where it occurs.

One important problem with much of contemporary functional neuroimaging research relates to how data are analyzed and interpreted. A typical functional neuroimaging study can involve analysis of hundreds of thousands of datapoints, each representing a different point in the brain, measured across multiple timepoints. Because each of these datapoints is often treated as independent and because of the large number of points involved, it is likely that many false associations will be observed simply by chance. This is exacerbated by the fact that, in many studies, researchers tend to select the largest associations from the pool that were observed, which mathematically are guaranteed to be inflated on average, owing to their chance nature (Vul, Harris, Winkielman, & Pashler, 2009). The problems are further compounded when one considers that such imaging studies are often conducted with very small numbers of individuals, which limits the replicability of findings and increases the likelihood that any observed association is due to chance (Button et al., 2013).

Vul et al. (2009), for example, demonstrated that it is not uncommon in the functional neuroimaging literature to observe correlations that are essentially impossible in nature given the psychometric reliabilities of psychological measures and the neuroimaging signals involved in the correlations. They concluded that such correlations are inflated from chance and unlikely to be generalizable, owing to the issues discussed here. Button et al. (2013) similarly concluded that many neuroimaging studies lack the statistical power to detect the effects they are reporting, and as a result, report inflated associations that are unlikely to replicate (see also David et al. 2013). Similarly, in our own work (Jonas & Markon, 2014), we have found that neuroimaging studies are likely to report larger genetic associations than studies involving behavioral measures, but are also more susceptible to inflation due to publication bias.

Functional neuroimaging methodology has contributed significantly to our understanding of the neurobiological substrates of psychopathology. Care is warranted, however, in interpreting results and proffering conclusions that are likely to generalize across samples. Meta-analyses of functional neuroimaging studies can be helpful in identifying which findings replicate across studies and which are chance findings due to the large number of associations being examined. Additional research is also needed on new approaches to analysis of functional neuroimaging data to ameliorate these problems.

Methods of Gene Discovery Decades of behavioral genetics research have clearly converged upon the notion that genetic influences are important for nearly every psychological disorder (and, importantly, nearly every behavioral dimension related to psychopathology). However, twin studies do not tell us about which genetic variants are specifically implicated. Molecular genetics research over the past 20 years has increased exponentially with this exact goal in mind. Much of this work began by identifying candidate gene polymorphisms based upon the (hypothesized) action of particular pharmacological agents used in treatment. This work typically proceeded by examining samples of cases and controls to determine if the proportion of a particular allele was significantly higher among cases versus controls. For disorders with an earlier age of onset, family studies were also employed to examine whether preferential transmission of particular alleles from parent to child occurred more frequently for affected versus unaffected individuals (Waldman, Robinson, & Rowe, 1999). While some initial results were promising, the accumulation of findings from candidate gene studies has indicated that effects are likely very small and quite heterogeneous across samples (odds ratios of approximately 1.5; see Gizer, Ficks, & Waldman, 2009 for a review of candidate gene studies of ADHD). Lack of replication of these effects (see Zheng et al., 2012), as well as the increasing availability of genetic technologies and statistical methods, then paved the way for more advanced molecular genetic methods of gene discovery.

Technological improvements in genotyping platforms along with recognition of the potential importance of considering variation throughout the genome (and not just within particular candidate genes) have fueled the progression to genome-wide association (GWA) studies. These studies use large platforms to genotype as many as 500,000 single nucleotide polymorphisms (SNPs) among cases and controls. Unlike candidate gene studies that build upon neurobiological theories of psychopathology, GWA investigations make no prior assumptions about the types of genes that may be involved in any particular disorder. Furthermore, it is important to note that GWA studies are examining statistical association with numerous markers and thus must employ large corrections for multiple tests; thus, the only findings that reach "genome-wide" significance are those with a p-value less than 10^{-8}.

Multiple large-scale GWA studies have been conducted for a variety of psychological disorders, including schizophrenia, ADHD, depression, bipolar disorder, and autism. While some studies have found markers in particularly novel genes (i.e., circadian rhythm genes and depression; Hek et al., 2013) and some findings have been replicated, it is important to note that relatively few markers within each study have survived correction for "genome-wide significance." However, combined efforts such as the Enhancing Neuro Imaging Genetics through Meta-Analysis (ENIGMA) project and meta-analyses of genome-wide data appear to be discovering novel

genetic variants that may contribute to a variety of disorders (Thompson et al., 2014). For example, a recent meta-analysis of GWA studies of schizophrenia suggests that approximately 8,300 different, common SNPs contribute to the development of schizophrenia, and in total, account for approximately 32% of the genetic liability (Ripke et al., 2013). At the close of the chapter, we briefly discuss how GWA consortium and collaborative efforts likely represent the way forward in gene discovery for psychopathology.

In addition to GWA, other models of genetic discovery are also underway. For example, copy-number variants (i.e., long strands of the DNA that are copied a certain number of times which are then duplicated or deleted among some individuals) have been implicated in studies of both ADHD and autism spectrum disorder (Matsunami et al., 2014; Yang et al., 2013). Genetic studies of autism spectrum disorder have revealed that some of these genetic variants may be *de novo* mutations. Alterations in these copy-number variants appear to originate in the parental gametes (i.e., not present in the parental DNA) that are then transmitted to their offspring. That is, within a single generation, there may be some mutations occurring within these copy-number variants that increase the liability for autism spectrum disorders. These mutations that are present in the offspring (but not in parents) are referred to as *de novo* mutations. Furthermore, it may be the case that these *de novo* variants may contribute to sex differences in risk for autism spectrum disorders (Ronemus, Iossifov, Levy, Wigler, 2014).

In addition to examining association between changes in the structural DNA and behavioral phenotypes of psychopathology, molecular genetic work has also discovered the "epigenome," which is likely also relevant to the development of psychopathology. Epigenetic effects involve the ways in which *gene expression* can be modified by environmental experiences. That is, through a variety of chemical transactions, the ways in which genes are encoded and ultimately expressed appear to be sensitive to environmental experience as well (Reik, 2007). Regulation of gene expression via epigenetics can occur via several different mechanisms—one of the most commonly studied is chromatin remodeling (for a review of epigenetic mechanisms, see Portela & Esteller, 2010). Animal and human work has suggested that chromatin remodeling may occur as a function of environmental experiences, including stress, diet, early deprivation, and potentially via prenatal exposures to teratogens (Weder et al., 2014). Additionally, epigenetic modifications to gene expression may be long lasting and subsequently inherited by future generations (Youngson & Whitelaw, 2008). Thus, alterations within the structural DNA (discovered via GWA and association methods), as well as epigenetic changes, are likely involved in increasing liability for developing for psychological disorders. While future work will likely be important for illuminating the specific genes and biological mechanisms that underlie the development of psychopathology, these methods will likely be most informative for constructing comprehensive models of etiology and for identifying potential novel targets for intervention and prevention of psychological disorders.

The Characterization and Measurement of Personality and Psychopathology Genetic polymorphisms, neurophysiological processes, and brain lesions are increasingly drawn on not only to understand psychopathology, but to define it. Psychiatric classification is currently in a period of substantial transition, with a fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), and ongoing revisions to the *International Classification of Diseases* (World Health Organization, 1992; at the time of writing, ICD-11 is currently in development and is due for release in 2017). In addition, new classification systems are being proposed, such as the National Institute of Mental Health (NIMH) Research Domain Criteria (RDoC; National Institute of Mental Health 2011; Insel et al., 2010). During this period of transition, the role of biological constructs in defining and classifying psychopathology has been a major focus of discussion, with some arguing that it should largely form the basis for how we define psychopathological constructs (e.g., Insel et al., 2010).

Central to many of these discussions is the concept of an *endophenotype*, loosely defined as a marker of mental disorder that is "closer" in some sense to underlying etiology (e.g., gene products, neural circuits) than clinical status itself. Working memory or BOLD response in an fMRI design, for example, might be considered endophenotypes for schizophrenia, as those markers are related to schizophrenia and also may be seen as more strongly related to underlying etiologic factors than a schizophrenia diagnosis. If the etiology of a particular mental disorder is seen as a network of paths from underlying liability factors to illness, at different levels of analysis from the molecular to the behavioral, endophenotypes include those constructs that are closer in the network to the liability factors than the illness itself (Figure 3.12, top).

The term endophenotype has its roots in psychiatric genetics (Gottesman & Shields, 1972), although it has since entered widespread use in other fields of psychopathology. There have been many attempts to precisely define the term, and related concepts are often used (e.g., *intermediate phenotype* or *biomarker*; see Lenzenweger, 2013 for a critical review). Endophenotypes are traditionally defined as phenotypes having genetic relationships with the illness (e.g., co-segregating with the illness within affected families and between affected and nonaffected families), as well as state-independence (i.e., observability in some way in affected or at-risk individuals regardless of current clinical status; Gottesman & Gould, 2003). Endophenotypes are not necessarily biomarkers, nor are they themselves necessarily putative causal factors (Miller & Rockstroh, 2013). The use of the term has loosened over

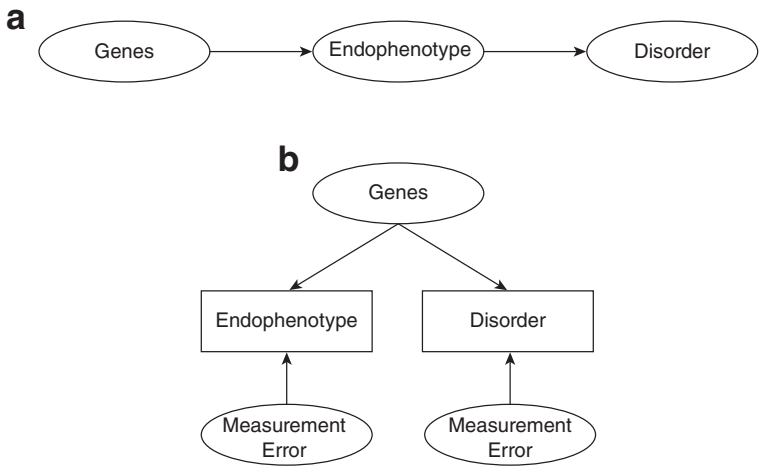

Figure 3.12 Mediational and spurious endophenotype models. These are simplified examples; other etiologic factors could be substituted for genes, and there could be many genes or endophenotypes acting in a pathway. (a) A typical endophenotype model, where an endophenotype mediates relationships between genes and disorder, or is otherwise intermediate between the two. (b) A spurious endophenotype, where the putative endophenotype is not actually closer to genetic influences, but appears to be because it is measured with less error.

time, however, and it is sometimes applied in ways that are different from its classical meaning.

Although the endophenotype concept has found widespread use in a number of different areas of psychopathology research, concerns and cautions have also been noted. Kendler and Neale (2010) have noted, for example, that putative endophenotypes may show stronger relationships with etiologic variables than clinical variables if the endophenotypes are more reliable than the clinical variables. This would result in spurious identification of variables as endophenotypes, owing to differences in measurement error among variables (Figure 3.12, bottom). Flint and Munafo (2007), similarly, examined proposed endophenotypes for schizophrenia (attentional shifting, working memory, and electrophysiological indices) using meta-analysis and found that the endophenotypes did not relate to genotypes any more strongly than schizophrenia itself. Reviewing the broader genomics literature, they argued that phenotypes involved in complex disease are not likely to differ in their etiologic simplicity, calling into question the assumptions underlying endophenotype theory.

Despite these caveats, endophenotype theory has come to play a substantial role in discussions of how to define focal psychopathology constructs of interest. The NIMH, for example, has largely structured its proposed classification system, RDoC, around endophenotype or intermediate phenotype theory (Insel & Cuthbert, 2009). In contrast to DSM and ICD, which NIMH views as clinical nosologies that do not necessarily map onto underlying etiology, RDoC constructs are intended to reflect underlying etiology more closely, in part by using measurement of hypothesized neurobiological contributors. RDoC is based on three guiding principles (National Institute of Mental Health, 2011): that its constructs vary continuously from the normal to abnormal range; that it be agnostic with regard to traditional diagnostic categories, and instead aim to "generate classifications stemming from basic behavioral neuroscience"; and that it include multiple levels of analysis, from the molecular to the behavioral. RDoC delineates psychopathology constructs in five broad areas: negative valence systems (e.g., fear, anxiety, loss), positive valence systems (e.g., reward valuation), cognitive systems (e.g., attention, language), social processing systems (e.g., attachment, self-knowledge), and arousal and regulatory systems (e.g., arousal, sleep and wakefulness; NIMH, 2011).

Disorders and Conditions

In this section, we demonstrate how these conceptual and methodological advances have been applied to understand the origins of some forms of psychopathology, with an emphasis on the large-scale neurobiological systems that are implicated in various disorders. These disorders are covered in detail in other chapters in parts II and III of this book; here, we focus on several exemplar disorders, with an emphasis on findings relating to the biological bases of each.

Schizophrenia Few areas of pathological behavior so clearly demonstrate the relevance of genetic and neurobiological processes than schizophrenia and psychosis. Schizophrenia is a heterogeneous disorder characterized by experiences of psychosis (hallucinations and delusions), as well as behavioral, motivational, and emotional deficiencies. As early as the 1930s, family studies of schizophrenia revealed a link between parental symptom severity and offspring risk for schizophrenia (Kallmann, 1938). Decades of twin and family work have consistently pointed toward sizable genetic influences in behavioral symptoms of schizophrenia (i.e., hallucinations, delusions, disorganized speech and behavior, negative symptoms;

Singh, Kumar, Agarwal, Phadke, & Jaiswal, 2014), with heritability estimates approaching 80%. Importantly, however, twin concordance rates are not at unity, and decades of research have implicated a variety of environmental influences as well, including season of birth, paternal age, and perinatal complications (Abdolmaleky & Thiagalingam, 2011). Thus, while genes likely play an important role in the development of schizophrenia, they are not deterministic.

As mentioned earlier, GWA studies have implicated as many as 8,300 common genetic variants in the etiology of schizophrenia (Ripke et al., 2013). Additionally, work in this area has begun to piece together these findings to form functional genomic network models based upon the type and number of genes implicated in GWA studies. Recent meta-analyses of 6 large-scale GWA studies (which included over 30,000 participants) implicated 43 genes in 14 different pathways, many related to neuronal function. The most significant pathway identified by Aberg and colleagues (2013) involved axon guidance, which included genes involved in the processes in which neurons send out axons to various targets. To simplify, many of the genetic effects implicated in schizophrenia may be related to abnormalities in basic neuronal architecture, organization, and communication. Additional genes implicated in schizophrenia include genes on chromosome 8 that encode the Neuregulin 1, a glycoprotein whose expression has been linked to creativity as well as psychosis (Murray & Castle, 2012). Additionally, genes encoding proteins involved in dopamine neurotransmission, including the catechol-o-methyl transferase gene on chromosome 22 have also been implicated in schizophrenia (Martin, Robinson, Dzafic, Reutens, & Mowry, 2014). This gene is one of many that are not expressed among individuals with 22-q deletion syndrome, who are also at increased risk for developing psychosis during adolescence. Furthermore, there has been long-standing interest in dopamine-related genes and processes and schizophrenia, due to neurobiological hypotheses of schizophrenia that suggested that the symptoms underlying the disorder were due to hyperdopaminergic function in key frontal brain regions (Javitt & Laruelle, 2006). More recent hypotheses instead point toward excessive stimulation of striatal dopamine D2 receptors (part of the basal ganglia within the brain) that are involved in movement and balance (Harrison, 2012) along with a deficiency in prefrontal dopamine D1 receptors (Howes & Kapur, 2009), which may account for the cognitive difficulties associated with the disorder.

Schizophrenia has long been described as a disorder of brain connectivity (Friston, 1988). It does not appear that schizophrenia develops as the result of isolated damage to a small number of brain regions. Instead, symptoms of schizophrenia likely arise as a result of problematic connectivity and activation in structures that span across multiple neural circuits. Clear structural and morphological differences in the brain have emerged in studies of patients with schizophrenia, including reduced white matter volumes (Hulshoff Pol et al., 2004). Developmental trajectories of white matter have also been shown to be disrupted among non-psychotic siblings of youth with childhood-onset schizophrenia (Gogtay et al., 2012). Studies of white matter density have implicated abnormalities in specific regions among individuals with schizophrenia, including the fasciculus and the corpus callosum (Kubicki et al., 2007), whereas tissue histology studies have suggested pathological myelination of tracts involving the prefrontal cortex (Uranova et al., 2013). Along these lines, the lateral ventricles may be enlarged among individuals with schizophrenia, a phenomenon that appears to be somewhat genetically influenced (Harrison, 2012; Staal et al., 2000). Similarly, meta-analytic investigations have reported reductions in gray matter volume bilaterally in regions spanning from the insula to the inferior frontal cortex (Bora et al., 2011). Reduced volumes have been reported in a number of structures involved in higher-order processing of cognition and emotion, including the hippocampus, amygdala, and frontal and temporal cortices, and importantly, are similar among first-episode patients as well as those with chronic symptoms (Levitt, Bobrow, Lucia, & Srinivasan, 2010). Thus, the morphological changes observed among individuals with schizophrenia appear to pre-date the onset of the symptoms themselves.

Many of the disturbances in functional activity and connectivity associated with schizophrenia involve the prefrontal cortex, including frontal-parietal (Repovs, Csernansky, & Barch, 2011), frontal-striatal (Filipi et al., 2013), and frontal-temporal networks (Hoffman et al., 2013). The consistent implication of impaired frontal processing in the pathology of schizophrenia has led to the hypothesis that a core deficit in the disorder involves deficiencies among “hub” regions and networks (Rubinov & Bullmore, 2013). Functional connectivity findings have implicated both increased and decreased connectivity of frontal-temporal and frontal-parietal (van den Heuvel & Fornito, 2014), although the majority of findings suggest reductions in connectivity. Given abnormalities in activation in networks involving the frontal regions, patients with schizophrenia have unsurprisingly demonstrated impaired performance on numerous neuropsychological tasks, including executive function, attention, and memory (Neill & Rossell, 2013). Such deficits have been identified during the prodromal phase of the illness (Woodberry et al., 2013) and have been found to be relatively stable over time (Dickerson et al., 2014). Further, deficits in these areas have also been observed in unaffected family members of patients with schizophrenia (Massuda et al., 2013) and severity of impairments in patients have been correlated with degree of gray matter reduction, suggesting that these measures may serve as useful intermediate or endophenotypes for the disorder.

Alterations in connectivity of these networks also appear to underlie the development of psychotic symptoms. For

example, auditory hallucinations (and proneness to experience auditory hallucinations) have been posited to result from misattributions of "internal" thoughts or speech and have been linked to reduced processing in frontal-temporal networks. This has led to the hypothesis that schizophrenia is ultimately a disorder of self-monitoring (Kanemoto, Asai, Sugimori, & Tanno, 2013). These findings are in line with past work suggesting that individuals with schizophrenia perform significantly worse than controls and exhibit reduced cortical activation on tasks of verbal self-monitoring (McGuire & Frith, 1996). In addition, deficiencies in processing associated with negative symptoms have also been linked to impaired functioning in frontal as well as limbic regions. For example, reductions in activation of connections involving the frontal cortex and amygdala have been found among individuals with schizophrenia during tasks requiring processing of emotional expressions in faces (Fakra, Salgado-Pineda, Delaveau, Hariri, & Blin, 2008). Patients with schizophrenia also showed reductions in activity in the fusiform gyrus, a region associated with holistic face processing, but instead evidenced *increased* activation in regions associated with feature analysis (inferior parietal regions, right precuneus, Fakra et al., 2008). Taken together, alterations in neural morphology, functionality, and connectivity appear to be widespread and persistent among individuals with schizophrenia and appear to underlie significant deficits in both cognition as well as processing of emotions.

Current work aiming to link specific putative genetic risk factors to these neurobiological and structural and functional differences is currently underway. The ENIGMA work group is currently collecting and combining data from over 30,000 patients with and without schizophrenia in order to tie novel genetic variants identified via GWA to specific patterns of morphological and functional changes associated with the onset and progression of the disease (Thompson et al., 2014). Such work can begin to link the complicated genetic influences on neuronal architecture to differences in brain structure and function and will likely play a large role going forward in the discovery of specific etiological pathways that give rise to the behavioral syndrome of schizophrenia.

Internalizing Disorders As a spectrum, different forms of internalizing psychopathology—for example, depression, anxiety, and fear—are strongly related to one another phenotypically, and are undergirded by many of the same neurobiological mechanisms. Internalizing disorders share common genetic and environmental etiologies with each other and with generalized negative emotionality (e.g., Hettema, Neale, Myers, Prescott, & Kendler, 2006), and have overlapping neurobiological correlates (e.g., Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012). Meta-analyses have identified numerous putative candidate genes, including those that may impact neurotransmission of monoamines (López-León et al., 2008). GWA investigations have also pointed toward networks of genes involved in neuronal development and architecture, similar to findings with schizophrenia (Jia, Kao, Kuo, & Zhao, 2011).

Many neurobiological models of internalizing psychopathology can be conceptualized in terms of stress response: either the processing of stressors, or the effects of that processing. Within these frameworks, different forms of internalizing are often framed in terms of different types of stressors—for example, depression and chronic stress or fear and acute stress—but within the same structural network. This network primarily involves the hypothalamic–pituitary–adrenal (HPA) axis, the amygdala and its nuclei, and their connections with other structures such as the hypothalamus, thalamus, hippocampus, prefrontal cortex, and basal ganglia. Importantly, the amygdala receives projections from areas of the brain associated with fast, nonconscious processing of threats, such as the thalamus, as well as cortical areas associated with higher-order processing, such as the vmPFC. It also projects onto a number of other brain regions involved in stress response (hypothalamus), memory (hippocampus), as well as attention and motivation (basal ganglia).

In many ways, the amygdala can be thought of as a central mediator of memory and learning about stressors and negatively valenced stimuli. The amygdala is active during processing of negatively valenced stimuli (Lindquist et al., 2012), for example, and disruptions of the amygdala impair learning of stressors and other negatively valenced stimuli (Bechara et al., 1995). Conversely, facilitations of input to the amygdala can enhance memory about such stimuli (Roozendaal, McEwen, & Chattarji, 2009). Lesions to the amygdala, while impairing fear learning and responses to phobic stimuli, do not appear to disrupt fear responses to internal stimuli, suggesting that the amygdala is more critical to responding to stimuli in the external environment than it is to the experience of negative emotion *per se* (Feinstein et al., 2013).

The development of emotional memories via the amygdala is partially moderated through HPA axis-related neurotransmitters and hormones such as noradrenaline and glucocorticoid hormones. Within the amygdala, noradrenaline appears to mediate acquisition of stress-related memory, and glucocorticoid hormones seem to be involved in consolidation of long-term memories. For example, infusion of noradrenaline into the basolateral amygdala enhances memory for negatively valenced stimuli (Roozendaal, McEwen, & Chattarji, 2009), and blockade of noradrenaline into the same region using adrenergic receptor antagonists impairs memory for stressful stimuli (Roozendaal, McEwen, & Chattarji, 2009; Rodrigues, LeDoux, & Sapolsky, 2009). Similar phenomena have been demonstrated for the effects of glucocorticoid hormones on consolidation of long-term emotional memories (Rodrigues, LeDoux, & Sapolsky, 2009).

Of course, the amygdala is part of a larger network of structures undergirding stress-related processing, and stress has other effects on other components of this

network. Connections with the vmPFC, for example, presumably mediate top-down, higher-order regulation of negative emotion, and, in turn, affect behavioral regulation itself (Grupe & Nitschke, 2013). In contrast to their role in facilitating long-term emotional memory in the amygdala, glucocorticoid hormones appear to inhibit working memory in the prefrontal cortex, and have a similarly impairing effect on memory retrieval in the hippocampus (Roozendaal, McEwen, & Chattarji, 2009; Rodrigues, LeDoux, & Sapolsky, 2009). These different effects of glucocorticoid hormones in the amygdala versus hippocampus and prefrontal cortex may partially help explain "tunnel memory" in post-traumatic stress responses, where memory for primary aspects of a trauma are enhanced, while memory for peripheral stimuli are reduced (Berntsen, Rubin, & Johansen, 2008).

Although internalizing disorders share certain common neurobiological substrates, such as amygdala hyperactivity (Etkin & Wager, 2007; Hamilton et al., 2012), different internalizing disorders also have unique features that differentiate them. Phenotypic studies have demonstrated a distinction between distress disorders—such as depression, generalized anxiety, and traumatic stress-related disorders—that are characterized by more ruminative behavioral patterns, and fear disorders—such as phobias and panic—that are characterized by more acute, paroxysmal response patterns (Krueger & Markon, 2006). Although these two forms of internalizing disorders strongly co-occur, they are differentiated by their behavioral patterns and neurobiological correlates. For example, although all the internalizing disorders are characterized by amygdala and insula hyperactivity in response to negative stimuli, fear disorders such as the phobias are characterized by even greater hyperactivity than distress disorders (Etkin & Wager, 2007). This is consistent with the acute fight-or-flight response typical of fear disorder, and the amygdala's role in automatic processing of threat stimuli via the thalamus. It is also consistent with the amygdala's mediation of the effects on emotional memory of stress-related signaling chemicals such as noradrenaline and glucocorticoid hormones.

Conversely, relative to fear disorders, distress disorders such as depression and traumatic stress-related disorders have been uniquely associated with hypoactivation of prefrontal cortex during processing of negatively valenced stimuli (Drevets et al., 1997; Etkin & Wager, 2007; Hamilton et al., 2012). In particular, lower dorsolateral PFC activation during processing of negative stimuli has been demonstrated for individuals with depression (Hamilton et al., 2012), and lower ventromedial PFC activation has been observed in individuals with post-traumatic stress disorder (Etkin & Wager, 2007). The significance of these findings is unclear, although it could reflect either deficient top-down regulation of emotional processing through the cortical areas, or disruption of areas involved in attention in working memory through rumination or processing of intrusive thoughts.

Clarifying genetic contributions to internalizing psychopathology has been complex. The heritability of internalizing disorders is approximately 0.3–0.4 (Hettema, Neale, & Kendler, 2001; Sullivan, Neale, & Kendler, 2000), which is lower than some rarer forms of psychopathology such as schizophrenia. Although many genes have been reported to be associated with internalizing disorder, few have replicated. Some associations have replicated, however, such as associations between depression and genes involved in serotonin and folate processing (López-León et al., 2008).

Although the monoamine hypothesis—that deficits in monoamines such as serotonin cause internalizing problems—has been a popular theory, especially in explaining depression, it is not supported by the empirical research overall (Krishnan & Nestler, 2010). More recent models for the pathogenesis of internalizing problems usually explain monoamine involvement through other more proximal mechanisms. Neurotrophic factors—substances involved in neural growth and development—are one example, positing that neural growth factors themselves are more primary in explaining internalizing problems, and that monoamines are involved secondarily. Vascular endothelial growth factor (VEGF), for example, has been shown to mediate the effects of a wide range of antidepressant treatments, including various antidepressant medications and electroconvulsive shock therapy: antidepressant treatments increase neural growth and levels of VEGF in the brain. VEGF signaling is necessary for these antidepressant effects on neural growth; and VEGF signaling also mediates the effects of antidepressants on behavior (Warner-Schmidt & Duman, 2007). Similar effects have been demonstrated with regard to fear learning (Licht et al., 2011; Warner-Schmidt, Madsen, & Duman, 2008). In this regard, antidepressants may not treat internalizing problems directly via monoamines such as serotonin, but through encouraging neural growth and proliferation.

Damage to the dorsolateral prefrontal region has also been associated with depression (e.g., Koenigs et al., 2008). Blumer and Benson (1975) referred to a pseudodepressed syndrome with decreased self-initiation following dorsolateral prefrontal lobe damage. Cummings (1985) described apathy, indifference, and psychomotor retardation as frequent features following dorsolateral prefrontal damage. Depressive symptomatology and social unease were found to be common in patients 3 months after dorsolateral prefrontal damage from stroke or trauma (Paradiso, Chemerinski, Yazici, Tartaro, & Robinson, 1999). Patients with lesions to the left hemisphere appear to be at the greatest risk of developing depression (e.g., Robinson, Kubos, Starr, Rao, & Price, 1984, 1985). For example, damage to the left dorsolateral prefrontal region resulting from stroke in the anterior distribution of the middle cerebral artery often has devastating consequences (paresis of the dominant side, aphasia), and unlike comparable lesions on the right, generally does not impair

awareness for the acquired impairments. However, during the acute phase following a stroke, depression cannot be fully explained by these factors, suggesting a neurophysiological basis for the relationship between left dorsolateral prefrontal damage and depression. Reaction to functional impairments is likely to play a more important role in depression that begins 6 months or more after a stroke (Robinson, Bolduc, & Price, 1987).

Psychopathy and "Pseudopsychopathy" Perhaps the greatest disturbances of personality and social behavior, absent a macroscopic brain lesion, are those that occur in psychopaths. In his pioneering profile of the psychopathic personality, Cleckley (1976) emphasized pervasive disregard for others as a primary feature. To facilitate the diagnosis of psychopathy, Hare (1991) created a specific and operationalized instrument: the Psychopathy Checklist-Revised (PCL-R), which highlights the fact that psychopathy is a syndrome and a cluster of behaviors defines the condition. There are some remarkable overlaps between the psychopathic traits noted in Hare's nomenclature and the personality manifestations shown by neurological patients following brain damage, especially damage to the prefrontal region, pointing toward a common neurobiological mechanism underlying "developmental" versus "acquired" psychopathy—the former designating the conventional condition that has been part of DSM nomenclature as a personality disorder; the latter designating an acquired form of the condition that occurs in premorbidly normal individuals who develop personality changes after a macroscopic brain lesion (Koenigs & Tranel, 2006).

As noted earlier, the association of the prefrontal lobes with psychopathic behavioral manifestations has been established since the landmark case of Phineas Gage, reported by Harlow in detail in 1868. Following the well-chronicled accident of having an iron bar shot through the front of his head, Gage changed from shrewd, persistent, and respectable to profane, capricious, and unreliable. Reports of similar types of personality changes following selected cases of frontal injury accumulated over the years. Blumer and Benson (1975) coined the term "pseudopsychopathy" to describe the personalities of a subset of patients with frontal lobe damage, emphasizing that such patients were best characterized by the "lack of adult tact and restraints." In contrast to conventional "developmental" psychopathy, in which psychopathic traits emerge in childhood and adolescence with no macroscopic structural brain lesion, pseudopsychopathic behaviors "follow injury to the orbital frontal lobe or pathways traversing this region" (Blumer and Benson, 1975). Our working definition of pseudopsychopathy is *a personality disturbance acquired in adulthood after the onset of brain damage, that entails antisocial behavior, including failure to conform to social norms, deceitfulness, impulsivity, irritability, consistent irresponsibility, and lack of remorse*. Our investigations of the nature of the personality disorder we have dubbed "acquired sociopathy" have pointed to several core identifying features (Barrash, Tranel, & Anderson, 2000): (1) general dampening of emotional experience (impoverished emotional experience, low emotional expressiveness and apathy, inappropriate affect); (2) poorly modulated emotional reactions (poor frustration tolerance, irritability, lability); (3) disturbances in decision making, especially in the social realm (indecisiveness, poor judgment, inflexibility, social inappropriateness, insensitivity, lack of empathy); (4) disturbances in goal-directed behavior (problems in planning, initiation, and persistence, and behavioral rigidity); and (5) marked lack of insight into these acquired changes. A well-studied example of this phenomenon is patient EVR, first reported by Eslinger and Damasio (1985). At age 35, EVR underwent resection of a bilateral orbitofrontal meningioma. Mesial orbital and lower mesial frontal cortices comprising the vmPFC were excised with the tumor, and profound personality changes ensued. Although EVR is skilled and intelligent enough to hold a job, he cannot report to work promptly or regularly and he is unacceptably unreliable in completion of various intermediate job steps. His ability to plan his activities, both on a daily and long-term basis, is severely impaired—in particular, he fails to take into account his long-term interests and often pursues peripheral interests of no value. He is a poor judge of character and is often socially inappropriate. He has had failed marriages since his lesion. In a casual interaction, though, he comes across as an avid conversationalist and as intelligent, charming, and witty. It is notable that many of EVR's most salient postoperative changes (short-term marriages, poor long-term planning, unreliability/irresponsibility, dependence) appear nearly verbatim on the PCL-R. Many additional cases of this nature have now been reported, and the main findings related to pseudopsychopathy are summarized next (see Koenigs & Tranel, 2006, for a summary).

The personality disturbances in patients with vmPFC damage are reminiscent of many of the core features of conventional "developmental" psychopathy, including shallow affect, irresponsibility, vocational instability, lack of realistic long-term goals, lack of empathy, and poor behavioral control (Hare, 1970). Also, general dysregulation of affect has been noted in both acquired and developmental psychopathy (Damasio et al., 2012; Scarpa & Raine, 1997; Zlotnick, 1999). Similar psychophysiological manifestations, including generally diminished autonomic responsiveness (especially to social stimuli), have been noted in patients with acquired sociopathy (Damasio et al., 1990; Tranel, 1994) and developmental psychopaths (Hare, 1970; Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000; Schmauk, 1970). Finally, developmental psychopaths manifest decision-making deficits on a reward-based decision-making task (the Iowa Gambling Task) that are reminiscent of those reported in adult-onset vmPFC patients (Mazas, Finn, & Steinmetz, 2000; Schmitt, Brinkley, & Newman, 1999).

We have studied a number of individuals who sustained damage to prefrontal cortices very early in life (Anderson, Bechara, H. Damasio, Tranel, & A. Damasio, 1999; Anderson, Barrash, Bechara, & Tranel, 2006; Anderson, Wisnowski, Barrash, Damasio, & Tranel, 2009; Taber-Thomas et al., 2014). The results of these investigations provide important corroboration of the notion that the vmPFC sector contains key structures supporting the acquisition of appropriate social conduct and moral behavior. The patients were studied with a variety of neuropsychological, neuroanatomical, and psychophysiological experiments, when they had grown up into adulthood. Notably, most of these patients were raised in stable, middle-class homes, and did not have siblings with behavior problems or any family history of psychiatric disease or risk factors for behavioral disturbance other than their brain injury. Thus, adverse genetic or environmental contributions to the patients' behavioral problems can be discounted. The patients with childhood-onset vmPFC lesions developed profound disturbances of social conduct and moral reasoning. Typical descriptions of these patients include: extreme interpersonal impairments; conspicuous lack of friends; stealing; lack of foresight; manipulative and shallow courteousness to adults; lack of concern or remorse; insensitivity to punishment; lewd and irresponsible sexual behavior; and egocentrism. The parallels between developmental psychopathy and acquired sociopathy suggest a common pathophysiologic mechanism. Dysfunction in lower mesial and orbital prefrontal cortices appears to be a likely culprit. However, it has been more difficult to flesh out the details of this story: clearly, neuronal damage in the critical regions (vmPFC generally) can lead to behavioral problems, but the mechanisms by which this occurs have not been identified. Nonetheless, there may very well be a genetically driven vulnerability that entails dysfunction of neurotransmitter systems in vmPFC that may be exacerbated by impoverished learning experiences and the addition of factors such as alcohol and substance abuse.

In both developmental psychopathy and the acquired variety, the condition or "syndrome" is not an all-or-none phenomenon. There are clear degrees of features and characteristics, and clear gradients in the manifestation of signs and symptoms. This is surely true of developmental psychopathy, where milder versions of the condition are not uncommon and in fact fairly adaptive for certain activities, such as pursuing a career in law enforcement or serving in the military. We have observed considerable variability as well in patients with acquired sociopathy, who tend to run a gamut of impairment from mild to severe.

Research on developmental precursors of psychopathy, such as callous-unemotional traits, has provided some insights into the genetic contributions to psychopathy. For example, evidence suggests that the heritability for conduct disorder with callous-unemotional traits is significantly higher than for conduct disorder without callous-unemotional traits (Viding, Jones, Frick, Moffitt, & Plomin, 2008). Genetic influences also appear to be large contributors to the stability of high levels of callous-unemotional traits across development (Fontaine, Rijsdijk, McCrory, & Viding, 2010). In molecular studies, GABRA2 has been repeatedly implicated in impulsivity, substance use, and psychopathy (Dick et al., 2013). Other novel variants, including DYRK1A, have also been implicated in adult psychopathy via GWA (Tielbeek et al., 2010).

Substance Use Disorders Substances of abuse encompass a diverse range, including narcotics, amphetamines, hallucinogens, alcohol, and innumerable other chemicals. This range, moreover, is only increasing with the rise in popularity of synthetic drugs, many of which are new and bridge different families of substances. With this diversity is an equally diverse range of neurobiological mechanisms of action—the precise neural substrates involved in one drug of abuse are different from that of another.

Despite this diversity of neurobiological substrates, many substance use problems are mediated through common pathways. Different substances often share similar biological risk factors, including genetic risk factors, and many of these risk factors are shared with other forms of psychopathology, especially externalizing psychopathology (Iacono, Malone, & McGue, 2008). Numerous substances act on a common set of neural substrates, moreover, and the development of substance use problems often involves common neurobiological pathways (e.g., Barrot et al., 2012; Everitt & Robbins, 2005; Hyman, Malenka, & Nestler, 2006).

Most neurobiological accounts of substance use disorder focus on dopaminergic pathways, especially the mesolimbic and mesocortical dopamine systems (including the nucleus accumbens, ventral tegmental area (VTA) and striatum). These areas are critical to reward processing, reinforcement learning, attention, and habit formation, consistent with the craving and seeking aspects of substance use problems. Dopaminergic neurons originate in the VTA and project onto the nucleus accumbens, where dopamine acts on its receptors. These regions also have numerous connections to brain regions involved in learning, emotion, and behavioral control, such as the amygdala, hippocampus, and prefrontal cortex.

Many drugs of abuse have direct or indirect effects on the mesolimbic dopamine pathway, such as keeping dopamine in synapses by inhibiting its reuptake (e.g., cocaine), activating production of dopamine in the VTA (e.g., nicotine), or inhibiting "breaks" on dopamine production in the VTA (e.g., opiates; Barrot et al., 2012; Hyman, Malenka, & Nestler, 2006). The mesolimbic dopamine pathway has also been shown to be critical to reinforcement, especially motivation for reward. Studies involving inhibition of dopamine receptors in this pathway suggest that the pathway is important for the experience of reward and reinforcement learning, but not habit formation *per se*, which involves other areas of the brain (Everitt & Robbins, 2005). These studies suggest individuals can

continue habitual substance-seeking behavior even when the substance is no longer reinforcing, paralleling the experience of many individuals with drug use problems who continue to seek substances even when they no longer find them pleasurable.

Individuals with substance use problems have greater brain activity in the nucleus accumbens when exposed to substances of abuse, and exhibit lower dopamine binding in that region (e.g., Heinz et al., 2004). This lower dopamine binding in the nucleus accumbens occurs prior to any substance exposure, and is also associated with disinhibited, impulsive behavior. Rats bred to be impulsive, for example, exhibit lower dopamine binding in the nucleus accumbens prior to drug exposure, and are more likely to seek drugs after exposure (Dalley, Mar, Economidio, & Robbins, 2007). Similar findings have been found in humans as well (e.g., Buckholtz et al., 2010; Casey et al., 2014). These findings suggest that the lower dopamine binding associated with substance use problems reflects a risk for substance use, rather than a consequence, and is shared with multiple externalizing problems, such as impulsivity and decision-making deficits.

Not all neurobiological substrates of substance use are shared between disorders. Some evidence suggests that this may be especially true for initial use rather than problems, in that substance-specific genetic influences may act on initiation and recreational use rather than the problems themselves (Kendler, Jacobsen, Prescott, & Neale, 2003). One interpretation of this finding is that substance-specific genetic influences shape individual's preferences for one drug versus another through the experience of using that drug, whereas genetic influences on problems tend to act on general reward pathways such as the mesolimbic dopamine system. Examples of this are genes related to alcohol processing, such as the alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) genes: alcohol is processed via ADH into acetylaldehyde, a major contributor to the experience of hangover, which in turn is degraded by ALDH. Depending on their versions of ADH and ALDH genes, an individual may therefore experience better or worse hangovers, which may shape their choice to use alcohol at all (Whitfield, 1997). The extent of other substance use, or the transition to problems, however, might be governed by other genetic mechanisms that act on common pathways such as the mesolimbic dopamine system.

Delineating the effects of substance use on neuropsychological functioning can be challenging. It can be difficult to distinguish the effects of substances from preexisting risk factors and to isolate the effects of one substance from another (given that use of multiple substances is common, especially with severe use; Fernandez-Serrano, Perez-Garcia, & Verdejo-Garcia, 2011). For example, observational studies have indicated that chronic, long-term cannabis use is associated with declines in cognitive functioning, even after cessation of use (Grant, Gonzalez, Carey, Natarajan, & Wolfson, 2003; Meier et al., 2012). However, etiologically informative studies, such as twin and family studies, have indicated that there are no permanent effects of cannabis use on cognitive or other functioning once genetic and background environmental risk factors are controlled for (Eisen et al., 2002; Lyons et al., 2004). That is, the observed associations appear due to background factors, such as preexisting cognitive impairments, or variables that predispose one to use cannabis as well as develop cognitive deficits. Similar complications arise in interpreting the association between adolescent cannabis use and psychosis: although adolescent cannabis use is associated with later psychosis, it is unclear whether this association is limited to those with genetic liability for psychosis (i.e., as a form of gene-environment interaction), or the cannabis use reflects genetic liability for psychosis (i.e., as a form of gene-environment correlation; van Winkel & Kuepper, 2014).

Neurodevelopmental Disorders While nearly all disorders described in the DSM or other nosological systems can be considered developmental disorders in some way, DSM-5 describes a specific set as neurodevelopmental disorders, owing to their strong genetic and neurobiological influence, their emergence in early childhood, and relative persistence throughout the life course. These disorders include impairments in intellectual functioning, communication and language development, and learning. However, we next turn our attention to a common neurodevelopmental disorder, both in terms of prevalence as well as mental health referrals: ADHD.

ADHD is one of the most commonly diagnosed disorders that emerges in childhood and is currently the leading cause for referral of children to mental health services in the United States. ADHD comprises two correlated, but partially separable symptom dimensions that include inattention–disorganization and hyperactivity–impulsivity that are both developmentally inappropriate and cause significant impairments across contexts (American Psychiatric Association, 2013). Importantly, the clinical presentation of ADHD is quite heterogeneous (both within time and across development), although the condition has a relatively chronic course in most cases. This clinical heterogeneity is thought to reflect diversity in its underlying causal mechanisms, which likely involve numerous genetic and neurobiological processes (Nigg, 2005).

Genetic influences have been long suspected to play an important role in ADHD. Family studies (Faraone et al., 2000), as well as examination of twin concordance and heritability, have demonstrated that genetic contributions to ADHD symptom dimensions are moderate to large, accounting for approximately 70–75% of their variance (Nikolas & Burt, 2010; Sherman, McGue, & Iacono, 1997) and appear to be high at all levels of symptomatology (Willcutt, Pennington, & DeFries, 2000) and consistent across development (although some studies have reported substantially lower heritabilities for ADHD among adults, see Larsson, Chang, D'Onofrio,

& Lichtenstein, 2013). However, a recent developmental twin investigation revealed that genetic influences on ADHD symptom dimensions remain high from childhood to adulthood and that additional genetic factors appear to come "online" for ADHD during adolescence in particular (Larsson et al., 2013).

Regarding specific genetic markers, small but significant associations have been found between ADHD and genes related to dopamine neurotransmission, particularly the dopamine transporter gene (DAT1; SLC6A3) and the dopamine D4 and D5 receptor genes (Gizer, Ficks, & Waldman, 2009). The dopamine system has long been of interest, owing to the hypothesized action of psychostimulants on dopamine neurotransmission in the successful treatment of ADHD symptoms (Wilens, 2006). As mentioned previously, however, there is substantial variability in genetic association effect sizes across studies, and overall effects appear to be quite small (accounting for 1% or less of the variance). Multiple GWA studies have also been completed for ADHD (see Ebejer et al., 2013; Neale et al., 2010). Although meta-analyses of findings have not indicated any markers or regions surviving corrections for genome-wide significance (although samples of *n*~3,000 cases and *n*~3,000 non-ADHD controls remains relatively small for such investigations), several genetic regions and candidates of interest have been identified, including markers on chromosomes 7, 8 and 11 (Neale et al., 2010) that appear to influence the development of neuronal architecture (Lee & Song, 2014). Additionally, rare genetic variants, including large copy-number variants have also been associated with ADHD in large GWA studies (Yang et al., 2013); these regions of large deletions or duplications in the DNA appear to contain genes important for a variety of neurological function, including central nervous system development and synaptic transmission (Williams et al., 2010).

Future gene discovery work in ADHD is now proceeding in tandem with genetic investigation of other disorders (e.g., schizophrenia, bipolar, autism, depression) as part of the Psychiatric GWAS Consortium. In addition to molecular gene discovery and genome-wide association work, there is also substantial interest in gene-environment interplay and ADHD, including interactions between genetic and environmental factors (Nigg, Nikolas, & Burt, 2010), as well as the ways in which prenatal exposures may exert programming effects on the developing brain via epigenetic mechanisms. Both such mechanisms may be particularly relevant for understanding the emergence of neurodevelopmental disorders during early childhood (Nigg, 2012).

Decades of neurobiological investigations have also been informative for understanding the nature of ADHD. Alterations of numerous brain structures have been observed among individuals with ADHD relative to non-ADHD comparison individuals, including the frontal lobes, basal ganglia, corpus callosum, and cerebellum (Castellanos et al., 2002; Giedd et al., 1994). Imaging studies have also provided support for the involvement of right frontal-basal ganglia circuitry in ADHD, which also appears to involve modulatory inputs from the cerebellum (Giedd & Rapoport, 2010). Longitudinal investigations of brain maturation in ADHD have also provided evidence of a developmental delay in cortical thickness among youth with ADHD relative to their non-ADHD counterparts, as well as findings indicating the normalization of cortical volumes may be associated with parallel improvements in behavioral symptoms (Giedd & Rapoport, 2010). Multiple pathway models of ADHD etiology have been proposed which link inattention symptoms to executive dysfunction underpinned by frontal-striatal circuitry, whereas hyperactivity symptoms may be linked toward deficits in motivation and reward response related to frontal-limbic connections (Sonuga-Barke, 2005). Additionally, problems in environmental adaption observed among individuals with ADHD have been hypothesized to be linked to deficits in signaling from the prefrontal cortex to both subcortical and posterior systems (Nigg & Casey, 2005).

Concluding Comments and Emerging Trends

The impact of neurobiological and genetic processes on psychopathology is wide ranging and robust. Construction of comprehensive models that incorporate the neurogenetic origins of psychopathology will be crucial for further refinement and development of assessment and treatment approaches. Recognition of this trend is evident through large-scale investments aimed to investigate these mechanisms, whether it be the Human Genome Project, the RDoC matrix pushed out by NIMH, or the designation of a "Decade of the Brain." We conclude by providing a brief window into what these endeavors might look like in the future.

The Emergence of Social Neuroscience "Social neuroscience" is a hybrid term that has been used to describe a melding of the fields of social psychology and sociology, on the one hand, and neuroscience, on the other. Both parts of the rubric—"social" and "neuroscience"—refer to disciplines that are perhaps more timely and more relevant now than at any point in history. The evolution of humankind has led to dramatic increases in pressures on social functioning, as more people live in closer proximity and the demands for social acumen—even just to navigate through daily life—increase exponentially. In fact, it is estimated that more than two-thirds of the world population will live in urban areas by the year 2050 (Dye, 2008).

Densely populated urban areas make constant, profound demands on social functioning. In such environments, social skills are a necessity to function successfully in mainstream life. And it is no accident that psychiatric disorders of socialization—as seen especially prominently in autism spectrum disorders and various types of personality disorders—have pushed their way into the forefront

of public consciousness The effects of urbanization on mental health have been documented in meta-analyses, which show, for example, substantial increases in risks for mood and anxiety disorders in urban dwellers (Peen, Schoevers, Beekman, & Dekker, 2010). Schizophrenia, too, is far more common in persons raised in urban environments, and there is even evidence that this relationship is causal (Krabbendam & van Os, 2005). Neural correlates of the effects of urban upbringing on social stress processing have been identified—e.g., urban dwellers have distinct social stress responses (in fMRI experiments) in the amygdala and perigenual anterior cingulate cortex (Lederbogen et al., 2011). Thus, there is now clear evidence that increased demands on social skills not only take a toll on mental health, but also have an effect on neural processors linked to social and emotional functioning. As global society continues to urbanize in the decades ahead (if current trends continue, and there are few signs that they won't), the importance of social neuroscience will only continue to grow.

With the ongoing trends toward urbanization and high population density, and ever-increasing globalization, the importance and relevance of social aptitude may only increase. This, in turn, could be expected to put greater focus on the neural basis of social skills, especially in regard to various neurally based developmental and acquired disorders that can affect social functioning. In fact, one could envision that the domain of social functioning will gain a prominence—and level of detail—akin to what has long characterized the domain of language and aphasia. For example, what are now known as autism spectrum disorders might be understood as related, but distinct, specific disorders, with partially distinct neuroanatomical underpinnings. The same might become true of various syndromes of personality disorders, such as antisocial personality disorder and borderline personality disorder, which now comprise fairly heterogeneous sets of manifestations and diverse arrays of neuroanatomical correlates (Tranel, 2013).

Genetic Consortiums and Search for Pleiotropic Effects Technological improvements in genotyping platforms along with recognition of the importance of considering variation throughout the genome have fueled the progression to GWA studies and gene network identification. However, many of these efforts to identify novel genetic variants are now proceeding to examine genetic *pleiotropy*, or genetic effects that may be common to a range of psychiatric disorders. Efforts that concurrently examine association among as many as 1,000,000 SNPs and a range of psychiatric conditions (i.e., schizophrenia, bipolar disorder, depression, ADHD; Hall et al., 2014). Results from this work have implicated several genetic variants that may have pleiotropic effects—that is, these variants may influence vulnerability to psychiatric problems more broadly (e.g., L-type voltage-gated calcium channel subunits CACNA1C and CACNB2 within neurons), which may then exert their influence on psychopathology via changes in neuronal function and communication. Additionally, rare copy-number variants have also been identified which may have large effects for neurodevelopmental disorders, including autism spectrum disorders and ADHD (Menache et al., 2013).

Still, despite enormous gains in molecular genetic research, genes alone are unlikely to completely account for the etiology of psychopathology. Genetic variants identified in GWAS account for only a small proportion of the variability in psychiatric conditions. Now large-scale GWA studies can use data from multiple related individuals to estimate SNP heritability, or the proportion of genetic effects in different disorders, which are likely due to variation (in total) across these bases. Even though heritability estimates from twin studies are high, heritabilities due to variations in SNPs are much lower around 30–40% (Yang et al., 2013). This gap in heritability between twin estimates and from SNPs (or what has been termed "missing heritability") may be, in fact, due to gene–environment interplay processes, including gene–environment interaction, correlation, and epigenetic effects. Thus, future work in the genetics of psychopathology will likely require consideration genetic variants that increase general liability for psychopathology as well as the potential ways in which environmental experiences throughout development may then alter gene expression and brain development. Such work is already proceeding through the efforts of the ENIGMA consortium.

Attempts to Move Toward a More Biologically Informed Classification of Psychopathology As mentioned earlier, official psychiatric nosology is undergoing a period of significant transition. Correspondingly, there is significant disagreement about this transition—how much change should occur and what the nature of that change should be. However, one major change often advocated is that psychiatric classification more closely reflect advances in our understanding of the biological basis of psychopathology, such as those discussed here. Some have even gone so far as to argue that classification of psychopathology should be primarily based on biological considerations (e.g., Insel et al., 2010). NIMH's RDoC initiative, discussed previously, is a major example of this push to organize description and classification of mental illness around its biological substrates. Psychopathology constructs used in assessment, treatment, and research probably will become more biologically oriented, and less phenomenological or behavioral in nature.

Whether or not this occurs is uncertain, however. Similar calls in the 1970s for a more biologically informed nosology formed the basis for the *Research Diagnostic Criteria*, DSM-III, and by extension, our current diagnostic systems (e.g., Blashfield, 1982)—the very same systems that are now being criticized for not being biologically informed enough. Just as those approaches suffered from paradigmatic errors (e.g., overgeneralized

assumptions that psychiatric illness consists of monogenic, discrete diseases), it is likely current approaches to "pure" biological classification will be seen in the near future as suffering from their own limitations as well.

As many have noted, it is unlikely that a description of psychiatric illness focused solely on one level of explanation will ever be productive (Kendler, 2012, 2014). The causes of psychopathology operate at multiple levels, from the molecular, neurobiological, and physiological, to the developmental, social, and cultural. Furthermore, factors operating at different levels may dynamically influence one another. As such, an authoritative description and classification of psychopathology (such as the DSM, ICD, or RDoC) focused on one level exclusively will almost certainly be limiting, if not in accounting for what is known, then in accommodating progress in theory and research. Science generally "finds a way" around such limitations, regardless of whether or not official nosology recognizes it; the burden is on those who develop formal nosologies to accommodate science in this regard, not the other way around (Markon, 2013).

Nevertheless, as research into causes of psychopathology progresses, and paths from molecular to cultural influences on mental illness are delineated more clearly, the constructs of focus will certainly shift as well. Although a focus on biological levels of description and explanation may not be sufficient for a comprehensive understanding of psychopathology, it is necessary. In this regard, the set of constructs used to discuss mental illness will likely diversify and increase in the future, if not in the precise manner articulated by initiatives such as RDoC, then along similar lines. The task for those who study psychopathology is to understand how to accommodate and integrate these different levels of explanation to produce a fuller account of mental illness.

Notes

1. This arrangement would apply to the typical left-verbal, right-nonverbal hemispheric specialization found in the vast majority (about 90–95%) of right-handed persons and also the majority (about 65–70%) of left-handed persons.
2. "Afferents" (inputs) are neurons that carry information into or toward a particular structures; "efferents" (outputs) are neurons that carry information out of or away from a particular structure.
3. We have used the term "ventromedial prefrontal cortex" to designate the medial parts of the orbitofrontal cortex and the ventral parts of the mesial prefrontal cortices. These terms are not interchangeable, but they do overlap in their anatomical implications (see Naqvi, Tranel, & Bechara, 2006).

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4

Cultural Dimensions of Psychopathology

The Social World's Impact on Mental Disorders

Steven R. López and Peter J. Guarnaccia

Over the past several decades, researchers have increasingly examined cultural influences in psychopathology. However, for much of this period, the study of culture and mental disorders was a marginal field of inquiry. As we demonstrate in this chapter, cultural issues have moved to the fore in the study of psychopathology. A landmark event marking this transition came when Kleinman (1977) heralded the beginning of a "new cross-cultural psychiatry," an interdisciplinary research approach integrating anthropological methods and conceptualizations with traditional psychiatric and psychological approaches. Mental health researchers were encouraged to respect indigenous illness categories and to recognize the limitations of biomedical illness categories, such as depression and schizophrenia. The new cross-cultural psychiatry also distinguished between disease, a "malfunctioning or maladaptation of biological or psychological processes", and illness, "the personal, interpersonal, and cultural reaction to disease" (p. 9). The perspective that Kleinman and others (Fabrega, 1975; Kleinman, Eisenberg & Good, 1978) articulated in the 1970s reflected an important direction for the study of culture and psychopathology—to understand the social world within mental illness (see also Marsella, 1980; Draguns, 1980).

Many advances were made during the first decade of the new cross-cultural psychiatry. One was the establishment of the interdisciplinary journal, *Culture, Medicine, and Psychiatry*. This journal, in conjunction with *Transcultural Psychiatry* in Canada (formerly *Transcultural Psychiatric Research Review*), provides an important forum for cultural research in psychology and psychiatry. In addition, during the 1980s, large-scale epidemiologic studies were carried out. The second multinational WHO study of schizophrenia was launched and preliminary findings were reported (Sartorius et al., 1986) and the Epidemiological Catchment Area (ECA) studies were conducted (Regier et al., 1984). Some may question how culturally informed these classic studies were (Edgerton & Cohen, 1994; Fabrega, 1990; Guarnaccia, Kleinman, & Good, 1990). However, most reviews of culture, ethnicity, and mental disorders still refer to the findings from the WHO and ECA studies to address how social, ethnic, and cultural factors are related to the distribution of psychopathology. Also during this time, the National Institute of Mental Health funded research centers with the sole purpose of conducting research on and for specific ethnic minority groups (African Americans, American Indians, Latino Americans, and Asian Americans). Some of the research from these centers contributed to the growing cultural psychopathology database (e.g., Cervantes, Padilla, & Salgado de Snyder, 1991; King, 1978; Manson, Shore & Bloom, 1985; Neighbors, Jackson, Campbell, & Williams, 1989; Rogler, Malgady, & Rodriguez, 1989; Sue, Fujino, Hu, Takeuchi, & Zane, 1991).

Dialogues across disciplines were also initiated during this time. For example, Kleinman and Good's (1985) influential volume, *Culture and Depression*, brought together the research of not only anthropologists, but also psychologists and psychiatrists as well as historians and philosophers. Another significant indicator of the field's development was and continues to be its success in attracting new investigators. In sum, these first 10 years can be characterized as an exciting and fertile time for the emergence of the field of cultural psychopathology.

Despite the many advances, the field's main messages were not reaching larger audiences. Investigators were communicating primarily among themselves in their specialty journals and books. On a rare occasion, one would find a special issue on cultural research in a mainstream journal. Those findings that did manage to

be published in widely distributed journals were scattered among a broad array of journals. Thus, from the perspective of mainstream investigators, the developments of the new cross-cultural psychiatry went largely unnoticed. A telling example of this occurred at a joint meeting of the DSM-IV Task Force and the Culture and Diagnosis Work Group in 1991. After two days of intensive discussion of the potential contributions of cultural psychopathology research to the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994), the chair of the DSM-IV Task Force commented that this was really interesting work and that the members of the Culture and Diagnosis Work Group should publish this research—this 3 years after Kleinman's (1988) *Rethinking Psychiatry* had appeared! In this book, Kleinman provided a comprehensive review of culture, psychopathology, and clinical research. Drawing on empirical data and theory, he argued that culture matters for the study and treatment of mental disorders. This volume serves as a significant marker in the development of the new cross-cultural psychiatry or cultural psychopathology.

Key Developments

Conceptual Contributions

Definition of Culture Central to the study of cultural psychopathology is the definition of culture. Much of the past and even current research relies on a definition of culture that is outdated. In fact, Betancourt and López (1993) wrote a critical review of cultural and psychological research in which culture was defined as the values, beliefs and practices that pertain to a given ethnocultural group. The strength of this definition is that it begins to unpack culture. Instead of arguing that a given expression of distress resides within a given ethnocultural group, for example, researchers argue that the expression of distress is related to a specific value or orientation. We see this as a significant advancement. It helps researchers begin to operationalize what matters about culture in the specific context. Further, it recognizes the heterogeneity within specific ethnocultural groups. Knowing that someone belongs to a specific ethnic group suggests the possibility of cultural issues in psychopathology, but it does not imply that that person adheres to the prototypic cultural values and practices of that group.

At the same time, this definition of culture as values, beliefs, and practices has significant limitations (see Good, 1994 on beliefs; Lewis-Fernandez & Kleinman 1995). One limitation is that this definition conceives of culture as residing largely within individuals. The emphasis on values and beliefs points out the psychological nature of culture. In contrast, we argue that culture is manifested in the interaction between people and is highly social in nature. Situating practices (customs and rituals) within values and beliefs gives the impression that the practices in the social world are a function of values and beliefs. For example, people are thought to rely on their family in times of crisis because they are high in *familism* or family orientation. Investigators rarely examine what about the social world facilitates or impedes reliance on family members. Perhaps harsh environmental conditions contribute to families coming together to overcome adversity. When applying the values and beliefs definition of culture, the social world is subjugated to the psychological world of the individual. Contrary to this perspective, we argue that it is action in the social world that produces culture as much as people's ideas about the world. In our view, the social world interacts on an equal footing with the psychological world in producing human behavior.

A second important limitation of this frequently used definition of culture is that it depicts culture as a static and bounded phenomenon. We assert that culture involves process and change and that culture is constantly in flux both in its regions of origin and as people bring their cultures with them as they move around the globe (Garro, 2001; Greenfield 1997; Guarnaccia & Rodriguez 1996). Attempts to freeze culture into a set of generalized value orientations or behaviors will continually misrepresent what culture is. Culture is a dynamic and creative process, some aspects of which are shared by large groups of individuals resulting from particular life circumstances and histories. Given the changing nature of our social world and given the efforts of individuals to adapt to such changes, culture can best be viewed as an ongoing process, a system or set of systems in flux (see also Hong, Morris, Chiu, & Benet-Martinez, 2000).

A related limitation of the values-based definition of culture is that it depicts people as recipients of culture from a generalized "society" with little recognition of the individual's role in negotiating their cultural worlds (Garro, 2000). More recent approaches to culture in anthropology, while not discarding the importance of a person's cultural inheritance of ideas, values, and ways of relating, have focused equally on the emergence of culture from the life experiences and interactions of individuals and small groups. People can change, add to, or reject cultural elements through social processes such as migration and acculturation. A viable definition of culture acknowledges the agency of individuals in establishing their social worlds.

In sum, current views of culture attend much more to people's social world than past views of culture that emphasized the individual. Of particular interest are people's daily routines and how such activities are tied to families, neighborhoods, villages and social networks. By examining people's daily routines one can identify what matters most to people (Gallimore, Goldenberg, & Weisner, 1993), or what is most at stake for people (Kleinman, 2006). Furthermore, this perspective captures the intersubjective and interactional dimensions of culture as it is both a product of group values, norms and experiences, as well as individual innovations and life histories. The use of this broader definition of culture should

help guide investigators away from flat, unidimensional notions of culture, to discover the richness of a cultural analysis for the study of psychopathology. An important component of this perspective is the examination of intra-cultural diversity (Pelto, P. J. & Pelto, G. H. 1975). In particular, social class, poverty and gender continue to affect different levels of mental health both within cultural groups and across cultural groups.

Goals of Cultural Research Culture is important in a number of domains within psychopathology research. One domain is the expression of disorder and distress. A cultural analysis can point out the variability in the manner in which mental illness is manifested. Social and cultural factors can also affect the etiology and prevalence of disorder by differentially placing some at more risk than others for developing psychopathology. In addition, the course of disorder, as reflected in the degree of disability, or in the number of clinical relapses is also related to important cultural factors. We want to encourage all of these lines of inquiry.

Regardless of the specific domain of research, there are two meta-goals of cultural research. Some writers imply that cultural research should test the generality of given theoretical notions. For example, in a thoughtful analysis of cultural research, Clark (1987) noted: "Conceptual progress in psychology requires a unified base for investigating psychological phenomena, with culture-relevant variables included as part of the matrix." (p. 465). From Clark's point of view, cross-cultural work can serve to enhance the generality of given conceptual models by adding, when necessary, cultural variables to an existing theoretical model to explain between group and within-group variance. Although Clark acknowledges the possibility that a construct developed in one country may not have a counterpart in another country, at no time does she discuss the value of deriving models of distinct clinical entities found in only one country or ethnocultural group. This suggests that, for Clark, the main purpose of studying culture is to reinforce the universality of existing psychological models by subsuming cross-cultural variations into the mainstream models.

In contrast, both Fabrega (1990) and Rogler (1989) criticize researchers for attending insufficiently to the cultural specificity of mental illness and mental health. Fabrega examines researchers' use of mainstream instruments and conceptualizations in studying mental disorders among Latinos and challenges such researchers to be bold in their critiques of "establishment psychiatry." Rogler recommends a framework for mainstream psychiatric researchers that attend more fully to culture. For both Fabrega and Rogler, the risk of overlooking cultural variations is much greater in current psychopathology research than the risk of overlooking cultural similarities. Focusing on culture-specific phenomena at this time is of central importance to the further development of the field.

An important conceptual advancement is the recognition of both positions; that is, studying culture to identify general processes, and studying culture to identify culture-specific processes. By focusing only on generalities, we overlook the importance of culture-specific phenomena. On the other hand, by emphasizing culture-specific phenomena we overlook the possibility of generalities. The overall purpose of cultural research, therefore, is to advance our understanding of general processes, culture-specific processes, and the manner in which they interact in specific contexts (see also Beals et al., 2003; Draguns, 1990). Our aim is to identify culture's mark amidst the ubiquity of human suffering.

Major Advances We now turn to selected developments over the past 25 years in the study of culture and psychopathology. We begin with a discussion of four of the most important projects that were carried out since 1988: the incorporation of cultural factors in DSM-IV, and the continued development of cultural issues in the fifth edition (DSM-5; American Psychiatric Association, 2013); the publication of the *World Mental Health Report* (Desjarlais, Eisenberg, Good, & Kleinman, 1996); the release of the U.S. Surgeon General's Supplemental report on *Mental Health: Culture, Race and Ethnicity* (U.S. Department of Health and Human Services, 2001); and the completion of the Collaborative Psychiatric Epidemiology Surveys.

The National Institute of Mental Health (NIMH) funded the establishment of a Culture and Diagnosis Work Group to inform the development of the DSM-IV. The work group's efforts resulted in three main contributions to DSM-IV: (a) inclusion of some discussion of how cultural factors can influence the expression, assessment, and prevalence of disorders in each of the disorder chapters; (b) an outline of a cultural formulation of clinical diagnosis to complement the multiaxial assessment; and (c) a glossary of relevant cultural-bound syndromes from around the world. A more complete documentation of the Work Group's findings and contributions is available from three sources: (a) a special volume derived from the group's deliberations (Mezzich et al., 1997); (b) volume 3 of the *DSM-IV Sourcebook* (Widiger et al., 1997); and (c) other publications (e.g., Alarcón, 1995; Kirmayer 1998; and Mezzich, Kleinman, Fabrega, & Parron, 1996). Another major impact of the Culture and Diagnosis Work Group was the bringing together of a large number of junior and senior investigators that brought new energy to the field of cultural psychopathology. Without a doubt the attention given to culture in DSM-IV is a major achievement in the history of the classification of mental disorders. Never before had classification schemas or related diagnostic interviews addressed the role of culture in psychopathology to this degree (López & Núñez 1987; Rogler 1996; see Kirmayer, 1998, Lewis-Fernandez & Kleinman, 1995, Mezzich et al., 1999, for a discussion of the limitations of how DSM-IV considered culture).

The cultural considerations for DSM-5 build on the foundation provided by DSM-IV, particularly in the areas of the conceptualization of cultural concepts of distress and the cultural formulation interview. The introductory section to the new manual (see pp. 14–15 of DSM-5) includes a brief, but important section on cultural issues. This section makes important points about culture's role in shaping the patient and family's acceptance of diagnosis and treatment and the conduct of the clinical encounter. The introduction also previews a new conceptualization of cultural conceptions of distress.

Cultural conceptions of distress are divided into three categories (see pp. 758–9 of DSM-5). One broad category includes cultural syndromes, which are defined as a cluster or group of co-occurring symptoms that represent a cultural pattern of distress. *Ataques de nervios*, which we discuss later in this chapter, is a prototypical cultural syndrome. Cultural idioms of distress are ways of talking about suffering, but they are not associated with a core set of syndromes or causes. They are a more diffuse experience that communicate personal and social distress, such as *nervios* among Latinos. The third category is cultural explanation or perceived cause of illness, which involves an explanatory model of distress focused on specific causal factors rather than on a core of symptoms or experiences; *susto* is a quintessential example of this type of idiom. The DSM-5 also includes a glossary of cultural concepts of distress (pp. 833–7) that includes nine well-researched concepts from a range of cultures.

The DSM-5 also incorporates a much more developed cultural formulation interview (CFI; see pp. 749–59 of DSM-5) than noted in DSM-IV. The interview is highly structured so that it is much more useable by clinicians. The interview also has been divided into several modules so that it can be used more flexibly by clinicians. The core interview is a set of 16 questions focused on clinical assessment. In addition to a version of this interview for the patient, there is another version for a key informant to answer about the identified patient. The sections of the core interview include: cultural definition of the problem; cultural conceptions of cause, context and support, including cultural identity; cultural factors affecting self-coping and past help seeking; and cultural factors affecting current help seeking. Supplementary modules include more detailed versions of the subsections of the core interview, as well as an explanatory model interview, a culturally sensitive interview about levels of functioning, a module to assess social networks, and a section on spirituality, religion and moral traditions. Modules have also been developed for special population groups such as children and adolescents, older adults, immigrants and refugees, and caregivers. These modules are available from the American Psychiatric Association for research and evaluation purposes. The CFI has undergone field tests in several sites in the United States as well as in India, Peru, the Netherlands, Canada and Kenya (New York State Psychiatric Institute, 2011). These features of the DSM-5 represent important advances in the conceptualization and study of cultural psychopathology.

A second major development within the last two decades was the publication of the *World Mental Health Report* (Desjarlais et al., 1996). Desjarlais and colleagues compiled research from across the world to identify the range of mental health and behavioral problems (e.g., mental disorders, violence, suicide), particularly among low-income countries in Africa, Latin America, Asia, and the Pacific. The authors derived several conclusions. Perhaps the most significant was that mental illness and related problems exact a significant toll on the health and wellbeing of people worldwide, and produce a greater burden based on a "disability-adjusted life years" index than that from tuberculosis, cancer, or heart disease (see Murray & Lopez, 1996). Among all physical and mental disorders, depressive disorders alone were found to produce the fifth greatest burden for women and seventh greatest burden for men.

Another important observation was that mental disorders and behavioral problems are intricately tied to the social world and occur in clusters of intimately linked social and psychological problems. For example, the authors identified the social roots of the poor mental health of women. Among the many factors include hunger (undernourishment afflicts more than 60% of women in developing countries), work (women are poorly paid for dangerous, labor-intensive jobs), and domestic violence (surveys in some low-income communities worldwide report that up to 50% and 60% of women have been beaten). The research on women's mental health illustrates that psychopathology is as much pathology of the social world as pathology of the mind or body.

Based on their findings, Desjarlais and colleagues make specific recommendations to advance both mental health policy and research to help reduce the significant burden of mental illness across the world. Their consideration of the social world leads easily to recommending specific interventions to address not only mental health problems but also the social conditions that surround and greatly influence such problems. In addressing the poor mental health of women, for example, they call for coordinated efforts to empower women economically and educationally, as well as to reduce violence against women in all its forms. In addition, women's mental health is identified as one of the top five research priorities worldwide. They call for research to examine the social factors that influence women's health in specific cultural contexts and to identify effective community based interventions in improving their health status (see also Winstead & Sanchez, Chapter 5 in this volume).

The third development was the *Surgeon General's Supplemental Report on Mental Health* concerning culture, race, and ethnicity (U.S. Department of Health and Human Services, 2001). The Surgeon General first published a landmark report on the status of the nation's mental health (U.S. Department of Health and Human Services, 1999). Some observers were concerned that

insufficient attention was given to the mental health of the country's ethnic and racial minority groups (Chavez, 2003; López, 2003). In response to this concern, and under the leadership of the Substance Abuse and Mental Health Services Administration, the Surgeon General published a report on the mental health of the nation's four main minority groups: American Indians/Alaska Natives, African Americans, Asian Americans/Pacific Islanders, and Latino Americans. Although the report's focus was mental health care, considerable attention was given to our current understanding of the types of psychopathology common among these groups, based largely on epidemiological and clinical research. One of the major contributions of this report was the synthesis of literature on the mental health of these diverse groups.

The main message of the Surgeon General's report was that "culture counts." "The cultures from which people hail affect all aspects of mental health and illness, including the types of stresses they confront, whether they seek help, what types of help they seek, what symptoms and concerns they bring to clinical attention, and what types of coping styles and social supports they possess." (U.S. Department of Health and Human Services, 2001, p. ii) The Surgeon General's report compiled the best available research that culture matters in these domains. For example, evidence was reviewed regarding the relationship between racism and mental health (e.g., Kessler, Mickelson, & Williams, 1999), and ethnicity and psychopharmacology (e.g., Lin, Poland, and Anderson, 1995). In addition, the report outlined future directions to address the mental health needs of these underserved communities, including expanding the science base and training mental health scientists and practitioners. In all, this report served two important functions. It brought together the best available mental health research regarding the main U.S. minority groups. Also, because of the status and visibility of the Office of the Surgeon General, the report alerted the nation to the mental health needs of the four main ethnic and racial minority groups.

The Collaborative Psychiatric Epidemiology Surveys (CPES) represent the fourth and final major development that is highlighted. The CPES comprises three nationally representative surveys of both majority and minority groups in the U.S. Similar instruments and methods were used to foster cross-survey comparisons. The National Comorbidity Survey-Replication (Kessler et al., 2004) was carried out between 2001 and 2003 and was based on a probability sample (N = 9,282) of the United States (the 48 contiguous states) in which Euro-Americans were primarily represented (73%) with much smaller proportions of minority group members (non-Hispanic Blacks 12%, Hispanics 11%, and others 4%). This sample was composed of English-speaking individuals 18 years and older. The National Survey of American Life (Jackson et al., 2006) was conducted between 2001 to 2003, and focused on national samples of African origin adults including African Americans (N = 3,570), and Afro-Caribbeans (N = 1,623). In addition, an adolescent sample of 1,200 13- to 17-year-old African Americans and Afro-Caribbeans was studied. Including samples of African Americans and Afro-Caribbeans enabled the examination of the heterogeneity of persons of African origin. The National Latino and Asian American Study (NLAAS; Alegría, Takeuchi et al., 2006) was the first mental health survey of nationally representative samples of Latinos (N = 2,554) and Asian Americans (N = 2,095). These surveys were of persons 18 years and older and were conducted during 2002 to 2003 (Alegría et al., 2008; Abe-Kim et al., 2007). Among the strengths of these surveys were that non-English-speaking persons (Spanish, Tagalog, Chinese, and Vietnamese) were included, as well as individuals from several subethnic groups. This enabled the examination of language and within ethnic group differences.

The contributions of the CPES have been most significant. For the first time we have nationally representative samples of three of the major U.S. racial/ethnic minority groups (American Indians are notably missing) and can compare their prevalence rates with that of Euro-Americans with minimal concern for differences in methodologies or representativeness of samples. In addition, we have data for subracial or subethnic groups to examine the importance of within-group variability. A study of major depression carried out by Gonzalez, Tarraf, Whitfield, and Vega (2010) reflects both of these important strengths. First, they find that across most of the racial/ethnic groups there is a healthy immigrant effect in which immigrants have lower prevalence rates of depression than U.S.-born adults of the same ethnic/racial background. Second, as noted previously by other authors (e.g., Alegría et al., 2008), the healthy immigrant effect does not apply to all groups, especially Cubans and Puerto Rican Islanders. A third important finding is that the healthy immigrant effect does not hold for older adults; the immigrant effect appears to "yield to the overwhelming effects of socioeconomic disadvantages in later years" (p. 1050). Although these three findings concern only major depression, they reflect some of the richness of the CPES. Studies based on the CPES will likely continue to contribute to our understanding of the sociocultural context and psychopathology.[1]

Together, DSM-IV, DSM-5, the *World Mental Health Report*, the *Surgeon General's Supplemental Report on Mental Health*, and the CPES make major contributions to the study of culture and psychopathology, and give important visibility and credibility to this important domain of research. At the same time, they illustrate the range of conceptualizations of culture and the importance of the social domain. For DSM-IV, culture tends to be depicted as exotic, by referring to the noted culture-bound syndromes that reside largely among persons from "culturally different" groups. There is little attention paid to the influence of culture in every clinical encounter—influencing the client, provider, and the broader community context, regardless of the patient's ethnic or racial background.

These limitations have been corrected somewhat in DSM-5, both in the introductory materials and the development of the CFI. However, it will take time to see whether the CFI becomes widely used in clinical work and research or whether it remains a tool only for work with groups who are culturally different.

The *World Mental Health Repor*t, on the other hand, recognizes the dynamic, social processes linked to culture. Hunger, work and education, for example, are integrally related to how people adapt or fail to adapt. Clinical phenomena are recognized but so are behavioral problems not traditionally considered in psychiatric classification systems, such as, domestic violence and sexual violence. Throughout, the authors recognize cultural variability as a key dimension of culture and mental health and they highlight the moral and health implications of controversial practices, such as female genital mutilation (female circumcision).

The Surgeon General's supplemental report falls between both perspectives. It recognizes the importance of culture in specific clinical entities (both culture-bound syndromes and mainstream mental disorder categories). It also acknowledges the importance of the broader social world, although the emphasis tends to be more disorder based and less contextually based than the *World Mental Health Report*. The CPES also falls between both perspectives. On the one hand, the main findings focus on DSM-IV defined disorders with little attention to the different ways psychopathology is expressed given specific sociocultural contexts (for exemptions see Lewis-Fernandez et al., 2009; Guarnaccia et al., 2010). On the other hand, attention has been given to the sociocultural context and how that relates to disorder and distress (e.g., Gavin et al., 2010). Despite the differences in the treatment of culture, these four research developments indicate that culture as a subject matter is no longer the exclusive purview of cultural psychologists, psychiatrists, and anthropologists. It is now the subject matter of all users of DSM-5, worldwide policy makers, national health policy makers, mental health researchers, and those who care for people with mental disorders

Disorder-Related Research We now turn to the examination of selected psychopathology research. We chose the study of anxiety, schizophrenia, and childhood psychopathology, because within each of these areas there are systematic studies that examine the cultural basis of the expression of these disorders, as well as the social and cultural processes that underlie the development and course of illness.

Anxiety Although there are existing overviews of the study of culture and anxiety (Guarnaccia 1997; Guarnaccia & Kirmayer 1997), we chose to focus our attention on one line of research, the study of *ataques de nervios*, The study of *ataques de nervios* is an important line of research because it focuses on a culture-specific phenomenon for which the triangulation of ethnography, epidemiology, and clinical research has made important contributions. Thus, we are able to examine some ways in which ethnography informs mainstream research and to highlight the important ways in which culture influences psychopathology.

Ataque de nervios is an idiom of distress particularly prominent among Latinos from the Caribbean, but also recognized among other Latino groups. Symptoms commonly associated with *ataques de nervios* include: trembling, attacks of crying, screaming uncontrollably, and verbal or physical aggression. Other symptoms that are prominent in some *ataques*, but not others, are seizure-like or fainting episodes, dissociative experiences, and suicidal gestures. A general feature experienced by most sufferers of *ataques de nervios* is feeling out of control. Most episodes occur as a direct result of a stressful life event related to family or significant others (e.g., death or divorce). After the *ataque*, people oftentimes experience amnesia of what occurred, but then quickly return to their usual level of functioning (American Psychiatric Association, 1994, p. 845).

Guarnaccia and colleagues initiated a program of research by first carrying out open-ended, descriptive interviews in clinical settings with people who had experienced *ataques de nervios* (De La Cancela, Guarnaccia, & Carrillo, 1986; Guarnaccia, De La Cancela, & Carrillo, 1989). Drawing from the rich description of clinical cases and an understanding of the social history of Puerto Ricans living in the United States, these investigators pointed out an association between social disruptions (family and immediate social networks) and the experience of *ataques*. To build on the ethnographic base, Guarnaccia and colleagues turned to epidemiological research to examine its prevalence in Puerto Rico. They examined data from a large epidemiological study in Puerto Rico (Canino et al., 1987), in which respondents were directly queried as to whether they had suffered an *ataque de nervios* and what the experience was like (Guarnaccia, Canino, Rubio-Stipec, & Bravo, 1993). The prevalence rate was found to be high, 16% of the large community sample ($N = 912$), and *ataques de nervios* were found to be associated with a wide range of mental disorders, particularly anxiety and mood disorders. The social context continued to be important in understanding *ataques de nervios*. *Ataques* were found to be more prevalent among women, persons older than 45 years, people from lower socioeconomic background, and those with disrupted marital relations. Guarnaccia and colleagues then returned to the ethnographic mode to explicate the experience of *ataques* among those persons who had reported suffering an *ataque de nervios* in the epidemiological study (Guarnaccia, Rivera, Franco, & Neighbors, 1996). Through in-depth interviewing, the full range of symptoms and the specific social contexts were identified. This "experience-near" research approach enabled Guarnaccia and associates to

examine carefully how the social world can become part of the physical self as reflected in *ataques de nervios*.

Clinical research has further examined the relationship between *ataques de nervios* and psychiatric diagnoses. Liebowitz and colleagues (1994) carried out clinical diagnostic interviews of 156 Latino patients from an urban psychiatric clinic that specializes in the treatment of anxiety. They explicated the relationship between patients having an *ataque de nervios* and meeting criteria for panic disorder, other anxiety disorders, or an affective disorder. Their fine-grained analysis suggests that the different expressions of *ataque de nervios* interact with different co-existing psychiatric disorders. Persons with an *ataque de nervios* who also suffer from panic disorder present largely anxiety symptoms. However, in those with an affective disorder, *ataque de nervios* are characterized by emotional lability, especially anger (Salmán et al., 1998). Thus, in addition to the social factors previously noted, these findings suggest that the clinical context may also play a role in understanding *ataque de nervios*. Another clinical study from the same site, but with different data, more clearly delineates the borderlines between *ataque de nervios* and panic disorder (Lewis-Fernandez et al., 2002). The investigators note that key distinguishing features of *ataques* as compared to panic attacks are that *ataques* are most often triggered by some stressful event, that people feel relieved after their *ataques*, and that *ataques* do not follow the same rapid crescendo as panic attacks. The most recent clinical study from this group looks at the association of *ataques de nervios* with trauma and dissociation (Lewis-Fernandez et al., 2010; see also Hinton, Chong, Pollack, Barlow, & McNally, 2008).

Work on *ataques de nervios* has been extended to children (Guarnaccia, Martinez, Ramirez, & Canino, 2005). In a study of the mental health of children in Puerto Rico, the authors included a question on *ataques de nervios*. They found that *ataques* continue to be reported by Puerto Rican children, particularly adolescent girls. As in adults, *ataques* are more common in children who meet criteria for a range of psychiatric disorders in the anxiety and depression spectrum.

The most recent work on *ataques de nervios* comes from the NLAAS, one of the CPES studies discussed earlier (Guarnaccia et al., 2010). The NLAAS provided the first opportunity to systematically study *ataques de nervios* across the range of Latino groups in the United States. Key findings include that *ataques* are recognized by all Latino groups, but reported more frequently by Puerto Ricans (15% for Puerto Ricans compared with between 7% and 9% for other Latinos). At the same time, once Latinos endorsed the *ataque* screener question, their pattern of symptomatic reports were quite similar, indicating that the cultural syndrome of *ataque de nervios* appears to be relatively similar across Latino groups. As in previous studies, *ataques* were strongly associated with anxiety and depression disorders, suicidal symptoms, and disability due to a mental health problem. One surprising finding was that *ataques* were more frequently reported by those born in the United States who spoke more English. The authors' interpretation of these findings fits with the Latino mental health paradox that the more acculturated Latinos become, the more distress they experience (Vega & Sribney, 2011). What is interesting is that *ataques de nervios* remain an important idiom for expressing that distress. These findings, and the broader program of research, led Guarnaccia and colleagues (2010) to conclude that *ataques de nervios* are best conceptualized as an indicator of psychological and social vulnerability among Latinos who suffer these experiences. For clinicians, they provide an important window into a range of mental health and social issues among Latinos. For researchers, a question about *ataques* provides a simple, yet powerful indicator of a range of mental health needs.

The study of *ataque de nervios* is exemplary in the field of cultural psychopathology for many reasons. What is most striking is the systematic ongoing dialogue among ethnographic, epidemiological, and clinical research methods to advance our understanding of *ataques of nervios* and how the social world interacts with psychological and physical processes in the individual. With the multiple approaches, one observes the shifting of the researchers' lenses (Kleinman, A., & Kleinman, J. 1991). In the early ethnographic work, Guarnaccia and colleagues drew from a small number of clinical cases and interpreted their findings with broad strokes, focusing on the larger social contexts of the individuals, particularly their migration experience and experiences of marginal social status. In the epidemiological research, the investigators used large, representative samples to identify people with *ataques* and the social and psychiatric correlates of that experience. In this research, the social context is reduced to single questions concerning gender, age, educational level, and marital status, which provides some basis for interpretation but certainly lacks the richness of ethnographic material. The clinical studies provide an in-depth profile of the symptom patterns of those with and without an *ataque*, but they provide less information about the social world of the sufferer. Each approach has its strengths and limitations. What matters though is not a given strength or limitation of a specific study but the weaving of multiple studies with multiple approaches to understand the given phenomenon in some depth.

In addition to the ongoing dialogue among research approaches, the research is also exemplary by placing *ataque de nervios* and related mental disorders in their social context. In almost all studies, *ataque de nervios* is presented not as a popular illness (an illness defined by the community), or clinical entity (a psychologically defined disorder) that resides within individuals, but as a cultural syndrome that reflects the lived experience of women with little power and disrupted social relations. In adopting multiple approaches, the emphasis given to the social domain is likely to shift. Nevertheless, over the several studies, Guarnaccia and his colleagues have maintained

considerable attention to the social context. In so doing, they have demonstrated how to include the social world in epidemiological (e.g., Guarnaccia et al., 1993, 2010), clinical (Salmán et al., 1998; Lewis-Fernandez et al., 2002), as well as ethnographic studies (Guarnaccia et al., 1989, 1996). This integration resulted in a proposal for a parallel categorization of psychopathology for Puerto Ricans using popular categories that bring together both the psychiatric and social dimensions of distress (Guarnaccia, Lewis-Fernandez, Rivera Marano, 2003). Overall, the study of *ataque de nervios* provides a model for the investigation of culture and psychopathology, particularly for research that begins with a cultural concept of distress (Guarnaccia & Rogler, 1999).

Schizophrenia The cultural conception of the self can influence the manner in which disorders are expressed and understood by others. This is articulated in Fabrega's (1989) overview of how past anthropologically informed research contributed to the study of psychosis and how future studies can advance our understanding of the interrelations of culture and schizophrenia. According to Fabrega, the effect of schizophrenia on individuals and communities depends on whether they conceive of the self as autonomous and separate from others or as connected and bound to others (Shweder & Bourne 1984; Markus & Kitayama 1991). The research that most directly addresses this notion is that which examines the role of social factors in the course of schizophrenia. Two prominent lines of inquiry include the WHO cross-national study of schizophrenia, and a series of studies examining the relationship of families' emotional climate to the course of illness (for an examination of culture and symptom expression in schizophrenia, see Brekke and Barrio, 1997; Weisman et al., 2000).

The WHO's International Pilot Study on Schizophrenia (IPSS) and the follow-up study Determinants of Outcomes of Severe Mental Disorder (DOSMD) represent the largest multinational study of schizophrenia to date (IPSS: 9 countries and 1,202 patients, World Health Organization, 1979; DOSMD: 10 countries and 1379 patients; Jablensky et al., 1992). Many contributions have been made by these investigations, including finding evidence of the comparability of schizophrenia's core symptoms across several countries (for a critique, see Kleinman, 1988). The finding that has received the most attention by cultural researchers is that schizophrenia in developing countries has a more favorable course than in developed countries (Weisman, 1997). Some investigators have referred to this basic finding as "arguably the single most important finding of cultural differences in cross-cultural research on mental illness." (Lin & Kleinman, 1988, p. 563). Others have been most critical of the studies' methods and interpretations (see Cohen, Patel, Thara, & Gureje, 2008; Edgerton & Cohen, 1994; Hopper, 1991). For example, Edgerton and Cohen (1994) point out that the distinction between "developed" and "developing" countries is unclear. Moreover, they argue that the cultural explanation for the differences in course is poorly substantiated. They also suggest that such research could be more culturally informed through the direct measure of specific cultural factors in conjunction with observations of people's daily lives (see also Hopper, 1991). What is clear is that the WHO findings have provided the basis for an important discussion of method and theory regarding how the course of schizophrenia can be shaped by the social world.

Another line of research addressing culture's role in the course of schizophrenia focuses on families' emotional climate. Based on the early work of George Brown and associates (e.g., Brown, Birley & Wing, 1972), research has found that hospitalized patients who return to households marked by high *expressed emotion* (criticism, hostility, and emotional involvement) are more likely to relapse than those who return to households that are not so characterized (Bebbington & Kuipers 1994; Butzlaff & Hooley, 1998; Leff & Vaughn, 1985). This line of investigation is important to the study of culture because it points out the importance of the social world. More specifically, cross-national and cross-ethnic studies have uncovered interesting differences in the level and nature of expressed emotion (Jenkins & Karno, 1992). These studies show that cultural factors in the definitions and experiences of schizophrenia affect the emotional climate of families where ill individuals live. Furthermore, these cultural differences have important effects on mental health outcomes.

The most systematic cultural analysis of families' role in schizophrenia has been carried out by Jenkins and her colleagues. In using both clinical research methods based on the prototypic contemporary study of expressed emotion (Vaughn & Leff, 1976) and ethnographic methods based on in-depth interviews, Jenkins and associates extended this line of study to Mexican American families in Los Angeles. In the first major report, Karno and colleagues (1987) replicated the general finding that patients who return to high expressed emotion families are more likely to relapse than patients who return to low expressed emotion families. Jenkins (1988a) then carried out an in-depth examination of Mexican American families' conceptualization of schizophrenia, specifically *nervios*, and how this differed from a comparable sample of Anglo American families who viewed schizophrenia largely as a mental illness (see also Guarnaccia, Parra, Deschamps, Milstein, & Argiles, 1992). Based on both quantitative (coded responses to open-ended questions) and qualitative data, Jenkins (1988b) suggested that Mexican Americans' preference for labeling the family member's schizophrenia as *nervios* is tied to the family members' efforts to decrease the stigma associated with the illness and also to promote family support for the ill individual. In subsequent papers, Jenkins (1991, 1993) critiqued the cultural basis of the expressed emotion construct in general, as well as its components of criticism and emotional over-involvement

in particular. An important theoretical contribution to the study of the course of schizophrenia is that Jenkins situates families' expressed emotion not only in the family members' attitudes, beliefs or even feelings (the perspective taken in most studies), but also in families' expressed emotion in the patient-family social interaction. Overall, Jenkins' work has brought much needed attention to how serious mental illness is embedded in specific social and cultural contexts.

Building on Jenkins work, López and colleagues have further critiqued the notion of expressed emotion with its focus on negative family functioning, particularly criticism (López, Nelson, Snyder, & Mintz, 1999). They point out that at an early juncture in the study of families and relapse, investigators (Brown et al., 1972) opted to focus on aspects of family conflict that predict relapse rather than the prosocial aspects of family functioning that buffer relapse. In a reanalysis and extension of the Mexican American sample (Karno et al., 1987) and a comparable Anglo American sample (Vaughn, Snyder, Jones, Freeman, & Falloon, 1984), López and associates (2004) found that family warmth predicted relapse for Mexican Americans, whereas criticism predicted relapse for Anglo Americans. In other words, Mexican American patients who returned to families marked by high warmth were less likely to relapse than were those who returned to families characterized by low warmth. For Anglo Americans, high criticism was positively related to relapse, but warmth was unrelated to relapse. Subsequent analyses of the Mexican American sample (Breitborde, López, Wickens, Jenkins, & Karno, 2007) provided direct evidence that the presence of high warmth and a moderate degree of emotional over-involvement were associated with less relapse than the usual relapse rate in the study of expressed emotion (Butzlaff & Hooley, 1998). Moreover, a high level of emotional over-involvement was associated with a greater-than-usual rate of relapse. Aguilera and associates replicated the relationship between emotional overinvolvement and relapse with an independent sample of Mexican Americans (Aguilera, López, Breitborde, Kopelowicz, & Zarate, 2010). Together, these findings point out that, for largely immigrant Mexican Americans, there is an ongoing tension between maintaining close family ties (high warmth and moderate degrees of involvement) without becoming too involved (Jenkins, 1993). Although close family connections may be valued (and beneficial), too much closeness can be problematic. (See Kopelowicz et al., 2006, for a behavioral observation study of Mexican American families' interactions with their ill relatives that found a similar dynamic.) In addition to identifying this family dynamic, López and colleagues (2009) found that there is a relatively high number of Mexican American caregivers who are identified as emotionally overinvolved. Together, these findings suggest that family interventions with this ethnic group should not simply address family negativity, the emphasis of most family based treatments (e.g., Falloon et al., 1982). Attention to caregivers' emotional overinvolvement is likely to be helpful as well (see also Singh, Harley, and Suhail, 2013).

López and colleagues (2004) did not attribute the observed ethnic differences in family processes related to relapse to a set of presumed cultural beliefs associated with the "Mexican culture" or a collectivist culture. Instead, they noted that most of the families and patients of Mexican origin were immigrants and that maintaining family ties was crucial to the survival of low-income immigrants living in a foreign and at times hostile environment. Their maintenance of the value of family support was extended to relatives who developed serious mental illness. The latter interpretation is consistent with a view of culture as embedded in the social world, rather than as a set of individual values or beliefs.

In conjunction with two other international studies (Italy: Bertrando et al., 1992; Yugoslavia: Ivanović, Vuletić, & Bebbington, 1994), these findings are consistent with the hypothesis that culture plays a role in the manner in which families respond to relatives with schizophrenia, which in turn influences the course of illness. A limitation of most of these findings is that there was no direct measure of cultural processes (for an exception, see Aguilera et al., 2010). Nevertheless, the importance of this research is that the exploration of possible cultural variability led to the beginning of a line of inquiry that examines what families do to prevent relapse. Such research has the potential to add a much needed balance to family research by focusing on both positive and negative aspects of families' behaviors (see also Weisman, Gomes, and López, 2003). The study of caregiving (e.g., Guarnaccia, 1998; Lefley 1998; Ramirez Garcia, Chang, Young, López & Jenkins, 2006) and families' day to day interactions with ill family members will likely shed further light on the importance of families' prosocial functioning.

Childhood Disorders The study of child psychopathology is a rich field of inquiry for those interested in culture. Child psychopathology requires attention to the behavior of children as well as the views of adults—parents, teachers, and mental health practitioners—for it is the adults who usually decide whether or not a problem exists. The fact that others determine whether children's behavior is problematic underscores the importance of the social world in defining mental illness and disorders of children and adolescents.

John Weisz and his colleagues have carried out the most systematic research on culture and childhood psychopathology (for a review see Weisz, McCarty, Eastman, Chaiyasit, & Suwanlert, 1997). In their first study, conducted in Thailand and the U.S., Weisz and associates found that Thai children and adolescents who were referred to mental health clinics reported more internalizing problems than U.S. children and adolescents (Weisz, Suwanlert, Chaiyasit, Weiss, & Walter, 1987). In contrast, U.S. children and adolescents reported more externalizing

problems than Thai children and adolescents. In follow-up community studies, where the mental health referral process was not a factor in the identification of problem behaviors, the cross-national differences were confirmed for internalizing problems but not for externalizing problems (Weisz, Suwanlert, Chaiyasit, Weiss, Achenbach et al., 1987; Weisz, Suwanlert et al.,1993). The prevalence of externalizing problems among U.S. and Thai youth identified in their respective communities did not differ. Weisz and colleagues argue that the findings with regard to internalizing problems are consistent with the idea that culture shapes the manner in which children and adolescents express psychological distress. Because they come from a largely Buddhist religious and cultural background that values self-control and emotional restraint, Thai youth may be more likely than U.S. youth to express psychological distress in a manner that does not violate cultural norms (for an analysis of specific somatic and affective symptoms in the U.S. and Thai samples, see Weiss et al., 2009).

In addition to these intriguing findings, two other factors stand out in Weisz and colleagues' research: the systematic nature of the research and the care with which the research has been conducted. Weisz and colleagues began this line of investigation in mental health clinics, and then used a community survey to rule out the possibility of the influence of referral factors (Weisz, Suwanlert, Chaiyasit, Weiss, Achenbach et al., 1987). Based on these findings, Weisz and Weiss (1991) derived a referability index for specific problem behaviors (e.g., vandalism and poor school work) that specifies the likelihood that a given problem will be referred for treatment, taking into account the problem's prevalence in a given community. In this study, they demonstrated how gender and nationality influence whether or not a problem is brought to the attention of mental health professionals. Subsequently, Weisz and colleagues examined teachers' reports of actual children (Weisz et al., 1989) and both parents' and teachers' ratings of hypothetical cases (Weisz, Suwanlert, Chaiyasit, Weiss, & Jackson, 1991). In a study of teachers' report of problem behaviors, Weisz and colleagues (Weisz, Chaiyasit, Weiss, Eastman, & Jackson, 1995) found that Thai teachers report more internalizing and externalizing problem behaviors among Thai children than U.S. teachers report among U.S. children. Each of Weisz and colleagues' studies systematically builds on their previous work in advancing an understanding of how adults with differing social roles define children's problem behaviors. Multiple cross-cultural studies using different methods with different research participants provide a rich network of findings to advance our understanding of how the social context shapes the identification of youths' mental health problems.

The care with which Weisz and colleagues carry out their research is best illustrated in the study of teachers' ratings of problem behaviors (Weisz et al., 1995). They found that Thai teachers rate more internalizing and externalizing problem behaviors for Thai students than U.S. teachers rate of their own students. Given that this finding runs counter to the previous clinical and community studies which only found differences for internalizing problems, they devised an innovative observational methodology to assess whether it was something about the children or the teachers that contributed to this contradictory finding. Weisz and associates (1995) employed independent observers of children's school behavior and obtained teacher ratings of the same children who were observed in Thailand and in the U.S. One of the independent raters was a bilingual Thai psychologist who had received graduate training in the U.S. His participation in both teams of independent observers was critical to assessing the reliability of the Thai and U.S. observers. The relationship between his ratings and those of the other U.S. and Thai raters were equally high, suggesting that the ratings were reliable across both national sites. Interestingly, the observers rated Thai children as having less than half as many problem and off-task behaviors as U.S. children, yet Thai teachers rated the observed students as having many more problem behaviors than U.S. teachers rated their students. These data suggest that Thai teachers have a much lower threshold than U.S. teachers for identifying problem behaviors in their students. Findings in cross-cultural research are often open to multiple interpretations. By using careful methodology across multiple studies, Weisz and collaborators have discerned the specific meaning of their complex set of findings. In doing so, they have highlighted the importance of contextual factors in the assessment of child psychopathology and demonstrate that it is untenable to view the assessment of child psychopathology as culture-free.

Developmental researchers are examining more closely the influence of culture on the type and degree of problem behaviors of children and adolescents. Weisz and associates have extended their research in Thailand and the U.S. to Jamaica and Kenya (Lambert, Weisz, & Knight, 1989; Weisz, Sigman, Weiss, & Mosk, 1993). Other investigators have compared rates of internalizing and externalizing problems in other parts of the world (e.g., Denmark, Arnett & Balle-Jensen, 1993; Puerto Rico, Achenbach et al., 1990). Achenbach and colleagues (2008) have embarked on the most ambitious project thus far, drawing on hundreds of cross-cultural studies using the Achenbach System of Empirically Based Assessment and delineating sociocultural norms based on nationality. The assumption in such research is that the syndromes being measured are similar across cultural contexts. In a reanalysis of the Thai and U.S. data, Weisz and associates provide data to suggest that the syndromes are not equivalent pointing out some Thai specific ways in which children express distress (Weisz, Weiss, Suwanlert, & Chaiyasit, 2006).

Other researchers have examined specifically internalizing type problem behaviors (Greenberger & Chen, 1996) or externalizing type problem behaviors (e.g., Weine, Phillips, & Achenbach, 1995) in cross-national or cross-ethnic samples. An important trend in this research is that epidemiological research that compares groups cross-nationally and suggests possible cultural explanations is now being complemented by research that

examines the psychosocial processes associated with children and adolescents' adjustment or psychopathology. For example, Chen and associates examined risk factors (parent–adolescent conflict and perceived peer approval of misconduct) and protective factors (parental warmth and parental monitoring) associated with acting-out problems across four groups of adolescents: European Americans, Chinese Americans, Taipei Chinese and Beijing Chinese (Chen, Greenberger, Lester, Dong, & Guo, 1998). They found both cross-cultural similarities and differences in the way both family and peer factors contributed to misconduct. Generally, the predicted risk and protective factors accounted for a significant amount of variance for misconduct in each of the four groups, however, peer influences were less relevant for the Taipei and Beijing Chinese youth. (See also Polo and Lopez, 2009, as they examined the sociocultural mediators that explain some of the difference in internalizing distress among U.S. born and immigrant Mexican origin youth.)

The strength of the more recent studies is that they examine processes that may explain potential cross-national differences and similarities, including social (family and peers) and psychological (values) processes. Thus, an important step has been taken to understand why differences and similarities may occur in behavior problems cross-nationally. Although the conceptual models used to frame such research are rich, include social processes, and have a strong empirical tradition in psychological research, they are not well informed by cultural-specific processes of the non-U.S. groups under study. Investigators typically apply models developed largely in the United States. Ethnographic research that attempts to identify what about the social and cultural world might play a role in the expression of distress and disorder among children would be especially welcome. This research could then lead to the direct examination of those culture-specific factors that are believed to affect psychopathology within a given conceptual framework, as evidenced in the work of some developmental researchers (e.g., Fuligni, 1998), and as advocated by others (e.g., Schneider, 1998). The growing interest of researchers in studying internalizing and externalizing problem behaviors cross-nationally and cross-ethnically attests to the utility of this approach for enhancing our understanding of culture and childhood psychopathology.

Research Areas With Potential to Advance Our Understanding of the Social World

Immigration There has been a longstanding interest in the role of immigration or acculturation as it relates to mental health and mental disorders. Important studies indicate that among adults of Mexican origin, immigrants have lower prevalence rates than those who are born in the United States (Burnam, Hough, Karno, Escobar, & Telles, 1987; Vega et al., 1998). More recently, Grant and colleagues (2004) reported that the acculturation effect applied not only to Mexican-origin residents but also to non-Latino White immigrants when compared with non-Latino Whites born in the United States. The same effect has also been reported for African Caribbean people from the NSAL; immigrant African Caribbean people had lower rates of substance abuse disorders (Broman, Neighbors, Delva, Torres, & Jackson, 2008) and major depression (Williams et al., 2007) than U.S.-born African Caribbean people. Alegría and colleagues (Alegría, Canino et al., 2006; Alegría et al., 2008) caution that the immigration/acculturation effect does not universally apply to immigrant groups. In particular, they point out that, among Latinos, the immigrant/acculturation effect pertains largely to adults of Mexican origin, not Cuban Americans or Puerto Ricans, although Puerto Ricans are not really immigrants as they are U.S. citizens whether they live on the Island or the mainland. For Asian Americans, also, the immigrant effect primarily applies to women (Takeuchi et al., 2007). Thus, available literature suggests that there is strong evidence for an immigration effect; however, it does not apply universally to all immigrant groups.

The social and psychological mechanisms that are responsible for the differing prevalence rates for the immigrant groups at this time are poorly understood. Nevertheless, available studies indicate that research on immigration and acculturation can contribute much to our understanding of how the social world and psychopathology interrelate (see also Rogler, 1994). A particularly wide-open area of study is the examination of immigration and mental health disorders among children and adolescents (see Gil & Vega, 1996; Guarnaccia & López, 1998; Polo & López, 2009). Not only will immigration/acculturation research be able to address important conceptual and methodological issues in the study of culture, but it will also have important policy implications for the delivery of mental health services to underserved communities (e.g., Salgado de Snyder, Diaz-Perez, & Bautista, 1998).

Immigration status is also related to incidence rates of schizophrenia. Unlike the prior studies, immigration status is associated with elevated incidence rates of schizophrenia and other psychotic disorders. The initial impetus of this line of study began with Caribbean migrants to the United Kingdom (e.g., Harrison et al., 1997), and has extended to other parts of the world (e.g., the Netherlands; Veling et al., 2006). Most of this research has been carried out in Europe. Two meta-analyses (Cantor-Graae & Selten, 2005; Bourque, van der Ven, & Malla, 2011) provide convincing evidence that immigrants and their offspring have a higher incidence of psychotic disorders than most people. In fact, Bourque and colleagues concluded that

> migrant status, either FGI (first generation immigrant) or SGI (second generation immigrant), cannot be disregarded as an important risk factor for psychotic disorders, with a risk magnitude within the same range as that associated with cannabis use, urbanicity or perinatal complications.
>
> (Bourque et al., 2011)

The fact that the elevated risk persists to the second generation suggests that post-migration factors may be particularly important. Adverse social environments such as discrimination are considered to be one possible contributory factor as well as other social (substance use) and biological factors (exposure to viruses; Bourque et al., 2011). This line of study promises to be a challenging as well as an exciting research direction in cultural psychopathology.

American Indians We are encouraged by the growing interest in the study of psychopathology among U.S. ethnic minority groups. To highlight this area, we have chosen to focus on research with American Indians, because we have given little attention to this growing body of literature and because researchers in this area have given considerable attention to social factors. A systematic series of studies has examined the mental health problems of American Indian children (e.g., Beiser, Sack, Manson, Redshirt, & Dion, 1998; Dion, Gotowiec, & Beiser, 1998) and adults (Maser & Dinges, 1992/1993). Although many of these studies use mainstream models of disorder and distress (O'Nell, 1989), there are growing efforts to contextualize the mental health problems within these communities. In one study, Duclos and colleagues (1998) found that nearly 50% of the Indian adolescents detained in the juvenile justice system met criteria for mental disorders, ranging from substance abuse/dependence (38%) to major depression (10%). These rates were much higher than community surveys of Indian youth and non-Indian youth. These researchers argue that the juvenile justice system has the potential to serve as an important site for treating these high-risk youth, many of whom would go untreated. In another study, O'Nell and Mitchell (1996) found that the line between normal and pathological drinking among adolescent Indians is contextually based. Their ethnographic findings suggest that a rigid model of alcohol abuse defined by biology (frequency and amount of alcohol) or psychology (distress), without significant attention to the sociocultural context (e.g., when and with whom one drinks) is limited in distinguishing between normative and pathological drinking (see also Klostermann & Kelley, Chapter 14 in this volume.)

The largest study to date of the mental health of Americans Indians is the American Indian Services Utilization, Psychiatric Epidemiology, Risk and Protective Factors Project (Beals et al., 2003). This project is based on representative samples of adult residents within two reservations (Southwest tribe, N = 1446, and Northern Plains tribe, N = 1638). Among the early findings reported from this research is that, when controlling for various demographic correlates, the American Indian samples reported greater lifetime prevalence rates of alcohol abuse and post-traumatic stress disorder and lower lifetime rates of major depression than a national sample of the United States across ethnicities (Beals et al., 2005). Other studies have examined the role of adversity (e.g., disruptions to children's lives such as parental divorce and traumas such as witnessed violence) in the development of mental disorders. Whitesell and colleagues (2007), for example, found that both proximal and cumulative distal experiences of adversity were associated with an increased risk of the onset of substance dependence symptoms. Similarly Beals and colleagues (2013) found that the higher rates of post-traumatic stress disorder among the two reservation communities is likely due to higher rates of trauma exposure. Given that ethnographic interviews were also carried out in this large scale project, we expect in the future more nuanced research reports that examine the social world as it relates to trauma and alcohol disorders, for example.

Conclusion

Cultural psychopathology, the study of culture and the definition, experience, distribution, and course of psychological disorders, is now "on the map." Articles are being published in culture-focused journals as well as mainstream journals, Substantive areas of psychopathology research are being shaped by cultural research. Efforts to integrate cultural concepts of distress with psychological and psychiatric constructs are well underway. Examples include studies of *ataques de nervios* and anxiety and affective disorders, and *nervios* and families' conceptualization of serious mental illness. Guarnaccia and Rogler (1999) provide a program for moving some of this research forward. Kleinman's important 1988 book got the message out that culture matters. As evidenced in the Surgeon General's report and the CPES, the message has been received; cultural research is providing an innovative and fresh perspective to our understanding of several important aspects of psychopathology.

For cultural researchers to build on the empirical and conceptual foundation that has been established, they need to continue to be critical of how culture is conceptualized and how such conceptualizations guide their research (see Kagawa Singer et al., 2013, for a recent statement on the conceptualization and measurement of culture in health research). It is clear from this review that culture can no longer be treated solely as an independent variable or as a factor to be controlled for. Rather, culture infuses the full social context of mental health research. Culture is important in all aspects of psychopathology research—the design and translation of instruments, the conceptual models that guide the research, the interpersonal interaction between researcher and research participants and clinician and patient, the definition and interpretation of symptom and syndromes, and the structure of the social world that surrounds a person's mental health problems. Cultural psychopathology research requires a framework that incorporates culture in multifaceted ways. It is crucial that cultural research not obscure the importance of other social forces such as gender, class, poverty and marginality that work in conjunction with culture to shape people's everyday lives. The examination of both social and cultural processes is one way to help guard against superficial

cultural analyses that ignore or minimize the powerful political economic inequalities that coexist with culture.

A corollary of the need for a broad framework for research is the need for approaches that integrate qualitative and quantitative methods. Cultural psychopathology research can serve as an important site for integrating ethnographic, observational, clinical, and epidemiological research approaches. Mental health problems cannot be fully understood through one lens. Ethnographic research provides insights into the meaning of mental health problems and how they are experienced in their sociocultural context. Observational research captures people's functioning in their daily lives. Clinical research can provide detailed descriptions of psychopathological processes and can contribute to developing treatments to alleviate suffering at the individual as well as social levels. Epidemiological research can identify who is most at risk for psychopathology and broaden perspectives to more generalized processes and populations. It is the integration of these perspectives, through new mixes of methodologies and in the composition of research teams, that will make the cultural psychopathology research agenda succeed (Guarnaccia, 2009).

The ultimate goal of cultural psychopathology research is to alleviate suffering and improve people's lives. This requires attention to the multiple levels of individual, family, community, and the broader social system. Our enhanced notion of culture leads to analysis of the expression and sources of psychopathology at all of these levels. Our commitment to making a difference in peoples' everyday lives argues for the development of treatment and prevention interventions at these multiple levels as well. The increasing cultural diversity of the United States, and the massive movements of people around the globe, provide both an opportunity and imperative for cultural psychopathology research.

Note

1. For an extensive list of all CPES publications, see http://www.icpsr.umich.edu/icpsrweb/CPES/BIBLIO/STudies/20240/resources 9accessed April 23, 2015).

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5

The Role of Gender, Race, and Class in Psychopathology

Barbara A. Winstead and Janis Sanchez

When we consider the occurrence of psychopathology, its etiology, and the effectiveness of its treatment, we generally treat clients and clinicians involved as if they all belong to the same group. We are not unaware of group differences, but the majority of the literature on psychopathology refers to clients and clinicians without reference to their social identities and with the mistaken assumption that diagnoses, treatments, and research outcomes can be generally applied to one and all.

This chapter focuses on differences rather than universalities. It addresses these questions:

- Do gender, race, and class affect which psychological disorders individuals experience or how they experience them?
- Are individuals from different groups treated differently by the mental health system?
- Do recommended treatments work equally well for individuals from these different groups?
- Should treatments take the demographic characteristics of the client into account?
- Does the gender or race of the therapist matter?
- What difference do gender, race, and class make?
- And, if they make a difference, why?

In this chapter, we discover that these questions raise more questions. Clear-cut answers are scarce, but the questions themselves shed light on the process of psychological diagnosis and treatment of psychological disorders.

First a word about terminology: *Race* may be considered an outdated term; scientists are in agreement that biological variation among humans does not fall neatly into "racial" groups. The assumption, however, that group differences in physical or mental health have biological foundations remains prevalent; even as the argument that these categories are socially constructed and represent social relationships, typically between those with more power and privilege and those with less, gains currency (Mullings & Schulz, 2006). The use of the term *race*, then, as opposed to *ethnicity* or *culture*, is meant to remind us of these historical and current assumptions and of the advantages (increasing understanding across groups) and hazards (distancing and pathologizing the "other") of using race as an identifier. *Gender* is also a problematic term (Pryzgoda & Chrisler, 2000). A distinction has often been made between "sex," meaning biological differences between girls and boys, women and men, and "gender," referring to psychological and sociological differences. But how does one sort these out? Gender reminds us again that a simple biological explanation will rarely if ever suffice in understanding human mental health or disorder.

Recent Initiatives Focused on Gender, Race, and Class

Concern about the impact of gender, race, and class on mental health and mental health care is expressed in at least three recent and ongoing efforts. One is the attention currently being paid to racial and ethnic disparities in health and mental health. The second is both the approach taken to gender and culture in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) and critical reactions to it. The third is the focus in the training of clinical and counseling psychologists and other mental health professionals in cultural competence.

That inequities exist in health and health care based on ethnicity is hardly news, but the attention paid to these inequities at the national level is a relatively recent phenomenon. Nelba Chavez, then Administrator of Substance Abuse and Mental Health Services Administration, refused to sign *Mental Health: A Report of the Surgeon*

General (U.S. Department of Health and Human Services, 1999) on the grounds that insufficient attention was paid to cultural diversity and disparities in mental health care (Chavez, 2003). This led to the development of a supplement to the report, *Mental Health: Culture, Race, and Ethnicity* (U.S. Department of Health and Human Services, 2001) that summarized research on the impact of ethnicity on mental health.

One of the goals of the U.S. health agenda, Healthy People 2020, is the achievement of health equity and the elimination of disparities. The Office of Minority Health (part of Health and Human Services), the National Institute on Minority Health and Health Disparities, the Center for Disease Control, the National Institutes of Health, and other federal agencies, all support research and programs aimed at understanding and eliminating disparities. In 2003, The President's New Freedom Commission on Mental Health issued a report, *Achieving the Promise: Transforming Mental Health Care in America*, which speaks explicitly to problems for ethnic minorities in accessing quality mental health care and outlines a plan for research and practice that can close the gap. Safran et al. (2009) notes that publications on mental health disparities showed a small increase between 1995 and 2000 and then a linear increase from around 20 publications per year in 2000 to 180 in 2007.

The long-awaited fifth edition of DSM, the standard used by mental health professionals for diagnosis of psychological disorders, was published in May 2013. One of the six preplanning white papers in *A Research Agenda for DSM-V* (Kupfer, First, & Regier, 2002) dealt specifically with the influence of culture on psychiatric diagnosis, including the influences of ethnicity, language, education, gender, and sexual orientation (Alarcón et al., 2002). DSM-5 also includes "cultural formulation" (pp. 749–59), which provides interview tools and guidelines for understanding the cultural context in which the client experiences mental illness. Each diagnostic category also includes "gender-related diagnostic issues" and/or "culture-related diagnostic issues" where these are deemed relevant. However, concerns about gender and ethnic bias in the DSM criteria for various psychological disorders are longstanding and various (Caplan & Cosgrove, 2004; Maracek & Gavey, 2013). Critics of DSM have argued that any classification system of psychological disorders represents an ethnocentric construction of what is an idealized, or nonpathological, self. Gaines (1992) suggested that the essential feature of DSM diagnoses appears to be the absence of self-control, a Western/European ideal not held by all cultures. Others argued that women are pathologized by DSM diagnostic criteria (Ali, Caplan, & Fagnant, 2010; Caplan & Cosgrove, 2004). Histrionic personality disorder (HPD) and dependent personality disorder are cited as cases where feminine traits are used to establish the presence of psychopathology (Ali et al., 2010). The creators of DSM-5 have also been criticized for expanding diagnostic categories, making more behaviors pathological, and for being too closely aligned with the pharmacology industry. The inclusion of premenstrual dysphoric disorder (PMDD, moved from an appendix in DSM-IV to the main text in DSM-5) has been criticized for these reasons. PMDD pathologizes an aspect of women's experience, and the DSM website actually mentioned the use of medication as a treatment for PMDD in justifying its inclusion as a diagnosis (Cosgrove & Wheeler, 2013).

In the disciplines of counseling and clinical psychology, there has been a persistent, but increasing, focus on the cultural competence of practitioners. Training in cultural diversity was recommended at the 1973 Vail conference on graduate education in clinical psychology (Korman, 1974); questions regarding training in cultural diversity became a part of the American Psychological Association (APA) accreditation domains and standards in 1986 and continue to be a focus of consideration in accreditation; APA published *Guidelines for providers of psychological services to ethnic, linguistic, and culturally diversity populations* (American Psychiatric Association, 1990), and incorporated awareness of "cultural, individual, and role differences, including those related to age, gender, race, ethnicity, national origin" in its Ethics code in 1992 (American Psychiatric Association, 1992a, p. 1598). In 2002, APA approved *Guidelines on multicultural education, training, research, practice, and organizational change for psychologists* (American Psychiatric Association, 1992b). The Council of National Psychological Associations for the Advancement of Ethnic Minority Interests published *Psychological treatment of ethnic minority populations* in 2003. A joint task force of APA Divisions 17 (Society of Counseling Psychology) and 35 (Society for the Psychology of Women) published *Guidelines for Psychological Practice with Girls and Women* in 2007. The APA has also published books and special issues of APA journals devoted to multicultural practice and assessment of cultural competency (Bernal & Domenech Rodríguez, 2012; Comas-Diaz, L., 2011; Roberts, Barnett, Kelly, & Carter, 2012).

Despite this widespread interest in gender, race, and class, and numerous publications, understanding whether these variables make a difference and, if they do, how they influence diagnosis and treatment of psychopathology is far from complete. Of great significance when considering race is whether or not social class, as represented by income and education, is included. In the United States, class and ethnicity are highly interrelated. U.S. Census data from 2011 show that 9.8% of non-Hispanic Whites live below the poverty line, while 12.3% of Asian Americans, 25.3% of Hispanics, and 27.6% of Blacks live in poverty (U.S. Census Bureau, 2013). Analyses of ethnicity that do not include social class may confound differences among ethnic groups with differences attributable to income. Even more often overlooked is the connection between gender and poverty. In 2011, the poverty rate for married couple families was 6.2%, for single male head of household,

16.1%, and for single female head of household, 31.2%. (U.S. Census Bureau, 2013).

Even in this brief introduction it may be apparent that, although we continually refer to the intersection among gender, race, and social class, many other documents refer to some of these, all of these, or these plus various other demographic characteristics. The critical issue is placing the individual within the sociocultural context that best helps us to understand and effectively intervene. So, although this chapter focuses on gender, race, and social class, it is understood that these interact with age, sexual orientation, ability, and other variables to form unique experiences for individuals and that the intersection of these variables with others represent combinations that are perceived differently by society and by mental health professionals.

The Role of Gender, Race, and Class in Diagnosis

The process of diagnosis and treatment is less than perfect, but the goal can be clearly stated: We wish to identify the problem, treat it, and thereby permit the client to lead a more productive and rewarding life. But how do we identify the problem? With physical illness, physicians are aided by diagnostic tests that identify abnormalities in biochemistry, histology, or anatomy that signal specific disease processes. With psychological problems, there are rarely physical signs such as an abnormal cell count. We may use diagnostic tests and/or interviews, but these are likely to be based on self-reports of clients, not blood chemistry or cell cultures. What the DSM provides is agreed-upon criteria that help clinicians to make diagnoses based on sets of symptoms. Although psychological diagnosis leads to a present/absent decision, there is growing recognition, including in DSM-5 (pp. 12–13), that a continuous rather than a categorical model is better for understanding psychological disorders (Maddux, 2002; see also Maddux, Gosselin, & Winstead, Chapter 1 in this volume). With a perfectly reliable and valid system, we could easily discover whether gender, ethnicity, and social class were related to different psychological disorders, if the etiologies or disease courses of these illnesses were similar or different, and ultimately, whether individuals with different characteristics benefit from similar or different treatments. But with less than perfect diagnostic systems, our questions about gender, race, and class become more difficult to answer.

Questions of gender bias in the diagnostic categories themselves (Ali et al., 2010) and of failure to appreciate ethnic and racial variations in perspectives on normality and abnormality (Gaines, 1992) have been consistently raised. Furthermore, the gathering of pertinent information to make this determination may also be influenced by the gender, ethnicity, and class of the client and the interviewer. Even items on standardized scales may introduce bias, as words or phrases may have different meanings to different groups of people. Green et al. (2012) outline ways in which diagnostic validity may be affected by race/ethnicity: (a) There may be racial/ethnic differences in comfort with the interview/testing process, rapport with the diagnostician, and/or familiarity with responding to questions and describing mental health symptoms; (b) questions or test items may be interpreted differently than they were intended; (c) cultural context may provide different standards for what is considered maladjustment or impairment; (d) culturally influenced conceptions of time may affect questions of duration, frequency, or recency; and (e) phenomenology of mental disorders may vary such that perceptions of normality and abnormality do not correspond with those of the interviewer. Although they focused on race/ethnicity, many of these concerns might also affect how gender interacts with the diagnostic process. Finally, putting all the symptoms together to arrive at a diagnosis may be influenced by clinician expectations and beliefs about the client based on her or his demographic characteristics.

DSM-5 Information on Gender, Race, and Class

A fundamental issue is whether or not the prevalence rates of disorders vary by gender or race. For 89 disorders, DSM-5 includes some discussion of cultural issues. Often, the text asserts that the diagnosis is seen around the world and/or that cultural/social context is critical to understanding whether "symptoms" are culturally congruent or problematic; but it also provides for some diagnoses fairly specific information on prevalence among typical U.S. ethnic categories; for example, 12-month prevalence rates for alcohol use disorder for Hispanics, Native Americans/Alaska Native, White, African Americans, and Asian American/Pacific Islanders. DSM-5 also makes reference to specific cultural syndromes, such as *ataque de nervios*, *khyâl*, or *taijin kyofusho*. Finally, a chapter is devoted to "cultural formulation," which expands on the guidelines for assessing the cultural context of the patient, as provided in DSM-IV, with a 16-question cultural formulation interview.

While there is interesting but rarely detailed information in DSM-5 on ethnic differences, there is more frequent information on gender. For DSM-IV, Hartung and Widiger (1998) concluded that "[m]ost of the mental disorders diagnosed with the DSM-IV do appear to have significant differential sex prevalence rates" (p. 280). Excluding disorders that are, by definition, specific to one sex or the other (e.g., female orgasmic disorder, male erectile disorder), 84% of the DSM-IV disorders, for which information on prevalence by sex was reported, were described as occurring at different rates in females and males. Of the 123 similar disorders in DSM-5, 93 have some information on sex prevalence and 89% are described as occurring at different rates in females and males. However, DSM-5 shows clear evidence of increased research on the topic and several cases with less than clear-cut presentation of gender differences. For example, while autism

spectrum disorder is reported to be four times more prevalent in boys than in girls, it is noted that girls with the diagnosis generally have intellectual disability, suggesting "that girls without accompanying intellectual impairments or language delays may go unrecognized" (p. 57). HPD is more frequently diagnosed in women in clinical settings, but "some studies using structured assessments report similar prevalence rates among males and females" (p. 668).

A general summary of gender differences noted in DSM-5 indicates that boys and men are more likely to be diagnosed with neurodevelopmental disorders, including autism, attention-deficit hyperactivity disorder (ADHD), and learning disorders; disruptive, impulse-control and conduct disorders; substance-related and addictive disorders; neurocognitive disorders; and paraphilias. Girls and women are more likely to be diagnosed with depressive disorders, anxiety disorders, trauma- and stressor-related disorders, dissociative disorders, and feeding and eating disorders. Generally, boys and men are more likely to have externalizing disorders and girls and women, internalizing disorders. Diagnoses of personality disorders tend to parallel expected sex differences in personality traits or even gender stereotypes: Women are reported to have higher rates of borderline, histrionic (but it may depend on type of assessment), and dependent personality disorders; men are reported to have higher rates of schizoid, schizotypal, antisocial, and obsessive-compulsive personality disorders. Of particular interest is the fact that while substance-use disorders are still generally more prevalent in men, the size of this difference is smaller and in some cases reversed, especially in adolescence. Tobacco use is now reported as equal for women and men. Shifts from gender differences to gender similarities in substance use highlight the role of social norms in the prevalence of some psychological disorders.

DSM-5 statements concerning ethnic or gender differences, although presumably based on research findings, are made without references. However, extensive information on prevalence rates of disorders based on the National Institute of Mental Health's Collaborative Psychiatric Epidemiology Surveys (CPES) is available.[1]

Gender, Race, and Class Bias

Much has been written about whether sex differences in prevalence rates represent true differences or bias. Garb (1997) concluded that there was race bias for diagnosis of schizophrenia and psychotic affective disorders such that Black and Hispanic patients were more likely than White patients to be misdiagnosed with schizophrenia when symptoms suggest psychotic affective disorders and gender bias in that a diagnosis of antisocial personality disorder (ASPD) is more likely to occur for males and HPD for females. Social class was not found to affect diagnoses.

Widiger (1998) delineated six ways in which diagnoses may reflect bias: "(a) biased diagnostic constructs, (b) biased diagnostic thresholds, (c) biased application of the diagnostic criteria, (d) biased sampling of persons with the disorder, (e) biased instruments of assessment, and/or (f) biased diagnostic criteria" (p. 96). Although specific labels and indicators have changed from DSM-IV to DSM-5, the issue of bias remains. Using these categories as a basis for understanding bias in diagnosis, we discuss biases in diagnostic standards (e.g., constructs, criteria, and thresholds), biased assessment instruments, biased applications of these diagnostic standards, and biased sampling.

Biased Diagnostic Standards Critics of DSM have argued that any classification system of psychological disorder represents an ethnocentric and possibly phallocentric construction of what an idealized, or nonpathological, self is. To the extent that this psychologically healthy self is independent, self-sufficient, rational, emotionally regulated, even unemotional, then women and individuals from less individualistic, more collectivistic cultures by virtue of gender and cultural socialization and expectations are likely to be more prone to be diagnosed with a mental disorder using the DSM.

Ross, Frances, and Widiger (1995) describe how DSM-III (American Psychiatric Association, 1980) personality disorder criteria were constructed "for the most part, by males with little input from systematic empirical research" (p. 212) and, thus, it was hardly surprising that stereotypically feminine traits were more likely to be labeled as abnormal than masculine traits. But, over time, efforts were made to make diagnostic criteria more gender neutral. For example, the HPD item "Inappropriately sexually seductive in appearance or behavior" (DSM-III-R, American Psychiatric Association, 1987, p. 349) was changed to "interaction with others is often characterized by inappropriate sexually seductive or provocative behavior" (p. 714). Removing the reference to "appearance" was intended to reduce the possibility that the normal female response to social pressure to appear physically attractive might be viewed as "inappropriately sexually seductive." Such efforts may be in part responsible for the observation made in DSM-5 that, while HPD is still seen more in women in clinical settings, structure assessments have found no gender differences (see below for biased application of the diagnostic criteria).

Somatization disorder was a disorder reported to range from "0.2% to 2% among women and less than 0.2% in men" (DSM-IV, p. 447). Although substantial revisions were made to make criteria less sex biased, DSM-IV retained criteria that made a diagnosis in men unlikely. One of the required diagnostic criteria was "one sexual or reproductive symptom other than pain." Four of the six examples given included symptoms that apply exclusively to women: "irregular menses, excessive menstrual bleeding, vomiting throughout pregnancy" (DSM-IV, p. 449). Somatic symptom disorder is the replacement for somatization in DSM-5. Although it is still reported as "likely to

be higher in females" (p. 312), the criteria for diagnosis refer only to somatic symptoms that are disruptive of daily life, are accompanied by excessive thought, feelings, or behaviors, and are persistent. This then is a diagnosis that potentially can apply equally to women and men.

The question of criterion bias is further complicated by the issue of the diagnostic validity of specific symptoms for different groups. It may be the case that certain symptoms are predictive of a diagnostic category, future symptoms, or responsiveness to treatment for some groups, but not for others. In a study assessing physical aggression as an indicator of antisocial behavior in young children, Spilt, Koomen, Thijs, Stoel, and van der Leij (2010) found that physically aggressive girls used more verbal threats, while physically aggressive boys were more likely to engage in fighting, suggesting that different behaviors were indicative of a common problem in need of detection and intervention. Indeed, DSM-5 notes that

> Males with a diagnosis of conduct disorder frequently exhibit fighting, stealing, vandalism, and school discipline problems. Females . . . are more likely to exhibit lying, truancy, running away, substance use, and prostitution. Whereas males tend to exhibit both physical aggression and relational aggression . . . females tend to exhibit relatively more relational aggression"
>
> (DSM-5, p. 474)

Another example involves diagnostic criteria for depression. In general, women are twice as likely to be diagnosed with depression in community samples in nearly all cultures. While many studies focus on the stressors or sociocultural processes that might account for women feeling more depressed than men, others have focused on the possibility that feelings of sadness and worthlessness and being tearful (noted as symptoms in DSM-5) are not consistent with societal standards of masculinity and therefore may not be reported or expressed by men, even when they are depressed (Addis, 2008). The proposed "depressive equivalent" symptoms include irritability, anger, and self-distracting or numbing behaviors, such as substance abuse, gambling, or workaholism. In a secondary analysis of the National Comorbidity Survey Replication, Martin, Neighbors, and Griffith (2013) created a male symptoms scale, which included irritability, anger attacks/aggression, sleep disturbance, alcohol/other drug abuse, risk-taking behavior, hyperactivity, stress, and loss of interest in pleasurable activity, and a gender inclusive depression scale, which included the male symptoms and traditional symptoms of depression. In support of their hypotheses, they found that the male symptoms scale was more strongly correlated with a diagnosis of major depression episode than with alcohol abuse, drug abuse, or intermittent explosive disorder, and that it led to higher prevalence in men (26.3%) than in women (21.9%). Furthermore, although endorsement of individual symptoms varied by sex, the gender inclusive scale, as a whole, yielded percentages of mild, moderate and severe depressive disorder that showed no significant sex differences. The authors conclude that irritability, anger, and substance abuse might be considered when assessing depression.

Being able to identify a criterion that is differentially valid for women and men requires a diagnostic system that is independent of this criterion. In the case of many physical illnesses, the presenting symptoms suggest a diagnosis, but this can be further assessed with blood work or other diagnostic tests. For psychological disorders, confirmation is more difficult. In reference to establishing criteria for personality disorders, Robins and Guze (1970) advocated for validation studies that would "assess the extent to which the criteria select persons who have a history, present, and/or future consistent with the construct of a (particular) personality disorder" (p. 17). One might also suggest a validity test that considers the effectiveness of standard interventions for persons with and without a particular diagnostic criterion.

According to DSM-5, "A mental disorder is a syndrome characterized by clinically significant disturbance in an individual's cognition, emotional regulation, or behavior" (p. 20). Thresholds for diagnoses should represent the point at which the accumulation of symptoms reaches this level of impairment or distress. But where is it? The issue of thresholds is one of the arguments for a dimensional rather than categorical view of psychopathology (Maddux, 2002; see also Chapter 1 in this volume), but it remains the case that clinicians and clinical researchers are regularly faced with the task of a present/absent decision in regard to the diagnosis of pathology.

Prevalence of a disorder for any particular group will be affected by the setting of this threshold. An example of possible gender bias in terms of threshold was discovered by Boggs et al. (2009). They tested for sex bias in the DSM-IV criteria for four personality disorders by comparing level of diagnosis (absent, present but of uncertain clinical significant, and definitely present) for borderline personality disorder (BPD), obsessive-compulsive personality disorder, avoidant personality disorder, and schizotypal personality disorder with functional impairment (i.e., one's level of functioning in work, social life, relationships, parenting, etc.). In general, they found that level of diagnosis predicted level of functioning similarly for both women and men. However, when they looked at the intercepts they found that diagnostic threshold set for nine of the ten BPD criteria underestimated the level of global functioning for women. This means that women with a BPD diagnosis are likely to be functioning better than men with the diagnosis. Criteria for BPD have not changed from DSM-IV to DSM-5, suggesting that we may continue to diagnosis BPD in women with less serious functional impairment compared with men.

Green et al. (2012) examined the diagnostic validity of assessment of adolescent DSM-IV disorders across racial and ethnic groups. They found that sensitivity varied for four diagnoses (and specificity for one of these as well) of the ten diagnostic categories studied. Panic disorder,

posttraumatic stress disorder (PTSD), and ADHD were underdiagnosed in non-Latino Black youth and agoraphobia was overdiagnosed and inaccurately diagnosed. Adjustments to criteria were attempted. Tightening the diagnosis of agoraphobia for Latino and non-Latino Blacks by requiring that the adolescent consider their symptoms bad or disappointing reduced inflated rates. For ADHD, removing the modification intended to adjust for parental over-reporting improved the diagnosis, suggesting that parents of Latino and non-Latino Black adolescents, compared with non-Latino Whites, do not over report ADHD symptoms. In conclusion, the authors assert that their findings "underscore the importance of testing measurement validity by race and ethnicity" (p. 318).

Biased Assessment Instruments Diagnosis generally involves objective tests or structured or semi-structured interviews. Although bias often suggests subjectivity in judgment, standardized tests can also contain bias. Differential item functioning is a term for what happens when a test item has different measurement properties for different groups. Woods, Oltmanns, and Turkheimer (2008) evaluated five scales from the Schedule for Nonadaptive and Adaptive Personality (SNAP) that demonstrated potential bias in previous research. They used a sample with women and men from five ethnic groups. They concluded that gender, ethnicity, or both, do indeed affect many SNAP items and that scores on these scales do not mean the same thing for individuals from different groups. Research of this sort reminds us that "objective" test scores are not bias free and, further, that there are accepted statistical tools for evaluating assessment instruments and improving them.

In a 2005 special issue of *Psychological Assessment* focused on evidence-based assessment (EBA), Hunsley and Mash (2005) specify, "EBAs need to be sensitive to gender, ethnicity, and cultural factors. Scientific evidence for the applicability of assessment tools needs to be demonstrated, not simply assumed on the basis of generalizations from nonrepresentative samples" (p. 252). Anthony and Rowa (2005) admit that, for many of the instruments used to assess anxiety disorders, utility across different populations remains an unanswered question. Similarly, in their article on assessment of couple distress, Snyder, Heyman, and Haynes (2005) conclude: "Given that nearly all measures of couple distress were developed and tested on White middle-class married couples, their relevance to, and utility for, assessing ethnic couples, gay and lesbian couples, and low-income couples is unknown" (p. 302).

Joiner, Walker, Pettit, Perez, and Cukrowicz (2005) cite several studies that have demonstrated no evidence for gender or ethnic bias in the commonly used assessment measures for depression. On the other hand, Sprock and Yoder (1997) reviewed studies that suggest that circumstances and age may affect the validity of these instruments. When scales were described as measures of depression, items were endorsed less often by men than by women, but there was no sex difference when the instruments were described as hassles scales (Page & Bennesch, 1993). Allen-Burge, Storandt, Kinscherf, & Rubin (1994) found that the Beck Depression Inventory and the Geriatric Depression Scale were more likely to detect major unipolar depression in elderly women than in elderly men. In addition, depressed women report more symptoms of depression than do depressed men, even when clinicians' judgments of severity of depression do not differ, leading Sprock and Yoder (1997) to suggest that the number of criteria for diagnosis of depression should perhaps be greater for women than for men.

Not surprisingly, the assessment of personality disorders presents a more complex picture (Widiger & Samuel, 2005). Problems occur when self-report instruments use responses that do not reflect dysfunction but do apply to one sex or gender more than the other (Lindsay & Widiger, 1995; Widiger, 1998; Widiger & Samuel, 2005). Bias occurs when men or women (sex bias) or masculine or feminine persons (gender bias) are more likely to endorse the item. Using college students, Lindsay and Widiger (1995) determined that 13–31% of the items from three standardized measures of personality disorder showed some evidence of sex or gender bias according to these criteria. In a follow-up study, Lindsay, Sankis, and Widiger (2000) used outpatients from mental health clinics and updated versions of the instruments: Millon Clinical Multiaxial Inventory-III (MMCI-III), Minnesota Multiphasic Personality Inventory-II, and Personality Diagnostic Questionnaire-Revised. Four personality disorder scales (histrionic, dependent, antisocial, and narcissistic) from these instruments were analyzed for sex or gender bias. None of the scale scores from any of the inventories was related to biological sex. Only three scale items showed sex bias (women or men were more likely to endorse them but the item was unrelated to a measure of psychopathology); whereas 36 items suggested gender bias (the item correlated with a measure of femininity or masculinity but not with the measure of psychopathology). Although the few items (three) demonstrating sex bias were all from the histrionic scales, more examples of gender bias were from the narcissistic scale, suggesting that masculine individuals may receive a higher score by endorsing items that reflect adaptive, rather than pathological, characteristics.

Less attention has been paid to the role of ethnicity in assessment of personality disorder, although Widiger and Samuel (2005) note the finding of higher scores for African Americans compared with Whites on the paranoid personality disorder scales of the MCMI-III. As they suggest, it is possible (but not empirically established), that experiences of discrimination and prejudice lead to greater endorsement of items such as "I am sure I get a raw deal in life," which contributes to the paranoid personality score but may be a reasonable reflection on a life lived facing barriers imposed by racism.

Biased Application of the Diagnostic Criteria Regardless of the care taken to establish reliable and valid diagnostic tools, clinicians using the tools or other sources of clinical information may be biased in making diagnoses. López (1989) found evidence of overpathologizing for cases with low social class and diagnostic category biases for race and, to some extent, gender. But he also argued that clinicians can both overpathologize and underpathologize in their judgments of patients and that errors in judgment affect both oppressed and nonoppressed groups and result from information processing biases not just prejudice or discrimination. Luebnitz, Randolph, and Gutsch (1982) found that lower-class African American patients were more likely to receive a diagnosis of chronic alcohol abuse than White lower-class, or African American, or White higher-class patients. However, given the symptoms presented, the error was more in underdiagnosing alcohol abuse in the White and higher-class African American patients. Similarly, a study by Leinhardt, Seewald, and Zigmond (1982) suggests that White boys are disproportionately identified as learning disabled compared with Black and female children. Diagnoses of depression may also be affected by clinician bias. Potts, Burnam, and Wells (1991) compared clinicians' judgments with diagnoses made with a standardized interview assessment and found that there were discrepancies between standardized assessment and clinician judgments, in that medical practitioners were less likely to diagnose depression in men and mental health practitioners were more likely to diagnose depression in women. Overpathologizing can lead to unnecessary interventions and stigma; underpathologizing leads to lack of treatment and failure to intervene where needed.

López (1989) noted that 45% of experimental studies examining severity judgments and 55% examining diagnostic judgments revealed bias. Warner (1978) gave clinicians ambiguous patient profiles, with a mixture of hysterical and antisocial symptoms (e.g., suicide attempts, no close relationships, self-centered, shoplifting with no remorse for crime, flirtatious). Female profiles were more likely than male profiles to be labeled hysterical (76% vs. 49%) and male profiles were more likely than female to be labeled antisocial (41% vs. 22%). It can be argued that with ambiguous information, clinicians rely on base rates (women are more often diagnosed with HPD; men are more likely to be diagnosed with ASPD). The base rates themselves, however, may also represent a biased accumulation of data on women and men. If clinicians use stereotyped perceptions of women and men in their diagnoses (as these studies indicate), then the data based on their clinical judgments will be biased.

To circumvent the issue of base rates, subsequent studies presented clinicians with cases that meet the DSM criteria for one or more disorders. Ford and Widiger (1989) created cases that met DSM-III criteria for HPD or ASPD, or failed to reach diagnostic criteria for either. HPD was more frequently diagnosed in women, even when the case contained more antisocial than histrionic criteria; and ASPD was less often diagnosed in women. Adler, Drake, and Teague (1990) developed one case history that met the explicit criteria of four diagnoses: histrionic, narcissistic, borderline, and dependent. Men were more likely to receive a diagnosis of narcissistic personality disorder; women, HPD. There were no differences for borderline or dependent (which was rarely used) personality disorder. Fernbach, Winstead, and Derlega (1989) created separate vignettes describing clients who met diagnostic criteria for antisocial and somatization disorders and found that there were no sex-of-vignette differences for somatization disorder; but, for ASPD, although most clients were accurately diagnosed, men (73%) were significantly more likely than women (53%) to receive the diagnosis; 19% of clinicians made an inaccurate diagnosis of BPD; of these, 68% were for the female clients and 32% for the male clients. Crosby and Sprock (2004) further explored the influence of patient sex on diagnosing antisocial personality disorder. They provided clinical psychologists with two cases, one meeting minimum criteria for ASPD, the other used as a distracter. Female clients were underdiagnosed for ASPD and overdiagnosed for BPD.

If the diagnostic criteria for a disorder are built into the case description, then clinicians are coming up with the "wrong" answer when they indicate a diagnostic category other than the one intended. Ford and Widiger (1989) demonstrated that at the level of individual criteria there is no sex bias. Specific behaviors were rated as indicative of HPD and ASPD with equal frequency for men and women, but they did find sex bias when assigning clients to one of these diagnostic categories. They concluded that HPD was overdiagnosed in women and underdiagnosed in men (Ford & Widiger, 1989). Morey and Ochoa (1989) and Blashfield and Herkov (1996) looked at clinicians' ratings of DSM criteria for their own patients and compared ratings on individual criteria with actual diagnoses of these patients. They found weak associations between clinicians' ratings of criteria and the clinicians' diagnoses, suggesting that giving a diagnosis does not necessarily involve an objective formula based on the presence or absence or the total number of certain criteria. Morey and Ochoa (1989) found that clinicians overdiagnose ASPD in men and BPD in women; and Blashfield and Herkov (1996) replicated these findings. The absence of sex bias for specific criteria in contrast to the presence of sex bias in assigning DSM-based diagnoses suggests that dimensional approaches to diagnosis may be less prone to bias than categorical labels, as judgments in the dimensional approach remain at the level of behavioral indicators rather than a summary diagnostic decision.

A great deal has been written about the overdiagnosis of schizophrenia in African Americans and the underdiagnosis of affective disorders (Baker & Bell, 1999; Simon, Fleiss, Gurland, Stiller, & Sharpe, 1973). Neighbors, Trierweiler, Ford, and Muroff (2003) cite numerous studies that have found higher rates of schizophrenia in African Americans and mood disorders in Whites. In a

study of race disparities in emergency rooms, Kunen, Neiderhauser, Smith, Morris, and Marx (2005) found that African American patients were less likely than Whites to receive a diagnosis for psychiatric disorders. Anxiety and depression were more likely to be diagnosed for White than for African American patients, and even though Whites were more likely to be diagnosed as psychotic, African Americans were more likely than Whites to be diagnosed for schizophrenia. Although differences in prevalence do not necessarily indicate bias, no racial differences in prevalence rates for psychotic disorders were reported in a major epidemiological study (Robins & Regier, 1991) where researchers use structured interviews in community samples to establish, as objectively as possible, actual rates of psychological disorders.

Unlike research on gender bias, which has relied on analog studies, most studies supporting racial bias have compared diagnoses by hospital or clinic staff with diagnoses from research investigators using chart reviews or structured interviews (e.g., Mukherjee, Shukla, Woodle, Rosen, & Olarte, 1983; Pavkov, Lewis, & Lyons, 1989). Neighbors et al. (1999) found the agreement between hospital and research diagnoses to be low for both African American and White patients, but the higher rates of schizophrenia diagnoses for African Americans and of mood disorders for Whites remained, even with research diagnoses. Nevertheless, racial discrepancies were smaller in the more structured assessment (using a DSM-III checklist) than in the less structured assessment. In a subsequent study using research interviews, Neighbors et al. (2003) found the same race differences in schizophrenia (African American higher) and bipolar disorder (Whites higher), but no differences in major depression or schizoaffective disorder. There were no socioeconomic status differences among patient groups. In this study, they also measured ratings of symptoms and relations between symptoms and diagnoses. Similar profiles were used for diagnosing bipolar disorder, although White patients received higher ratings for hypertalkativeness, which may have led to their higher rate of bipolar diagnosis. But, for schizophrenia, more individual symptoms were related to the diagnosis for African Americans than for Whites; and of particular interest, inappropriate affect, although equally observed in the two groups, was considered an indicator of schizophrenia for African Americans but not for Whites (Neighbors et al., 2003).

Examining diagnoses of psychotic patients discharged from a state psychiatric hospital, Strakowski, Shelton, and Kolbrener (1993) found more diagnoses of schizophrenia, especially the paranoid subtype, and fewer diagnoses of affective disorders for African American patients than for White patients. Similarly, in a psychiatric emergency clinic, Strakowski et al. (1995) found more diagnoses for schizophrenia, particularly for African American men. African-American patients (Strakowski, McElroy, Keck, & West, 1996) and African-American men (Arnold et al., 2004) have been found to exhibit more severe psychotic symptoms, especially first-rank symptoms (i.e., hallucinations, delusions, disordered thinking), even when diagnostic interview transcripts contained no information about race. Gara et al. (2012) looked again at the influence of patient race and ethnicity on clinical assessment with ethnicity-blinded and unblinded interview transcripts and including a Latino sample, as well as African American and White patients. They found, yet again, that African Americans were more likely to receive a diagnosis of schizophrenia, whether their ethnicity was known or not. They did not, however, vary in terms of diagnoses of affective disorders. Latino patients did not differ from Whites. Males and those with low income were also more likely to receive a diagnosis of schizophrenia. The authors suggest that African Americans may present with higher levels of psychotic symptoms that are overvalued by clinicians leading to a diagnosis of schizophrenia. They also argue that in cases where the schizophrenia diagnosis overrides a proper diagnosis of mood disorder, treatment may be inappropriate and the more negative prognosis may also affect clinician, patient, and patient's family in their view of the likelihood of treatment success.

An exception to the absence of analog studies to explore racial/ethnic bias in diagnosis and the exclusive focus on gender in most analog studies is the work of Kales et al. (2005) and Loring and Powell (1988). Kales et al. (2005) used video vignettes of elderly African-American and White women and men describing symptoms that met criteria for major depression but also included cognitive problems, alcohol use, and paranoid thoughts. The vignettes were viewed by primary-care physicians. In this well-controlled study neither gender nor race nor the interaction affected diagnosis or treatment recommendations. These physicians were generally accurate in their diagnoses; 70–80% of cases were diagnosed with major depression. They were even more likely to recommend antidepressants as the first-line treatment, 82–90%, although interestingly they were unlikely to refer the patient to a psychiatrist or other mental health professional, 9–19.5%.

Using written vignettes that included both personality disordered (dependent) and psychotic (undifferentiated schizophrenia) symptoms in the cases to be diagnosed, Loring and Powell (1988) manipulated race (African America vs. White) and gender as demographic characteristics. Vignettes of clients with no information about ethnicity or sex were also included. Finally, they examined gender and race of the psychiatrists as influences on the diagnostic process. They found that modal diagnoses were most accurate in cases without information on gender and ethnicity. When this information was introduced, complex patterns of interaction between characteristics of the case and characteristics of the diagnosing psychiatrist occurred. Generally, similarity in race and gender between clinician and client produced the most accurate diagnoses, but male clinicians overdiagnosed depression in women and HPD in White women, and all clinicians

overdiagnosed paranoid schizophrenia and paranoid personality disorder in African American men. This study reinforces the conclusion that the interaction of race and gender has a powerful influence on diagnosis.

Loring and Powell (1988) suggested that clinicians are more accurate in their diagnosis when the gender and race of the case are the same as those of the clinician. An exception was diagnosis of a White female client by White female clinicians that suggested favoring a less severe diagnosis. Garb (1997) presents further evidence that clinicians may see less pathology in similar others and more pathology in dissimilar others. For example, Li-Repac (1980) found that White clinicians compared with Chinese American clinicians gave more negative ratings on depression and interpersonal variables to Chinese American clients and Chinese American clinicians rated White clients as having more severe psychopathology. Tseng, McDermott, Ogino and Ebata (1982) found that American psychiatrists' ratings of Japanese fathers were more negative than Japanese psychiatrists' ratings. Russell, Fujino, Sue, Cheung, and Snowden (1996) found that, for African American, Asian, White, and Hispanic clients, when therapist and client were ethnically matched, therapists' ratings of overall client functioning were more positive. A possible source of bias in diagnosis is the actor-observer effect (Jones & Nisbett, 1987), by which we are more likely to explain our own behavior, or the behavior of those with whom we can identify, in terms of situational factors, but the behavior of others in terms of inner causes, such as a psychological disorder (Poland & Caplan, 2005). This may lead clinicians to overpathologize others, perhaps especially those whose sociocultural context is difficult for the clinician to understand, but it can also lead to underdiagnosing similar others.

These studies indicate that sex and race of clients does influence clinician judgments about diagnosis. Some diagnoses are more prone to this bias than others. Although analog studies allow the researcher to present clinicians with identical information while varying characteristics of the client, the responses of clinicians to paper and pencil, or even audio or video versions of a patient, cannot completely capture the more interactive process that actually occurs during diagnosis, a process which may be even more susceptible to bias. On the other hand, evidence also suggests that the use of structured interviews, compared to unstructured interviews, can diminish the effects of bias.

Biased Sampling The most convenient data for analysis of prevalence rates are data obtained in clinical settings. But do all individuals with a particular disorder show up in a clinical setting? Factors bringing adults into a clinical setting include willingness to acknowledge the symptoms, willingness to seek treatment, or the persuasive or coercive influence of others. Factors bringing children into a clinical setting include parents' and teachers' perceptions of the severity of the problem and of the possibilities for treatment. These factors are likely to be influenced by the gender, race, and class of the affected individual. Women may be more likely to recognize and less resistant to acknowledging that symptoms are indicative of an emotional problem that needs treatment (Yoder, Shute, & Tryban, 1990). Non-Whites are less likely to have access to mental health care, in part due to lack of health insurance, and less likely to use specialized care, often seeking help from primary care services (U.S. Department of Health and Human Service, 2001). Arnold et al. (2004) suggest that one reason first-rank symptoms of schizophrenia may be more pronounced in African American men is that they have delayed seeking mental health treatment. To the extent that rates of psychological disorder seen in clinical populations are the result of differences in help seeking, problem recognition, tolerance/intolerance for symptoms by self or others or attitudes toward mental health care, then differences in prevalence rates in clinical settings will reflect these differences and not just "true" differences in the occurrence of the psychological disorder.

The solution to this dilemma is epidemiological studies with community samples. In these studies, interviewers contact individuals in representative samples and acquire information about symptoms. Diagnosis is accomplished independently of the participants' acknowledgment of or concern about the disorder. Whereas differences in clinical populations reflect differences in the psychological disorders plus differences in various non-disordered behaviors such as help seeking, differences in community samples will not have this problem. For example, Miltenberger, Rapp, and Long (2006) suggest that the gender difference in trichotillomania (compulsive hair pulling), which is seen far more often in women than men in clinical settings, may result in part from women being more inclined than men to seek treatment for such an appearance-altering problem. More men than women seek help for gambling, but female gamblers may feel stigmatized and unwilling to seek treatment (see Hartung & Widiger, 1998).

The National Institute of Mental Health's CPES combine three nationally representative data sets: the National Survey of American Life, the National Comorbidity Survey Replication and the National Latino and Asian American Study.[2] The website lists two books and 392 journal articles based on these data, many focusing on gender, race, and class. Ideally, every disorder would have an adequate and current base of epidemiological data from community samples but, often, statements about differences in prevalence rates are based on potentially biased clinical samples.

The Role of Gender, Race, and Class in Treatment

When we consider disparities in mental health care, we must take into account the fact that the majority of individuals identified as having psychological disorders do not receive care for these disorders. Using data from the CPES,

Wang et al. (2005) found that, within a 1-year period, only 41% of those identified with a psychological diagnosis received any care; and González, Tarraf, Whitfield, and Vega (2010) found that only 50.6% of respondents who met major depression criteria had received either pharmacotherapy or psychotherapy and many fewer (21.2%) received therapy consistent with APA guidelines. Nevertheless, low income and minority status are barriers to receiving mental health care (Cooper-Patrick et al. 1999; U.S. Department of Health and Human Service, 2001, González et al., 2010). Wells, Klap, Koike, & Sherbourne (2001) found that among those with perceived need for treatment for alcoholism, drug abuse, or mental health disorders, African American and Hispanics were more likely to report unmet need and were less likely to be in active treatment. Additionally, Hispanics, compared with Whites, reported significantly more delays in treatment and less satisfaction in all components of health care. Cook, Zuvekas, and colleagues (2013), using the Medical Expenditure Panel Survey, reported that, controlling for need, African Americans and Latinos initiated mental health care less often, were less likely to have minimally adequate care, and had episodes of care that were shorter in duration, compared with Whites. Cook, Barry, and Busch (2013) found significant disparities in mental health care use for African American and Latino children and these disparities did not change from 2002 to 2007. Since expenditures on mental health care were comparable across groups once children initiated care, the authors focus on the need to reduce barriers to access, such as stigma, losing pay from work, and lower satisfaction with care (Cook, Zuvekas et al., 2013). Using the National Comorbidity Survey Replication data, Wang et al. (2005) found that unmet need was greatest for those with low incomes and without insurance and for minorities, the elderly, and rural residents. Lack of access to transportation or inflexible work schedules can also work against using mental health resources, even when they are available through public mental health services (cf. Armistead et al., 2004, who increased retention in a community intervention program by providing childcare and transportation). Because publicly funded programs, such as Medicaid, are intended to provide mental health care for the uninsured, the connection between medial insurance and access to care is complicated. For example, racial disparities in use of publicly supported services are generally small but data from the National Medical Expenditure Survey revealed that African Americans with private insurance are less likely than Whites to use mental health care (Snowden & Thomas, 2000). It is likely that the impact of the Affordable Care Act on access to and use of mental health care will be closely followed.

Pingitore, Snowden, Sansone, and Klinkman (2001) reported that, for individuals with depressive symptoms as their main reason for a medical visit, men, African Americans, other persons of color, and those over 65 were more likely to seek care from a primary-care physician, whereas women, Whites and individuals between 45 and 64 years were more likely to visit a psychiatrist. The quality of care provided by primary-care physicians was significantly lower than the care provided by a psychiatrist. Alegría et al. (2002) found that, in a low socioeconomic status sample, Latinos, compared with Whites, and in a higher socioeconomic sample, African Americans, compared with Whites, were less likely to use specialty care for mental health problems. Other studies confirm that Latinos (Cabassa, Zayas, & Hansen, 2006) and African Americans (Cooper-Patrick et al., 1999; Snowden & Pingitore, 2003) are more likely to receive mental health care in a general medical setting than from a mental health specialist. In addition, Kung (2003) found that about 15% of Chinese Americans (in Los Angeles) received mental health specialty care, a figure even lower than that reported for other minority groups. Wang, Berglund and Kessler (2000) found no ethnic or racial differences in access to treatment, but they did find that being African American and not having insurance predicted not receiving evidence-based care. Young, Klap, Sherbourne, and Wells (2001) also found that African Americans and Hispanics suffering from anxiety or depression were less likely than Whites to receive care congruent with established guidelines. Cabassa et al. (2006) reviewed 16 articles based on seven epidemiological studies and concluded that Hispanic adults compared with non-Latino Whites both underutilize mental health services and are less likely to receive guideline congruent care.

Chow, Jaffee, and Snowden (2003) examined clinical characteristics and mental health service use patterns of Whites, African American, Hispanic, and Asians in low and high poverty communities and found that socioeconomic status does moderate the relationship between ethnicity and service access and use. In high poverty areas, Hispanics and Asians were more likely than Whites to use emergency services, and Whites were more likely to receive a diagnosis of schizophrenia, perhaps reflecting the downward social mobility of poorly functioning Whites. In low poverty areas, African American and Hispanics were more likely than Whites to be referred into mental health services by law enforcement, perhaps reflecting the fact that deviant behavior by minorities in low poverty neighborhoods are more likely to be attended to and constrained. Results were complex but clearly indicate that paths to mental health care differ by both ethnicity and the economic status of one's community.

Kuno and Rothbard (2005) examined quality of care provided by community mental health centers in neighborhoods with different income and racial profiles. Using quality indicators, such as use of atypical antipsychotic prescriptions and intensive case management, they found that community mental health centers in high-income, White areas had higher quality of care than those in low-income, African American neighborhoods. This suggests that even when mental health care is available; it may not be of similar quality.

These last two studies also illustrate an inherent problem in the ways in which socioeconomic status is generally used in mental health research. Socioeconomic status is most commonly measured at the individual or family level. Although there are complex and sophisticated ways to create such a participant specific measure, neighborhood level measurement is often superior to these. Corral and Landrine (2010) make a strong argument for area-based measures of socioeconomic status, including the fact that low socioeconomic status neighborhoods contain poorer-quality services and more stressors than high socioeconomic status neighborhoods. This is an important consideration in research examining social class and psychopathology.

When gender is included in analyses, the results tend to support the general finding that women are more likely to seek mental health care than men, and this holds true in Hispanic (Cabassa et al., 2006) and Chinese American (Kung, 2003) samples. Men are also more likely than women to delay seeking treatment (Wang et al., 2005). These studies support the general consensus that women are more likely than men to seek psychological treatment (Petry, Tennen, & Affleck, 2000).

Beyond issues of access and general quality of care, we also want to know if gender, race, and class affect the outcomes of treatment. The complexity of the question is revealed if we consider some of the other treatment-relevant variable: Sociodemographic characteristics of the therapist (e.g., gender, race, and class), type of therapy, length of therapy, type of problem being treated. Many studies do not have the methodological controls that allow for random assignment of clients to therapists and therapists to clients, making therapist or client characteristics nonexperimental variables. In these cases the possibility that unmeasured variables account for the outcomes must always be kept in mind.

Influence of the Client on Therapy Outcomes In reviewing the impact of client variables on psychotherapy outcomes, Clarkin and Levy (2004) cite studies showing that higher socioeconomic status is related to staying longer in psychotherapy and that higher levels of education are related to longer treatment for substance abuse programs, although they also found studies in which socioeconomic status was not related to remaining in treatment. Wierzbicki and Pekarik (1993), in a meta-analysis of psychotherapy dropout, found that race (minority status), low level of education, and low socioeconomic status all significantly predicted dropping out of therapy. Reis and Brown (1999), in their review of three decades of research on the unilateral terminator (i.e., a "client who terminates treatment before the clinician believes the client is ready," p. 123), concluded that socioeconomic status is a reliable predictor of unilateral termination, and that, despite some nonsignificant results, non-White clients are also more likely to terminate unilaterally. In a more recent study of individual therapy in a substance abuse clinic, researchers found that being female and African American were independent predictors of early treatment dropout (King & Canada, 2004).

Clarkin and Levy (2004) conclude that neither race nor gender have much impact on therapy outcome. On the other hand, a review by Lam and Sue (2001), while finding that "improvement in therapy is independent of client gender" (p. 480), reported equal numbers of studies that find no difference in outcomes for African Americans compared with Whites and studies that find that African Americans gain less in therapy. They also reference a study that found that American Indian youth did not benefit as much from substance abuse treatment as Whites (Query, 1985), but they conclude that outcomes for Asian Americans and Hispanics may be similar to those of Whites. Rosenheck, Fontana, and Cottrol (1995) found that African American veterans in treatment for PTSD were more likely to drop out but also less likely to benefit from therapy than White veterans.

Despite general conclusions of no sex-of-client effects on therapeutic outcomes, some studies suggest otherwise. Recent studies of substance abuse (Galen, Brower, Gillespie, & Zuker, 2000; Kosten, Gawin, Kosten, & Rounsaville, 1993) and substance abuse plus PTSD (Triffleman, 2001) have found greater severity for women than for men at the beginning of treatment, but equal levels of functioning at the end of treatment, suggesting that women made greater gains during therapy than did men. Ogrodniczuk, Piper, & Joyce (2004) looked at outcomes of short-term group therapy for depression and complicated grief. Women were found to benefit more from this therapy, but they were also found to be more committed to the therapy groups and to be perceived by other group members as more compatible. Both a meta-analysis of child and adolescent outcome research (Weisz, Weiss, Han, Grnager, & Morton, 1995) and a study of the psychotherapy outcomes for youth treated in public outpatient programs (Ash & Weis, 2009) found that girls were more likely to benefit from treatment than boys, although Suveg and colleagues (2009) found no effect for gender on outcomes for individual and family-based interventions for anxiety disorders in a randomized clinical trial.

Issues about women's special needs in substance abuse treatment have been discussed (Wilke, 1994). Treatments included in the studies reviewed above were all outpatient programs. Many interventions for substance abuse or addiction rely, however, on inpatient treatment. Some women may be unable to participate in inpatient treatment or need to terminate treatment prematurely because they may be the sole caregiver for their children (Wilke, 1994). Questions have also been raised about differences in the effectiveness of single-sex and mixed-sex treatment groups. Dahlgren and Willander (1989) found that women in a single-sex group reported better social adjustment and lower alcohol consumption at a 2-year follow-up

than did women in a mixed-sex group. Similarly, in a randomized controlled trial, a manual-based 12-session Women's Recovery Group was compared with a mixed-gender Group Drug Counseling. Although no differences appeared at the end of treatment, women in single-sex group showed continued gains and greater reduction in drinking at the 6-month follow-up. Client satisfaction was also significantly higher for women in the single-sex group (Greenfield, Trucco, McHugh, Lincoln, & Gallop, 2007).

Client–Therapist Matching Many studies have examined client–therapist matching in terms of race or gender, on the assumption that greater understanding or credibility will occur when therapist and client share these critical social statuses. The review of client variables by Petry et al. (2000) reports mixed results from studies of matching for race. Some find that clients stay in treatment longer, others that there is no effect on retention or outcomes. In their review, Lam and Sue (2001) report evidence that matching for African Americans is related to number of sessions (Rosenheck et al., 1995), but not to outcomes, and that matching for Asian American and Hispanics is related to less dropout and more therapy (e.g., Gamst, Dana, Der-Karabetian, & Kramer, 2001; Sue, Fujino, Hu, Takeuchi, & Zane, 1991). In subsequent studies, Gamst et al. (2003, 2004) again found no advantage for ethnic matching for African American children and adolescents, but greater client satisfaction and better child outcomes for ethnically matched Asian Americans. Sterling, Gottheil, Weinstein, and Serota (2001) found no effects for either race- or sex-matching, in terms of retention or outcome in a study of substance abuse treatment for African American cocaine-dependent clients. In a general review of the relationship between ethnic match and termination, Maramba and Hall (2002) concluded that while ethnic similarity reduces rates of treatment dropout and increases the length of time in treatment, the effects are small and do not improve clinical outcome.

In a study examining the outcomes from an empirically based treatment, multisystemic therapy for youth with antisocial behavior, Halliday-Boykins, Schoenwald, and Letourneau (2005) found that caregiver ethnic similarity did matter. The very large sample was diverse and assignment to therapist was to next available. The analyses revealed that "when caregivers are ethnically matched with therapists, youths demonstrate greater decreases in symptoms, stay in treatment longer, and are more likely to be discharged for meeting treatment goals" (p. 814). This effect was partly mediated by therapists' greater adherence to the multisystemic therapy protocol in ethnically matched cases.

A diverse sample of children and adolescents seen in outpatient services in mental health facilities also found that race and language matching matter (Hall, Guterman, Lee, & Little, 2002). Ethnic match, but not gender match, was related to lower dropout rates for Asian American, Mexican American, and African American children. Language match predicted only for Mexican Americans. Ethnic match predicted more treatment sessions for Mexican Americans and Asian Americans; and language and gender match were additional predictors for Mexican Americans. In a meta-analysis of culturally adapted mental health interventions, Griner and Smith (2006) found that studies with no report of matching had a higher average affect size than studies that did attempt to match, although matching for language had a positive effect. In some studies matching for ethnicity and matching for common language may be confounded.

Sue, Zane, and colleagues have argued that the advantages of ethnic matching, when they occur, probably accrue from similarities in problem perception, coping orientation and goals for treatment (Zane et al., 2005). In a sample of Asian American and White outpatients they assessed client and therapist for these factors independently and prospectively and found that

> treatment-goals match was predictive of session effect and comfort. Coping-orientation match was predictive of less dysphoria after four sessions of treatment [and] . . . similarities . . . in perceived distress associated with the problem appeared to result in higher psychosocial functioning at the end of the fourth session"
>
> (Zane et al., 2005, p. 582)

This important study suggests mechanisms by which matching may affect outcomes.

Gender matching has also been a focus of research. Jones and Zoppel (1982) obtained self-reports from clients and therapists regarding the results of therapy. They found that female clients paired with female therapists were perceived by both clients and therapists as having experienced the most positive outcomes, suggesting that sex matching made the difference, but other research fails to support benefits from gender matching. Zlotnick, Elkin, and Shea (1998) took advantage of an exceptional opportunity to investigate sex of therapist effects in a well-controlled study, the National Institute of Mental Health Treatment of Depression Collaborative Research Program. In this research, all clients were experiencing major depression and were assigned randomly to different treatment conditions, but also to either a female or male therapist. Attrition, treatment outcome (based on the Hamilton Rating Scale for Depression), and client-reported therapist empathy were assessed. Analyses revealed that

> among depressed patients, a male or female therapist, or same-versus opposite-gender pairing, was not significantly related to level of depression at termination, to attrition rates, or to the patient's perceptions of the therapist's degree of empathy early in treatment and at termination."
>
> (Zlotnick et al., 1998. p. 657)

Clients' beliefs about whether a female or male therapist would be more helpful were also assessed prior to therapy, but these beliefs had no impact on outcomes whether they were assigned to the person they believed would be more helpful or not. Similarly, in a study of clients randomly assigned to one of three cognitive behavioral treatment conditions for panic disorder, neither gender of the therapist or gender match had an effect on therapy outcomes (Huppert et al., 2001).

Certain deleterious effects of psychotherapy are, however, more likely to occur in some sex of therapist–sex-of-client combinations. For example, data suggest that sexual relationships occur between therapist and client in 5–6% of therapy relationships, with 85% of these involving a male therapist with a female client (Lamb & Catanzaro, 1998). This particular negative therapeutic experience is clearly more likely to affect women.

Culturally Adapted Therapy The current emphasis in psychological treatment is on empirically supported treatments (Stewart and Chambless, Chapter 8 in this volume). Once a treatment has been found to be effective in clinical research, we may hypothesize that it will be effective universally, in similarly diagnosed but ethnically different populations; but data suggest that this assumption is not being empirically tested. Weisz, Doss, and Hawley (2005) identified 236 studies of randomized trials testing 386 treatments for children and adolescents. They report that "60% of all the articles failed to include any report on the race or ethnicity of their samples, and more than 70% failed to provide any information on family income or socioeconomic status" (p. 348). Numbers that were reported indicated that while African American youth are generally represented in these samples; other minorities are not. Finally, samples of minority youth are generally too small to adequately test the effectiveness for these groups in comparison with Whites. A major review of treatments for depression failed to address the question of effects for gender, race, or class at all, except to acknowledge that since women are prone to depression in the childbearing years, the relative success of psychotherapy without medication is encouraging (Hollon, Thase, & Markowitz, 2002).

The assumption that effective treatments need no further adaptations to be universally useful is challenged by a meta-analysis of 76 studies that investigated outcomes of culturally adapted treatment (Griner & Smith, 2006). For the 62 studies with stronger experimental or quasi-experimental designs, the effect size was $d = .45$. Examination of moderators demonstrated that older clients and higher percentages of Latino participants led to higher effect sizes. Furthermore, language matching for Latino groups greatly increased the effect size, suggesting that reducing or eliminating the language barrier for adult Latino clients greatly improves their mental health outcomes. Looking at types of outcomes, the researchers found that client satisfaction was most affected by the adaptation. Of note is the fact that in a burgeoning literature on treatment outcomes so few studies looking explicitly at planned cultural adaptations of therapy could be found. Nevertheless, this meta-analysis strongly suggests that programs which deliberately take the culture of clients into account will improve their therapeutic outcomes.

Gender, Race, and Class and Pharmacotherapy In previous decades, the exclusion of women and minorities from clinical trials led to a lack of information about the differential responses of women and minorities to psychopharmacological treatment. In 1994 (amended in 2001) the federal government issued "NIH Guidelines on the Inclusion of Women and Minorities as Subjects in Clinical Research" (National Institutes of Health, 2014), including both biomedical and behavioral research.

Results concerning the effects of race on pharmacotherapy are inconsistent. In a study of visits to primary-care providers, Lasser, Himmelstein, Woolhandler, McCormick, & Bor (2002) found that African Americans were less likely to receive antidepressant, anti-anxiety, or any drug therapy and similar results were found for visits to psychiatrists, except that African American clients were also less likely to receive antipsychotic prescriptions. Similarly, Snowden and Pingitore (2003) found that visits to a primary-care physician with a mental health concern led to less pharmacotherapy for African Americans than Whites. On the other hand, in a sample of clients with a primary diagnosis of major depression for whom drug treatments were provided only by specialty mental health clinics, African Americans were more likely than Whites to receive pharmacotherapy.

Results have also indicated that African Americans may be less likely than other ethnic groups to receive the current standard of care. Kuno and Rothbard (2002) found that African Americans treated for schizophrenia were significantly less likely than Whites to be prescribed nontraditional, second-generation, antipsychotics, even when controlling for funding and service types. Similarly, Herbeck et al. (2004) found that African American men, adjusting for clinical, socioeconomic status, and health-system characteristics, were significantly less likely to receive these drugs and more likely to receive medications that have a greater risk of producing tardive dyskinesia and extrapyramidal side effects. Garb (2005) found that adherence with recommended treatment for schizophrenia was poor and that African American clients often received "excessively high dosages of antipsychotic medicine, and [were] less likely to be on atypical psychotics" (p. 82). These results suggest that African American patients are being overmedicated on traditional drugs and are not benefiting from advances in psychopharmacology.

There are also documented racial differences in attitudes toward psychiatric medication. Schnittker (2003) found that African Americans reported less willingness to use psychiatric medications or to give them to children

in their care. These attitudes about pharmacotherapy were predicted by beliefs about efficacy and side effects, and were not related to socioeconomic status, knowledge, religiosity, or medical mistrust. On the other hand, Haekyung (2012) found that income level was associated with use of selective serotonin reuptake inhibitors, but not in a linear fashion. The low-income respondents were less adherent than high- or middle-income respondents, but very-low-income respondents were not. They suggest that very-low-income patients may have access to public health insurance that the low-income patients do not have. A study investigating maintenance pharmacotherapy for bipolar disorder found that fear of addiction and medication as a symbol of mental illness contributed to nonadherence in African American patients (Fleck, Keck, Corey, & Strakowski, 2005).

Women are more likely than men to be prescribed psychotropic drugs, even after controlling for other demographics, health status, socioeconomic status, and diagnosis (Simoni-Wastila, 2000). But this may reflect women's mental health care-seeking behaviors, as Garb's (1997) review of numerous studies using case vignettes did not find gender effects on recommendations for use of psychotropic medications.

Sex and ethnicity may also affect responses to drug treatment. Differences may include absorption, distribution, metabolism, and elimination, all of which affect the bioavailability of the therapeutic substance. Women, for example, tend to have more fat tissue than men, which affects the metabolism and storage of drugs. Women's endogenous (e.g., menstrual cycle, pregnancy, postpartum, menopause) and exogenous (e.g., birth control pills, hormone replacement therapy) hormone levels also affect drug response (Yonkers & Hamilton, 1995). Estrogens tend to increase the effectiveness of some antipsychotic drugs (Seeman, 1995), meaning that women may be treated with lower doses; but they may also experience more side effects or toxicity from doses that are safe in men (Yonkers & Hamilton, 1995). Estrogen affects metabolic processes and drug response, and estrogen has been tried with mixed to positive results, alone and combined with antidepressants, in treatment for women with refractory depression and for postmenopausal women (Casper, 1998; Kornstein, 1997). Research on the effectiveness of antidepressant medication, however, suggests that women respond more poorly than men to tricyclics and better than men to selective serotonin reuptake inhibitors and monoamine oxidase inhibitors (Kornstein, 1997). On the other hand, females with schizophrenia respond better and more rapidly to pharmacological treatment and require lower doses than men in both acute episodes (Szymanski, Lieberman, Alvir, & Mayerhoff, 1995) and ongoing treatment (Tamminga, 1997). Women are also more likely than men to be exposed to more than one psychoactive drug, owing to a greater incidence of comorbidity, and so drug interaction effects must be considered (Casper, 1998).

Different ethnic groups may have different responses to drug treatments (Baker & Bell, 1999; Lin & Smith, 2000; Snowden, 2001). For example, African Americans, compared with Whites, may have reduced responsiveness to beta blockers and may be more likely to be nonresponders to fluoxetine, but may respond better and more rapidly to tricyclic antidepressants and to certain benzodiazepines and to need less lithium to control manic symptoms (Baker and Bell, 1999). The presence of sickle cell anemia in some African Americans may also complicate pharmacotherapy. Ethnic differences in therapeutic levels of lithium, clozapine, and antidepressants have been reported as well as differences between Asian Americans and Whites to haloperidol (Lin & Smith, 2000).

Finally, psychological disorders, especially depression, frequently occur in women 20–45 years of age, years when women are likely to bear children. The effects of psychopharmacological agents on maternal health, the health of the developing fetus, and lactation must be understood. To date, research suggests that benzodiazepines (anti-anxiety drugs) lead to craniofacial abnormalities and, when taken in late pregnancy, "floppy infant" syndrome; lithium (used in treating bipolar disorder) has been associated with cardiovascular, central nervous system, and mental and physical abnormalities; antidepressant medications have shown no adverse effects in some investigations, but small increased risks of miscarriage or deformities in others; antipsychotics have yielded mixed outcomes (Casper, 1998). Clearly research clarifying the effects of medication during various stages of pregnancy and during lactation is critical.

Understanding Gender, Race, and Class

Chang (2003) presents two basic research perspectives for understanding racial (and, we would add, gender) differences in mental health and mental health care. The first approach seeks to discover and describe the common factors that predict sociodemographic differences, such as discrimination, poverty, and low social status. The second approach assumes that each group is embedded in a complex and unique set of historical, social, and cultural factors and that even when common factors are identified, each group may respond differently to them.

Common factors that affect women and minorities are discrimination and poverty. A series of studies by Landrine, Klonoff, and colleagues have found that sexist discrimination accounted for more variance in depressive and somatic symptoms than life event or daily hassles (Landrine, Klonoff, Gibbs, Manning & Lund, 1995), and that recent and lifetime sex discrimination accounted for the difference between women and men in depressive, anxious, and somatic symptoms (Klonoff, Landrine, & Campbell, 2000). Moradi and DeBlaere (2010) provide an extensive review of the research literature on women's experiences of sex

discrimination and its links to psychological and physical health. For African Americans, experiences of racial discrimination are related to low self-esteem, depression, anxiety, and stress-related somatic symptoms (Landrine & Klonoff, 1996; Klonoff, Landrine, & Ullman, 1999). Recently, the concept of *race-based trauma* has been discussed as an additional diagnostic category (Carter, 2007). Perceived racial discrimination predicted psychological distress in Hispanic women (Salgado de Snyder, 1987). In a study of urban adolescents in mental health treatment, perceived racism was associated with exposure to risks, such as violence, sexual abuse and assault, drug use, and to worry about self and others (Surko, Ciro, Blackwood, Nembhard, & Peake, 2005). Recent research on racial microaggressions, "brief, commonplace, and daily verbal, behavioral, and environmental slights and indignities directed toward Black Americans, often automatically and unintentionally" (Sue, Capodilupo, & Holder, 2008, p. 329) is exploring how these subtle but frequent incidents contribute to experiences or powerlessness and invisibility, and may also be a barrier to effective psychotherapy when these occur in the psychotherapeutic relationship (Sue et al., 2007). Racial minorities and women are also more likely than nonminorities and men to live in poverty or have fewer economic resources. Poverty and low income are well-established correlates of depression (Belle & Doucet, 2003).

Other research, however, suggests that belief systems, values, coping strategies, and other factors will vary by gender, race, and culture (Brown, Abe-Kim, & Barrio, 2003). Corral and Landrine (2010) warn us that "researchers rarely measure and control the social contextual correlates of membership in ethnic (and other status) groups" and further that "the plethora of potentially relevant, social contextual correlates of ethnic (and other status) group membership cannot be known a priori" (p. 86). In other words, while we focus on race or socioeconomic status as the risk factor for a disorder, it may, in fact, simply be living in a neighborhood where safety is a day-to-day concern. In this example, if all neighborhoods were equally secure, the effects of race and socioeconomic status would go away. Sue and Chu (2003) suggest that "rather than developing broad theories to explain the mental health of ethnic minority groups, it may be wiser to begin an inductive process in which separate ethnic groups are studied and examined before construction of a more general theory" (p. 461).

In this era of evidence-based practice, an equally important debate concerns the universality versus specificity of treatment effectiveness. Miranda, Nakamura, and Bernal (2003) argue that although evidence-based treatments have generally been developed and tested on White, middle-class samples, they are likely to be effective for minorities as well. They acknowledge that these therapies should continue to be studied to determine what cultural, social, or environmental conditions affect the quality or acceptability of these treatments.

Sue and Zane (2006) disagree with this argument. They criticize the lack of attention paid to ethnic and cultural minorities in the research on evidence-based interventions and call for not only more research that is inclusive but also research aimed at explaining the effects of cultural variables. Muñoz and Mendelson (2005) describe the development and evaluation of prevention and treatment manuals created for low-income minority populations. They used five principles in developing these manuals: (1) Including members of the target group in developing the manual; (2) incorporating relevant cultural values; (3) using religion and spirituality when appropriate; (4) dealing with the stress of acculturation (for those adapting to a new culture); and (5) acknowledging the reality of racism, prejudice, and discrimination. Empirical evaluations of the effectiveness of these manuals were positive (Muñoz & Mendelson, 2005).

Cultural Competence No review of the research literature can completely capture the complexities of gender, race, and class. Indeed to improve diagnosis and treatment of individual clients we might focus less on the client and more on the therapist. Strategies for developing individual and cultural competence in therapists are critical. The APA's *Guidelines on Multicultural Education, Training, Research, Practice, and Organizational Change for Psychologists* American Psychiatric Association (1992b) present six guidelines for enhancing cultural competence: (1) being aware of one's own attitudes and beliefs that can be detrimental in interacting with others who are different; (2) recognizing the importance of multicultural sensitivity, knowledge, and understanding; (3) employing the constructs of multiculturalism and diversity in educating others; (4) recognizing the importance of conducting culture-centered and ethical research with diverse participants; (5) applying culturally appropriate skill in clinical and applied settings; and (6) supporting culturally informed policy development and practices. Daniel, Roysircar, Abeles, and Boyd (2004) review research on four elements of cultural competence: self-awareness of attitudes, biases, and assumptions; knowledge about diversity; understanding of gender's interaction with other statuses; and the reality of multiple identities for all individuals. They discuss how these can be incorporated into graduate education and training of clinical psychologists.

Conclusions

This chapter has reviewed the research literature on gender, race, and class as they affect diagnosis and treatment of psychological disorders. Evidence shows that certain diagnoses are more likely to be given to some clients (schizophrenia in the case of African Americans; histrionic and, perhaps, BPD in the case of women; ASPD in the case of men). Research on access to mental health care

has produced complex findings on the effects of gender, race, and class on use of different services for mental health treatment. Research on the impact of gender, race, and class on therapy outcomes has shown that these statuses matter and that cultural adaptations to mental health interventions also improve outcomes. Research on quality and type of treatment and on pharmacotherapy suggests inequities in treatment and a need for better information about the connections between individual characteristics (including gender and race) and effectiveness of and reactions to psychotropic medications. In an era of establishing and promoting empirically supported treatments, we need to be mindful of the population being treated and we need to ask: Do therapies work equally well with different groups and can psychological interventions be improved by culturally based adaptations? Finally, we need to consider how best to seek greater understanding of the role of gender, race, and class on psychopathology and psychotherapy and we need to continue to focus on developing cultural competence in clinicians.

This chapter makes it abundantly clear that there are very few studies that actually meet the challenge of considering the intersection of gender, race, and class. Even where all of these sociodemographic variables are assessed, they are often analyzed as individual factors or possibly two-way interactions and the possibilities of higher-order interactions are not pursued. Research that takes context into account, such as Chow et al.'s (2003) examination of mental health service use by diverse populations in low and high poverty communities and Kuno and Rothbard's (2005) study of quality of care at community mental health centers in neighborhoods that vary by income and racial composition, demonstrate the value of doing more than collecting individual reports on sociodemographic variables. Snowden and Yamada (2005) acknowledge that we do not yet understand disparities in mental health care. They call for "more studies of treatment-seeking pathways that favor nonspecialty sources of assistance, improve trust and treatment receptiveness, eliminate stigma, and accommodate culturally distinctive beliefs about mental illness and mental health and styles of expressing mental health-related suffering" (pp. 160–1). A better understanding of interactions among gender, race, and class may require a more qualitative approach (Mullings & Schulz, 2006), partly because reporting of quantitative differences among groups may serve to further marginalize minority groups without helping us to understand them (López, 2003).

Mullings and Schulz (2006) point out that "it is often difficult to pinpoint how the interaction, articulation, and simultaneity of race, class, and gender affect women and men in their daily lives, and the ways in which these forms of inequality interact in specific situation to condition health" (p. 6). They suggest that gender, race, and class should be viewed as social relationships rather than as characteristics of individuals. Building on this theme, Martin criticizes the supplement to the surgeon general's report and its assumption that

> there is something concrete and real in the world (and increasingly in the brain) that corresponds one to one with the major psychiatric diagnosis. Identifying this real thing . . . is the proper first step in getting [patients] the proper treatment. Identifying observer bias and removing it will clear the way to more frequently correct diagnosis. But there are . . . troubling aspects of this view. First, what if the categories into which psychiatry divides disorders themselves already have cultural assumptions embedded in them, not fixed for all time . . . ? Second, what if our only route to the real is always through linguistic categories that are necessarily saturated with culturally constituted sets of meanings?
>
> (Martin, 2006, p. 85)

These closing remarks remind us that gender, race, and class are more than sociodemographic characteristics. They constitute essential aspects of the lived experience of individuals who may come to us as clinicians in need of understanding and assistance. We must determine how to treat these individuals in culturally sensitive and appropriate ways.

Notes

1. Collaborative Psychiatric Epidemiology Surveys 2001–2003 (United States): http://www.icpsr.umich.edu/icpsrweb/CPES/biblio/studies/20240/resources (accessed April 24, 2015).
2. Collaborative Psychiatric Epidemiology Surveys: http://www.icpsr.umich.edu/icpsrweb/CPES (accessed April 24, 2015).

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6

Classification and Diagnosis

Historical Development and Contemporary Issues

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Dysfunctional, impairing and/or maladaptive thinking, feeling, behaving, and relating are of substantial concern to many different professions, the members of which will hold an equally diverse array of beliefs regarding etiology, pathology, and intervention. It is imperative that these persons be able to communicate meaningfully with one another. The primary purpose of an official diagnostic nomenclature is to provide this common language of communication (Kendell, 1975; Sartorius et al., 1993).

Official diagnostic nomenclatures, however, can be exceedingly powerful, impacting significantly many important social, forensic, clinical, and other professional decisions (Frances, 2013; Schwartz & Wiggins, 2002). People think in terms of their language and the predominant language of psychopathology is the fifth edition of the American Psychiatric Association's (2013) *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) and the 10th edition of the World Health Organization's (WHO) *International Classification of Diseases* (ICD-10; WHO, 1992; ICD-11 is under construction).

The DSM and ICD are largely congruent nomenclatures. The ICD is published by the WHO. The United States, as a member of the WHO, is required to use the coding system of the ICD. The code numbers used in all clinics and hospitals within the United States (and included within DSM-5) are the ICD code numbers. For example, 307.51 is the ICD-9 code number for bulimia nervosa; F50.2 is the ICD-10 code number. The United States is scheduled to shift from the ICD-9 codes to the ICD-10 codes on October 1, 2014 (DSM-5 includes both the ICD-9 and ICD-10 code numbers).

DSM-5 is America's version of ICD-10. Each member country of the WHO can modify the diagnostic criteria for a respective disorder, as long as the modification does not result in an entirely different disorder. Each country can also decline to include a particular disorder within its version of the ICD. Finally, each country can also add a disorder to its own version of the ICD that is not included in the ICD, as long as that country does not use a code number that is already in use within the ICD. The primary distinction between DSM-5 and ICD-10 is that the latter has two versions, one for clinicians and the other for researchers (WHO, 1992). The research version includes specific and explicit criterion sets, albeit most international researchers have been using DSM-IV-TR (American Psychiatric Association, 2000) rather than ICD-10. The clinician version of the ICD uses narrative paragraph descriptions because it is considered that clinicians find the criterion sets to be too complex and cumbersome. This chapter gives an overview of the DSM-5 diagnostic nomenclature, beginning with historical background, followed by a discussion of the major issues facing the current and future revisions.

Historical Background

The impetus for the development of an official diagnostic nomenclature was the crippling confusion generated by its absence (Widiger & Mullins-Sweatt, 2008). "For a long time confusion reigned. Every self-respecting alienist [the 19th century term for a psychiatrist], and certainly every professor, had his own classification" (Kendell, 1975, p. 87). The production of a new system for classifying psychopathology became a rite of passage in the 19th century for the young, aspiring professor:

> To produce a well-ordered classification almost seems to have become the unspoken ambition of every psychiatrist of industry and promise, as it is the ambition of a good tenor to strike a high C. This classificatory ambition was

> so conspicuous that the composer Berlioz was prompted to remark that after their studies have been completed a rhetorician writes a tragedy and a psychiatrist a classification. (Zilboorg, 1941, p. 450)

In 1908, the American Bureau of the Census asked the American Medico-Psychological Association (which subsequently altered its title in 1921 to the American Psychiatric Association) to develop a standard nosology to facilitate the obtainment of national statistics:

> The present condition with respect to the classification of mental diseases is chaotic. Some states use no well-defined classification. In others the classifications used are similar in many respects but differ enough to prevent accurate comparisons. Some states have adopted a uniform system, while others leave the matter entirely to the individual hospitals. This condition of affairs discredits the science. (Salmon, Copp, May, Abot, & Cotton, 1917, pp. 255–6)

The American Medico-Psychological Association, in collaboration with the National Committee for Mental Hygiene, issued a nosology in 1918, titled *Statistical Manual for the Use of Institutions for the Insane* (Menninger, 1963). This nomenclature, however, failed to gain wide acceptance. It included only 22 diagnoses that were confined largely to psychoses with a presumably neurochemical pathology. "In the late twenties, each large teaching center employed a system of its own origination, no one of which met more than the immediate needs of the local institution" (American Psychiatric Association, 1952, p. v). A conference was held at the New York Academy of Medicine in 1928 to develop a more authoritative and uniformly accepted manual. The resulting nomenclature was modeled after the *Statistical Manual* but it was distributed to hospitals within the American Medical Association's *Standard Classified Nomenclature of Disease*. Many hospitals used this system but it eventually proved to be inadequate when, during World War II, the attention of the profession expanded well beyond psychotic disorders.

ICD-6 and DSM-I The Navy, Army, and Veterans Administration developed their own, largely independent, nomenclatures during World War II due in large part to the inadequacies of the *Standard Classified*. "Military psychiatrists, induction station psychiatrists, and Veterans Administration psychiatrists, found themselves operating within the limits of a nomenclature specifically not designed for 90% of the cases handled" (American Psychiatric Association, 1952, p. vi). World War II was also instrumental in convincing the profession that a common language of psychopathology was necessary. Effective communication on the battlefield regarding the many psychological casualties of the war was sorely handicapped by the absence of a uniform nomenclature. The WHO therefore, accepted the authority in 1948 to produce the sixth edition of the *International Statistical Classification of Diseases, Injuries, and Causes of Death* (now the ICD) and to include a section devoted to mental disorders (Kendell, 1975). The United States Public Health Service commissioned a committee, chaired by George Raines (with representations from a variety of professions and public health agencies beyond simply the American Psychiatric Association) to develop a variant of the mental disorders section of ICD-6 for use within the United States. The United States, as a member of the WHO, was obliged to use ICD-6, but, as noted earlier, adjustments could be made to maximize its acceptance and utility for local usage. Responsibility for publishing and distributing this nosology was given to the American Psychiatric Association (1952) under the title *Diagnostic and Statistical Manual: Mental Disorders* (hereafter referred to as DSM-I).

DSM-I was generally successful in obtaining acceptance by the major medical centers within the United States, due in part to its expanded coverage, including somatoform disorders, stress reactions, and personality disorders. However, the New York State Department of Mental Hygiene, which had been influential in the development of the Standard Nomenclature, continued for some time to use its own classification. DSM-I also included narrative descriptions of each disorder to facilitate understanding and more consistent applications. Nevertheless, fundamental criticisms regarding the reliability and validity of psychiatric diagnoses were also being raised (e.g., Zigler & Phillips, 1961). For example, a widely cited reliability study by Ward, Beck, Mendelson, Mock, and Erbaugh (1962) concluded that most of the poor agreement among psychiatrists' diagnoses was due largely to inadequacies of DSM-I.

ICD-8 and DSM-II ICD-6 had been revised to ICD-7 in 1955 but there were no revisions to the mental disorders. In 1965, the American Psychiatric Association appointed a committee, chaired by Ernest M. Gruenberg, to revise DSM-I to be compatible with ICD-8 and yet also be suitable for use within the United States. The final version was approved in 1967, with publication in 1968 (American Psychiatric Association, 1968).

The diagnosis of mental disorders, however, was again receiving substantial criticism (e.g., Rosenhan, 1973; Szasz, 1961). A fundamental problem continued to be the absence of empirical support for the reliability, let alone the validity, of its diagnoses (e.g., Blashfield & Draguns, 1976). Researchers, therefore, began developing their own criterion sets (Blashfield, 1984). The most influential of these efforts was produced by a group of neurobiologically oriented psychiatrists at Washington University in St. Louis. Their criterion sets generated so much interest that they were published separately in what has become one of the most widely cited papers in psychiatry (Feighner et al., 1972). Research has since indicated that mental disorders can be diagnosed reliably and do provide valid information regarding etiology, pathology, course, and treatment (Kendler, Munoz, & Murphy, 2010).

ICD-9 and DSM-III By the time that Feighner and colleagues' (1972) paper was published, work was nearing completion on the ninth edition of the ICD. The authors of ICD-9 had decided to include a glossary that would provide more precise descriptions of each disorder, but it was apparent that ICD-9 would not include the more specific and explicit criterion sets used in research (Kendell, 1975). In 1974, the American Psychiatric Association appointed a task force, chaired by Robert Spitzer, to revise DSM-II in a manner that would be compatible with ICD-9, but which would also incorporate many of the current innovations in diagnosis. DSM-III was published in 1980 and was remarkably innovative, including: (1) Specific and explicit criterion sets for all but one of the disorders (schizoaffective); (2) a substantially expanded text discussion of each disorder to facilitate diagnosis (e.g., age at onset, course, complications, sex ratio, and familial pattern), and (3) removal of terms (e.g., "neurosis") that appeared to favor a particular theoretical model for the disorder's etiology or pathology (American Psychiatric Association, 1980; Spitzer, Williams, & Skodol, 1980).

DSM-III-R An ironic advantage of the specificity and explicitness of the DSM-III criterion sets was that a number of mistakes quickly became apparent (e.g., panic disorder in DSM-III could not be diagnosed in the presence of a major depression). "Criteria were not entirely clear, were inconsistent across categories, or were even contradictory" (American Psychiatric Association, 1987, p. xvii). Many of these errors and problems were due to the fact that there was insufficient research to guide the authors of the criterion sets. The American Psychiatric Association authorized the development of a revision to DSM-III to make corrections and refinements. Fundamental revisions were to be tabled until work began on ICD-10.

However, it might have been unrealistic to expect the authors of DSM-III-R to confine their efforts to refinement and clarification, given the impact, success, and importance of DSM-III. More persons were involved in making corrections to DSM-III than were used in its original construction and, not surprisingly, there were many proposals for major revisions and even new diagnoses. Four of the diagnoses approved by the task force for inclusion (i.e., sadistic personality disorder, self-defeating personality disorder, premenstrual dysphoric disorder, and paraphiliac rapism) generated so much controversy that a special ad hoc committee was appointed by the Board of Trustees of the American Psychiatric Association to reconsider their inclusion. A concern common to all four was that their inclusion might result in harm to women. For example, paraphiliac rapism might be used to mitigate criminal responsibility for a rapist, and self-defeating personality disorder might be used to blame female victims for having been abused. Another concern was the lack of sufficient empirical support to address or offset these concerns. A compromise was eventually reached in which the two personality disorders and premenstrual dysphoric disorder were included in an appendix (American Psychiatric Association, 1987; Endicott, 2000; Widiger, 1995); paraphiliac rapism was deleted entirely.

ICD-10 and DSM-IV By the time the work was completed on DSM-III-R, work had already begun on ICD-10. The decision of the authors of DSM-III to develop an alternative to ICD-9 was instrumental in developing a highly innovative manual (Kendell, 1991; Spitzer et al., 1980). However, its innovations were also at the cost of decreasing compatibility with the ICD-9 nomenclature that was used throughout the rest of the world, which was problematic to the stated purpose of providing a common language of communication. In 1988, the American Psychiatric Association appointed a DSM-IV task force, chaired by Allen Frances (Frances, Widiger, & Pincus, 1989). Mandates for DSM-IV included better coordination with ICD-10 and improved documentation of empirical support.

The DSM-IV committee aspired to use a more conservative threshold for the inclusion of new diagnoses and to have decisions be guided more explicitly by the scientific literature (American Psychiatric Association, 1994). Proposals for additions, deletions, or revisions were guided by 175 literature reviews that used a required format that maximized the potential for critical review, containing (for example) a method section that documented explicitly the criteria for including and excluding studies and the process by which the literature had been reviewed (Frances et al., 1989). It was not unusual in the development of DSM-IV to find that proponents of a proposed revision attempting to limit their review largely to the studies that supported their proposal, neglecting to acknowledge issues and findings inconsistent with their position (Frances & Widiger, 2012; Widiger & Trull, 1993). The reviews were published within a three-volume DSM-IV Sourcebook (e.g., Widiger et al., 1994). Testable questions that could be addressed with existing data sets were also explored in 36 studies, which emphasized the aggregation of multiple data sets from independent researchers, and 12 field trials were conducted to provide reliability and validity data on proposed revisions. The results of the 36 studies and 12 field trials were published in the fourth volume of the *DSM-IV Sourcebook* (Widiger et al., 1998). Critical reviews of these 223 projects were obtained by sending initial drafts to advisors or consultants to a respective work group, by presenting drafts at relevant conferences, and by submitting drafts to peer-reviewed journals (Widiger, Frances, Pincus, Davis, & First, 1991).

DSM-IV-TR One of the innovations of DSM-III was the inclusion of a relatively detailed text discussion of each disorder, including information on age of onset, gender, course, and familial pattern (Spitzer et al., 1980). This text was expanded in DSM-IV to include cultural and ethnic group variation, variation across age, and laboratory and

physical exam findings (Frances, First, & Pincus, 1995). Largely excluded from the text was information concerning etiology, pathology, and treatment, as this material was considered to be too theoretically specific and more suitable for academic texts. Nevertheless, it had also become apparent that DSM-IV was being used as a textbook, and the material on age, course, prevalence, and family history was quickly becoming outdated as new information was being gathered.

Therefore, in 1997, the American Psychiatric Association appointed a DSM-IV text revision work group, chaired by Michael First (Editor of the text and criterion sets for DSM-IV) and Harold Pincus (Vice Chair for DSM-IV) to update the text material. No substantive changes in the criterion sets were to be considered, nor were any new additions, subtypes, deletions, or other changes in the status of any diagnoses to be implemented. In addition, each of the proposed revisions to the text had to be supported by a systematic literature review that was critiqued by external advisors. The DSM-IV text revision (DSM-IV-TR) was published in 2000 (American Psychiatric Association, 2000).

The outcome, however, was not entirely consistent with the original intentions. Revisions to the criterion sets for tic disorders and the paraphilias that involved a nonconsenting victim were implemented (First & Pincus, 2002), albeit no acknowledgments of these revisions were provided within the manual. In addition, no documentation of the scientific support for the text revisions was provided, owing to the inconsistency in the quality of the effort. Rather than have inconsistent and/or inadequate documentation, it was decided to have none at all.

DSM-5 and Continuing Issues for Future Editions

DSM-5, chaired by David Kupfer and Darrel Regier, was published in 2013. The development of DSM-5 generated a substantial body of controversy (Decker, 2010; Frances, 2013; Greenburg, 2013). The difficult issues that beset the development of DSM-5 are discussed herein, as they will likely continue to bedevil the authors of the next edition. Five issues are: (1) the empirical support for proposed revisions; (2) the definition of mental disorder; (3) the impact of culture and values; (4) shifting to a neurobiological model; and (5) shifting to a dimensional model.

Empirical Support Frances and colleagues (1989) had suggested that "the major innovation of DSM-IV will not be in its having surprising new content but rather will reside in the systematic and explicit method by which DSM-IV will be constructed and documented" (p. 375). Frances (2013), the Chair of DSM-IV, feels that the authors of DSM-5 flipped this priority on its head, with emphasis being give to new content and inadequate attention to first conducting systematic, thorough, and balanced reviews. Concerns with respect to the process with which DSM-5 was being constructed were perhaps first raised by Robert Spitzer, Chair of DSM-III and DSM-III-R, after having been denied access to the minutes of DSM-5 work group meetings (Decker, 2010). Frances and Spitzer eventually submitted a joint letter to the American Psychiatric Association Board of Trustees on July 7, 2009, expressing a variety of concerns with respect to the process with which DSM-5 was being constructed (Decker, 2010; Greenburg, 2013).

The Chair and Vice Chair of DSM-5 stated that the development of DSM-5 followed the procedure used for DSM-IV, including literature reviews, data reanalyses, and field trials (Regier, Narrow, Kuhl, & Kupfer, 2010). However, the letter by Frances and Spitzer was initiated by the fact that the field trial for DSM-5 was about to begin before the proposals had received any critical review or even been revealed. Frances followed this joint letter with additional letters of his own and a series of articles which eventually led to the decision to postpone the field trial until after all of the proposals had been posted on a website, thereby allowing for at least some external review and public awareness (Decker, 2010).

Kendler, Kupfer, Narrow, Phillips, and Fawcett (2009) developed guidelines for DSM-5 work group members. These guidelines indicated that any change to the diagnostic manual should be accompanied by "a discussion of possible unintended negative effects of this proposed change, if it is made, and a consideration of arguments against making this change should also be included" (p. 2). Kendler and colleagues further stated that "the larger and more significant the change, the stronger should be the required level of support" (p. 2). This was a very commendable and demanding set of guidelines, but it did not appear that work group members were actually required to adhere to them.

The initial literature reviews that provided the empirical support for DSM-5 proposals were first posted on the DSM-5 website in February, 2010. Revisions to these literature reviews continued to be posted through 2012. Many of the DSM-5 literature reviews did appear to meet the spirit of the Kendler and colleagues (2009) guidelines, such as the reviews for hypersexual disorder (Kafka, 2010), dissociative disorders (Spiegel et al., 2013), and a callous-unemotional specifier for conduct disorder (Frick, Ray, Thornton, & Than, 2014), but others did not. For example, one major revision to the manual was the creation of a new class of behavioral addiction disorders, subsuming substance use disorders and pathological gambling, and allowing for the recognition of additional disorders, such as Internet and shopping addiction. The posted literature review that provided the rationale and empirical support for this major revision consisted of just two sentences: "Pathological (disordered) gambling has commonalities in clinical expression, etiology, comorbidity, physiology and treatment with Substance Use Disorders. These commonalities are addressed in the following selected papers from a relatively large literature" (American Psychiatric

Association, 2010). There was no discussion of the potential costs or risks of developing a new diagnosis of behavioral addiction. It is readily conceivable that a thorough and compelling literature review could have been generated that would have supported the proposal (e.g., Shaffer, LaPlante, & Nelson, 2012), but none was posted on the DSM-5 website.

The literature review for a new diagnosis, mood dysregulation disorder (previously temper dysregulation disorder of childhood) was thorough in its coverage, but acknowledged that the empirical support for the proposal was, at best, sorely limited. The impetus for this proposal was the increasing number of children with temper tantrums who were being diagnosed with bipolar mood disorder and provided with medications in the absence of adequate scientific support for the validity of this diagnosis in children (Fawcett, 2010). It was felt that the overdiagnosis of bipolar mood disorder in children might be addressed through the development of specific and explicit diagnostic criteria that would be sufficiently restrictive. Of course, the existence of the diagnosis within DSM-5 will likely have the opposite effect of increasing the medication of children with temper tantrums by providing the diagnosis with substantial credibility and perceived validity that it does not really deserve (Frances & Widiger, 2012; Hyman, 2010).

In addition, the Childhood and Adolescent Disorders Work Group (2010) acknowledged that the research "has been done predominately by one research group in a select research setting, and many questions remain unanswered" (p. 4). This point was reiterated within a joint statement by the Mood Disorders and Childhood and Adolescent Disorders Work Groups (2010) that "both work groups are concerned by the fact that the work on severe mood dysregulation [in children] has been done predominately by one research group in a select research setting" (p. 6). Kendler et al. (2009) had stated explicitly that a new diagnosis "should rarely if ever be based solely on reports from a single researcher or research team" (Kendler et al., 2009, p. 5), yet this proposal was approved.

The authors of the proposal suggested that its inclusion in DSM-5 would help to generate the research needed for its validation.

> Indeed, it can be argued that one of the major 'take-home' messages from the controversy about the diagnosis of pediatric bipolar disorder is the fact that the research needs of a large population of children with severe irritability are not being met, particularly with respect to clinical trials.
>
> (Mood Disorders and Childhood and Adolescent Disorders Work Groups, 2010, p. 8)

As expressed by the Childhood and Adolescent Disorders Work Group (2010) the inclusion of this new diagnosis will help to "'jump-start' research on severe irritability in youth" (p. 9). In other words, not only was there an acknowledgment of inadequate empirical support, the proposal was approved for inclusion in order to generate the research that would (hopefully) support its validity. This would appear to be quite inconsistent with the guidelines of Kendler et al. (2009), yet, again, the proposal was approved.

Blashfield and Reynolds (2012) reviewed the reference list of the final review posted by the DSM-5 Personality Disorders Work Group and concluded that it was slanted heavily toward the publications of work group members with an inadequate representation of significant contributions by other investigators. And, it is perhaps worth noting that the review studied by Blashfield and Reynolds was the final version that had the potential advantage of having been informed by the many published critiques of the earlier versions that generated substantial controversy (Widiger, 2013). The initial reviews posted by Skodol (2010) and Clark and Krueger (2010) were considerably more limited than the final version. All of the proposals for the personality disorder section of DSM-5 were ultimately rejected by a scientific oversight committee that was developed during the course of DSM-5, owing to concerns regarding the process with which it was being constructed (Decker, 2010; Frances, 2013; Greenburg, 2013).

One of the more unusual revisions for DSM-5 involved the diagnosis of cyclothymia. The diagnosis of cyclothymia in DSM-III and DSM-III-R required that the symptoms of hypomania meet the diagnostic threshold for a hypomanic episode. This was loosened somewhat in DSM-IV by requiring only that there be "numerous periods with hypomanic symptoms" (p. 365). Persons above threshold for a hypomanic episode would, of course, still be given the diagnosis of cyclothymia. However, in DSM-5, it is stated that the "hypomanic symptoms . . . do not meet criteria for a hypomanic episode" (p. 139). This is a very clear and significant change, and it is one that is difficult to understand, but it is even more difficult to find the rationale, let alone the empirical support.

It was stated in the first posting on the DSM-5 website that there would be no revisions for the diagnosis of cyclothymia. No reference was made to proposed change in the criteria for cyclothymia in the published literature review for the DSM-5 mood disorders (Fawcett, 2010). In the final posting on the DSM-5 website, there was no summary of the rationale or empirical support for the revision. In the section of DSM-5 for "highlights of changes from DSM-IV to DSM-5" (pp. 809–16), there is no mention of any change for cyclothymia. Requiring that the hypomanic symptoms be below the threshold for a hypomanic episode for the diagnosis of cyclothymia is a rather significant revision to the criterion set, and one whose rationale is even difficult to understand. This revision is comparable to changing the criteria for a bipolar mood disorder to require that the manic symptoms be below the threshold of a manic episode. Such a revision would obviously be a major change to bipolar mood disorder and, again, difficult to understand. In sum, no literature review was ever provided to justify this odd change to the criterion set for

cyclothymia, nor even an explanation for its rationale. This would again appear to be quite inconsistent with the guidelines of Kendler et al. (2009).

The field trials for DSM-5 were also very different from the DSM-IV field trials. The purpose of the field trials for DSM-IV was to test empirically the validity of proposed revisions, compare alternative proposals, and indicate their potential impact (Widiger et al., 1991). This was not the intention of the DSM-5 field trials, which were confined largely to questions of feasibility, reliability, and face validity (Clarke et al., 2013). "The main interest was to determine the degree to which two clinicians would agree on the same diagnosis" (p. 44). This question is interesting, but it has already been well established that psychiatric diagnoses tend to be unreliable in the absence of systematic clinical assessments (Garb, 2005; Spitzer et al., 1975). More importantly, it is not clear how answering that question would be relevant to a decision whether or not to implement a particular proposal. Of more importance for a field trial concerned with new diagnostic proposals is addressing questions of validity (Kendler, 1990), comparing alternative proposals with one another, and with the existing nomenclature (Widiger et al., 1991), and addressing the potential costs and concerns specific to each proposal (Frances & Widiger, 2012; Kendler et al., 2009). The DSM-5 field trials largely ignored what probably should have been their primary concerns. In fact, the field trial was unable to obtain any information for some of the proposals (e.g., attenuated psychotic symptoms, bipolar II, hoarding, mild neurocognitive disorder, narcissistic personality disorder, and schizotypal personality disorder) owing to the sites not having available a sufficient number of cases for data analysis (Clarke et al., 2013; Regier et al., 2013). It is unclear why sites were chosen if it was apparent that they would not have enough cases over the course of the DSM-5 field trial data collection (Jones, 2012).

Definition of Mental Disorder The boundaries of the *Diagnostic Manual* have been increasing with each edition and there has long been vocal concern that much of this expansion represents an encroachment into normal problems of living (Folette & Houts, 1996; Frances, 2013; Horwitz & Wakefield, 2007; see also Maddux, Gosselin, & Winstead, Chapter 1 in this volume.) Coverage has again increased with DSM-5, with such new diagnoses as disruptive mood dysregulation disorder, excoriation disorder, hoarding disorder, illness anxiety disorder (a significant expansion of hypochondriasis), premenstrual dysphoric disorder, and binge eating disorder, as well as the expansion of substance use disorder to include behavioral addictions, the addition of a muscle dysmorphia subtype to body dysmorphic disorder, and the removal of the bereavement exclusion criterion from the diagnosis of a major depressive disorder.

How the authors of a diagnostic manual can know when a behavior is the result of some form of psychopathology and should then be included within the diagnostic manual remains unclear. There is no laboratory measure to document objectively the existence of a mental disorder (Kapur, Phillips, & Insel, 2012). The decision to consider a condition or behavior pattern a mental disorder is a matter of opinion, supported, it is hoped, by compelling research.

DSM-III was the first edition of the *Diagnostic Manual* to include an explicit definition of what constitutes a mental disorder, the result of an effort by the authors of DSM-III to develop specific and explicit criteria for deciding whether a behavior pattern (homosexuality in particular) should be classified as a mental disorder (Spitzer & Williams, 1982). The intense controversy over homosexuality has largely abated, but the issues raised in this historical debate continue to apply, as the definition of mental disorder included within the diagnostic manual is sufficiently vague that it does not really help determine what is or is not a mental disorder (see also Maddux, Gosselin, & Winstead, Chapter 1 in this volume.) Two illustrative examples of the debatable boundaries between normality and abnormality are provided by the diagnosis of paraphilia and the bereavement exclusion criterion for major depressive disorder.

Paraphilia In order to be diagnosed with pedophilia, DSM-III-R required only that an adult have recurrent intense urges and fantasies involving sexual activity with a prepubescent child over a period of at least six months, and have acted on them or be markedly distressed by them. Every adult who engaged in a sexual activity with a child for longer than six months would meet these diagnostic criteria. The authors of DSM-IV were therefore concerned that DSM-III-R was not providing adequate guidance for determining when persistent deviant sexual behavior is the result of a mental disorder versus when it is a willful criminal act. Presumably, some persons can engage in deviant, aberrant, and even heinous activities, without being compelled to do so by the presence of psychopathology. The authors of DSM-IV, therefore, added the requirement for all the paraphilias that "the behavior, sexual urges, or fantasies cause clinically significant distress or impairment in social, occupational, or other important areas of functioning" (DSM-IV, p. 523).

Spitzer and Wakefield (1999), however, argued that the impairment criteria included in DSM-IV were inadequate. They concurred with a concern raised by the National Law Center for Children and Families that DSM-IV might have contributed to a normalization of pedophilic and other paraphilic behavior by allowing the diagnoses not to be applied if the persons who have engaged in these acts were not themselves distressed by their behavior or did not otherwise experience impairment. Frances et al. (1995) had argued that pedophilic sexual "behaviors are inherently problematic because they involve a nonconsenting person (exhibitionism, voyeurism, frotteurism) or

a child (pedophilia) and may lead to arrest and incarceration" (p. 319). Therefore, any person who engaged in an illegal sexual act (for longer than six months) would be exhibiting a clinically significant social impairment and would therefore meet the DSM-IV threshold for diagnosis. However, one should not use the illegality of an act to help determine when an illegal act is a disorder (Spitzer & Williams, 1982). This undermines the rationale for the inclusion of the impairment criterion to help distinguish immoral or illegal acts from abnormal or disordered acts, and it is inconsistent with the DSM-IV (and DSM-5) definition of a mental disorder that states that neither deviance nor conflicts with the law are sufficient to warrant a diagnosis.

The diagnostic criteria for pedophilia were revised in DSM-IV-TR to return to what was provided in DSM-III-R, in order to try to avoid the misunderstanding that an absence of personal distress or impairment would suggest that the behavior is considered to be normal (First & Pincus, 2002). However, the DSM-IV-TR criteria again allowed simply the presence of the behavior for longer than six months to indicate the presence of the disorder, thereby providing no meaningful distinction between pedophilic behavior that is willful and volitional from pedophilic behavior that is driven by some form of organismic pathology.

The threshold for a paraphilic disorder has been further revised and effectively lowered in DSM-5. DSM-5 continues to allow a person to be diagnosed with a paraphilic disorder even if the behavior is neither harmful nor distressing to the person. One revision, though, is to now allow the diagnosis if the behavior is simply harmful to others. Willful, voluntary behavior that is not driven by an underlying psychopathology constitutes a mental disorder if that behavior is harmful to someone else. This is comparable to stating that all cases of theft constitute the disorder of kleptomania because thefts are harmful to others. In fact, actual harm to others is not even required. Simply "risk of harm" to others is sufficient for the diagnosis (DSM-5, p. 685).

Bereavement The DSM-IV criterion set for major depressive disorder excluded most instances of depressive reactions to the loss of a loved one (i.e., uncomplicated bereavement). It was considered to be normal to be significantly depressed after the loss of a loved one. Depression after the loss of a loved one could be considered a mental disorder however if "the symptoms persist for longer than two months" (p. 327). Allowing two months to grieve before one is diagnosed with a major depressive disorder might be as arbitrary and meaningless as allowing a person to engage in a sexually deviant act only for six months before the behavior is diagnosed as a paraphilia.

Wakefield (2011; Wakefield & First, 2003) has further argued that it was logically inconsistent to exclude the diagnosis of a mental disorder if the loss was a loved one, but then allow the diagnosis of major depression in response to the loss of physical health, job, or employment. DSM-IV allowed clinicians to diagnose a mental disorder if one became depressed in reaction to a terminal illness, but did not allow the diagnosis to be made if the loss was the death of a close relative.

The DSM-5 Mood Disorders Work Group agreed with Wakefield (2011) with regard to the existence of this inconsistency. Depression in response to the loss of a loved one does not appear to be meaningfully different from depression in response to any other loss (Kendler, 2010). However, rather than add exclusion criteria for these other losses, as Wakefield suggested, the work group decided to delete the exclusion criterion for bereavement (Zisook, Shear, & Kendler, 2009).

This decision, albeit controversial, is perhaps quite reasonable. What is currently considered to be a normal depression in response to the loss of a loved one does include pain, suffering, and meaningful impairments to functioning, and is often outside of the ability of the bereaved person to fully control. It is normal and reasonable to respond to the loss of (for instance) a son or daughter (i.e., a psychological trauma) with depression, but many physical disorders and injuries are themselves reasonable and understandable responses to a physical trauma (e.g., impairment secondary to the experience of a car accident). The loss is perhaps best understood as part of the etiology for the disorder, not a reason for which a disorder is not considered to be present. "Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability)" (DSM-5, p. 161) can all be diagnosed now as a mental disorder. However, in the face of substantial public and professional opposition to the implication that grieving would now be considered a mental illness, the American Psychiatric Association (pp. 125–6) decided to just leave the decision up to the clinician, who can choose to consider it to be a mental disorder or choose not to. However, the authors of DSM-5 may have neglected to also revise the definition of mental disorder, as it continues to state that "an expectable or culturally approved response to a common stressor or loss, such as the death of a loved one, is not a mental disorder" (p. 20).

Alternative Definitions of Mental Disorder Wakefield (1992) developed an alternative "harmful dysfunction" definition of mental disorder wherein dysfunction is a failure of an internal mechanism to perform a naturally selected function (e.g., the capacity to experience feelings of guilt in a person with antisocial personality disorder) and harm is a value judgment that the design failure is harmful to the individual (e.g., failure to learn from mistakes results in repeated punishments, arrests, loss of employment, and eventual impoverishment). Wakefield's model has received substantial attention and was considered for inclusion in DSM-5 (Rounsaville et al., 2002).

However, Wakefield's proposal has also received much critical review (e.g., Bergner, 1997; Kirmayer & Young, 1999; Lilienfield & Marino, 1999; Widiger & Sankis, 2000). A fundamental limitation is its reliance on evolutionary theory, thereby limiting its relevance and usefulness to alternative models of etiology and pathology (Bergner, 1997). Wakefield's model might even be inconsistent with some sociobiological models of psychopathology. Cultural evolution may at times outstrip the pace of biological evolution, rendering some designed functions that were originally adaptive within earlier time periods maladaptive in many current environments (Lilienfeld & Marino, 1999; Widiger & Sankis, 2000). For example,

> the existence in humans of a preparedness mechanism for developing a fear of snakes may be a relic not well designed to deal with urban living, which currently contains hostile forces far more dangerous to human survival (e.g., cars, electrical outlets) but for which humans lack evolved mechanisms of fear preparedness.
>
> (Buss, Haselton, Shackelford, Bleske, & Wakefield, 1998, p. 538; see also Maddux, Gosselin, & Winstead, Chapter 1 in this volume)

Missing from Wakefield's (1992) definition of mental disorder, as well as from the definition included in DSM-5, is any reference to dyscontrol. Mental disorders are perhaps best understood as dyscontrolled impairments in psychological functioning (Kirmayer & Young, 1999; Klein, 1999; Widiger & Sankis, 2000). "Involuntary impairment remains the key inference" (Klein, 1999, p. 424). Dyscontrol is one of the fundamental features of mental disorder emphasized in Bergner's (1997) "significant restriction" and Widiger and Sankis' (2000) "dyscontrolled maladaptivity" definitions of mental disorder. Including the concept of dyscontrol within a definition of mental disorder also provides a fundamental distinction between mental and physical disorder, as dyscontrol is not a meaningful consideration for a physical disorder.

Fundamental to the concept of a mental disorder is the presence of impairments secondary to feelings, thoughts, or behaviors, over which a normal (healthy) person has adequate self-control to alter or adjust in order to avoid these impairments, if he or she wishes to do so (Widiger & Sankis, 2000). To the extent that a person willfully, intentionally, freely, or voluntarily engages in harmful sexual acts, drug usage, gambling, or child abuse, the person would not be considered to have a mental disorder. Persons seek professional intervention in large part to obtain the insights, techniques, skills, or other tools (e.g., medications) that would increase their ability to better control their mood, thoughts, or behavior.

Dyscontrol as a component of mental disorder does not imply that a normal person has free will, a concept that is, at best, difficult to scientifically or empirically verify (Bargh & Ferguson, 2000; Howard & Conway, 1986). A person with a mental disorder could be comparable to a computer lacking in the necessary software to combat particular viruses or execute effective programs. Pharmacotherapy alters the neural connections of the central nervous system (the hardware), whereas psychotherapy alters the cognitions (the software) in a manner that increases a person's behavioral repertoire, allowing the person to act and respond more effectively. A computer provided with new software has not been provided with free will, but has been provided with more options to act and respond more effectively.

A definition of mental disorder confined to the presence of dyscontrol and maladaptivity would, however, allow for a considerable expansion of the diagnostic manual. Persons critical of the nomenclature have objected to this expansion over the past 50 years (e.g., Follette & Houts, 1996; Frances, 2013; Kirk, 2005). Alternatively, perhaps the assumption that the diagnostic manual is expanding too rapidly is the more questionable position. It is to be expected that scientific research and increased knowledge have led to the recognition of more instances of psychopathology (Wakefield, 1998, 2001). In addition, perhaps the assumption that only a small minority of the population currently has, or will ever have, a mental disorder (Regier & Narrow, 2002) is also questionable. Very few persons fail to have at least some physical disorders, and all persons suffer from quite a few physical disorders throughout their lifetime. It is unclear why it should be different for mental disorders, as if most persons have been fortunate to have obtained no problematic genetic dispositions or vulnerabilities and have never sustained any psychological injuries or have never experienced significant economic, environmental, or interpersonal stress, pressure, or conflict that would tax or strain psychological functioning.

Optimal psychological functioning, as in the case of optimal physical functioning, might represent an ideal that is achieved by only a small minority of the population. The rejection of a high prevalence of psychopathology may reflect the best of intentions, such as concerns regarding the stigmatization of mental disorder diagnoses (Kirk, 2005) or the potential impact on funding for treatment (Regier & Narrow, 2002), but these social and political concerns could also hinder a more dispassionate and accurate recognition of the true rate of a broad range of psychopathology within the population (Widiger & Sankis, 2000; see also Maddux, Gosselin, & Winstead, Chapter 1 in this volume).

Culture and Values It was the intention of the authors of ICD-10 to provide a universal diagnostic system, but diagnostic criteria and constructs can have quite different implications and meanings across different cultures. DSM-5 addresses cultural issues in three ways. First, the text of DSM-5 provides a discussion of how each disorder is known to vary in its presentation across different cultures. Second, an appendix provides a culturally informed diagnostic formulation that considers the cultural identity

of the individual and the culture-specific explanations of the person's presenting complaints (Lim, 2006). DSM-IV-TR had a similar appendix, but DSM-5 goes further through the inclusion of an outline for a cross-cultural clinical interview that helps glean the relevant information. DSM-IV-TR had also included an appendix of "culture-bound" syndromes that briefly described a number of disorders that were thought to be specific to a particular culture. These were replaced in DSM-5 with a more general discussion of how and why certain disorders might be relatively unique to a particular culture.

There is both a strong and a weak cross-cultural critique of current scientific understanding of psychopathology. The weak critique does not question the validity of a concept of mental disorder, but does argue that social and cultural processes affect and potentially bias the "science of psychopathology and diagnosis: a) by determining the selection of persons and behaviors as suitable material for analysis; b) by emphasizing what aspects of this material will be handled as relevant from a (clinical) standpoint; c) by shaping the language of diagnosis, including that of descriptive psychopathology; d) by masking the symptoms of any putative 'universal' disorder; e) by biasing the observer and would-be diagnostician; and f) by determining the goals and endpoints of treatment" (Fabrega, 1994, p. 262). These concerns are not weak in the sense that they are trivial or inconsequential, but they do not dispute the fundamental validity of a concept of mental disorder or the science of psychopathology. The strong critique, in contrast, is that the construct of mental disorder is itself a culture-bound belief that reflects the local biases of western society, and that the science of psychopathology is valid only in the sense that it is an accepted belief system of a particular culture (see also, Maddux, Gosselin, & Winstead, Chapter 1 in this volume, and López & Guarnaccia, Chapter 4 in this volume).

The concept of mental disorder does include a value judgment that there should be necessary, adequate, or optimal psychological functioning (Wakefield, 1992). However, this value judgment is also a fundamental component of the construct of physical disorder (Widiger, 2002). In a world in which there were no impairments or threats to physical functioning, the construct of a physical disorder would have no meaning except as an interesting thought experiment. Meaningful and valid scientific research on the etiology, pathology, and treatment of physical disorders occurs because in the world as it currently exists there are impairments and threats to physical functioning. It is provocative and intriguing to conceive of a world in which physical health and survival would or should not be valued or preferred over illness, suffering, and death, but this form of existence is unlikely to emerge anytime in the near future. Placing a value on adequate or optimal physical functioning might be a natural result of evolution within a world in which there are threats to functioning and survival.

Likewise, in the world as it currently exists, there are impairments and threats to adequate psychological functioning. It is again provocative and intriguing to conceive of a society (or world) in which psychological health would or should not be valued or preferred, but this form of existence is also unlikely to emerge anytime in the near future. Placing a value on adequate, necessary, or optimal psychological functioning might be inherent to and a natural result of existing in our world. Any particular definition of what would constitute adequate, necessary, or optimal psychological functioning would likely be biased to some extent by local cultural values, but this is perhaps best understood as only the failing of one particular conceptualization of mental disorder (i.e., a weak rather than a strong critique). The value judgment that is inherent to the concept of a mental disorder is not reflective of local, cultural values; it is perhaps an inherent and necessary result of current existence. Valuing adequate, necessary, or optimal psychological functioning is likely a logical and natural result of existing in a world in which there are threats to psychological functioning, just as placing a value on adequate, necessary, or optimal physical functioning would be a logical and natural result of existing in a world in which there are threats to physical functioning (Widiger, 2002).

Different societies, cultures, and even persons within a particular culture will disagree as to what constitutes optimal or pathological biological and psychological functioning (López & Guarnaccia, Chapter 4 in this volume; Sadler, 2005). An important and difficult issue is how best to understand the differences between cultures with respect to what constitutes dysfunction and pathology. For example, simply because diagnostic criterion sets are applied reliably across different cultures, this does not necessarily indicate that the constructs themselves are valid or meaningful within these cultures. A reliably diagnosed criterion set can be developed for an entirely illusory diagnostic construct.

On the other hand, it is unclear why it should be necessary for the establishment of a disorder's construct validity to obtain cross-cultural (i.e., universal) acceptance. A universally accepted diagnostic system will have an international social utility and consensus validity (Kessler, 1999), but it is also apparent that belief systems vary in their veridicality. Recognition of and appreciation for alternative belief systems is important for adequate functioning within an international community, but respect for alternative belief systems does not necessarily imply that all belief systems are equally valid (Widiger, 2002). Simply because a behavior pattern is valued, accepted, encouraged, or even statistically normative within a particular culture, this does not necessarily mean that it is conducive to healthy psychological functioning. In sum, it is important for research on cross-cultural variation to go beyond simply identifying differences in behaviors, belief systems, and values across different cultures. This research also needs to address the fundamental question of whether differences in beliefs actually question the validity of any universal conceptualization of psychopathology

or suggest instead simply different perspectives on a common, universal issue (see also Maddux Gosselin, & Winstead, Chapter 1 in this volume, and López & Guarnaccia, Chapter 4 in this volume).

Shifting to a Neurobiological Model The authors of DSM-III removed terms (e.g., neurosis) that appeared to refer explicitly to psychodynamic constructs, in order to have the manual be atheoretical, or at least be reasonably neutral with respect to alternative models of psychopathology (Spitzer et al., 1980). However, it appears that all theoretical perspectives have found the atheoretical language to be less than optimal for their own particular perspective. Interpersonal and systems theoretical perspectives, which consider dysfunctional behavior to be due to a pathology of a wider social system, rather than simply within the individual, consider the organismic diagnoses of DSM-5 to be fundamentally antithetical (Reiss & Emde, 2003). Psychodynamically oriented clinicians bemoan the fact that the succeeding editions of the manual have become increasingly objective, descriptive, and atheoretical, lacking an explicit reference to the inferred dynamic mechanisms of psychoanalytic theory and practice. Therefore, they have developed their own manual, the *Psychodynamic Diagnostic Manual* (PDM Task Force, 2006). Behaviorists argue that the organismic perspective of DSM-5 is inconsistent with the situational context of dysfunctional behavior (Folette & Houts, 1996). Even neurobiologically oriented psychiatry is unhappy. "Although there is a large body of research that indicates that a neurobiological basis for most mental disorders, the DSM definitions are virtually devoid of biology" (Charney et al., 2002, pp. 31–2).

DSM-I favored a psychodynamic perspective (Spitzer et al., 1980). In striking contrast, the American Psychiatric Association and the National Institute of Mental Health (NIMH) are now shifting explicitly toward a neurobiological orientation. For example, a reading of the table of contents of any issue of the two leading journals of psychiatry (the *American Journal of Psychiatry* and the *Archives of General Psychiatry*) will evidence a strong neurobiological orientation. DSM-IV included a new section of the text devoted to laboratory and physical exam findings (Frances et al., 1989). All of the laboratory tests included therein were concerned with neurobiological findings, with no reference to any laboratory test that would be of particular relevance to a cognitive, psychodynamic, or interpersonal-systems clinician. This has not changed for DSM-5.

The most explicit embracing of the neurobiological perspective has been proclaimed by the NIMH, which has become disgruntled with the lack of progress in the identification of specific etiologies, pathologies, and treatments (Cuthbert & Insel, 2013). NIMH blames this failure on the APA's *Diagnostic Manual*; more specifically, its failure to embrace a neurobiological reductionism. "Unlike our definitions of ischemic heart disease, lymphoma, or AIDS, the DSM diagnoses are based on a consensus about clusters of clinical symptoms, not any objective laboratory measure" (Insel, 2013). NIMH has taken the position that "mental disorders are biological disorders involving brain circuits that implicate specific domains of cognition, emotion, or behavior" (Insel, 2013) and they have indicated that they will be reluctant to fund studies that are concerned with the syndromes of DSM-5. "It is critical to realize that we cannot succeed if we use DSM categories" (Insel, 2013). "The first step is to inventory the fundamental, primary behavioral functions that the brain has evolved to carry out, and to specify the neural systems that are primarily responsible for implementing these functions" (Cuthbert & Insel, 2013, p. 4). NIMH has identified five broad areas of research they prefer to fund (i.e., negative valence systems, positive valence systems, cognitive systems, systems for social processes, and arousal/modulatory systems), collectively known as the Research Domain Criteria (RDoC; Sanislow et al., 2010). It is evident that NIMH will favor those studies with a neurobiological orientation. It will be very difficult for psychodynamic, cognitive-behavioral, or social-interpersonal systems researchers to obtain funding unless they can present their study within a neurobiological or cognitive neuroscience orientation (Insel, 2009).

Psychopathology involves, in part, dysregulation along various neurochemical pathways, but this neurochemistry interacts with and occurs within the context of psychological, sociological, and cultural variables that also play a significant role in the development of psychopathology. Their contribution is unlikely to be understood well through a biological reductionism (Satel & Lilienfeld, 2013). A person is a psychological being existing in a social world as well as a neurochemical mechanism. As suggested by Kendler (2005), mental disorders need to be understood at all levels of explanation, from the biological to the psychological to the cultural. "It is unlikely that cultural [and psychological] forces that shape psychopathology can be efficiently understood at the level of basic brain biology" (Kendler, 2005, p. 436). Biology, psychology, and culture interact and influence one another, and each level of explanation probably should be acknowledged if a comprehensive and complete understanding of psychopathology is to occur.

It is difficult, if not impossible, to create a diagnostic manual that is entirely neutral or atheoretical. However, the diagnostic manual should probably make an effort to remain above the competitive fray of competing theoretical models rather than embrace one particular team. As expressed by the APA (2013), DSM-5 is "used by clinicians and researchers from different orientations (biological, psychodynamic, cognitive, behavioral, interpersonal, family/systems), all of whom strive for a common language to communicate the essential characteristics of mental disorders" (p. xlii). A language that purposely favors one particular perspective will not provide an equal playing field and will subtly if not explicitly bias scientific research and discourse (Frances et al., 1989; Wakefield, 1998; Widiger &

Mullins-Sweatt, 2008). Nevertheless, NIMH has taken the position of embracing one particular theoretical model. With respect to NIMH funding, the biological theoretical perspective has essentially usurped the playing field. Not surprisingly, NIMH has received criticism for this decision (e.g., Frances, 2014; Wakefield, 2014; Weinberger & Goldberg, 2014), but these objections are unlikely to cause any fundamental change to their commitment to a neurobiological model of psychopathology.

Shifting to a Dimensional Model of Classification "DSM-IV-TR [was] a categorical classification that divides mental disorders into types based on criterion sets with defining features" (American Psychiatric Association, 2000, p. xxxi). This categorical classification is consistent with the medical tradition in which it is believed (and often confirmed in other areas of medicine) that mental disorders have specific etiologies, pathologies, and treatments (Zachar & Kendler, 2007). The intention of the *Diagnostic Manual* is to help the clinician determine which particular disorder is present, the diagnosis of which would indicate the presence of a specific pathology that would explain the occurrence of the symptoms and suggest a specific treatment that would ameliorate the patient's suffering (Kendell, 1975; Frances et al., 1995).

It is evident, however, that the diagnostic manuals of the APA have routinely failed in guiding a clinician to the identification of one specific disorder. Despite the best efforts of the authors of each edition to revise the criterion sets to increase their specificity, multiple diagnoses remain the norm (Widiger & Samuel, 2005). As expressed by Kupfer, First, and Regier (two of whom were the Chair and Vice Chair of DSM-5):

> In the more than 30 years since the introduction of the Feighner criteria by Robins and Guze, which eventually led to DSM-III, the goal of validating these syndromes and discovering common etiologies has remained elusive. Despite many proposed candidates, not one laboratory marker has been found to be specific in identifying any of the DSM-defined syndromes. Epidemiologic and clinical studies have shown extremely high rates of comorbidities among the disorders, undermining the hypothesis that the syndromes represent distinct etiologies. Furthermore, epidemiologic studies have shown a high degree of short-term diagnostic instability for many disorders. With regard to treatment, lack of treatment specificity is the rule rather than the exception.
>
> (Kupfer, First, & Regier, 2002, p. xviii)

In other words, the Chair and Vice Chair of DSM-5 (Drs. Kupfer and Regier, respectively) shared the frustration of the head of NIMH (Dr. Insel) regarding the failure of the existing diagnostic system to identify specific etiologies, pathologies, or treatments. However, the Chair and Vice Chair of DSM-5 placed the blame on the illusion that there are homogeneous, distinct conditions that have specific etiologies, pathologies, or treatments (rather than a "failure" to focus research on neurobiology). Most (if not all) mental disorders appear to be the result of a complex interaction of an array of interacting biological vulnerabilities and dispositions with a number of significant environmental, psychosocial events that often exert their progressive effects over a period of time (Rutter, 2003). The symptoms and pathologies of mental disorders appear to be highly responsive to a wide variety of neurobiological, interpersonal, cognitive, and other mediating and moderating variables that help to develop, shape, and form a particular individual's psychopathology profile. This complex etiological history and individual psychopathology profile are unlikely to be well described by single diagnostic categories that attempt to make distinctions at nonexistent discrete joints (Widiger & Samuel, 2005).

In 1999, a DSM-5 Research Planning Conference was held under joint sponsorship of the APA and the NIMH, the purpose of which was to set research priorities that would optimally inform future classifications. An impetus for this effort was the frustration with the existing nomenclature (Kupfer et al., 2002). At this conference, research planning work groups were formed to develop white papers that would set a research agenda for DSM-5. The Nomenclature Work Group, charged with addressing fundamental assumptions of the diagnostic system, concluded that it is "important that consideration be given to advantages and disadvantages of basing part or all of DSM-V on dimensions rather than categories" (Rounsaville et al., 2002, p. 12).

The white papers developed by the research planning work groups were followed by a series of international conferences whose purpose was to further enrich the empirical data base in preparation for the eventual development of DSM-5. The first conference was devoted to shifting personality disorders to a dimensional model of classification (Widiger, Simonsen, Krueger, Livesley, & Verheul, 2005). Chapter 12 in this volume, by Crego and Widiger, discusses the dimensional model of personality disorder proposed for DSM-5. The final conference was devoted to dimensional approaches across the diagnostic manual, including substance use disorders, major depressive disorder, psychoses, anxiety disorders, and developmental psychopathology, as well as the personality disorders (Helzer et al., 2008).

The introduction to DSM-5 explicitly acknowledges the failure of the categorical model: "the once plausible goal of identifying homogeneous populations for treatment and research resulted in narrow diagnostic categories that did not capture clinical reality, symptom heterogeneity within disorders, and significant sharing of symptoms across multiple disorders" (American Psychiatric Association, 2013, p. 12). It is further asserted that dimensional approaches will "supersede current categorical approaches in coming years" (p. 13). It was not the intention of the authors of DSM-5, though, to replace the diagnostic categories with a dimensional model of classification. Opposition to this shift is substantial, even for

the personality disorders (e.g., Gunderson, 2010; Shedler et al., 2010). "What [was] being proposed for DSM-V is not to substitute dimensional scales for categorical diagnoses, but to add a dimensional option to the usual categorical diagnoses for DSM-V" (Kraemer, 2008, p. 9).

Nevertheless, many of the revisions that occurred with DSM-5 reflect the preference to eventually shift the manual to a dimensional model of classification. Autism and schizophrenia are explicitly conceptualized as spectrum disorders, with different variants existing along a common spectrum of underlying pathology (see White & Conner, Chapter 21 in this volume). The problematic distinction between substance abuse and dependence was abandoned in favor of a level of severity (see Klosterman & Kelley, Chapter 14 in this volume). Reference is made within the introduction of the manual to the broad dimensions of internalizing and externalizing dysfunction that cut across existing categories. Included in Section 3 of DSM-5 for emerging models and measures is a five-domain 25-trait dimensional model of maladaptive personality functioning (Krueger et al., 2011) that is aligned conceptually and empirically with the five-factor model of general personality structure (DSM-5, p. 773).

Conclusions

Nobody was fully satisfied with, or lacked valid criticisms of, DSM-IV-TR. This is unlikely to change with DSM-5. Zilboorg's (1941) suggestion that budding 19th-century theorists and researchers cut their first teeth by providing a new classification of mental disorders still applies, although the rite of passage today is to provide a critique of the DSM.

Nobody, however, appears to suggest that all official diagnostic nomenclatures be abandoned. The benefits do appear to outweigh the costs (Salmon et al., 1917). Everybody finds fault with this language, but there is at least the ability to communicate disagreement. Communication among researchers, theorists, and clinicians of common or different theoretical persuasions would be much worse in the absence of this common language. The DSM-5 is perhaps to be commended for resisting the pressure of neurobiological psychiatry to favor this theoretical model to the detriment of all other theoretical perspectives. DSM-5 will be considerably more user-friendly for the psychodynamic, cognitive-behavioral, and interpersonal-systems clinicians and researchers than the RDoC system of NIMH.

Nevertheless, as an official diagnostic nomenclature, DSM-5 is an exceedingly powerful document with a considerable impact on how psychopathology is not only diagnosed, but also understood and treated. Persons think in terms of their language and DSM-5 will govern the manner in which clinicians think about psychopathology for many years to come, for better or for worse.

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7

Psychological Assessment and Clinical Judgment

Howard N. Garb, Scott O. Lilienfeld and Katherine A. Fowler

What major advances have occurred in the assessment of psychopathology over the past 30 years? Many psychologists would argue that the most important breakthroughs include the development of explicit diagnostic criteria, the growing popularity of structured interviews, the proliferation of brief measures tailored for use by mental health professionals conducting empirically supported treatments, and the use of measures for monitoring treatment progress. But they may also feel that progress has been frustratingly slow (e.g., McNally, 2011; Frances & Widiger, 2012, see also Widiger, Chapter 6 in this volume.) Controversies abound in the domain of assessment, and most beginning readers of this literature are left with little guidance regarding how to navigate the murky scientific waters.

Conceptual and methodological issues in the assessment of psychopathology, together with substantive results, are described in this chapter. We compare different types of assessment instruments, including structured interviews, brief self-rated and clinician-rated measures, projective techniques, self-report personality inventories, and behavioral assessment and psychophysiological methods. We also review research on the validity of assessment instruments and research on clinical judgment and decision making.

Our principal goal in this chapter is to assist readers with the task of becoming well-informed and discerning consumers of the clinical assessment literature. In particular, we intend to provide readers with the tools necessary to distinguish scientifically supported from unsupported assessment instruments and to make valid judgments on the basis of the former instruments.

Psychometric Principles

Before describing the validity of assessment instruments and research on clinical judgment and decision making, we must first clarify the meaning of three key terms: reliability, validity, and treatment utility (see also the *Standards for Educational and Psychological Testing*, American Educational Research Association, American Psychological Association, & National Council on Measurement in Education, 1999).

Reliability refers to consistency of measurement. It is evaluated in several ways. If all the items of a test are believed to measure the same trait, we want the test to possess good internal consistency: Test items should be positively intercorrelated. If a test is believed to measure a stable trait, then the test should possess test–retest reliability: on separate administrations of the test, clients should obtain similar scores. Finally, when two or more psychologists make diagnoses or other judgments for the same clients, interrater reliability should be high: Their ratings should tend to agree.

Traditionally, interrater reliability was evaluated by calculating the percentage of cases on which raters agree. For example, two psychologists might agree on diagnoses for 80% of the patients on a unit. However, percentage agreement is unduly affected by base rates. In this context, a *base rate* refers to the prevalence of a disorder. When the base rates of a disorder are high, raters may agree on a large number of cases because of chance. For example, if 80% of the patients on a chronic psychiatric inpatient unit suffer from schizophrenia, then two clinicians who randomly make diagnoses of schizophrenia for 80% of the patients will agree on the diagnosis of schizophrenia for about 64% of the cases (.8 × .8 = .64). Thus, a moderately high level of agreement is obtained, even though diagnoses are made arbitrarily. By calculating kappa or an intraclass correlation coefficient (ICC), one can calculate the level of agreement beyond the chance level of agreement. This calculation is accomplished by taking into account the base rates of the disorder being rated.

When interpreting kappa and ICC, one generally uses the following criteria: Interrater reliability is poor for values below .40, fair for values between .40 and .59, good for values between .60 and .74, and excellent for values above .75 (Fleiss, 1981, p. 218). However, these criteria are typically used for making judgments (e.g., diagnoses or predictions of behavior), not for scoring test protocols. For scoring protocols, a reliability coefficient of at least .90 is desirable (Nunnally, 1978, pp. 245–6). Reliability should be higher for scoring tests than for making clinical judgments because if a test cannot be scored reliably, judgments based on those test scores will necessarily have poor reliability.

According to the *Standards for Educational and Psychological Testing* (American Educational Research Association et al., 1999), *validity* refers to "the degree to which evidence and theory support the interpretations of test scores" (p. 9). Put another way, validity is good if the use of a test allows us to draw accurate inferences about clients (e.g., if the use of test scores helps us to provide correct descriptions of traits, psychopathology, and diagnoses).

Reliability and validity differ in important ways. Reliability refers to the consistency of test scores and judgments; validity refers to the accuracy of interpretations and judgments. When reliability is good, validity can be good or poor. For example, two psychologists can agree on a client's diagnosis, yet both can be wrong. Or a client can obtain the same scores on two administrations of a test, yet inferences made using the test scores may be invalid. In contrast, when reliability is poor, validity is necessarily also poor. For example, if two psychologists cannot agree on a client's diagnosis, at least one of them is not making a valid diagnosis.

Different types of evidence can be obtained to evaluate validity. First, evidence can be based on test content (*content validity*). For example, a measure of a specific anxiety disorder, such as obsessive-compulsive disorder, should include an adequate representation of items to assess the principal features of this disorder, in this case obsessions and compulsions. To ensure content validity, many structured interviews contain questions that inquire comprehensively about a particular mental disorder. Second, evidence can be based on the relation between tests. A test should show *moderate or high* correlations with other tests that measure the same attribute or diagnosis (*convergent validity*) and *low* correlations with tests that measure other attributes and diagnoses (*discriminant validity*). When test scores are used to forecast future outcomes, psychologists refer to this as *predictive validity*. When these scores are correlated with indices or events measured at approximately the same time, psychologists refer to this as *concurrent validity*.

The validity of an assessment instrument can also be evaluated by examining its internal structure (*structural validity*; see Loevinger, 1957). If a test generates many scores that do not intercorrelate as expected, this result may suggest that inferences based on those test scores are wrong.

When we describe the content validity, convergent validity, or structural validity of an assessment instrument, we are also describing its construct validity. A construct is a theoretical variable that cannot be measured perfectly. More specifically, constructs are hypothesized attributes of individuals that cannot be observed directly, such as extraversion or schizophrenia (Cronbach & Meehl, 1955; but see Borsboom, Mellenbergh, & Van Heerden, 2004, for a critique). Construct validity is a broad concept that subsumes content validity, convergent validity, and structural validity.

A number of statistics can be used to evaluate the validity of assessment instruments. Researchers commonly calculate correlations between a test and other measures, including scores on related tests. Other statistics can yield even more useful information. For example, results on sensitivity and specificity are presented routinely in the literature on medical tests, but only infrequently in the literature on psychological assessment (Antony & Barlow, 2010). *Sensitivity* is the likelihood that one will test positive if one has a specified mental disorder. *Specificity* is the likelihood that one will test negative if one does not have the specified disorder. Ideally, one attempts to maximize both sensitivity and specificity, although there may be cases in which one elects to emphasize one statistic over the other. For example, if one were attempting to predict suicide in a large group of patients, one would probably be more concerned with sensitivity than specificity (it would be better to identify too many individuals for follow-up than to miss someone who may commit suicide). Other important statistics are positive and negative predictive power. *Positive predictive power* describes the likelihood of a disorder given the presence of a particular result on an assessment instrument. *Negative predictive power* describes the likelihood of the absence of a disorder given the absence of the particular result on the assessment instrument.

The concepts of sensitivity, specificity, positive predictive power, and negative predictive power are illustrated in Table 7.1. In this scenario, provisional diagnoses of a mood disorder are made when the *T*-score for scale 2 (depression) of the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) is ≥ 65 (the standard cutoff for psychopathology on the MMPI-2).[1] In the sample with a base rate of 50% (50% of the clients are depressed), 500 clients are depressed, and 500 clients are not depressed. For the 500 clients who are depressed, 425 have a *T*-score ≥ 65. Thus, sensitivity is equal to 425/500 = 85.0%. Computations for specificity, positive predictive power, and negative predictive power are also presented in the table.

Depending on the statistic used to evaluate validity, accuracy can vary with the base rate of the behavior being predicted (Meehl & Rosen, 1955; also see Greene, 2000, pp. 365–6). As can be seen from Table 7.1, when

TABLE 7.1
Effect of Base Rate on Positive Predictive Power (PPP), Negative Predictive Power (NPP), Sensitivity, and Specificity

Scale 2 (D)	Depressed (*n*)			PPP		NPP		Sensitivity		Specificity	
	Yes	No	Total (*N*)	(*n*)	(%)	(*n*)	(%)	(*n*)	(%)	(*n*)	(%)
Base rate = 50%											
T ≥ 65	425	150	575								
T < 65	75	350	425								
Total	500	500	1000	425/575	73.9	350/425	82.4	425/500	85.0	350/500	70.0
Base rate = 2%											
T ≥ 65	17	294	311								
T < 65	3	686	689								
Total	20	980	1000	17/311	5.0	686/689	99.6	17/20	85.0	686/980	70.0

percentage correct, positive predictive power, and negative predictive power are used to describe validity, accuracy varies with the base rate. For example, percentage correct is 425 + 350 divided by 1000 = 77.5% when the base rate is 50%, and 17 + 686 divided by 1000 = 70.3% when the base rate is 2%. In contrast, when sensitivity and specificity are used to describe validity, accuracy does not vary with the base rate. Put another way, when percentage correct, positive predictive power, and negative predictive power are used to describe accuracy, the same test will be described as having varying levels of accuracy depending on the base rates in different samples. In general, positive predictive power tends to decrease when base rates decrease, whereas negative predictive power tends to increase when base rates decrease. Thus, statistically rare events (e.g., suicide) are difficult to predict, whereas common events (e.g., no suicide) are relatively easy to predict.

When reading about positive findings for a test score, psychologists should be aware that the score may not work well in their work setting if the base rate in their work setting differs widely from the base rate in the study. If the base rate for a disorder is .5 in a study but .01 in a clinic, one can expect results for positive predictive power to be much less favorable in the clinic. Nevertheless, a large body of psychological research indicates that psychologists tend to (a) neglect or greatly underuse base rates when making judgments and predictions; and (b) focus too heavily on whether a client falls above or below a test's cutoff score (Finn & Kamphuis, 2002). As a consequence, clinician judgments can sometimes be grossly inaccurate when the base rates of the phenomenon in question are extreme (e.g., very low).

Signal detection theory (SDT) often provides the most useful information regarding the validity of an assessment instrument. SDT is a statistical approach that is used when the task is to detect a signal, such as the presence of major depression in a client. By using SDT, we can describe the validity of an assessment instrument across all base rates and across all cutoff scores (the signal is said to be present when a client's score exceeds the cutoff score). Different clinicians may set different cutoff scores for the same test; it is important that our estimate of the validity of a test not be influenced by the placement of the cutoff score. As observed by McFall and Treat (1999): "There is no longer any excuse for continuing to conduct business as usual, now that SDT-based indices represent a clear and significant advance over traditional accuracy indices such as sensitivity, specificity, and predictive power" (p. 227). Although such indices as sensitivity, specificity, and predictive power are informative, we agree with McFall and Treat that in many cases the use of SDT is more appropriate and comprehensive.

Other important psychometric concepts are norms, incremental validity, and treatment utility. *Norms* are scores that provide a frame of reference for interpreting a client's results. For the assessment of psychopathology, normative data can be collected by administering a test to a representative sample of individuals in the community. If a client's responses are similar to the normative data, psychologists should be very cautious about inferring the presence of psychopathology. When norms for a test do not accurately reflect the population of interest, clinicians may make inaccurate and potentially harmful judgments even if the test is valid. For example, a test may be a good measure of thought disorder, but a clinician interpreting a client's test results may erroneously conclude that the client has a thought disorder if the norms give a false impression of how people in the community perform on the test. Historically, problems with norms have been as serious as, if not more serious than, problems with validity (Wood, Garb, & Nezworski, 2007).

Incremental validity describes the extent to which an instrument contributes information above and beyond already available information (e.g., other measures). For example, the use of a psychological test may allow clinicians to make judgments at a level better than chance, but judgments made using interview and test information may not be more accurate than judgments based on interview information only. One major criticism of the Rorschach inkblot test is that it does not provide useful information beyond that which can be obtained using more easily administered instruments, such as questionnaires (Lilienfeld, Wood, & Garb, 2000).

Treatment utility describes the extent to which an assessment instrument contributes to decisions about

treatment that lead to better outcomes. An assessment instrument could have good validity and good incremental validity, yet not lead to improved treatment outcome. Surprisingly, few researchers have examined the treatment utility of assessment instruments (Harkness & Lilienfeld, 1997; Garb, Lilienfeld, Nezworski, Wood, & O'Donohue, 2009).

Assessment Instruments

Interviews *Unstructured interviews* are used predominantly in clinical practice, whereas *structured* and *semistructured interviews* are used predominantly in research. One exception is that structured and semistructured interviews are used for clinical care in a growing number of university-based clinics. When conducting an unstructured interview, a psychologist is responsible for deciding what questions to ask. In contrast, when conducting a structured interview, questions are standardized. As one might surmise, semistructured interviews represent a balance between structured and unstructured interviews, providing guidance for interviewers but affording them some flexibility.

Reliability Field trials were conducted to learn about the reliability of diagnoses using the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013). In the field trials, the same patients were interviewed by different clinicians on separate occasions, in clinical settings, and with usual (that is, unstructured) clinical interview methods (Regier et al., 2013). Reliability ranged from unacceptable (three diagnostic categories) to very good (five diagnostic categories). The other two levels of reliability were "good" (nine diagnostic categories) and "questionable" (six diagnostic categories). Although reliability was good or very good for 14 of the 23 mental health diagnoses, it was questionable or unacceptable for the remaining nine. For example, posttraumatic stress disorder was one of the most reliable mental diagnoses in the field trials, while diagnoses of major depressive disorder was in the questionable range and mixed anxiety-depressive disorder was in the unacceptable range.

When mental health professionals do not adhere to diagnostic criteria, interrater reliability is often poor. Studies indicate that this is often likely to happen. Instead of attending to diagnostic criteria, clinicians often make diagnoses by comparing patients with their concept ("prototype") of the typical person with a given mental disorder (e.g., Blashfield & Herkov, 1996; Garb, 1996; Morey & Ochoa, 1989).

The use of semistructured and structured interviews tends to lead to both good adherence to diagnostic criteria and good interrater reliability (Antony & Barlow, 2010). For example, the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Brown, Di Nardo, & Barlow, 1994) requires interviewers to inquire about the DSM-IV criteria for anxiety disorders. Favorable reliability results have been found for this instrument: kappa values have ranged from .60 to .86 for diagnoses of the DSM-IV anxiety disorders (Brown, Di Nardo, Lehman, & Campbell, 2001).

Validity Virtually all mental disorders are open concepts marked by (a) intrinsically fuzzy boundaries, (b) an indicator list that is potentially infinite, and (c) an unclear inner nature (Meehl, 1986). Although diagnostic criteria sometimes change in response to changing social norms and political pressures, they also change as more is learned about a disorder. For example, we may eventually learn that certain individuals who meet the DSM criteria for schizophrenia actually have disorders that have not yet been identified. Still, the construct of schizophrenia can be useful, even though we are aware that the meaning of the term is somewhat imprecise and will change as more research is conducted. DSM diagnoses can be valid and useful even when we possess an incomplete understanding of the nature, etiology, course, and treatment of the conditions categorized.

There are several reasons to believe that structured interviews are, on average, more valid than unstructured interviews. First, it does make a difference whether one conducts an unstructured or structured interview, as agreement between structured interview diagnoses and diagnoses made in clinical practice, where unstructured interviews are the norm, is generally poor (Rettew, Lynch, Achenbach, Dumenci, & Ivanova, 2009). Second, reliability tends to be better for structured and semistructured interviews, so all other things being equal they are more likely to be valid. Third, many structured interviews are designed to inquire comprehensively about the DSM criteria. To the extent that the DSM criteria have been validated, the validity of these structured and semistructured interviews will be supported. Fourth, when interviews and self-report instruments are used to diagnose personality disorders, clinicians using unstructured interviews typically show the lowest agreement with other assessment instruments. In their review of the literature, Widiger and Lowe (2010) noted that "validity coefficients decrease when at least one of the two methods of assessment is an unstructured clinical interview" (p. 586).

Although structured interviews generally appear to be more valid than unstructured interviews, their limitations need to be recognized. Because these limitations are shared with unstructured interviews, they do not indicate that unstructured interviews possess advantages over structured interviews. First, it is relatively easy for respondents to consciously underreport or overreport psychopathology on structured interviews (Alterman et al., 1996). Moreover, few structured interviews contain validity scales designed to detect dishonest or otherwise aberrant responding. Second, because memory is fallible, reports in

interviews are often inaccurate or incomplete, even when clients are not intentionally trying to deceive the interviewer (Henry, Moffitt, Caspi, Langley, & Silva, 1994). Third, when clinicians are instructed to make use of medical records and other information in addition to structured interviews, considerable clinical judgment is required to describe and diagnose the client because information from different sources may conflict. This requirement is a potential limitation because clinical judgment is fallible.

Finally, an important methodological advance in evaluating the validity of interviews is the LEAD standard. LEAD is an acronym for *longitudinal*, *expert*, and *all data*. When using the LEAD standard (Spitzer, 1983), diagnoses made by using interviews are compared with diagnoses made by collecting longitudinal data. Using this approach, clients are followed over time to provide longitudinal data for making diagnoses, and diagnoses are made by expert clinicians using all relevant data. Although the LEAD standard can help us learn about the validity of diagnoses, it has rarely been used. Using this approach, one can more accurately evaluate the validity of diagnoses based on unstructured, structured, or semistructured interviews.

Brief Self-Rated and Clinician-Rated Measures Brief measures have been developed for (a) monitoring psychotherapy progress, and (b) providing information necessary to deliver standardized evidence-based interventions. To an extent seldom found for other assessment instruments, treatment utility has been established for measures used to monitor psychotherapy progress.

A number of systems have been developed for monitoring the progress of psychotherapy, and in particular for identifying clients who have made little or no progress during psychotherapy and who are at risk for deteriorating. For example, research suggests that it can be helpful to administer the Outcome Questionnaire-45 (OQ-45; Whipple & Lambert, 2011) to clients before every session. The OQ-45 takes about 5–7 minutes to complete, and measures constructs related to level of functioning including: (1) distress and symptoms; (2) interpersonal problems; and (3) problems related to social role performance. Normative data have been collected, making it possible to identify clients who have made less progress than 95% of the clients who obtained the same initial OQ-45 score (Lambert, Harmon, Slade, Whipple, & Hawkins, 2005). Results obtained in Germany and the United States demonstrate that clients tend to have better outcomes when therapists are alerted to poorly progressing clients (e.g., Lambert et al., 2003; Percevic, Lambert, & Kordy, 2004). For example, for clients identified as being at risk, deterioration rates decreased from 21% to 13% when therapists were provided with feedback information (Lambert et al., 2003).

In addition, brief self-rated and clinician-rated measures have been constructed to provide information necessary to deliver standardized, evidence-based interventions. For example, the Beck Depression Inventory-II, the Beck Anxiety Scale, and other scales are frequently used to assess depression and anxiety (Beck & Steer, 1990; Beck, Steer, & Brown, 1996). In addition, psychologists who use cognitive behavioral techniques to treat panic disorder frequently use brief measures to describe: (a) the severity and frequency of panic-related symptoms; (b) cognitions or beliefs that are frequently associated with panic disorder; (c) clients' perceptions of their control over threatening internal situations; and (d) panic-related avoidance behaviors (e.g., Baker, Patterson, & Barlow, 2002). Because psychologists who use cognitive behavioral techniques are not usually concerned with measuring broad areas of psychopathology and personality, brief self-rated and clinician-rated measures are typically best suited for their needs.

The reliability and validity of many of these measures appears to be adequate. For example, the Panic Disorder Severity Scale (PDSS; Shear et al., 1997) is a seven-item scale that can be completed by either clients or clinicians to describe key features of panic disorder with agoraphobia. When completed by a clinician, the ratings are based on information that has been gathered in interview and therapy sessions. Ratings are made for frequency of panic, anxiety about future panic attacks, magnitude of distress during panic, interference in social functioning, interference in work functioning, and avoidance behaviors. The PDSS possesses excellent interrater reliability (kappa = .87; Shear et al., 1997). In addition, PDSS ratings are correlated significantly with other measures of features of panic disorder. For example, Shear et al. (1997) obtained a correlation of $r = .55$ for the relation between total scores on the PDSS and severity ratings for panic disorder on the ADIS-IV. Equally important is evidence related to the content of the scale items: The PDSS was designed to assess problems that are important for treatment planning. In fact, a reason that brief self-rated and clinician-rated measures are popular is that they evaluate dimensions believed to be important for treatment planning. That is, they are designed to (a) evaluate problems that need to be addressed in treatment; and (b) provide information that is required to implement empirically supported treatment interventions (see Stewart & Chambless, Chapter 8 in this volume). Finally, the PDSS can be used to track a client's progress. This use bears important implications for treatment use. For example, if the measure is administered during the course of treatment and indicates that a client is not improving, the clinician should consider trying a different intervention. In one study (Shear et al., 1997), clients were classified as treatment responders and nonresponders on the basis of ratings made by independent evaluators. A different group of evaluators made PDSS ratings for all clients before and after treatment. In contrast to nonresponders, responders showed statistically significant improvement on the PDSS.

Although reliability and validity appear to be fair for many self-rated and clinician-rated measures, the

evaluation of their validity has been limited. Rather than compare the results from one of these measures with the results of a structured interview or with another self-rated or clinician-rated measure, it would be helpful to use behavioral assessment methods to evaluate validity. This approach has rarely been used, but the results from one study are provocative. When behavioral assessment methods were used to evaluate the validity of clients' statements, clients were found to overestimate the frequency and intensity of their panic attacks on a structured interview and on a brief self-rated test (Margraf, Taylor, Ehlers, Roth, & Agras, 1987).

Behavioral Assessment Methods and Psychophysiological Assessment Behavioral assessment methods and psychophysiological assessment can provide valuable information. For example, by using diary measures, one can ask a client to record and rate the frequency and intensity of panic attacks shortly after symptoms occur. Or one can monitor a client's eating or smoking habits. By making ratings shortly after symptoms or behaviors occur, the results are more likely to be accurate than when based on retrospective reports. Cell phones have made it easier to collect and analyze self-monitoring data (data describing clients' ongoing behavior are recorded by the clients themselves).

Behavioral assessment tests and other behavioral observation techniques can also provide valuable information. Behavioral assessment tests (also known as behavioral approach tests and behavioral avoidance tests) involve asking clients to enter situations that typically make them anxious, that they avoid, or both. For example, a client with a phobia can be instructed to approach a feared object (e.g., a spider) and a client with an obsessive-compulsive disorder can be instructed to switch off an electrical appliance and leave the room without checking it. During and after behavioral assessment tests, clients rate their level of anxiety. Other techniques include observing clients in roleplay situations and in natural settings (without their being instructed to confront feared situations). For example, observations can be made of children in classrooms or of patients on psychiatric units.

Psychophysiological techniques can also provide valuable information. For example, a polysomnographic evaluation, which is conducted in a sleep laboratory, can provide valuable information about how well a client is sleeping (Savard, Savard, & Morin, 2010). Similarly, measures of psychophysiological arousal can provide important information in the assessment of posttraumatic stress disorder, especially with respect to treatment process and outcome (Litz, Miller, Ruef, & McTeague, 2002).

Global Measures of Personality and Psychopathology *Projective techniques* and *self-report personality* inventories are designed to measure broad aspects of personality and psychopathology. Projective techniques include the Rorschach, Thematic Apperception Test (TAT), and human figure drawings. Self-report personality inventories include MMPI-2 (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989), the MMPI-2-Restructured Form (MMPI-2-RF; Ben-Porath & Tellegen, 2008), the Personality Assessment Inventory (PAI; Morey, 2007), and the Millon Clinical Multiaxial Inventory-III (MCMI-III; Millon, 1994). Projective techniques are relatively unstructured: Stimuli are frequently ambiguous (e.g., inkblots, as in the case of the Rorschach) and response formats are typically open-ended (e.g., telling a story in response to a drawing of individuals interacting, as in the case of the TAT). In contrast, self-report personality inventories are relatively structured: Stimuli are fairly clearcut (e.g., statements with which a client agrees or disagrees) and response formats are constrained (e.g., on the MMPI-2-RF, one can make a response of "true" or "false"). In general, validity findings are more encouraging for self-report personality inventories than for projective techniques, although exceptions have been found (see Lilienfeld, Wood, & Garb, 2000; Wood, Nezworski, Lilienfeld, & Garb, 2003).

Projective Techniques A common argument for using projective techniques is that they can circumvent a client's purported defenses and therefore can be used to evaluate conscious and unconscious processes. For example, some psychologists believe that when clients look at Rorschach inkblots and report what they see, they cannot invent faked responses because they do not know the true meaning of their Rorschach responses. However, at least one study suggests that projective techniques are vulnerable to faking. In this study (Albert, Fox, & Kahn, 1980), normal participants were instructed to fake paranoid schizophrenia. Presumed experts in the use of the Rorschach (Fellows of the Society for Personality Assessment) were unable to detect faking of psychosis. Diagnoses of malingering were made for 4% of the psychotic participants, 9% of the informed fakers (they were informed about the nature of disturbed thought processes and paranoid schizophrenia, but not about the Rorschach), 7% of uninformed fakers, and 2% of normal participants who were not instructed to fake. Diagnoses of psychosis were made for 48% of the psychotic participants, 72% of the informed fakers, 46% of the uninformed fakers, and 24% of the normal participants who were not instructed to fake.

Several problems with projective techniques can be described. First, reliable scoring is often difficult to achieve.[2] For example, the Comprehensive System (Exner, 1993) continues to be the most popular system for scoring and interpreting the Rorschach (Meyer, Hsiao, Viglione, Mihura, & Abraham, 2013). Exner (1993, p. 23), the developer of the Comprehensive System, claimed that interrater reliability is better than .85 for all Comprehensive System scores. However, results from other studies indicate that this result is true for only about 50% of the Comprehensive

System scores (Acklin, McDowell, Verschell, & Chan, 2000; Guarnaccia, Dill, Sabatino, & Southwick, 2001; Nakata, 1999; Shaffer, Erdberg, & Haroian, 1999; but see Meyer et al., 2002). In comparison, scoring is typically excellent for other types of tests (e.g., intelligence tests and personality inventories). For example, interrater reliability coefficients for the scoring of the Wechsler Adult Intelligence Scale, third edition (WAIS-III) have a median value of .95 and a minimum value of .90. Reliability for scoring the MMPI-2 is even better because it is typically computer scored.

Another problem with projective techniques is that normative data have often been unavailable or inaccurate. Norms are crucial because they allow clinicians to compare a client's responses with results from a well-defined sample, such as a sample that is representative of the general population. If a client's results look just like those typically obtained for the general population, then one is unlikely to conclude that the client has unusual levels of psychopathology. Yet, psychologists rarely use normative data when interpreting TAT protocols and human figure drawings (largely because such data are unavailable for the substantial majority of indices derived from these measures), and serious problems have surfaced regarding the normative data for the Comprehensive System.

Problems with the normative data for the Comprehensive System are several. First, Exner (2001) reported that his 1993 Comprehensive System adult normative sample contained an error of enormous magnitude: The sample was described as being composed of 700 distinct protocols but it actually contained 479 distinct protocols with 221 protocols counted twice. Subsequently, the Comprehensive System adult normative sample has been revised (Exner, 2001), but even this sample has been reported to contain errors (Meyer & Richardson, 2001). Second, Exner (personal communication, December 8, 2000) refused to make the current Comprehensive System adult normative sample available for examination, even though J. M. Wood, a prominent critic of the Comprehensive System, offered to pay for any expenses that this would incur (J. M. Wood, personal communication, August 5, 2000).[3] Third, three of the strongest advocates for the Rorschach have subsequently discouraged clinicians from using the Comprehensive System norms for children, even though they have been used for many years (Meyer, Erdberg, & Shaffer, 2007). They also presented data that raise questions about the Comprehensive System norms for adults. Finally, and most important, despite scattered claims to the contrary (e.g., Butcher, 2010), the use of the Comprehensive System norms is likely to lead to the detection of psychopathology, even when no psychopathology is present (Hamel, Shaffer, & Erdberg, 2000; Shaffer et al., 1999; Wood, Nezworski, Garb, & Lilienfeld, 2001). For example, in one study (Hamel et al., 2000), the Rorschach was administered to a group of 100 relatively normal school children. Children were excluded from the study if they had a history of mental disorder. Even though an independent measure (the Conners Parent Rating Scale-92; Conners, 1989) revealed that the children were healthier than average, the results for the Rorschach indicated that the typical child in the sample suffered from "a distortion of reality and faulty reasoning approaching psychosis" and "an affective disorder that includes many of the markers found in clinical depression" (p. 291).

For evaluating validity, the following criteria were proposed by Wood, Nezworski, and Stejskal (1996): (a) test scores should demonstrate a consistent relation to a particular symptom, trait, or disorder; (b) results must be obtained in methodologically rigorous studies; and (c) results must be replicated by independent investigators. Historically, few scores for the Rorschach, TAT, and human figure drawings satisfy these criteria. For the Rorschach, the strongest support has been obtained for scores used to detect thought disorder and psychotic conditions marked by thought disorder (e.g., schizophrenia, bipolar disorder), predict psychotherapy outcome, and detect personality traits related to dependency (e.g., Acklin, 1999; Bornstein, 1999; Jorgensen, Andersen, & Dam, 2000; Meyer & Handler, 1997). For the TAT, the criteria have been satisfied for the assessment of achievement motives, object relations, and the detection of borderline personality disorder (Spangler, 1992; Westen, Lohr, Silk, Gold, & Kerber, 1990; Westen, Ludolph, Block, Wixom, & Wiss, 1990). For human figure drawings, the criteria have been satisfied only for distinguishing global psychopathology from normality, which is rarely a clinically useful demarcation (Naglieri & Pfeiffer, 1992).

Two notable meta-analyses, both on Exner's Comprehensive System, have recently been conducted (Mihura, Meyer, Dumitrascu, & Bombel, 2013; Wood, Garb, Nezworski, Lilienfeld, & Duke, 2015). Mihura and colleagues synthesized results for more than 50 Comprehensive System Rorschach scores from more than 200 published studies. They reported that there is "little to no validity support for over a third . . . of the targeted variables" even though those Rorschach scores have been interpreted by psychologists for decades (p. 27). They also reported that some scores were "strongly supported." A new meta-analysis on a subset of the Comprehensive System variables examined by Mihura and colleagues was conducted (Wood et al., 2015) using unpublished studies (e.g., dissertations) in addition to the published studies used by Mihura and colleagues (2013). Scores related to cognitive impairment (e.g., scores indicating aberrant speech patterns or odd thoughts) were supported. Other scores were not, even though they were described as being strongly supported by Mihura and colleagues (2013). For example, the validity coefficient for the *Anatomy and X-ray score*, a measure of "preoccupations with body vulnerability or its functioning" (p. 551), decreased from $r = .33$ to $r = .07$ when unpublished studies were included with the published results. Similarly, the validity coefficient for *Sum of Shading*, a measure of "distressing or irritating internal stimuli" (p. 550), decreased from .37

to .09 when the effect sizes were calculated using the International Norms (Meyer et al., 2007) and unpublished studies were included with the published results.

Personality Inventories Self-report personality inventories require clients to indicate whether a statement describes them. However, contrary to widespread claims, self-report personality inventories do not require clients to be able to accurately describe their symptoms and personality traits. The "dynamics" of self-report personality inventories were described by Meehl:

> A self-rating constitutes an intrinsically interesting and significant bit of verbal behavior, the non-test correlates of which must be discovered by empirical means. [The approach is free] from the restriction that the subject must be able to describe his own behavior accurately, ...The selection of items is done on a thoroughly empirical basis using carefully selected criterion groups.
>
> (Meehl, 1945, p. 297)

Thus, whereas the validity of brief self-rated tests rests on the content of items, the validity of self-report personality inventories rests on empirical research that relates test items (and test scales) to client characteristics. This approach has met with some success. For example, results from a meta-analysis, as well as data from more recent studies, indicate that the MMPI can be useful for detecting overreporting and underreporting of psychopathology and cognitive impairment (Berry, Baer, & Harris, 1991; see also Rogers, Gillard, Berry, & Granacher, 2011).

Scientific support for personality inventories has been mixed. One positive feature is that psychologists who use self-report personality inventories almost always use norms. Also, the norms of major self-report personality inventories, such as the MMPI-2-RF, are representative of American adults and adolescents in the community. There is no evidence that using the MMPI-2-RF and these norms would lead to judgments that normal individuals are pathological.

The validity of the primary scales of some tests (e.g., the MMPI-2, the MMPI-2-RF, the PAI) has generally been supported, whereas the validity evidence for the scales of other widely used tests (e.g., the MCMI-III) is weaker and less consistent. For example, Rogers, Salekin, and Sewell (1999) reviewed the research on the MCMI-III and concluded that it should not be used in forensic settings (also see Dyer & McCann, 2000; Rogers, Salekin, & Sewell, 2000). Even for the MMPI-2, half of the supplementary scales have not been consistently supported (e.g., Greene, 2000, pp. 218–69). Despite these negative findings, positive results on validity have been consistently replicated by independent investigators for a large number of MMPI-2 and MMPI-2-RF scores. For example, research has demonstrated that scale 4 (psychopathic deviate) is correlated positively with criminal behaviors and recidivism risk (see Greene, 2000, p. 148), whereas scale 9 (hypomania) is correlated with such characteristics as impulsiveness, extraversion, and superficiality in social relationships (e.g., Graham, Ben-Porath, & McNulty, 1997). In addition, although the new MMPI-2-RF scales are controversial in some quarters (Butcher, 2010), evidence suggests that they are valid for detecting many psychopathological characteristics and may even possess incremental validity for this purpose above and beyond the original MMPI-2 scales (e.g., Sellbom, Bagby, Kushner, Quilty, & Ayearst, 2012; Sellbom, Ben-Porath, Lilienfeld, Patrick, & Graham, 2005; van der Heijden, Rossi, van der Veld, Derksen, & Egger, 2013).

In clinical judgment studies, judgments of psychopathology have been more valid when psychologists have been given results from personality inventories than when given results from projective techniques (e.g., Garb, 1989, 1998, 2003). In fact, in several studies, validity actually decreased, at least slightly, when Rorschach results were made available in addition to brief biographical and/or questionnaire results (e.g., Whitehead, 1985).

On the negative side, evidence for the utility of personality inventories in treatment planning is scarce, and the *very* little evidence that exists has not been supportive (Garb et al., 2009). Specifically, when researchers have used manipulated assessment designs to randomly assign some clinicians to receive specific assessment information (e.g., the MMPI-2), and others to not receive such information, they have not detected significant differences in treatment outcome (Lima et al., 2005). Therapists in this study were 25 doctoral students in clinical psychology who each received about 3–5 hours per week of supervision with a licensed clinical psychologist.

Clinical Judgment and Decision Making

Experience, Training, and Clinical Judgment When confronted with evidence regarding the poor validity of specific assessment instruments, such as projective techniques, some clinicians report that their extensive clinical experience permits them to extract useful inferences from these instruments. In other words, the argument goes, validation studies fail to capture the rich and subtle information that highly seasoned practitioners can obtain from certain assessment measures. For example, in response to review articles demonstrating that a mere handful of Rorschach variables are empirically supported (e.g., Lilienfeld et al., 2000, 2001), several practitioners, in messages to the Rorschach Discussion and Information Group, maintained that the negative research evidence was largely irrelevant because their numerous years of clinical experience with the Rorschach endowed them with special judgmental and predictive powers.

Nevertheless, these arguments do not withstand careful scrutiny, because the relation between clinical experience and judgmental accuracy has been weak in most studies of personality and psychopathology assessment (for reviews,

see Dawes, 1994; Garb, 1989, 1998; Garb & Boyle, 2014). For example, in one study (Ebling & Levenson, 2003), judges were asked to watch 3-minute videos of 10 married couples. Judges rated marital satisfaction and predicted whether the marriages would end in divorce. Lay judges (e.g., newlyweds, recently divorced individuals) were more accurate than professional judges (marital therapists, marital researchers) for rating marital satisfaction, but there was no significant difference between groups for predicting divorce.

One potential exception to the finding that experience and accuracy are negligibly related was reported in a study by Brammer (2002). To help them make diagnoses, psychologists and psychology graduate students were allowed to ask questions and ask for specific items of information. Number of years of clinician experience was significantly associated with the number of correct diagnoses made (r = .33), as well as with the number of diagnostically specific questions asked (r = .51). Brammer's findings raise the intriguing possibility that experience is related to validity on tasks that require clinicians to structure complex tasks, such as formulating a psychiatric diagnosis by honing in on potential problem areas and then asking progressively more specific questions.

Some clinicians could contend that although the overall relation between experience and accuracy tends to be weak, many clinicians in real-world settings know which of their judgments are likely to be accurate. Nevertheless, for the use of projective techniques and some other assessment instruments, the relation between the validity of clinicians' judgments and their confidence in these judgments is generally weak. For example, Albert et al. (1980) found no significant relation between validity and confidence when practitioners were asked to use Rorschach protocols to detect malingering. For the MMPI, there is some evidence that confidence is positively related to the validity of clinicians' judgments, but only when these judgments are reasonably valid (e.g., Goldberg & Lewis, 1965). That is, when psychologists use the MMPI to assist in making diagnoses, they tend to be able to state correctly which of their diagnostic judgments are most likely to be correct.

In contrast to the dispiriting findings concerning the value of clinical experience for personality assessment judgments, the research literature supports the value of training for some judgment tasks. For example, neuropsychologists are more likely to detect neurological impairment than are clinical psychologists (Garb, 1998); psychologists with specialized training in gerontology are more likely to make essential age-related diagnoses and recommendations (Hillman, Stricker, & Zweig, 1997); psychologists with training in forensic psychology may be more likely than other psychologists to detect lying (Ekman, O'Sullivan, & Frank, 1999); and psychiatrists do better than other physicians at properly prescribing antidepressant medicine (e.g., making sure a client is on a therapeutic dose; Fairman, Drevets, Kreisman, & Teitelbaum, 1998).

Why Clinicians Often Do Not Benefit From Experience

Why are practitioners often unable to benefit from clinical experience? Although the reasons are manifold (see Arkes, 1981; Dawes, Faust, & Meehl, 1989; Garb, 1989), we focus on six here. The first three concern the nature of the feedback available to clinicians, and the remaining three reasons concern cognitive processes that influence the selection and interpretation of this feedback.

Nature of Feedback First, in contrast to physicians in most domains of organic medicine, psychologists rarely receive clearcut feedback concerning their judgments and predictions (Meehl, 1973). Instead, the feedback psychologists receive is often vague, ambiguous, open to multiple interpretations, and delayed. For example, if a clinician concludes that an adult client was sexually abused in childhood on the basis of an unstructured interview and Rorschach protocol, this judgment is difficult to falsify. If the client were to deny a past abuse history or express uncertainty about it, the clinician could readily maintain that the interpretation may still be correct because the client may have forgotten or repressed the abuse (although the scientific evidence for repression is controversial; see Holmes, 1990). Moreover, when clinicians receive feedback regarding their predictions (e.g., forecasts of violence), it is often substantially delayed, thereby introducing the potential distorting effects of memory.

Second, clinicians typically have access to only a subset of the data needed for accurate judgments, a quandary referred to by Gilovich (1991) as the "missing data problem." For example, clinicians may perceive certain psychological conditions (e.g., nicotine dependence) to be more chronic and unremitting than they are (see Schachter, 1982) because they are selectively exposed to individuals who remain in treatment. Cohen and Cohen (1984) referred to this effect as the "clinician's illusion."

Third, some feedback that clinicians receive from clients is misleading. Meehl (1956) referred to an individual's tendency to accept highly generalized but non-obvious personality descriptions as the *P. T. Barnum effect*, after the circus entrepreneur who quipped that "I like to give a little something to everybody" and "A sucker is born every minute." Numerous studies demonstrate that most individuals presented with Barnum descriptions (e.g., "You have a great deal of unused potential," "At times you have difficulty making up your mind") find such descriptions to be highly compelling, particularly when they believe that these descriptions were tailored for them (Logue, Sher, & Frensch, 1992; Snyder & Larson, 1972). The P. T. Barnum effect demonstrates that personal validation, the informal method of validating test feedback by relying on respondents' acceptance of this feedback, is a highly fallible barometer of actual validity. In addition, because clients are often impressed by Barnum feedback, such feedback can fool clinicians into believing that their interpretations are more valid than they really are. The same process can also explain why astrologers and palm readers are often

confident that their interpretations are accurate (see Wood, Nezworski, Lilienfeld, & Garb, 2003).

Cognitive Processes Fourth, a substantial body of literature documents that individuals are prone to confirmatory bias, the tendency to selectively seek out and recall information consistent with one's hypotheses and to neglect information inconsistent with these hypotheses. Several investigators have found that clinicians fall prey to confirmatory bias when asked to recall information regarding clients. For example, Strohmer, Shivy, and Chiodo (1990) asked counselors to read three versions of a case history of a client, one containing an equal number of descriptors indicating good self-control and poor self-control, one containing more descriptors indicating good control than poor self-control, and one containing more descriptors indicating poor than good self-control. One week after reading this case history, psychotherapists were asked to offer as many factors they could remember that "would be helpful in determining whether or not [the client] lacked self-control" (p. 467). Therapists offered more information that would be helpful for confirming than disconfirming the hypothesis that the client lacked self-control, even in the condition in which the client was described as characterized primarily by good self-control descriptors.

There also is evidence that clinicians are sometimes prone to *premature closure* in diagnostic decision making: they may tend to reach conclusions too quickly (Garb, 1998). For example, Gauron and Dickinson (1969) reported that psychiatrists who observed a videotaped interview frequently formed diagnostic impressions within 30–60 seconds. Premature closure may both reflect and produce confirmatory bias. It may reflect confirmatory bias because clinicians may reach rapid conclusions by searching only for data that confirm preexisting hypotheses. It may produce confirmatory bias by effectively halting the search for data that could disconfirm such hypotheses.

Fifth, investigators have shown that clinicians, like all individuals, are prone to *illusory correlations*, which has generally been defined as the perception of (a) a statistical association that does not actually exist, or (b) a stronger statistical association than is actually present. Illusory correlations are likely to arise when individuals hold powerful a priori expectations regarding the covariation between certain events or stimuli. For example, many individuals are convinced that a strong correlation exists between the full moon and psychiatric hospital admissions, even though studies have demonstrated repeatedly that no such association exists (Rotton & Kelly, 1985).

In a classic study of illusory correlation, Chapman and Chapman (1967) examined why psychologists perceive clinically meaningful associations between signs on the draw-a-person (DAP) test (e.g., the drawing of large eyes) and psychiatric symptoms (e.g., suspiciousness), even though research has demonstrated that these associations do not exist (Kahill, 1984). They presented undergraduate participants with DAP protocols that were purportedly produced by psychiatric patients with certain psychiatric symptoms (e.g., suspiciousness). Each drawing was paired randomly with two of these symptoms, which were listed on the bottom of each drawing. Undergraduates were asked to inspect these drawings and estimate the extent to which certain DAP signs co-occurred with these symptoms. Chapman and Chapman found that participants "discovered" that certain DAP signs tended to consistently co-occur with certain psychiatric symptoms, even though the DAP signs and symptoms had been randomly paired. For example, participants mistakenly perceived large eyes in drawings as co-occurring with suspiciousness and broad shoulders in drawings as co-occurring with doubts about manliness. Interestingly, these are the same associations that tend to be perceived by clinicians who use the DAP (Chapman & Chapman, 1967). Illusory correlation has been demonstrated with other projective techniques, including the Rorschach (Chapman & Chapman, 1969) and sentence completion tests (Starr & Katkin, 1969). Scientifically minded practitioners need to be aware of the phenomenon of illusory correlation, which suggests that clinicians can be convinced of the validity of assessment indicators in the absence of validity.

Finally, it can be difficult to learn from clinical experience unless one thinks in probabilistic terms (Einhorn, 1988). Unfortunately, this skill can be very difficult to acquire. For example, individuals tend to overestimate the likelihood that they would have predicted an outcome after they have become aware of the outcome, an effect termed *hindsight bias* (Arkes, Faust, Guilmette, & Hart, 1988; Fischhoff, 1975). Thus, if a client commits suicide and an investigation is conducted, the investigators may conclude that the suicide could have been predicted—in part because of hindsight bias. Similarly, when asked to rate the likelihood of (a) one event occurring, and (b) the same event plus another event occurring, individuals will often give a higher rating for the likelihood of both events occurring, an effect termed *conjunction bias* (Garb, 2006; Tversky & Kahneman, 1983). For example, for a particular client, if one diagnosis seems likely (e.g., major depressive disorder) and another diagnosis seems unlikely (e.g., antisocial personality disorder), then some clinicians may believe the likelihood of a client having both diagnoses is greater than the likelihood of the client have only the less likely disorder. However, according to probability theory, the probability of events A and B, P (A $\cap$ B), cannot be greater than the probability of event A, P (A), or the probability of event B, P (B). Because clinicians do not naturally think probabilistically, they sometimes engage in deterministic reasoning. Every clinician can generate explanations for why a client behaves a particular way or has a particular disorder, but these explanations may not be correct. Clinicians do not necessarily have complete information about every client, and some of the information they do have is likely to contain error. For this reason,

we should not unquestioningly accept a theory or case formulation simply because it seems compelling and seems to explain all of the available information.

Group Biases in Judgment A large number of studies have examined biases related to the demographic characteristics of clients (e.g., their race, sex, or socioeconomic status; for a review, see Garb, 1997; also Winstead & Sanchez, Chapter 5 in this volume). *Bias* occurs when the validity of a clinical judgment or test differs by demographic characteristics of clients (e.g., when the validity of judgments is better for White clients than Black clients). The most frequent type of bias discussed by psychologists is *slope bias*, which involves differences in validity coefficients across groups. When a test or clinical judgment yields a significantly higher validity coefficient in one group than another (slope bias), the test or judgment exhibits "differential validity" (Anastasi & Urbina, 1997). Note that the mere presence of group differences on a test is not sufficient to infer bias; bias requires that the clinical judgment or test be less valid for one group than another. We briefly describe results for sex bias, race bias, and social class bias below.

- **Sex bias**: Sex role stereotypes are a cause for concern in the diagnosis of psychopathology, especially for the diagnosis of personality disorders. When two groups of clinicians are given one of two case vignettes, and the case vignettes are identical except for the designation of gender, then histrionic personality disorder is more likely to be diagnosed in women and antisocial personality disorder is more likely to be diagnosed in men. Both male and female clinicians exhibit this bias (e.g., Adler, Drake, & Teague, 1990; Ford & Widiger, 1989). Ford and Widiger (1989) found that clinicians were not biased when they made ratings for the individual criteria making up these diagnoses. This finding suggests that the bias is linked to clinicians' perceptions of the diagnoses themselves, not to the DSM criteria for these disorders (see also Winstead & Sanchez, Chapter 5 in this volume.)
- **Race bias**: In a number of studies conducted in clinical settings, race bias has been shown to occur in psychiatric diagnosis, the prescription of psychiatric medications, violence prediction, and child abuse reporting (Garb, 1997, 1998). When the effect of social class was controlled, race still emerged as an important predictor. African American and Hispanic patients were less likely to be diagnosed with a psychotic mood disorder and more likely to be diagnosed with schizophrenia compared with White patients exhibiting similar symptoms (e.g., Mukherjee, Shukla, Woodle, Rosen, & Olarte, 1983; Simon, Fleiss, Gurland, Stiller, & Sharpe, 1973). This occurred even when a diagnosis of schizophrenia was inappropriate. Additionally, African American patients with bipolar disorder are less likely than White patients with bipolar disorder to receive lithium and SSRIs (Kilbourne & Pincus, 2006), African American patients with schizophrenia are less likely than White patients with schizophrenia to receive second-generation antipsychotics (e.g., Mallinger, Fisher, Brown, & Lamberti, 2006), and African American patients are at risk for being given excessive doses of antipsychotics (e.g., Walkup et al., 2000; see also Winstead & Sanchez, Chapter 5 in this volume.)
- **Social class bias**: Social class bias has been demonstrated only sparsely in psychiatric diagnosis and treatment (Garb, 1997, 1998). One finding that has emerged is the relation of social class to psychotherapy decisions. Clinicians were more likely to recommend middle-class individuals than lower-class individuals for psychotherapy, and expected them to do better in therapy, when both groups were recommended. Additionally, middle-class clients were more likely to be recommended for insight-focused therapy, whereas lower-class clients received more recommendations for supportive therapy (see Garb, 1997, for a review; see also Winstead & Sanchez, Chapter 5 in this volume.)

Methodological Recommendations

Several methodological steps can be taken to improve the quality of psychological assessment and the judgments derived from psychological tests. First, more sophisticated procedures can be used to evaluate validity. For example, to evaluate the validity of diagnoses, one can use the LEAD standard (Spitzer, 1983; also see Garb, 1998, pp. 45–53). Use of the LEAD standard, described earlier, allows researchers to ascertain the validity of structured interviews and other assessment instruments. In addition, the criteria proposed by Wood et al. (1996) should be used to determine if an assessment instrument is valid for its intended purpose. For example, if positive validity findings have been obtained in two studies but not in two others, one would conclude that the assessment instrument does not meet the Wood et al. (1996) criteria because the results were not consistent.

A second recommendation is that item response theory (IRT) be used to construct and evaluate tests. IRT is an alternative to traditional (classical) test theory. It can be used as a methodological and statistical tool for a number of purposes including test construction, evaluating a test, and using person-fit indexes to assess how well a trait (or construct) describes an individual. For example, using person-fit indexes, one may conclude that a trait is not relevant to a person if the person responds in an idiosyncratic manner (e.g., endorses severe but not moderate symptoms of depression). IRT also permits test constructors to determine which items are most discriminating at different levels of the trait in question. For example, IRT analyses could reveal that a measure of depression adequately distinguishes non-depressed from moderately

depressed individuals, but not moderately from severely depressed individuals. Although well established in achievement and aptitude testing, IRT has been applied infrequently to personality assessment. This is partly because cognitive constructs are better understood than personality constructs. Put another way, construct validity issues have been more formidable for personality measurement. Just the same, in a relatively few studies, IRT has been applied successfully to personality assessment. For example, historically, linear factor analyses have been used to describe the structure of the MMPI and MMPI-2. Because MMPI and MMPI-2 items are dichotomous (true–false), and because linear factor analysis assumes that ratings are normally distributed and not dichotomous, it is more appropriate to use nonlinear factor analysis or multidimensional IRT methods. Using IRT to uncover the factor structure of the MMPI-2, Waller (1998) found important differences between his results and those of previous factor analyses.

Third, the use of computers for making judgments and decisions is becoming increasingly important in the assessment of psychopathology. Findings from meta-analyses (Grove, Zald, Lebow, Snitz, & Nelson, 2000; Aegisdóttir et al., 2006) suggest that computer programs can be successfully developed for this purpose. The utility of these programs derives from well-established (although still largely neglected) findings that actuarial (statistical) formulas based on empirically established relations between predictors and criteria are almost always superior to or at least equal to clinical judgment (Dawes et al., 1989; Grove & Meehl, 1996). However, relatively few well-validated computer programs are available for clinical tasks. As observed by Wood, Garb, Lilienfeld, and Nezworski:

> Substantial progress has been made in developing computerized algorithms to predict violence, child abuse and neglect, and recidivism among juvenile offenders . . . However, there are still no well-validated algorithms for making diagnoses [or case formulations], . . . describing personality traits and psychopathology, or making treatment decisions.
>
> (Wood, Garb, Lilienfeld, and Nezworski 2002, p. 534)

Similarly, Snyder (2000) concluded that popular computer programs that have been used for years to interpret test results (e.g., for the MMPI-2 and the Rorschach) are inadequately validated. Research is needed to develop and validate new computer programs that provide valid descriptions of a client's personality and psychopathology.

Conclusions

Assessing psychopathology is an activity fraught with potential error and bias. However, by attending to research findings, psychologists can avoid using test scores that are invalid, and they can become familiar with the strengths and weaknesses of clinical judgment. In this way, errors that are potentially detrimental to clients can be avoided. For example, the use of the Comprehensive System norms for interpreting Rorschach protocols can lead to false positives in the assessment of psychopathology. By not using the Comprehensive System norms, or by using them with extreme caution, one can minimize the risk of harmful judgments such as misdiagnosing normal clients as pathological.[4]

Some psychologists argue that although scientific research is important, we should also rely on clinical experience to determine if an assessment instrument is valuable. Indeed, some even argue that when research and clinical experience conflict, we should place a higher premium on the latter. Psychologists are frequently encouraged to use the Rorschach and other projective techniques because they seem to provide rich clinical data (e.g., Karon, 2000). However, clinical experience can be fallible for a host of reasons, including biased feedback, illusory correlation, confirmatory bias, and deterministic reasoning (Dawes et al., 1989; Garb, 1998). The scientific method, not clinical experience, is the best method for minimizing error and resolving controversies. Scientific techniques, such as double-blind designs and control groups, are essential tools that researchers have developed to protect themselves from being misled (Lilienfeld, 2002, 2010). As McFall noted:

> [There is a] commonly offered rationalization that science doesn't have all the answers yet, and until it does, we must do the best we can to muddle along, relying on our clinical experience, judgment, creativity, and intuition (cf., Matarazzo, 1990). Of course, this argument reflects the mistaken notion that science is a set of answers, rather than a set of processes or methods by which to arrive at answers. Where there are lots of unknowns—and clinical psychology certainly has more than its share—it is all the more imperative to adhere as strictly as possible to the scientific approach. Does anyone seriously believe that a reliance on intuition and other unscientific methods is going to hasten advances in knowledge?
>
> (McFall, 1991, pp. 76–7)

Finally, as noted by McFall (cited in Trull & Prinstein, 2012, p. 65), one feature that should distinguish clinical and counseling psychologists from most other mental health professionals is their scientific training (see also Baker, McFall, & Shoham, 2009). To ignore research findings because they make us feel uncomfortable is to neglect our most distinctive and positive attribute: our training in, and our willingness to be guided by, science.

Notes

1. We use the term "provisional diagnoses" because formal diagnoses of psychiatric disorders should not be made on the basis of the MMPI-2 alone.
2. In fact, the TAT is rarely scored in clinical practice (see Lilienfeld et al., 2000).
3. There has also been a heated argument over the accessibility of studies that have been cited to support the Comprehensive System.

Unpublished studies sponsored by Rorschach Workshops are cited as evidence supporting the Comprehensive System, but attempts to obtain copies of papers describing the studies have frequently been unsuccessful (e.g., Wood, Nezworski, & Stejskal, 1996). Copies of all correspondence will be provided on request.

4. For a case history describing a client who was apparently harmed by interpretations of the Rorschach, see Garb, Wood, Lilienfeld, and Nezworski (2002).

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8

Psychotherapy Research

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The focus of this book is on the study of psychopathology and its assessment and treatment. Understanding psychopathology is an important part of the science of psychology in its own right, but it is more than pure science. Psychopathology research plays an important role in the development of interventions to ameliorate mental disorders and to promote wellbeing. Almost every chapter in Part II of this text includes material on treatment for the disorders in question, usually based on the outcome of treatment research. Where does this information come from? In this chapter, we describe the process by which psychotherapy research is conducted, and the controversies surrounding the proper nature and role of such research.

Psychotherapy Research

Psychotherapy research is a broad field encompassing a number of streams of research (for an extensive overview of such research, see Lambert, 2013). *Process research*, designed to identify and delineate the important events in therapy, typically addresses what happens in a therapeutic session, and investigates such variables as therapist behaviors, client behaviors, and interactions between the therapist and the client. For example, a common theme in psychoanalytic therapy research is the characteristics of transference (Connolly, Crits-Christoph, Barber, & Luborsky, 2000). *Outcome research* focuses on the effects of psychotherapy, both immediate and long-term changes in the problems for which a person seeks or is referred for treatment, as well as improvement on broader variables such as quality of life or interpersonal functioning. Not surprisingly, process research is often linked to outcome research so as to identify factors that may be important in treatment outcome. For example, meta-analyses indicate that the better the client–therapist alliance, the better the treatment outcome, with the alliance accounting for about 5% of the variance in improvement (Martin, Garske, & Davis, 2000).

Yet other questions in psychotherapy research include prediction of treatment outcome—what are the characteristics of clients who do well in this treatment—or, to answer a more sophisticated question, who will do well in this treatment but not in that treatment? For example, Barber and Muenz (1996) showed that in treatment of major depression, interpersonal therapy was better for clients with obsessive-compulsive personality traits, whereas cognitive-behavior therapy was better for clients with avoidant personality traits. Such findings demonstrate *moderation* of treatment outcome by a patient characteristic, in this case, specific personality traits. Finally, *mediation* of treatment outcome is an important area of study. Mediation research asks, what are the mechanisms underlying treatment efficacy? For example, in a study comparing several forms of treatment for attention deficit hyperactivity disorder, Hinshaw and colleagues (2000) found that the impact of treatment on children's social skills at school was mediated by parents' use of negative discipline, ineffective discipline, or their combination. In other words, to the degree that treatment reduced parents' use of negative or ineffective discipline, children's social skills at school improved. Although all of these forms of psychotherapy research are important, our focus in this chapter will be mainly be on psychotherapy outcome research.

A Prototypical Psychotherapy Outcome Study

There is no one method of outcome research accepted by all types of psychotherapy researchers. What we describe here is the reigning paradigm for major studies such as those funded by the National Institute of Mental Health

in which the efficacy of one or more treatments is tested, as well as the paradigm accepted for research on empirically supported treatments, which will be defined in a later section. We will address some of the controversies about this paradigm later in this chapter. To establish *efficacy*, a psychological intervention is tested under well controlled circumstances. If it proves to be beneficial in multiple studies, then researchers may turn their attention to *effectiveness* research, in which the generalization of the treatment's benefits to less well controlled, more real-world circumstances is examined. Hence, the first efforts have a strong focus on internal validity, whereas later efforts may concentrate on external validity (Moras, 1998). In yet later stages, researchers may focus on issues of cost effectiveness and the ease of dissemination of a treatment to a wide audience of clinicians, settings, and consumers (see Baker, McFall, & Shoham, 2009).

Research Designs Both single case experiments (Barlow & Hersen, 1973) and randomized controlled trials (RCTs) allow close controls on the internal validity of research. These experimental methods, properly conducted, permit the investigator to draw causal inferences about the efficacy of a treatment. Because RCTs are far more common, we focus on this approach here. In RCTs, patients are randomly assigned to the treatment of interest or to one or more control groups or alternative treatments. Without random assignment, it would be impossible to know whether any differences observed among treatments were due to the treatments or to systematic pre-existing differences among patients. Control groups are used to determine whether any improvement with treatment might be due simply to effects such as the passage of time, the assessment procedures (e.g., talking at length about your problems with the clinical assessor), or making a decision to do something about the problem. The simplest control group is a waiting list condition where clients receive the treatment under investigation after a prescribed delay during which they participate in assessment. Usually the delay period is for the length of the treatment, but for severely distressed clients, this may be shortened for ethical reasons. Waiting list conditions are similar to the delay many clients undergo when they seek treatment at clinics, but there are drawbacks, including the ethical concern about postponing treatment and the fact that a number of clients will seek treatment elsewhere or lose their resolve to undergo treatment during the waiting period.

Waiting list control conditions permit the researcher to draw an important conclusion that may be all many consumers want to know: Does the treatment work better than no treatment? However, such control groups are unsatisfactory if the researcher or consumer wants to know whether the treatment adds to the so-called nonspecific effects of psychotherapy. That is, does the treatment work because of placebo effects, the belief in and hope of change that occur when people think they are getting treatment? Does the treatment work simply because the clients get to talk with a sympathetic person about their problems? If so, there is nothing that is specifically helpful about this treatment versus any other, and no justification for having highly trained psychotherapists versus counselors with less training deliver the treatment. *Placebo control conditions* (e.g., a pill placebo combined with regular brief meetings with a supportive psychiatrist) and *alternative therapy conditions* (e.g., supportive counseling) provide for another level of analysis than the waiting list control group. However, these control groups are also not without their problems. For ethical reasons, patients given a placebo receive the real treatment at the end of the treatment period, but many may drop out before that time to seek other treatment if it is available. If patients who received supportive counseling are not satisfied with their improvement in treatment, they may get additional treatment during the follow-up period, which precludes controlled assessment of long-term outcome.

Finally, the researcher may choose to compare the treatment of interest to various medications or to other forms of psychotherapy known or believed to be efficacious for the types of problems being treated. Comparisons of two or more types of psychotherapy are less common at this point than is research designed to develop and test the efficacy of one treatment. For many disorders there are not yet two or more psychological approaches that have each been determined to yield satisfactory treatment benefits relative to waiting list or placebo control groups, making such comparisons premature. However, comparisons of psychotherapy with medication and with the combination of medication and psychotherapy are frequently conducted.

Typically, in the development of a particular treatment, the research follows this course: First, an uncontrolled study in which the effects of the treatment are tested by comparing clients' status before and after treatment; second, comparisons with waiting list control groups; third, comparisons with placebo or basic counseling; and fourth, comparisons with medication or other efficacious psychotherapies. At each stage, the comparisons become more stringent, and the number of participants required for the research increases. Statistically significant effects between a treatment and waiting list control group may be obtained with 25 patients per condition, but comparisons between two efficacious treatments or between psychotherapy and a medication will require 50 or more patients per group to be able to detect differences in efficacy that are clinically important (Kazdin & Bass, 1989). Consider that the typical psychotherapy medication trial includes the following conditions: psychotherapy plus placebo, medication alone, medication plus psychotherapy, pill placebo plus clinical management (meetings with a supportive, encouraging psychiatrist for medication monitoring), and perhaps psychotherapy without pill placebo (to control for any effects of the patients' thinking they are on medication, which may have a positive or negative impact). For such a trial, then, 250 or more patients are required, often requiring

multiple treatment sites to obtain enough participants. As a result, these studies are very expensive to mount and virtually impossible to conduct without major government grants. For this reason, psychotherapy research proceeds more slowly than pharmacotherapy research, which is funded largely by the pharmaceutical industry. There is, of course, no psychotherapy industry to fund research in this area.

Defining the Intervention For pharmacotherapy researchers, it is fairly easy to define the treatment intervention: Provide the drug (or pill placebo identical in appearance) in the proper therapeutic dosage and monitor its use and side effects. For psychotherapy researchers, the process is more difficult. The researcher must carefully describe the treatment approaches being studied such that the study therapists and the ultimate readers of the study (clinicians and other researchers) know exactly what treatment was tested. This is accomplished by writing a treatment manual that details the principles and procedures of the treatment. How structured such manuals are tends to vary with the sort of therapy tested. For example, cognitive-behavioral treatment manuals are often highly detailed, frequently with session-by-session outlines. In contrast, manuals for psychodynamic psychotherapy may rely on broader brush strokes, describing treatment principles and the sorts of conditions under which the therapist chooses one possible intervention over another, such as when to interpret the patient's behavior versus when to be supportive. Manuals often become more and more highly detailed across research trials and end up being book length.

Manuals are critical to treatment research in that they permit dissemination of efficacious treatments (others can read how the treatments were done) and because they provide the operational definition of the treatment. Labels for treatments are often not sufficiently informative. For example, there are many versions of psychodynamic treatment, some of which are radically different from others. Just which dynamic treatment approaches were followed in this study? Using manuals also tightens the internal validity of the study by reducing unwanted experimental variance. When therapists have a treatment manual to follow, there is less variability in the outcomes of the individual therapists employed in the treatment trial (Crits-Christoph et al, 1991).

It is not enough that the study therapists are provided with a treatment manual. They must also be trained in delivering this treatment, and then monitored during the study for their adherence to the treatment manual and their competence in carrying out the treatment (Perepletchikova, 2011). Trained raters watch videotapes of treatment sessions and use rating scales to determine whether therapists are following the key procedures of the treatment and are avoiding mixing in other treatments that are not to be included in this study. Expert raters also watch these tapes to code the therapists' skill. Ensuring that the therapists delivered the treatment in at least a minimally competent way is vital to the eventual acceptance of the study and the treatment. Readers can easily reject findings counter to their own beliefs if they can discount the results because therapist competence and adherence were not demonstrated.

Selecting the Treatment Sample and Assessing Outcome An early step in designing a treatment outcome study is selecting the target population and the sample. Again, an operational definition is required. Earlier in the history of psychotherapy research, the description of the study sample was often quite broad, for example, neurotic outpatients. The present practice is to define study samples more precisely, usually with a specific diagnosis from the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013), such as borderline personality disorder. However, samples may also be defined on the basis of characteristics other than formal diagnoses. For example, Markman and colleagues developed a program designed to prevent marital dissatisfaction and divorce and targeted unselected engaged couples (Markman, Renick, Floyd, Stanley, & Clements, 1993). Others have created programs to reduce drinking and problems associated with alcohol use in college students who drink heavily but do not meet the diagnostic criteria for an alcohol use disorder (e.g., Carey, Carey, Maisto, & Henson, 2006).

The researcher also determines the study's exclusion criteria. Who is omitted? Thus, for example, acutely suicidal patients who require immediate hospitalization are excluded from most outpatient studies for ethical reasons. Alcohol and substance dependent patients are usually excluded from trials for treatment of other disorders they may have until their chemical dependencies have been addressed, because these problems tend to interfere with progress in other areas. In the intake process, the typical RCT screens out about two-thirds of the people who initially contacted the project (Westen & Morrison, 2001). This means that for the 250-patient medication versus psychotherapy RCT described earlier, over 750 potential participants would need to be screened for the study.

To determine who enters the study and who improves in treatment, reliable and valid means of assessing the patients' problems and symptom severity are required. Questionnaire measures are often used to assess change in treatment, but interviews are commonly employed to make initial diagnoses as well as to monitor change over time. Interviewers must be carefully trained to be reliable in their assessments, and their reliability across the course of the study must be monitored by another trained rater who is uninformed as to the initial diagnosis or severity rating.

Empirically Validated Treatments Several of the chapters in Part II of this volume mention the terms

"empirically validated treatments" or "empirically supported treatments" (ESTs). In general, these terms are used interchangeably to refer to treatments that have met the standards set by one or more groups who have reviewed the psychotherapy literature to identify treatments that work for particular disorders or presenting problems (see Chambless & Ollendick, 2001, for a review). These efforts were initiated in the United States by Division 12 (Society of Clinical Psychology) of the American Psychological Association. Division 12's Task Force on the Promotion and Dissemination of Psychological Procedures (later the Committee on Science and Practice) defined ESTs as treatments that were probably efficacious or efficacious. Probably efficacious treatments are those that have been found to be superior to waiting list control groups in two or more studies, that have been found to be superior to another treatment in at least one study, or that have been found to be superior to another treatment in multiple studies but only tested by one research group. Efficacious treatments were defined as those that have proved more beneficial than placebo conditions or alternative treatments by more than one research group. (These are abbreviated criteria. A complete description can be found in Chambless et al., 1998.) In both cases, when multiple studies were available, the preponderance of the most exacting evidence had to favor the treatment's efficacy. Moreover, treatments had to be tested according to the methods described in this section for rigorous psychotherapy research. See DeRubeis and Crits-Christoph (1998) for examples of the efficacy evidence for a number of treatments of adult disorders.

At the time of their 2001 review, Chambless and Ollendick noted that the various review groups tackling the psychotherapy literature had identified 108 ESTs for adults and 37 for children. Although an updated comprehensive review has not yet been published, APA's Division 12 maintains an updated, online version of the list of empirically supported treatments, listing treatments with "strong," "modest," and "controversial" research support (www.psychologicaltreatments.org). With support from the National Institute for Health and Clinical Excellence (UK), an additional website has been developed to help consumers and psychotherapists access information about ESTs (www.nice.org.uk). The identification of ESTs may be categorized as a part of the *evidence-based practice* movement. According to the principles of evidence-based practice, clinicians are encouraged to integrate the best research evidence regarding possible treatment of a patient with their clinical expertise and consideration of the patient's characteristics and values (Levant, 2005; Sackett, Straus, Richardson, Rosenberg, & Haynes, 2000). Thus, evidence-based practice includes not only EST research but also any other sort of evidence the practitioner might bring to bear on treatment decisions; for example, knowledge of the importance of building positive expectations for change and of developing a strong working alliance with the client. Compilations of ESTs are intended to serve as quick guides for clinicians, students, and educators who want to learn more about how to effectively treat a variety of disorders, but who do not have time or the expertise to conduct extensive literature reviews themselves. Presently, the Commission on Accreditation of the American Psychological Association, which reviews and accredits doctoral programs and internships in professional psychology in the United States, requires that at least some of a student's didactic and practical training be devoted to the study of ESTs (Office of Program Consultation and Accreditation, 2013). The newly developed Psychological Clinical Science Accrediting System for clinical psychology programs places even heavier emphasis on training in empirically supported treatments for doctoral students (Baker et al., 2009; www.pcsas.org).

A Costly Endeavor We have covered but a partial list of the requirements for conducting a sound psychotherapy research trial (see Chambless & Hollon, 2012, for a more complete description). However, this should be sufficient for the reader to see that the psychotherapy researcher's efforts must be exhaustive and are exhausting! In addition, such research requires a great deal of patience. Psychotherapy trials often take 5 years to complete the evaluation of all patients' progress from the beginning to the end of treatment and another 2 years to complete assessment of treatment response at follow-up. The amount of time required to treat each participant is great, ranging from about 12 sessions of treatment of at least 50 minutes each to a year or more of treatment for severe psychopathology such as borderline personality disorder. The amount of effort that goes into treating and assessing each patient and the process of treatment and the requirements for highly trained personnel to carry out these activities makes such research a very expensive endeavor. Moreover, one study is not sufficient to demonstrate convincingly that a treatment is efficacious. Multiple replications, especially by other researchers not intimately involved in the development of the treatment, are essential.

Effectiveness Research Once a treatment's efficacy has been established, it is time to export or disseminate the treatment to the community to test whether it works outside of the hot house of the research clinic, that is, to conduct effectiveness research. Such tests include determining whether treatments work with the sorts of patients who may not have participated in university-based research trials (e.g., less-educated patients), in primary care practices and community mental health centers, when provided by less highly trained personnel, and so forth. Because the focus of such research is on external validity or generalization beyond the research clinic, the designs are often less tightly controlled and may be simple pretest-posttest studies with no control group. In such cases, benchmarking may be used. That is, the results of the effectiveness study

may be compared to those in published efficacy research. It typically takes many years to move from the initial uncontrolled pilot studies, through efficacy research, to the effectiveness stage of research. However, demonstrating effectiveness of research proven efficacious in RCTs is now a major thrust of the National Institute of Mental Health, and this has served to move research more quickly to tests of effectiveness. See Baker et al. (2009) for examples of ESTs with established effectiveness.

Given that psychotherapy research is difficult to conduct and very costly, why do it? We turn to this question next.

The Case for Psychotherapy Research

First, Do No Harm The Food and Drug Administration requires rigorous testing of medications before they are made available to the public through their physicians. The rationale is that it is important to determine before dissemination whether the drug has harmful effects, and whether its beneficial effects outweigh any harmful effects observed. There is no formal equivalent for testing new psychological interventions. Rather, psychologists' ethical code (American Psychological Association, 2010) exhorts them to let clients know whether a treatment is experimental and to avoid the use of treatments that might be harmful. That is, psychologists are largely expected to police themselves, although occasionally a state licensing board may take action against a psychologist using bogus treatments for which he or she has made unsubstantiated claims.

Some would argue that it is unnecessary to test psychological procedures before their widespread use because psychotherapy is undoubtedly beneficial or, at worst, innocuous. We take issue with this claim on two grounds. First, psychotherapy can be harmful. For example, Dishion, McCord, and Poulin (1999) found that peer-group interventions for delinquent teens increased adolescent problem behavior and negative life outcomes in adulthood, compared with control conditions. This research project, and others like it, was critical in determining that a seemingly logical treatment was actually harmful. Bootzin and Bailey (2005) and Lilienfeld (2007) provide other examples of treatments that may produce more harm than good, such as Critical Incident Stress Debriefing, a commonly used method of immediate (1 week post-trauma) crisis counseling designed to combat posttraumatic stress disorder (PTSD). In some studies participants in this intervention this approach have had more, rather than fewer, PTSD symptoms postintervention than participants in control groups.

Second, an ineffective treatment is in itself harmful. An example is facilitated communication for autistic children, which ostensibly enabled children with developmental disabilities to communicate using a computer keyboard and to demonstrate that they were far more cognitively capable than was apparent. This intervention generated great excitement until controlled research repeatedly demonstrated that the results either did not occur or were created, in all likelihood unknowingly, by the facilitator (e.g., Herbert, Sharp, & Gaudiano, 2002). Without this research, many parents would have continued to have their hopes cruelly and falsely raised, and resources for the treatment of these severely disabled children would have continued to be diverted from programs that might be genuinely helpful and assigned instead to a bogus intervention.

Some disorders worsen without effective treatment. Under these circumstances, providing a patient with an ineffective treatment when effective ones exist is not innocuous. For example, we have seen patients with severe anxiety disorders who had years, even decades of ineffective treatment while they lost their jobs, their friends, their avocations, and their life savings. When the efficacy of cognitive-behavioral treatments for such disorders has been repeatedly demonstrated (see Williams, Chapter 9 in this volume), withholding such treatment in the face of patient deterioration is not a neutral act. The ethics code of the American Psychological Association (2010) requires that psychologists refer clients who are not improving in their care for other treatment. Without psychotherapy research, clinicians cannot know what effective treatments are available.

Psychotherapists as Decision Makers Others claim that psychotherapy research is unnecessary because practicing clinicians know best how to treat their patients based on their clinical training, expertise, and lore (e.g., Silver, 2001; Silverman, 1991). There is a surprising dearth of research on psychotherapists' decision making. However, in a survey of psychologists in private practice, clinicians reported that they were most likely to rely on their clinical experiences when making treatment decisions (Stewart & Chambless, 2007). How accurate is clinical judgment? There is a large body of research on clinicians' decision making regarding psychological assessments, much of which suggests clinical experience does not increase practitioners' ability to reach valid conclusions if they rely on clinical judgment rather than empirical data in the assessment process (see Garb, Lilienfield, and Fowler, Chapter 7 in this volume). Making a decision about how to treat a new client or a client who is not responding to the present treatment plan is the result of an assessment process and is likely subject to the same errors in judgment if clinical experience is the only guide.

Kadden, Cooney, Getter, and Litt (1989) asked therapists of inpatients with alcohol dependence to predict which of two aftercare treatment programs would be better for their patients. The patients were randomly assigned to one of two treatments, and the authors found that patient data (e.g., severity of psychopathology) predicted which treatment would work better for which patients. In contrast, the inpatient therapists were no better than chance at predicting which treatments would work for which of their patients, despite their extensive

contact with these patients—much more contact than an outpatient therapist usually has before embarking upon a treatment plan. Schulte, Kunzel, Pepping, and Schulte-Bahrenberg (1992) randomly assigned patients with phobias to standardized treatment with exposure or to an individualized program of cognitive-behavior therapy of the therapist's device. The same therapists participated in both conditions. Therapists in this study were significantly more effective in treating patients with phobic disorders when they were constrained to use the EST of choice (exposure) than they were when they were allowed to devise their own treatment plan, which plans typically included less exposure than the mandated treatment. These therapists, then, although experienced in treating phobic disorders, thought they could do better than the research supported efficacious treatment by developing a treatment plan for their clients. They were wrong. Much more research is needed to test whether clinicians are more effective when they follow the treatment recommended by the research literature rather than when they follow their clinical intuitions, but so far the evidence indicates they should follow the data. This may be a distasteful thought to many practitioners (e.g., Silverman, 1991).

Why might clinical experience be a less accurate guide to treatment decisions than psychotherapists tend to believe? A variety of forces converge to make decisions about psychotherapy based on unsystematic observation vulnerable to error (Dawes, Faust, & Meehl, 1989). The amount of information that must be processed in psychotherapy is enormous and taxes the cognitive capacity of humans as information processors. This leaves clinicians open to the many cognitive biases that influence the attention, selection, and interpretation of feedback they may receive. It is easy for even the most well meaning of us to deceive ourselves under such circumstances.

An historical example is known to many a student of abnormal psychology. In the early 1770s, Franz Mesmer developed a technique based on his theory of animal magnetism, which posited the existence of palliative magnetic fluids in nature. With a combination of light, music, and chanting, Mesmer produced *mesmerism*, which allegedly moved the magnetic fluids, curing the body and mind of diseases (Pattie, 1994). Mesmer enjoyed great success and popularity in Europe. What may be less likely to be covered in undergraduate texts is the debunking of mesmerism. King Louis XVI commissioned the French Academy of Sciences, including Benjamin Franklin, then resident in Paris, to investigate Mesmer and his therapeutics. Using an early and literal example of the single blind study (wherein patients do not know which treatment they are receiving), the commissioners blindfolded patients so that they would not know whether they were being mesmerized. They discovered that patients' responses depended not on the treatment procedure, but on whether they believed they had been mesmerized; in short, on the placebo effect. Franklin concluded, "Some think it will put an end to Mesmerism, but there is a wonderful deal of credulity in the world, and deceptions as absurd have supported themselves for ages" (cited by Isaacson, 2004, p. 427). The case of mesmerism illustrates the dangers of reliance upon uncontrolled observations of practitioners and patients.

Thinking that a treatment works when it does not is one kind of mistake a therapist may make, but what about the other kind, when therapists think a treatment may be harmful when it is actually beneficial? Prolonged exposure to images, thoughts, and other stimuli associated with the trauma has by far the greatest evidentiary base for treatment of posttraumatic stress disorder (e.g., Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). However, despite its strong endorsement by experts, prolonged exposure is underutilized by clinicians in the field, who cite a variety of reasons for their reluctance, none of which are empirically based (Becker, Zayfert, & Anderson, 2004). In particular, clinicians feared that prolonged exposure would lead to symptom worsening. In contrast, the available evidence shows that patients who receive exposure therapy experience better outcomes and no reliable worsening of symptoms compared to patients in waiting list conditions and other treatments (Jayawickreme et al., 2014).

Garb and colleagues (Chapter 7 in this volume) describe a number of cognitive biases that may affect clinicians' decision making in the assessment process. These also come into play in practitioners' assessments of treatment efficacy. Psychotherapists are in a particularly difficult situation, in that unless they make concerted efforts to systematically collect information on their practice, they rarely receive clearcut feedback on their outcomes. Patients often leave without explanation—some because they are doing better, and some because they are dissatisfied. Others may leave saying they are feeling better because they are uncomfortable with telling their therapist that they are disgruntled. Moreover, many factors contribute to patients' improvement or deterioration, only one of which is the patient's psychotherapy, making causal attributions difficult even when it is clear that a patient is doing well or not. Such an information vacuum is fertile ground for cognitive errors. We will consider a few examples here.

First, many problems for which people seek treatment are subject to so-called *spontaneous remission*. That is to say, clients get better because the disorder has run its course or because other forces in their environment or their own efforts have led to improvement. The waiting list control group is designed to detect such improvement and to prevent researchers from concluding that a treatment is efficacious when change would have occurred without the intervention. Practitioners, however, have no control group and thus are subject to the *illusory correlation*, believing that their interventions led to the change when, in fact, there was no causal connection. In addition,

once clinicians, like anyone else, begin to believe that an intervention is beneficial for a certain sort of client, they may tend to look for evidence that supports their hypothesis, and ignore evidence to the contrary. This is called the *myside bias.*

The operation of the *availability heuristic* means that certain types of memory errors are likely. When clinicians search their memory bank for an intervention that was helpful in the past for a particular situation, they may recall a salient example of a time the intervention was associated with dramatic improvement and forget all the times it did not help. The availability heuristic may also account for the *clinician's illusion*. Cohen and Cohen (1984) coined this term to describe psychotherapists' beliefs that clinical disorders are more severe and enduring than epidemiological evidence indicates. Vessey, Howard, Lueger, Kächele, and Mergenthaler (1994) demonstrated that most clients stay in psychotherapy for 6 months or less, but that those clients who do remain long-term take up an increasing part of the therapist's practice as they accrue. Thus, it is easy for practitioners to erroneously conclude that most patients who enter treatment are long-term and that psychopathology is generally unremitting because these are the cases that readily come to mind. Absent systematic records, it appears that practitioners cannot accurately calculate the characteristics of their caseloads (Knesper, Pagnucco, & Wheeler, 1985). Yet how long clients stay in treatment seems relatively simple, discrete information compared with the assessment of clients' response to treatment interventions.

Do psychotherapists really know how well their patients are doing? Perhaps not. Hannan et al. (2005) compared the judgment of clinicians in a college counseling center with a research-derived algorithm for prediction of treatment failure, and found that clinicians were quite poor at predicting outcome, whereas the actuarial method worked very well. Hannan and colleagues noted that therapists rarely predicted deterioration even though it occurred in 42 of 550 patients. In contrast, the empirical method identified all of the patients who would become reliably worse, although it also generated numerous false-positive results, that is a prediction of deterioration when deterioration did not occur. Nevertheless, the false alarms did have poorer outcomes than those not identified as likely treatment failures. These data speak to the importance of collecting data in clinical practice rather than relying on unsystematic observation.

The purpose of this discussion is not to insinuate that clinicians are incompetent or unresponsive to facts, but rather to make the point that clinicians are subject to the same illusions, biases, and memory distortions as anyone else, and that they deal with very complex data. Nor is this discussion meant to downplay the importance of clinical lore and intuition. Clinical experience is a rich source of hypotheses about disorders and their treatments that, when submitted to experimental testing, have led to a variety of efficacious treatments. Nonetheless, the reliance of many clinicians on clinical judgment is exceedingly problematic in light of the literature on the superiority of data-based predictions over clinical judgment.

Rejection of Psychotherapy Research

As noted previously, some practitioners reject the importance of psychotherapy research for their practice, believing clinical expertise is all that is required. Others object to specific aspects of EST research, the predominant paradigm at present. We will consider a few of those objections here (see also Chambless & Ollendick, 2001; Norcross, Beutler, & Levant, 2006)

Nonspecific Factors Rule According to some authors (e.g., Ahn & Wampold, 2001; Wampold et al., 1997), the only important factors in psychotherapy outcome are so-called nonspecific factors that are common to all treatments. These include hope, expectation of change, and a good relationship with the therapist. A great deal of research bears witness to the importance of such factors in treatment outcome (Orlinsky, Grawe, & Parks, 1994), and it is a rare psychologist who would argue that better treatment results are obtained by having an uncaring attitude toward one's clients and communicating to them that therapy is unlikely to work. The controversy, therefore, is not over the importance of the nonspecific factors but rather concerns whether different treatment interventions have an impact above and beyond these factors. In our view, this will depend on whether particular treatments rely on specific and effective treatment interventions attuned to the psychopathology of a disorder. For example, in a recent meta-analysis, Siev and Chambless (2007) demonstrated that cognitive-behavior therapy is significantly more effective for panic disorder than are relaxation-based therapies, whereas this is not the case for generalized anxiety disorder. This difference may arise because the psychopathology of panic disorder is now well understood, and effective treatment interventions have been carefully designed to address it. Treatment of generalized anxiety disorder leads to significant change, but the degree to which patients improve remains unsatisfactory in that fewer than half change to a clinically significant degree. Accordingly, researchers in that area are reconsidering the best approach based on having taken another look at core features of the disorder. For example, Dugas, Schwartz and Francis (2004) have highlighted the importance of focusing on these patients' intolerance of uncertainty and have reported very promising preliminary results of treatment that includes such a focus (Dugas & Koerner, 2005).

Adherents to the nonspecific factors model of therapy efficacy might assert that when cognitive-behavior therapy is found to be more effective than another treatment such as relaxation training, as in the Siev and Chambless (2007) meta-analysis, it must be because the nonspecific

components of cognitive-behavior therapy were stronger than in the comparison treatment. Unfortunately, researchers in cognitive-behavior therapy have not always included measures of nonspecific factors such as the therapeutic relationship. When they have, the superiority of cognitive-behavior therapy to applied relaxation training is evident despite their equivalence on expectations of improvement and the quality of the therapeutic relationship (e.g., Clark et al., 2006).

Treatment Manuals Are Rigid and Rob Therapists of Creativity Psychotherapists differ as to whether they consider psychotherapy to be an art, a science, or a mixture of those things. Those who believe therapy is an art have expressed contempt for the constraint implied in treatment that is guided by a manual, a necessary component of EST research (Silverman, 1996). Such practitioners often indicate that they approach each client as an individual and avoid being guided by nomothetic treatment research as channeled through treatment manuals. There seem to be two misconceptions at play here. One is that the practitioner really can approach each client *de novo*, without any ideas about what might be helpful for him or her. After all, if there is nothing a therapist can be taught about how to treat particular clients, then there is no point to training in psychotherapy. Few are likely to agree this is the case. Rather, therapists likely draw on informal observations they learned from their supervisors or conclude from their own clinical experience to be helpful with a particular kind of case. As soon as a psychotherapist says, "In my experience this sort of client is best approached in this way," she or he is making a probabilistic statement. We would argue that there is great value in clearly articulating these clinical observations and testing their validity. This is precisely what is done when a treatment manual is constructed, and an RCT is conducted.

The second misconception is the idea that psychotherapists who use ESTs must follow treatment manuals in a robotic fashion such that they are robbed of their therapeutic creativity and not allowed to use their skills. Certainly some manuals are structured more than others, but it is impossible for the thousands of decisions that each therapist must make to be codified. Although manuals provide extensive descriptions of specific procedures within a treatment, flexibility is inherently necessary so that the therapist can respond to the patient and maintain a good therapeutic relationship (Kendall, Chu, Gifford, Hayes, & Nauta, 1998). Nonetheless, the goal is to detail as many of the treatment decisions as possible so that therapists who are not highly expert in the treatment of a particular disorder are able to learn from the experience of experts to carry out the treatment competently.

ESTs' Efficacy Does Not Generalize to Clinical Practice

Characteristics of Research Participants The claim is often made that ESTs will not work in clinical practice settings, usually because the clients in practice settings are purported to be more severe or to have more comorbid conditions than clients treated in research studies (e.g., Silberschatz, in Persons & Silberschatz, 1998). Westen and Morrison (2001) estimated that the average inclusion rate for studies of depression, panic disorder, and generalized anxiety disorder ranged from 32% to 36%. They inferred from these data that the more difficult cases were being excluded from research trials and thus the efficacy of ESTs for these disorders was likely overblown. Stirman and colleagues have conducted several studies challenging this conclusion.

In the first of these studies, Stirman, DeRubeis, Crits-Christoph, and Brody (2003) developed the concept of the virtual clinic. That is, they hypothesized that many patients might be excluded from one RCT because they did not have the disorder in question, whereas they would fit inclusion criteria for another RCT being run down the virtual hallway by another researcher. For example, the patient may have applied for treatment of panic disorder and was excluded because she had a primary diagnosis of social phobia. However, she would be eligible for a different RCT in the virtual clinic, the study for social phobia. To test this idea, these authors mapped information from charts of patients seeking treatment through a managed care program to the inclusion and exclusion criteria of nearly 100 RCTs for individual therapies. Of those patients who had diagnoses represented in the RCT literature, 80% of these patients would have been eligible for at least one study. Patients who failed to match to studies in the existing literature mostly would have been excluded not because their problems were too complex but because they failed to meet minimum severity criteria.

Another charge is that patients with comorbid conditions are excluded from RCTs. Because people with comorbid conditions are more likely to receive treatment than those with a single disorder (Kessler et al., 1994), it would be difficult indeed to conduct RCT research if this were the case. Nonetheless, the psychotherapy researcher must make a hierarchy of disorders so that the problem most in need of treatment is addressed. In a second study, using records of patients who had been screened out of RCTs, Stirman, DeRubeis, Crits-Christoph, and Rothman (2005) found that all patients who had a primary diagnosis of a disorder represented in the RCT literature would have been included in at least one RCT, regardless of their Axis I or II comorbidity.

Transportability of ESTs to Clinical Settings A second claim regarding generalization is that ESTs will not work once taken from their ivory tower settings into the real world of clinical practice. This is the domain of effectiveness research, and there is a burgeoning literature on the effectiveness of ESTs. Examples include research demonstrating the benefits of ESTs with ethnic minority clients (e.g., Carter, Sbrocco, Gore, Marin, & Lewis, 2003), ESTs administered by clinic personnel instead of highly trained

research therapists (e.g., Foa et al., 2005), ESTs in community mental health and primary care settings (Bedi et al., 2000; Merrill, Tolbert, & Wade, 2003), and ESTs with patients who either have not been asked to agree to random assignment to treatment (e.g., Juster, Heimberg, & Engelberg, 1995) or have refused to be randomized (e.g., Stiles et al., 2006). In one meta-analysis, Stewart and Chambless (2009) synthesized 56 effectiveness studies of cognitive-behavioral therapy (CBT) for adult anxiety disorders tested in less controlled, real-world circumstances. They found that this approach is effective in clinically representative conditions and that the results from effectiveness studies are in the range of those obtained in major efficacy trials. While more research on effectiveness is clearly needed, and meta-analyses of effectiveness studies for other disorders have yet to be completed, based on the available data, the arguments that ESTs do not generalize to clinical settings and the clients seen therein do not hold up.

EST Lists Are Unfair Psychologists of some theoretical orientations believe that the EST approach is unfair to their preferred psychotherapy method and argue that, because not all treatments have been tested, the playing field is not level (Stiles et al., 2006). This represents some confusion about what the designation of a treatment as an EST means. That a treatment is termed empirically supported does not constitute a claim that it has been shown to be superior to other treatments. Rather, it has been shown to be efficacious in comparison with control conditions, which might or might not include another type of psychotherapy. Moreover, an untested treatment may be effective; its benefits are simply unknown. If one prefers a treatment with efficacy evidence (and, admittedly, we do), then ESTs are superior to treatments without such evidence, but this is a different sort of superiority.

Cognitive-behavioral researchers have been at the forefront of developing treatment manuals and protocols, and the preponderance of EST research has been in cognitive-behavioral treatments. This is not surprising, given the traditional emphasis of cognitive-behavioral treatments on specifying procedures and identifying symptoms and treatment goals. Although researchers of other orientations, for example, psychodynamic psychotherapy, have also produced manuals (e.g., Milrod, Busch, Cooper, & Shapiro, 1997), the research on these interventions is limited compared with research on CBT. In part, this might account for Stewart and Chambless's (2007) findings that private practitioners with a psychodynamic orientation have less positive attitudes about the utility of EST-type treatment research for their practice than CBT and eclectic therapists. In the decades since the publication of the first Division 12 EST Task Force report (Task Force on Promotion and Dissemination of Psychological Procedures, 1995), research has provided some support for the efficacy of short-term psychodynamic therapy. The Division 12 Committee on Science and Practice currently lists two forms of short-term psychodynamic therapy as possibly efficacious: short-term psychodynamic psychotherapy for depression and short-term psychoanalytic therapy for panic disorder (www.psychologicaltreatments.org). More short-term dynamic treatments will undoubtedly appear on lists of ESTs in the future as they are updated. This may make the EST concept more acceptable to psychologists of this orientation.

The prospect for rigorously controlled outcome research on long-term psychodynamic psychotherapy is less hopeful. Certainly, patients cannot ethically be kept in control conditions for years. Accordingly, the remaining approach to controlled research (see section on Research Designs, above) would require very large studies in which long-term dynamic psychotherapy is compared with another active treatment. Such research would be extremely expensive, and few patients or public health systems would be able to afford access to this treatment even if it proved effective, making the public health significance low. In addition, drop-out rates in long-term treatment research are very high, threatening the internal validity of the research. Perhaps for these reasons, funding agencies are reluctant to support research on long-term therapy. As a result, psychodynamic therapists who focus on long-term treatment may rely upon case reports based on clinical experience and upon uncontrolled research (e.g., Leichsenring & Rabung, 2008) to inform the theory and practice of this form of therapy.

Conclusions

We began this chapter with a statement that psychopathology research serves psychotherapy research. We end with the observation that psychotherapy research serves psychopathology research. That is, there is a reciprocal feedback loop between these two forms of research that is mutually beneficial. For example, cognitive theory of panic disorder stresses the importance of frightening misinterpretations of bodily sensations to the development and maintenance of this problem (Clark, 1986). Treatment aimed at changing these cognitions has proved to be highly effective (see Siev & Chambless, 2007 for a meta-analytic review), and, closing the feedback loop, Teachman, Marker, and Clerkin (2010) have determined that changing beliefs about bodily sensations is critical to the efficacy of cognitive therapy for panic disorder. Thus, psychopathology research informs treatment, and treatment research informs the understanding of the psychopathology of this disorder.

The reciprocal feedback loop can also identify when something is wrong with either the theory or the treatment. For example, it was originally postulated that the "sine qua non of marriage was the quid pro quo" (see Gottman, 1998, p. 181). In other words, in happy couples there was an equitable exchange of positive behaviors. This theory led to the use of contingency contracting in marital behavioral therapy, wherein spouses were trained

to contract for a desired behavior on the part of their partner by agreeing to reciprocate by doing something that their partner wanted. However, systematic research on couples' interactions revealed that this was precisely the wrong thing to do. In fact, insistence on the equitable exchange of behaviors actually characterized unhappy marriages. Thus, the increased knowledge about marital satisfaction led to improvements in marital therapy, namely the removal of contingency contracting as a basic intervention.

On the basis of these and many other such examples, we argue that progress in applied clinical psychology occurs when treatments are based on a solid understanding of the psychopathology of a given disorder, and when those treatments are rigorously evaluated not only for their efficacy and effectiveness but also for the causal factors underlying their benefits. In this fashion, treatments may be honed more precisely to concentrate on the critical elements in outcome, making psychotherapeutic interventions more efficient and more effective.

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Part II

Common Problems of Adulthood

9

Anxiety Disorders, Obsessive-Compulsive, and Related Disorders

S. Lloyd Williams

To understand anxiety disorders and obsessive-compulsive disorders one must understand their underlying theory, which is that severe mental problems are medical illnesses that express psychopathology (Robins & Guze, 1970). The formal principles and statistical standards that define psychopathology and mental disorder are neither well specified nor widely accepted (Maddux, Goselin, & Winstead, Chapter 1 in this volume; Widiger, Chapter 6 in this volume).

For more than 60 years, mental illnesses have been defined in research and practice by the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-I; American Psychiatric Association, 1952) and its subsequent revisions (DSM-II, 1968; DSM-III, 1980; DSM-III-R, 1987; DSM-IV, 1994; DSM-IV-TR, 2000; DSM-5, 2013), and by the similarly conceived *International Classification of Diseases* (ICD) now in its 10th edition (World Health Organization, 1992). These manuals group proposed mental disorders into broader classes. The disorders named in the title of this chapter were psychoneurotic disorders in DSM-I, neurotic disorders in DSM-II, and anxiety disorders in DSM-III through DSM-IV-TR.

The DSM-5 removed obsessive-compulsive disorder from the anxiety disorders to a new separate disorder class (described below), and removed post-traumatic stress disorder and acute stress disorder to a new class called trauma and stress-related disorders, now discussed in Chapter 10 of this volume. This chapter takes as its scope the psychosocial problems that define most DSM-5 anxiety disorders, plus obsessive-compulsive disorder, but it does not address some newly designated specific anxiety disorders and obsessive-compulsive and related disorders such as selective mutism, trichotillomania, and separation anxiety disorder.

Despite this title, I reject the concept of mental disorder and its subsidiary concepts of anxiety disorders and obsessive-compulsive disorders without reservation. Far more important than DSM's latest reorganization of its proposed mental disorders is that its basic concept of mental disorder has never changed. The very point of DSM-III in 1980 was to salvage the psychopathology concept of DSM-II by defining its proposed mental disorders more precisely. Although DSM-III introduced many changes in details and a few changes in disorder classes, it made essentially no change to the core DSM concept of mental disorder itself (Carson, 1991). That legacy has now been passed on intact in DSM-5, despite its creators' intention, expressed often since DSM-IV-TR, to integrate into DSM-5 mental measurements based on graduated dimensions (Kupfer, First, & Regier, 2002; Regier, Narrow, Kuhl & Kupfer, 2009). That bold intention resulted in less than bold action: DSM-5 suggests optional dimensional measures but it continues to require DSM mental disorder diagnoses as before, and thus it did not integrate dimensions into disorders.

The present chapter maintains, as before (Williams, 2012), that people's mental problems are painfully real but mental disorders and psychopathology are scientific illusions. Behavioral scientists are increasingly observing that DSM has failed and that alternatives are needed (e.g., Hofmann, 2014; Insel, 2013; Smith, McCarthy, & Zapolski, 2009). But with few exceptions they continue to accept DSM's ideological core: the concepts of mental disorder and psychopathology. I dissent by holding that all human mental states and behaviors, including all severe mental problems, are best measured by the psychometric principle of mental variability (e.g., Nunnally & Bernstein, 1993; Spearman, 1904), which has no place within it for

mental pathology. In short, DSM fails empirically because mental disorder is an entirely false idea.

Severe anxiety, obsessions, and compulsions certainly exist. Every mental disorder is defined mainly by one or a few such very real psychosocial problems, the disorder's so-called "symptoms;" for example, panic disorder by its main symptom panic attacks, obsessive-compulsive disorder by obsessions and compulsions, and social anxiety disorder by social fears. The symptoms exist in painful abundance but the *disorders* said to produce the symptoms have never been shown to exist.

Scientists have accepted illusory validating evidence from what Persons (1986) called the *diagnostic category design*, which shows that people diagnosed as having a given mental disorder differ mentally from people diagnosed as not having it. However, that *between-persons* evidence logically cannot validate a mental disorder because people with literally any mental problem (for example any single symptom) differ mentally from people without the problem, whether or not that problem is a mental disorder. The diagnostic category test's inevitable finding of intergroup differences led Carson (1991) to characterize DSM validation research as having "the character of self-fulfilling prophecy" (p. 303).

The appropriate minimum standard of a mental disorder's validity is the *within-persons* main symptom test (Williams & Margraf, 2014); namely, that the diagnosis of a mental disorder must convey more information about a person's mental problems than does the disorder's main symptom alone. The mental disorder diagnostician must weigh many kinds of information besides the presence of the main symptom(s) of each disorder. The additional information (such as each disorder's exclusion criteria) seeks to amplify the information revealed by the main symptom. But that assumed amplification of main symptom information, and hence the very existence of mental disorders and of psychopathology, has never been verified even minimally by empirical evidence. Below I summarize reasons and evidence that no mental disorder will ever pass the main symptom test.

DSM-5 states, "A mental disorder is a syndrome characterized by a 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" (American Psychiatric Association, 2013, p. 20). This syndromal (multi-behavioral) definition does not distinguish mental problems that are mental disorders from mental problems, such as single behaviors (e.g., single symptoms) that decidedly are *not* mental disorders. Instead DSM-5 defines mental disorders by pointing at them one by one, each with an accompanying operational definition. DSM-IV-TR claimed that this method should "reveal the boundary between normality and pathology" (American Psychiatric Association, 2000, p. xxxi).

Influentially proposed by Robins and Guze (1970) were five validity criteria of a mental disorder: it must have a coherent description, generate consistent laboratory findings, exclude distinct disorders, have a common time course through follow-up, and run in families. These criteria do not explain the reasons that single main symptoms alone are not mental disorders. The DSM-5 supplemented the Robins–Guze list by proposing 11 validators (see American Psychiatric Association, 2013, p. 12) but still without saying why a single symptom is not a mental disorder or why one symptom alone cannot fulfill disorder validity criteria like having a coherent description, running in families, having a particular time course, having a demonstrable genetic basis, and the like (Rounsaville et al., 2002). Entirely escaping scientific notice is that the call for disorder exclusion criteria by Robins and Guze (1970) was a major psychometric error, as we will see below. The single main symptom of each mental disorder will fulfill validity criteria better than could the diagnosis of the disorder, because the main symptom alone contains more information about a person's mental problems than does the diagnosis.

Anxiety, Obsessions, and Compulsions: Mental Disorders or Psychosocial Problems?

Both pathology and its complement "normality" of behavior vanish in light of the psychometric principle of quantitative variability of behavior (Nunnally & Bernstein, 1993). Mental variability and mental pathology are as mutually exclusive as oil and water, and cannot both be true. A given kind (category) of mental problem can vary in its severity only quantitatively, in degree, whereas variation in kind rather than degree can occur only between different kinds of problems, each of which, because they are qualitatively different, must be measured quantitatively by its own severity scale (Williams & Margraf, 2014). The longed-for integration in DSM-5 of disorders with dimensions (i.e., of pathology with variability) came to almost nothing (i.e., to merely optional dimensions without useful cutoff scores) because the variability and pathology ideas are logically irreconcilable.

The 20 proposed DSM-5 anxiety disorders and obsessive-compulsive and related disorders to which this chapter's title points are listed in the left-hand column of Table 9.1 and each disorder's set of main defining psychosocial problems is listed in the right-hand column. This chapter's position is that the DSM diagnoses in the left-hand column divert us from, rather than inform us about, the psychosocial problems listed in the right-hand column (Williams & Margraf, 2014).

We consider five broad kinds of psychosocial problem in particular: (a) Phobic behavior, (b) compulsive behavior, (c) anxiety, (d) panic, and (e) troubling thoughts such as obsessions and worries. These problems are concrete mental realities in their own right, whereas anxiety disorders and obsessive-compulsive disorders are abstractions created by committees.

The failings of mental disorder theory are clear in the light of the simpler more powerful *psychosocial problem*

TABLE 9.1

The DSM-5 Anxiety Disorders, Obsessive-Compulsive and Related Disorders, and their Main "Symptoms" (Main Defining Psychosocial Problems)

	Mental Disorder	Main Symptoms/Defining Psychosocial Problems
Anxiety disorders		
1	Separation anxiety disorder	Anxiety about separation from attachment figures
2	Selective mutism	Failure to speak in some situations despite speaking in others
3	Specific phobia	Anxiety about and avoidance of a specific object or activity
4	Social anxiety disorder (social phobia)	Anxiety about and avoidance of certain social situations
5	Panic disorder	Recurrent unexpected panic attacks
6	Agoraphobia	Avoidance/anxiety of public transportation, open or closed places, standing in lines, bridges, being far from home, being outside alone or in a crowd
7	Generalized anxiety disorder	Frequent anxiety and worry about multiple events/activities
8	Substance/medication-induced anxiety disorder	Panic attacks or anxiety resulting from substance use
9	Anxiety disorder due to another medical condition	Panic attacks or anxiety resulting from a medical condition
10	Other specified anxiety disorder	Limited symptom (panic-like) attacks, generalized anxiety
11	Unspecified anxiety disorder	Symptoms characteristic of an anxiety disorder
Obsessive-compulsive and related disorders (OC&RD)		
12	Obsessive-compulsive disorder	Obsessions and/or compulsions
13	Body dysmorphic disorder	Repetitive behaviors or mental acts in response to perceived but objectively slight flaws in physical appearance
14	Hoarding disorder	Hoarding of possessions regardless of their value
15	Trichotillomania (hair-pulling disorder)	Hair pulling resulting in hair loss, repeated tries to stop it
16	Excoriation (skin-picking) disorder	Skin picking resulting in skin lesions, repeated tries to stop it
17	Substance/medication-induced OC&RD	Obsessions, compulsions, skin picking, hair pulling, after substance/medication use
18	OC&RD due to another medical condition	Obsessions, compulsions, appearance preoccupation, hoarding, skin picking, or hair pulling after substance/medication use
19	Other specified OC&RD	Symptoms characteristic of OC&RD or obsessional jealousy
20	Unspecified OC&RD	Symptoms characteristic of OC&RD

theory (Williams, 2012; Williams & Margraf, 2014), in which people's anxieties, obsessions, compulsions and all other problematical behaviors (i.e., problematical mental states and outward actions) are quite different from mental disorders. Every kind of psychosocial problem is a single category of behavior measured by a zero-to-maximum severity dimension. Psychosocial problem categories are structured in nested hierarchies of dimensions. This concept is shown simply and schematically in Table 9.2. Vertically arrayed for illustration are several possible categories (kinds) of psychosocial problems: Compulsion, depression, agoraphobia, panic attacks, and alcohol misuse. Indented are three subcategories (subproblems) of agoraphobia: Bridge phobia, bus riding phobia, and open spaces phobia. Horizontally arrayed in Table 9.2 is each problem/subproblem's continuous dimension of possible severity, which always ranges from zero (no problem) to maximum (a large problem). A superordinate category's severity dimension is the sum or average of its defining subcategories' severity dimensions.

The principles of psychosocial problems and their measurement can be summarized by observing that every kind of psychosocial problem:

- varies widely in its possible severity in finely graduated increments;
- has a possible severity that includes zero (no problem) then increases in increments of possible severity to as extreme, intense, disabling, frequent, distressing, costly, or otherwise bad as the problem can be;
- is seamlessly continuous with the whole psychological person, in whom no division exists between pathological and normal; the same universal psychometric principles and the same basic psychobiological dimensions apply whether or not a behavior is judged to be problematical;
- can stand alone or in subordinate or superordinate relationship to another problem;

TABLE 9.2

Psychosocial Problem Theory Depicted Schematically as a Category X Dimension Array

Category of Psychosocial Problem	Dimension of its Possible Severity
Compulsion	0 _____ maximum
Agoraphobia	0 _____ maximum
Bridge phobia	0 _____ maximum
Bus riding phobia	0 _____ maximum
Open space phobia	0 _____ maximum
Panic attacks	0 _____ maximum
Alcohol misuse	0 _____ maximum

- is always a single behavior, either a single response *per se* or multiple defining responses (subordinate behaviors) summed/averaged into a single score;
- has defining subproblems (if any) that empirically cohere sufficiently to justify treating their sum/average as a single behavior;
- is measured flexibly, with freedom within psychometrically permissible limits to name and define a problem, to set cut points to its severity dimension, and to dichotomize, truncate, or rescale its dimension as fits the assessment context;
- is defined only by behaviors it includes, i.e., without exclusion criteria.

A psychosocial problem can be indexed as a single behavior or observation: for example, a single task performance or a single anxiety rating given during a task. Often, a psychosocial problem is indexed as the sum of multiple behaviors. For example, any self-report anxiety inventory's (e.g., the Beck Anxiety Inventory; Beck, Epstein, Brown, & Steer, 1988) total score combines multiple observations (sums the inventory items' separate scores). And behavioral tests of phobic avoidance (e.g., Williams, Kinney, & Falbo, 1989) are dimensional measurements that may be scored either continusly (e.g., 0% to 100% performance) or dichotomously (e.g., failure = 0, success = 1).

Mental disorders deplete information about their main symptoms Psychometric principles imply that every DSM diagnosis undermines its power to predict what it wants to predict because it fails to capture, and thus it effectively depletes, information about the main defining behavior(s) of the disorder.

Disorders deplete information by stringent thresholds Disorders have arbitrarily stringent but fixed diagnostic thresholds that spawn an abundance of neither-fish-nor-fowl "subthreshold disorders" (Kessler, Chiu, Demler, Merikangas, & Walters, 2005), a term that seems to mean disorders that are not disorders. Subthreshold disorders arise, for example, from the DSM-5 rule that, to count an anxiety attack as a panic attack for diagnosing panic disorder, some attacks must be accompanied by at least four responses from a longer list, although an attack with only two or three such responses (for example, a smothering sensation and a feeling of imminent death) can be intense and have serious psychological sequelae (Margraf, Taylor, Ehlers, Roth, & Agras, 1987). The arbitrary threshold simply removes some potent anxiety attacks from the panic disorder diagnostic scheme.

Disorders deplete information by mandatory dichotomization A diagnosis is a forced dichotomy, whereas a psychosocial problem can be measured on a finely graduated, coarsely graduated, or dichotomous scale, as suits the purpose. Almost everybody agrees that each human mental and behavioral problem can come in many different levels of possible severity (see Maddux, Gosselin and Winstead, Chapter 1 in this volume; Widiger, Chapter 6 in this volume; Kupfer, First, & Regier, 2002). DSM-5 endorses dimensional measurement when it says that we need to know the number of panicky sensations a person has, the amount of time she or he spends in compulsive ritualizing, and the like. DSM-5 even provides optional new psychometric-style questionnaires to enable dimensional measurement of single symptoms on a 0–4 severity scale, and of anxiety disorder-relevant syndromal dimensions on six severity scales of 10 items each, scored 0–40. But then DSM-5 tells us that we have to exchange that graduated information for a dichotomous illness judgment: Disordered or not disordered.

This is a bad trade. To transform a finely graduated dimension into a dichotomy is, in Cohen's (1990) words, "willful discarding of information. It has been shown that when you so mutilate a variable, you typically reduce its squared correlation with behavior about 36% (Cohen, 1983). Don't do it" (p. 1307). Circumstances sometimes call for (or tolerate) dichotomization, but greater accuracy potentially can be achieved by more finely scaled measurement. DSM's new simple graduated dimensional measures foreshadow the death of DSM because researchers should quickly discover that these measures of single behaviors predict everything DSM wants to predict far better than does any DSM diagnosis, as this chapter explains in more detail below.

Disorders deplete information by tainting it The "syndrome" concept is the idea that a mental disorder is a combination of otherwise distinct behaviors. The very combining of dissimilar behaviors into one diagnosis taints main symptom information by mixing it inseparably with information about dissimilar mental problems. This robs the diagnosis of interpretable meaning (Smith et al., 2009) and limits its potential predictive power.

Disorders deplete information by diluting it A diagnosis dilutes main symptom information when the symptom features prominently in other, non-comorbid mental disorders. For example, anxiety features in the definitions of many disorders, thereby greatly limiting the capacity of any one diagnosis to inform us much about a person's anxiety. The same is true of depression. In psychosocial problem theory, anxiety and depression are measured in their own right, not as symptoms of a hypothetical but never validated mental disorder.

Disorders deplete information by exclusion Perhaps the largest information-depleting feature of DSM and ICD diagnoses is their exclusion criteria; for example, requirements that the main symptom be "not better accounted for by" or "not occur only during the course of" another particular disorder or condition, that the main symptom

must occur within certain time frames but not others, and the like. In some cases, another problematical behavior flatly excludes a diagnosis. For example, if panicky people are depressed, phobic, or highly anxious between panic attacks, as panicky people often are, they do not have ICD-10 panic disorder. Applying any DSM or ICD exclusion criterion in diagnosing any disorder simply erases the person's main symptom information from the disorder being diagnosed, thereby enfeebling the diagnosis as a measure of that symptom/behavior. In the case of panic disorder, the enfeeblement is further amplified by DSM-5's curious designation of panic attacks as a possible "specifier" for any mental disorder. This leaves the panic disorder diagnosis conveying little information about people's panic attacks. Psychosocial problem theory measures panic attacks irrespective of other things, which conserves the information about panic attacks in that single measurement.

The Main Symptom Hypothesis and Its Empirical Support

The preceding information-depleting psychometric errors in DSM and ICD inspire the *main symptom hypothesis* that the diagnosis of every mental disorder conveys less information about a person's mental problems than does the mental disorder's main symptom alone (or each of its main symptoms alone). To test this hypothesis, Williams and Margraf (2014) analyzed archival data from 19,257 participants in three community surveys of mental disorders. Survey 1 was the National Comorbidity Survey (Kessler, 2008); Survey 2 its replication (Alegria, Jackson, Kessler & Takeuchi, 2008), and Survey 3 was the Dresden Predictor Study (Trumpf et al., 2010). In each survey, all participants (1) received a DSM diagnosis (present or absent) of various mental disorders; (2) answered questions on the presence or severity of each main symptom of each disorder (i.e., each of its main defining behaviors); and (3) answered questions on the presence or the severity of diverse other DSM-mentioned mental problems. Analyses considered the ability of each diagnosis and each corresponding main symptom to predict each and every index of another DSM-mentioned problem within the survey. Each test of the main symptom hypothesis is an ordinary multiple regression analysis, in which two independent variables (one disorder diagnosis and one main symptom of the disorder) jointly predict one dependent variable (one other DSM-mentioned mental problem or related outcome such as mental treatment received). This R^2 is then further analyzed for the percentage relative importance of the main symptom to it compared with the percentage relative importance of the diagnosis to it, the two together equaling 100% (Grömping, 2007). Within each survey, we performed every main symptom test possible, yielding a three-survey total of 22,843 main symptom tests.

The results showed that the single main symptom of every mental disorder measured/predicted people's other DSM-mentioned mental problems more accurately (i.e., yielded a higher *r* and a higher percentage relative importance) than did the diagnosis of the mental disorder. When the main symptom and the other mental problem were each measured by a simple yes–no question then, in Survey 1, the main symptom's median relative importance was 77%, in Survey 2 it was 83%, and in Survey 3 it was 85%, showing that the DSM diagnoses explained, respectively, only 23%, 17%, and 15% of the jointly explained variance in participants' DSM-mentioned mental problems. A simple yes–no question asked to reach a diagnosis reveals much more about a person's mental problems than does the diagnosis.

When a diagnosis was made both with and without applying exclusion criteria (as in Studies 1 and 2), the relative importance of the diagnosis was consistently higher without exclusion criteria, and often substantially higher. This result shows directly that exclusion criteria simply damage diagnoses as measures of problematical behaviors, a fact that, surprisingly, has escaped previous scientific notice.

When, as in Survey 3, main symptoms and other mental problems were measured by precise multi-item scales rather than only by imprecise single yes–no questions, the median relative importance of the main symptoms increased to 99% and the median relative importance of the corresponding diagnosis dropped to 1%. These findings show that if people's mental problems are measured imprecisely, then main symptoms reveal far more about those problems than do disorder diagnoses; but if mental problems are measured precisely, then mental disorders essentially vanish while single main symptoms become strongly informative. A disorder's diagnosis thus tells us almost nothing compared with the disorder's well-measured single main symptom.

Therefore, psychosocial problem theory recommends that we discard the empty idea of mental disorders, including anxiety disorders and obsessive-compulsive and related disorders, but take some of their main defining behaviors, namely: (a) phobic avoidance, (b) compulsion, (c) anxiety, (d) panic, and (e) troubling thoughts, as starting points for a new dimension-based model of people's mental problems.

Psychosocial Problems Described

Phobic Behavior Avoidance behavior, including avoidant outward actions and avoidance of inward experiences, thoughts, and emotions, hampers people with many kinds of problems, and so is an important "transdiagnostic" human problem (Barlow et al., 2011). Needless avoidance in mild and transitory forms is widespread in psychological life, but when behavioral avoidance is marked and persistent one speaks of *phobia*. Phobia illustrates the "neurotic paradox" of self-defeating behavior, in which a person is unable to do an ordinary activity although she is otherwise capable, wishes to function normally, and is often aware that her

limitation is senseless. Phobias can involve gross disability, such as outright inability to cross a street, or speak to a stranger, or be in a room with a small spider. Some people with phobias refrain from doing things that they could do but would rather not. In DSM-5, but not ICD-10, unwarranted fear towards an activity or object is a phobia, even if the person does not avoid it. This chapter and DSM-5 loosely refer to either excessive anxiety or avoidance of something as phobia, but the classical meaning and the ICD-10 meaning of phobia is senseless behavioral avoidance.

Avoidance in phobia usually is accompanied by some degree of anxiety towards the avoided activity, but one of the most important and well-established findings in phobia research is that anxious feelings and avoidant behaviors are not highly correlated (Carr, 1979; Lang, 1985; Rachman, 1976), just as panic attacks and agoraphobic behavior are not highly correlated (Craske & Barlow, 1988; Wittchen, Gloster, Beesdo-Baum, Fava, & Craske, 2010). I discuss this lack of anxiety-behavior (or panic-behavior) correlation in more detail below. The present section emphasizes phobic behavior, while later sections address the anxious and panicky feelings and the troublesome thoughts that can variably accompany phobic behavior, and that are problems in their own right.

Phobias vary widely in object, severity, and generality. Some phobias are of highly specific objects or activities (e.g., riding elevators, or encountering cats or loud people), whereas generalized phobic patterns can encompass a wide range of seemingly dissimilar activities. Any specific phobia is a generalized problem when it is severe enough, or when the feared object/activity is found in diverse places (e.g., spiders or strangers). Specific phobias tend to occur with other phobias (LeBeau et al., 2010).

Phobias can occur in constellations such as agoraphobia, in which a person is simultaneously phobic of at least a few and possibly many or all of about 15 or so distinct community activities such as being away from home, using public transportation, shopping, ascending heights, crossing bridges, riding elevators or escalators, driving a car, or being in an audience. These activities are typically more difficult for the person when unaccompanied. A person need have only two such phobias to earn the agoraphobia label in DSM-5 and ICD-10.

Phobias of social scrutiny, or of being embarrassed, or of causing embarassment to others can vary endlessly in form, generality, and severity, between individuals and across different cultural contexts (Bögels et al., 2010). Great variation exists in the number, kinds, and patterning of individual phobias and in the diverse other problems that can accompany phobias, such as sad mood, panic attacks, somatic concerns, and many others (Barlow, 2002). Even a specific phobia can be heterogeneous: A person with dental phobia might dread the confinement, the needle, the drilling, the scents, allergic reactions, panic attacks, or various scary social possibilities (Oosterink, de Jongh, & Aartman, 2009), so the term "dental phobia" conveys only a limited amount of information.

Phobic avoidance and/or fearful feelings about particular things are common far beyond the problems that diagnostic manuals deem official phobic disorders. Dysfunctional avoidance is common among people diagnosed with any anxiety disorder. Moreover, some sexual phobias are called sexual dysfunctions; some disease phobias, hypochondriasis (or in DSM-5 "illness anxiety disorder", *not* an official anxiety disorder; e.g., Fergus & Valentiner, 2010); some phobias of gaining weight are called anorexia nervosa, and so on. Although the term "phobic avoidance" refers only to overt behavior, one can maladaptively avoid in the emotional and cognitive sphere too. Such "covert" avoidance has a negative impact psychologically (Solomon & Mikulincer, 2007).

Compulsive Behavior An important variation on phobic behavior is compulsive behavior. A person with a compulsion carries out one or more actions far beyond reason and with a feeling of being compelled to do so. Common compulsions include excessive cleaning, handwashing, repeating, checking, counting, arranging, or hoarding (now its own specific DSM-5 disorder). Compulsions can involve elaborate time-consuming rituals that seem to have a symbolic function rather than any practical effect (e.g., magically decontaminating an object). One can hardly overstate the heartbreaking extent of impairment and bizarreness of behavior that can be observed in people with severe compulsions (Steketee & Barlow, 2002). Less debilitating but still troubling compulsions appear to be common (Fullana et al., 2009). In recognition that diverse problem behaviors other than those included in obsessive-compulsive disorder can be compulsive in nature (Frost & Steketee, 2002), DSM-5 created a new class of "obsessive-compulsive and related disorders," which still overlooks compulsions common in eating-related and psychotic problems.

Compulsive behaviors tend to be accompanied by obssessions, by a feeling of fear or discomfort that declines with the compulsive behavior, by a sense that the compulsions prevent harm or danger, and by recognition that the behavior is excessive or unreasonable. But, importantly, not one of these several features is invariably present or necessary (Steketee & Barlow, 2002). Like phobic disorders, obsessive-compulsive disorder can be diagnosed despite an absence of negative emotions such as anxiety or discomfort; compulsions alone, defined by outward actions, are sufficient for the diagnosis.

Phobia and compulsion are related, and can be viewed for some purposes as passive and active variants, respectively, of avoidance, with phobia being avoiding by not doing and compulsion avoiding by doing. Phobic and compulsive behaviors often overlap (Rachman & Shafran, 1998). For example, a woman who dreaded burglars remained fully dressed in bed every night atop the bedcovers (phobia), and spent much time before bed checking and rechecking her window and door locks (compulsion). Importantly, though, not all compulsions serve perceived avoidant ends.

Although most people with phobias do not have marked compulsions, they often engage in ritual defensive activities when they do the things they would rather avoid (Williams, 1985; Williams & Zane, 1989). Most people with marked compulsions are phobic about compulsion-provoking circumstances, such as compulsive handwashers who phobically avoid contact with potential contaminating objects like door handles (Rachman & Shafran, 1998). Cognitive (or "covert") compulsions also exist, in which people perform rituals in thought, such as checking, counting, reciting, or arranging, sometimes in order to neutralize, undo, or counteract a previous unacceptable thought or action (Steketee & Barlow, 2002).

Troubling Thoughts Aversive intrusive thoughts, difficult to control or dismiss, can come as obsessive preoccupations, excessive worries, scary images, mad impulses, catastrophic expectations, or horrifying recollections, among others (e.g., Borkovec, Shadick, & Hopkins, 1991; Frost & Steketee, 2002; Rapee & Barlow, 1991). Obsessions and worry are experienced by many people who have no serious problems, and often occur in people who fear and avoid, regardless of their particular dreaded activities. People with specific phobias, for example, often perceive the phobic object/activity as dangerous and they worry, obsess, intrusively remember, fearfully anticipate, and even have nightmares about it. Noteworthy is the pronounced situational specificity of thinking; that is, it can vary markedly as a function of circumstances, with most individuals finding that particular settings or activities evoke more obsessions, worries, catastrophic thoughts, and the like, than do others (Salkovskis, 1996; Williams & Watson, 1985).

Some characteristic thought patterns are also proposed cognitive mechanisms of fear and avoidance (Bandura, 1997; Beck, 1976; Mathews & McLeod, 2005; Taylor et al., 2014), which we consider below in the section on causes. The present section considers troubling thoughts such as obsessions and worries that are problems in their own right.

Obsessions are intrusive unwanted thoughts, images, or impulses that can be experienced as aversive, alien, or frightening. Common obsessive themes involve moral, religious, sexual, contamination, disease, symmetry, ordering, and harming concerns (Williams, Mugno, Franklin, & Faber, 2013; Yovel et al., 2011). The relationship between obsessions and compulsions is complex. People sometimes engage in compulsive behavior in response to obsessions, such as the man obsessed with blasphemy who had to *not* think "Damn God!" when he stepped off a curb else he would be eternally damned; usually unsure if he had done it right, he had to step back and repeat it again and again.

Epidemiological studies have, however, observed substantial independence of obsessions from compulsions (Mayerovitch et al., 2003) and either can predominate or be entirely absent in a person with the other. Even in an individual who has both, the complulsions can appear unrelated to the obsessions (Yovel et al., 2013). Theories and treatments concerning each differ (Rachman, 1998; Frost & Steketee, 2002), because overt behaviors are available as treatment targets in compulsions but can be absent or minimal in obsessions. Symptom profiles in people diagnosed with obsessive-compulsive disorder are strikingly variegated (Williams et al., 2013; Yovel et al., 2012); factor analyses of obsessive-compulsive inventories obtain complex factor structures (e.g., Anholt et al., 2009), and twins' ratings of typical obsessive-compulsive behavior dimensions reveal heterogeneity of genetic influences on them (Iervolino, Rijsdijk, Cherkas, Fullana, & Mataix-Cols, 2011). These findings show that the conjoint "obsessive-compulsive" label can imply greater balance between the two problems than often exists.

Troubling thoughts and even hallucinations and delusions are common in psychological life generally (Michail & Birchwood, 2009; Vellante et al., 2012). Many patterns of troubled thinking show elements of both worries and obsessions (Fergus & Wu, 2010). Maladaptive worry is not always sharply distinct from constructive preparatory problem solving, because, in both, one thinks of possible dangers and how to prevent or manage them (Craske, 1999).

Nor are the boundaries between the excessive, the unreasonable, and the delusional entirely sharp. People with phobic avoidance, compulsions, panic, or obsessive worrying thoughts often recognize that these are out of proportion to objective reality, but such "insight" is highly variable between individuals, and even within individuals across different circumstances (Rachman & Shafran, 1998; Williams & Watson, 1985; Zimmerman, Dalyrmple, Chelminski, Young, & Galione, 2010). DSM-5 has generally dropped requirements that the person has to recognize that the phobias, obsessions, anxiety, and so on, are excessive. Some obsessions are plainly delusional (Frost & Steketee, 2002; O'Dwyer & Marks, 2000). People with psychotic problems frequently display obsessions, compulsions, phobias, panic, and anxiety (e.g., Michail & Birchwood, 2009; Foster, Startup, Potts, & Freeman, 2010). Psychosocial problems combine idiosyncratically and with little respect for official mental disorder boundaries.

Anxiety/Fear Nearly everyone has experienced anxiety at first hand. We speak of ourselves as feeling anxious, afraid, tense, nervous, worried, scared, and the like, which terms we use to describe our feelings and to explain our behaviors. We say "I left because I got anxious" or "I could hardly speak because I was too afraid." Scientists also hold that fear/anxiety causes many behaviors, normal and abnormal, adaptive and maladaptive. But the meaning of anxiety and its power to strongly influence behavior are far from clear.

Anxiety has long been a leading proposed cause of phobic avoidance behavior, but anxiety is potentially a serious

problem in its own right. Current usage of the term is variegated. DSM-5 distinguishes anxiety from fear: "Fear is the emotional response to real or perceived imminent threat, whereas anxiety is anticipation of future threat" (p. 189). Nevertheless, the present chapter uses the two terms as synonyms because the basic responses that define them differ mainly in temporal focus (imminent versus future) but not much in their psychological content, which in both is threat perception. Scientists have conceived anxiety in diverse ways (Hersen, 1973; Lang, 1985). It can be defined by a single kind of response, such as subjective fear intensity (Walk, 1956), or physiological arousal (Mowrer, 1960). The broadest definitions have expansive contents that include all the usual anxiety responses plus overt behavior and much else (Lang, 1985). Because anxiety responses in one mode (e.g., subjective fear) tend to be notably disassociated from anxiety responses in a different mode (e.g, physiological fear), the dissimilar responses are better kept separate terminologically rather than being lumped into an all-purpose but not very meaningful "anxiety" category (Williams, 1987).

Subjective Anxiety Being afraid means above all feeling afraid, consciously, without which anxiety has no meaning. Fear in consciousness is difficult to describe, but people can indicate how intensely afraid they feel by rating a simple scale, for example, from 0 (not anxious or afraid) to 10 (extremely anxious and afraid), or on a visual analog scale anchored similarly. Such scales are widely used and indicate straightforwardly how much a particular activity frightens someone (Williams, 1987). Subjective anxiety has meaning in relation to the psychological context in which people experience it. Multiple-item anxiety scales are also in wide use (e.g., the Beck Anxiety Inventory, Beck et al., 1988). Both single-rating and multi-item anxiety scales are psychosocial problem scales that measure a single dimension that ranges from no anxiety to high anxiety.

Panic Anxiety Panic attacks are sudden rushes of intense anxiety (or discomfort in DSM-5) that can be disturbing and can leave a lasting residue of dread and disability (Batelaan et al., 2009). People can have panic attacks but not develop serious problems in connection with them (Craske, 1999). Although panic, like ordinary anxiety, is conventionally conceived in part as bodily arousal, the actual relationship between bodily arousal and panic, like that between bodily arousal and anxiety, is not very strong. Many reported panic attacks come without autonomic arousal, and rapid high autonomic arousal often is not experienced as scary or panicky by an individual (Barlow, 2002; Ehlers, 1993; Margraf et al., 1987). As the essence of anxiety is the subjective feeling of anxiety, the essence of panic is the subjective feeling of panic.

Panic attacks vary widely in intensity, duration, and frequency, in the number and kinds of thoughts and sensations that accompany them, and along other graduated dimensions (Barlow, 2002). DSM-5 considers panic to be its own disorder ("panic disorder") and simultaneously to be a "specifier" for (a potential attribute of) every other DSM-5 mental disorder. This is a curious structural element of DSM-5, because many particular responses besides panic (for example, depressed mood, subjective anxiety, ruminating worry, autonomic arousal) can be found in people with diverse kinds of psychosocial problems, hence DSM-5's singling out of panic for special "specifier" status is odd. Why not also a depressed mood or an anxious feelings specifier?

Panic attacks vary in their apparent relationship to circumstances. Some panic attacks seem to come spontaneously, as if uncued, but people sometimes find panic attacks to be more likely in certain settings. To diagnose panic disorder in DSM-5 requires the person to have "unexpected" panic attacks. Apart from people's perception of a cue, expected and unexpected panic attacks are similar (Craske, 1991; Kessler et al., 2006). Early theories considered uncued panic to be a biological event (Barlow, 2002). The social cognitive view is that even unexpected anxiety and panic occur in relation to discrete events, but these can be cognitive or other internal events, such as catastrophic interpretations of perceived bodily states (e.g., Clark, 1986; Craske, 1991) or loss of self-efficacy for maintaining cognitive control (Casey, Oei, & Newcomb, 2004; Casey, Newcombe, & Oei, 2005; Williams & LaBerge, 1994). Panic can be highly responsive to situational and psychological manipulations and interventions (Barlow, 2002; Craske, 1999; Mitte, 2008).

Trait Anxiety Subjective anxiety can be viewed as a transitory feeling state and as an enduring personality trait, a disposition to see circumstances as threatening and to react with fear (e.g., Cattell & Scheier, 1961). Trait anxiety is usually measured by asking people to indicate the self-descriptiveness of various brief statements (Hersen, 1973). Trait conceptions and trait-like inventories abound that measure anxiety-in-general, without respect to context. Considerable attention has been paid recently to broad generalized higher-order traits such "internalizing" (Markon, 2010), "neuroticism" (Brown & Naragon-Gainey, 2013), or "negative affectivity" (Griffith et al., 2010). The personality trait of anxiety sensitivity, (i.e., being generally afraid of feeling anxious sensations) and its measure, the Anxiety Sensitivity Index, have become the objects of much interest in research (e.g., Naragon-Gainey, 2010).

Trait scores have some predictive ability but they rarely explain even 15% of the variance in behavior and often far less (Williams & Cervone, 1998). People certainly differ from one another in the amount that they are, on the average, distressed (or limited by phobias, bedeviled by troubling thoughts, frightened of being frightened, etc.), but in every case they are troubled by certain definite things and not by others. Small changes in context, like the approach

of a cat to a person with a cat phobia, or a chance reminder of childhood diseases to a worrying mother, can produce marked shifts in behavior, thought, and feeling (Beck, 1976). Idiosyncratic strong variation in an individual's mental states and actions across situations, which variation pervades the psychosocial problems we are considering in this chapter, is simply ignored by generalized traits (Salkovskis, 1996), as is the idiosyncratic configuration of problems from one person to another.

Psychological treatments involve the person coming into mental and physical contact with the specific things he or she fears, avoids, ritualizes over, or intrusively thinks about, and not with most other things. Therefore, someone's average tendency to be anxious-in-general (or anxiety-sensitive-in-general or anything-in-general) gives us limited useful information for helping the person change. Trait-like measures continue to inspire much research. But the pervasive situational specificities of behavior mean that no amount of inpiration will ever give us a personality trait that will tell us useful specifics about someone's problems. Social cognitive theory advocates measuring problematic responses *per se* when feasible, doing so directly in problematic circumstances when feasible, and otherwise doing so in a psychologically circumscribed context rather than as generalized context-free traits (Williams & Cervone, 1998). We will see below that non-trait, context-sensitive cognitions predict and explain people's context-sensitive problematical feelings and behaviors especially well.

Physiological "Anxiety" Anxiety is widely conceived in part as a physiological response, mainly autonomic arousal and its associated neurochemical mechanisms. Because autonomic arousal *per se* can be measured physically, without reference to people's consciousness, it is a favored index of fear among investigators chary of subjective mental measures. Defining anxiety as physiological arousal can be misleading because, although people commonly describe fear in part by describing how their heart raced or they began to sweat, their bodily perceptions often do not match their actual bodies (Hoehn-Saric, McLeod, Funderburk, & Kowalski, 2004; Andor, Gerlach, & Rist, 2008). Strong emotions of all kinds tend to come with bodily arousal, but that arousal does not distinguish between fears and other strong emotions (Hoehn-Saric, 1998). Frightened and panicky people show many patterns of physiological arousal, including no such arousal at all (Barlow, 2002; Ehlers, 1993; Margraf et al., 1987). Physiological arousal without subjective fear is common, as when people exercise, feel sexual interest, or hear a familiar voice (Lang, 1985). The large gap between autonomic physiology and subjective fear means that such bodily responses alone cannot be anxiety. Bodily responses and subjective fear are not two "systems of anxiety," but two separate domains of mental responding. Each must be considered on its own terms (Williams, 1987) and in relation to the other.

Anxiety as Perception of Physiological Arousal Social cognitive theory holds that physiological arousal *per se* has less impact on behavior than does the person's perception and interpretation of physiological arousal (Andor et al., 2008; Salkovskis & Warwick, 1986). Bodily perceptions can become a focus of obsessive worrying and panic, and can give rise to defensive actions, such as seeking medical help for a racing heart or ritually carrying a bottle of water against a possible dry mouth. Bodily perceptions are implicated in generalized anxiety, panic attacks and illness phobias (Clark, 1986; Salkovskis & Warwick, 1986; Wells, 2004). The perception of arousal can override the actual arousal (Andor et al., 2008).

Anxiety in DSM In DSM-5 anxiety comes in the form of fear, anxiety, and panic attacks. In the DSM-5 glossary, anxiety is "the apprehensive anticipation of future danger or misfortune accompanied by a feeling of worry, distress, and/or somatic symptoms of tension" (p. 818) and fear is "an emotional response to perceived imminent threat or danger associated with urges to flee or fight" (p. 821). DSM-5 states, "Panic attacks feature prominently within the anxiety disorders as a particular type of fear response" (p. 189). ICD-10's definition of anxiety is rather different: "Primary symptoms of anxiety . . . usually involve elements of (a) apprehension (worries about future misfortunes, feeling 'on edge', difficulty in concentrating, etc.); (b) motor tension (restless fidgeting, tension headaches, trembling, inability to relax); and (c) autonomic overactivity (lightheadedness, sweating, tachycardia . . . dizziness, dry mouth, etc." (p. 140). Neither definition incorporates avoidance behavior as such but DSM-5's definition of fear (but not anxiety) as prompting "urges to flee or fight" links fear to avoidance behavior by declaration, although the "fight" part of it is obscure.

A panic attack is defined in DSM-5 as a discrete period of rapidly mounting intense fear or discomfort accompanied by certain thoughts, feelings, perceptions, and physiological or other sensations, numbering about two dozen, grouped into 13 categories, with responses from at least four categories needed to call a fear attack a panic attack. Avoidance behavior is not part of this definition, but the motivation to avoid panic, or to avoid non-panic but "panic-like" fear attacks (or other incapacitating or embarassing reactions), weighs in the DSM-5 definition of agoraphobia. At the same time, DSM-5 separates panic disorder and agoraphobia as two diagnostically independent disorders. DSM distinguishes cued and uncued panic attacks, and it focuses the definition of panic disorder on uncued panic, although the distinction between cued and uncued panic is not hard and fast (Craske et al., 2010).

The DSM-5 requirement for thoughts of danger in both fear and anxiety, and their possible inclusion in ICD-10 anxiety, transform danger thoughts from the cause of anxiety into its very substance, and thereby transform Beck's (1976; Beck, Emery, & Greenberg, 1985) perceived danger theory of anxiety into the meaningless assertion that anxiety causes

itself. Exploring the possible role of danger perceptions in causing anxiety requires our keeping danger perceptions out of the definition of anxiety. The DSM/ICD mixing of danger perceptions and bodily arousal in "anxiety," "fear," and "panic" ensures that these concepts lack psychometric homogeneity and, hence, validity (Smith et al., 2009).

The Alleged Three Systems and Beyond The preceding views of anxiety define it by various nonbehavioral responses. The three-systems analysis (Lang, 1985) went further and threw in maladaptive behavior itself, the very phenomenon that anxiety was supposed to explain. Lang (1985) proposed that "the data of anxiety" consist of "verbal reports of distress, fear related behavioral acts," and "patterns of visceral and somatic activation" (Lang, 1985, pp. 133–4). People no doubt think, feel, act, and respond physiologically, all at once, in nearly everything they do. But, as a theory of avoidance behavior, the three systems were stillborn. The emotion of anxiety was to be the cause of problematic overt behavior. To assert that the emotion and the behavior are one and the same, namely "anxiety," robs both of meaning. The three-systems verbal report (or "language behavior") category is sometimes called the "cognitive system," ironically so, because Lang fashioned it after B. F. Skinner's adamantly non-cognitive idea of "verbal behavior". The three-systems analysis expanded anxiety to include avoidance, but it did not explain avoidance. To explain avoidance, bioinformational theory elaborated the alleged three systems into a vast polymodal fear network (Lang, 1985; Craske et al., 2008), discussed with causal theories, below.

Causes of Psychosocial Problems

This section focuses on current psychological causes of problem reactions. Nevertheless genetic, biochemical, developmental, historical, and environmental causes all have roles and have been studied extensively (Barlow, 2002; Rachman, 1977; Rapee & Spence, 2004; Taylor, 2011; van Houtem et al., 2013).

Causes of Phobic Avoidance Dysfunctional avoidance behavior is broadly important as a frequent element of mental problems generally (Barlow et al., 2011). Problematic avoidance is especially prominent in phobias, so they reveal well its nature and treatment. Some phobias develop straightforwardly; for example, springing full blown from a single brief traumatic experience (Ehring, Ehlers, & Gluckman, 2008). Scary verbal information alone can engender enduring fear (Field & Lawson, 2003). Most phobias lack discrete precipitating circumstances, and such circumstances (e.g., a severe automobile crash) typically produce phobias in relatively few people. Phobias, obsessions, and compulsions have complex genetic roots (Iervolino et al., 2011; Taylor, 2011; van Houtem et al., 2013) and can be traced to diverse social, psychological, and environmental circumstances (Rachman, 1977).

In the social cognitive approach, historical and biological causes operate mainly via conscious cognitive processes that prompt and sustain avoidance behavior in the here and now. Scientists took a very long time to find strong inner causes and correlates of avoidance behavior because, for decades, their research agenda focused on weak inner causes, such as anxious feelings or autonomic responses, or on external conditioning stimuli alone, while neglecting people's own inner frame of reference.

Anxiety Theory of Avoidance Maladaptive avoidance behavior was a principal psychological phenomenon that the concept of anxiety was originally intended to explain. Anxiety was the hypothesized cause and avoidance behavior the explained effect. Two-factor theory (Mowrer, 1960) proposed that anxiety comes to control avoidant behavior in a two-part process of classical conditioning plus operant conditioning. First, the person learns by classical conditioning to be afraid of a previously neutral stimulus, after having experienced it paired with an aversive stimulus. Then the anxiety provoked by the now-conditioned former neutral stimulus motivates the person to avoid that stimulus, which avoidance is rewarded (operant conditioning) by the decline in anxiety it leads to (Mowrer, 1960; Wolpe, 1958). Two-factor theory requires that "hot" (currently experienced) fear actually must decline to reinforce avoidance behavior. This need for hot fear and its reduction is a fatal flaw in two-factor theory.

Two-factor theory dominated the study of avoidance for decades and continues to have loyal adherents (e.g., Foa & McNally, 1995; cf. Craske et al., 2008), although a series of important reviews in the late 1960s and 1970s (e.g., Bandura, 1969; Carr, 1979; Rachman, 1976; Seligman & Johnston, 1973) pointed to fundamental problems that two-factor theory never solved. Because two-factor theory relies on currently experienced fear as both the instigator and the motivator of avoidance, it inherently cannot explain why maladaptive avoidance behavior so often takes place for prolonged periods with little or no fear, both in research animals (Schwarz, 1989; Seligman & Johnston, 1973) and in severely disabled phobic and compulsive people (Carr, 1979; Spitzer & Williams, 1985; Wittchen et al., 2010). The point is explicit in *ICD-10*: "It must be remembered that some agoraphobics experience little anxiety because they are consistently able to avoid their phobic situations" (World Health Organization, 1992, p. 136). DSM-5 makes the same point: "Sometimes the level of fear or anxiety is reduced by pervasive avoidance behaviors" (p. 189).

Most human avoidance behavior is fearless (Carr, 1979). People usually fill fuel tanks and lock doors in emotional tranquility. Height phobic people stroll calmly past tall buildings. Some severe agoraphobics remain always at home in serenity (Spitzer & Williams, 1985). Easily executed avoidance behaviors, even highly maladaptive ones,

can leave nothing to be afraid of. But sustained phobic avoidance without hot fear is bluntly incompatible with two-factor theory. In the social cognitive analysis discussed below, dysfunctional thinking provokes fear and avoidance. It is the dysfunctional thinking, not the fear, that causes the avoidance.

Panic Theory of Avoidance A variation on the anxiety theory of avoidance, embodied in DSM-5 but not in ICD-10, holds that agoraphobic avoidance is caused by panic attacks or by panic-like attacks. As panic and panic-like are similar to anxiety, so the panic theory of agoraphobia founders on the same rock that sank two-factor anxiety theory, namely, that panic does not correlate much with phobic behavior (Craske & Barlow, 1988). Agoraphobia is common without current panic and without any history of panic attacks (Wittchen et al., 2010).

Below I review findings that conscious cognitions, such as the anticipations of possible future panic, unlike panic *per se*, have a strong relationship to avoidance behavior. Similarly, the anticipation of reaching a high level of anxiety if one were to perform a certain task has a moderately strong capacity to predict whether a person will perform the task, and the judgment of one's ability to do the task (one's self-efficacy) is an even stronger predictor of task performance (Williams, 1996), but such conscious cognitions are not any part of two-factor theory.

Bioinformational Theory of Avoidance The three-systems idea was mainly a listing of proposed anxiety responses, but bioinformational theory (Lang, 1985; see Craske et al., 2008) sought to explain avoidance by a fear structure; that is, a largely unconscious associative network of memory nodes containing dozens of interacting language and meaning elements linked to diverse neurophysiological substrates. In bioinformational theory, "emotions . . . are fundamentally to be understood as behavioral acts" (Lang, 1985, p. 140) insofar as the fear structure initially produces a fear reaction unified across the three systems (i.e., overt behavior, language behavior, and physiology), but inhibitory neural pathways abort some fear responses before they are expressed (Lang, 1985). Transmuting fearful feelings into avoidant behaviors would be quite a feat of psychic alchemy, given their well-established empirical disassociation. Barlow (2002) wrote that accepting bioinformational theory's unity of fear and avoidance "demands a leap of faith" (p. 57). Bioinformational theory incorporated the latest terminology from neuroscience, cognitive science, and computer science, but it was a big step backwards. Its polyform phenomena governed by countless unconscious and neurobiological mechanisms could tell us little about people's problems. In contrast, social cognitive theories are readily testable and of proven value.

Social Cognitive Theory of Avoidance Social cognitive theory and a family of related cognitive theories explain phobic and compulsive behavior, anxiety, panic, and bothersome thoughts in terms of people's conscious appraisals of themselves and their circumstances (e.g., Bandura, 1997; Beck et al., 1985; Clark, 1999; Hofmann, Amundsson, & Beck, 2013; Rachman, 1998; Salkovskis, 1996; Williams & Cervone, 1998). I call these collectively "social cognitive theories" because they explain psychosocial problems by people's beliefs, expectations, and judgments about particular things, as representable to themselves in conscious awareness, and about which they potentially can communicate openly. Social cognitions are not generalized trait-like cognitive styles, like anxiety sensitivity, nor are they generalized information-processing biases, but context-specific thoughts, which context specificity enables them to predict the idiosyncratic patterning of avoidance and fear across situations and time.

Although social cognitive theories disagree with one another about many details, they all view conscious cognition as underlying both anxiety and avoidance. Different social cognitive theories emphasize different kinds of thought, of which thoughts of danger, anticipations of anxiety, and judgments of self-efficacy are especially important. People can imagine future dangers (such as possible physical, psychic, or social harms), and they can imagine becoming distressed (anxious or panicky). Alternatively, they can entertain doubts about their ability to execute courses of action irrespective of possible harm or distress that might result.

Perceived danger is measured, as Beck (1976) suggested, as the perceived likelihood, from 0% to 100%, of a harmful outcome resulting from a given action (Williams & Watson, 1985). Such perceptions of danger have a central causal role in theories derived from the work of Beck (1976), which holds them mainly responsible for the avoidance, fear, and other problems seen in phobia, panic, obsession, and compulsion (e.g., Beck, 1976; Clark, 1999; Salkovskis, 1996). *Anticipated anxiety* is measured as the level of anxiety people think they would reach if they did a certain task. *Anticipated panic* is measured as the likelihood from 0% to 100%, that doing a task would result in a panic attack (Williams, 1996). Such anticipated distress has been proposed as underlying dysfunctional avoidance (Smits, Powers, Cho, & Telch, 2004). *Perceived self-efficacy* is one's perceived ability to execute an action or a pattern of thought. Self-efficacy scales incorporate no mention of outcomes; the sole question is the extent the person thinks she or he can perform particular actions, overtly in behavior or in the case of thought control self-efficacy, covertly in consciousness. Self-efficacy theory (Bandura, 1997; Maddux, 1995; Williams, 1996) holds that avoidance, fear, and scary thoughts arise towards an object or activity largely because people have a diminished sense that they can act effectively and remain in control of circumstances and of themselves. Perceived control is intimately related to perceived self-efficacy because, to feel in control, a person must judge himself or herself able to enact controlling responses. A sense of control mitigates

anxiety and panic (Barlow, 2002; Borkovec et al., 1991; Gallagher, Naragon-Gainey, & Brown, 2014; Hofmann, 2005; Stapinski, Abbott, & Rapee, 2010).

The power of these particular social cognitions to predict phobic behavior has been compared in diverse studies (summarized in Williams, 1996; see also Öst, Ferebee, & Furmark, 1997). Both before and after phobia treatment, and in relation to the tasks of an upcoming behavioral avoidance test, people rated their perceived self-efficacy, anticipated anxiety, anticipated panic, and perceived danger. Self-efficacy was consistently the most accurate predictor of behavioral avoidance in the test, and usually accounted for more than half the variance in behavior. Anticipated anxiety and anticipated panic proved also to be strong predictors of avoidance that each accounted for nearly half the variance in behavior, but, importantly, they were consistently less accurate in predicting behavior than was self-efficacy (Williams, 1996). Perceived danger, rated as the percentage likelihood of a particular harmful outcome occurring, was a weak predictor of behavior.

Self-efficacy consistently remained strongly predictive of phobic behavior when any other cognitive factor (i.e., anticipated anxiety, anticipated panic, or perceived danger) was held constant, whereas each other factor consistently failed to significantly predict behavior when self-efficacy was held constant (Öst et al., 1997). Self-efficacy remains an accurate predictor of future behavior even when relevant past behavior is controlled statistically or does not exist, and when noncausal interpretations of the efficacy-behavior link can be ruled out (Williams, 1996). These findings make clear that people's self-perceptions of their capabilities causally affect their avoidance behavior.

Causes of Anxiety

Social Cognitive Causes of Anxiety In much of the phobia research described in the preceding paragraphs, one can examine the comparative ability of social cognitions to predict performance-related anxiety arousal; that is, how frightened people became during the behavioral tests. Analyses showed that anticipated anxiety and anticipated panic were strong predictors of people's anxiety, rated during the subsequent behavioral test. Anticipated anxiety and anticipated panic each accounted, separately, for just over half of the variance in performance-related subjective anxiety, whereas self-efficacy accounted for only about one-quarter of the variance in anxiety. The two "anticipated fear" measures (i.e., anticipated anxiety and anticipated panic) each continued to strongly predict people's performance-related subjective anxiety when either self-efficacy or perceived danger was held constant, whereas the latter two lost all significant predictiveness when either anticipated anxiety or anticipated panic was held constant.

Important to note is that people can express high self-efficacy for doing a task but high anticipated anxiety for it as well, in which case they are likely to do the task without great difficulty but with high anxiety (Williams, 1996). This result suggests that seeing oneself as vulnerable to anxiety (or to panic) causes one (makes one vulnerable) to actually experience anxiety, although the mechanisms of this connection are far from clear (cf. Maddux, 1995).

Research in the tradition of Beck's (1976) perceived danger theory has continued to examine how perceptions of danger can give rise to anxiety and avoidance (e.g., Stapinski et al., 2010), and appear to be involved in the development of fear reactions (e.g., Fentz et al., 2013). Yet danger perceptions were generally weak predictors of the level of anxiety people experience during phobia-related behavioral tests (Williams & Watson, 1985; Williams, 1996), and danger reappraisal was not a strong mechanism of cognitive-behavioral therapy treatment effects (Smits, Julian, Rosenfeld, & Powers, 2012). When people with agoraphobia thought aloud while attempting activities that frightened them, danger thoughts essentially never occurred to them (Williams, Kinney, Harap, & Liebmann, 1997). Danger ideation is hard to remove from fear and avoidance, but the relationship is complex and other cognitive processes especially self-reflective processes such as self-efficacy appear to be powerfully at work.

Cognitive Processing Bias Causes of Anxiety Information-processing approaches attribute anxiety partly to cognitive biases, both conscious and unconscious, in anxious people's attention to, perception of, interpretation of, and memory for fear-related information, as well as to biases in the contents of troubling thoughts and ruminations, compared with nonanxious people (Mathews & McLeod, 2005; McNally, 1999; Mitte, 2008, Van Bockstaele et al., 2013). In one typical kind of information-processing experiment, people selected for having or not having anxiety, such as a high trait anxiety score or a certain anxiety disorder diagnosis, view briefly presented words related to their anxiety themes or not, and their reaction times on subtasks ostensibly unrelated to the word meanings reveal biases in how they deployed attention as a function of those meanings. Complicating the picture is that different measures of threat bias (e.g. dot-probe versus eye-movement bias measures) may be little correlated with one another and with the severity of the type of problem being studied (e.g., social anxiety; Waechter, Nelson, Wright, Hyatt, & Oakman, 2014). Not all kinds of cognitive operations are equivalently biased in anxious individuals. For example, memory biases in anxiety are more likely for recall than for recognition (Mitte, 2008).

Social cognitive theories concur that people process information differently in relation to their problems than do people without those problems and that such biases can contribute to the development of problems and in turn be affected by them (Van Bockstaele et al., 2013). Interpretation biases towards threatening information predict new onsets of panic disorder (Woud, Zhang, Becker, McNally, & Margraf, 2014). Cognitive biases likely exist in processing information about possible threats and about

one's self-efficacy to manage threats, one's sense of control over or vulnerability to feeling anxiety and panic, and other self-reflective concerns. If the researcher focuses only on threat perceptions, then threat perceptions will indeed be found to be an important factor; but that does not establish that threat perception alone tells the whole story.

Bias effects can be complex, subject to divergent interpretations, and less than robust. For example, sometimes they can be eliminated or even reversed in direction by minor variations in experimental stimuli, participants, or responses (Lim & Kim, 2006; McNally, 1999). On the other hand, treatments that try to alter cognitive/attentional biases have shown promise in reducing people's anxiety (Mathews, 2006), a topic to which we return below in discussing treatments. Nevertheless, information processing biases cannot predict avoidance behavior or subjective fear reactions remotely as accurately as do conscious social cognitions such as perceptions of self-efficacy and anticipations of fear and panic.

Panic Attacks Most early research on panic focused on physiological causes (Barlow, 2002). Diverse physical influences, including sodium lactate, carbon dioxide, caffeine, vigorous exercise, and rapid breathing can induce panic in vulnerable individuals. The sheer variety of apparent bodily influences on panic suggests the possibility of a common psychological mechanism (Clark, 1986).

Psychological models of panic generally conceive it as resulting from perception of threat (Beck et al., 1985), in particular, a vicious cycle of perceiving bodily sensations, interpreting them catastrophically, therefore feeling afraid and apprehensive, which provokes more bodily sensations to be interpreted catastrophically, and so on (Clark, 1986; Rapee, 1993). A self-efficacy analysis also emphasizes the sense of control; that is, that one can prevent panic, can influence thoughts that otherwise lead to panic, and if necessary manage well depite panic (Williams & LaBerge, 1994). Evidence supports a role for cognition in panic, including a sense of control (Barlow, 2002; Casey et al., 2004, 2005; Gallagher et al., 2014; Williams & Falbo, 1996). Cognitive processes seem to mediate the effects of psychological and environmental manipulations on biological panic induction and the beneficial effects of panic treatments (Clark, 1993; Hoffman et al., 2007). "Panic self-efficacy" or belief that one can cope with panic, mediated treatment gains in people with panic disorder (Gallagher et al., 2014).

Troubling Thoughts

Social cognitive theories hold that troubling thoughts such as obsessions and worries occur in the normal stream of consciousness but become problematic as people interpret and respond to them maladaptively. General cognitive deficit theories of obsession fail because, like traits, they are unable to account for the pronounced situational specificity of obsessions (Salkovskis, 1996). Recent theorizing has emphasized an excessive sense of responsibility for preventing potential harmful effects and a corresponding impulse to take neutralizing actions.

When people try to avoid or suppress a bothersome intrusion, their neutralizing rituals can increase the frequency of such thoughts and undermine the sense of control, increasing anxiety and spurring greater efforts to exert control, continuing in a vicious cycle (Rachman, 1998; Salkovskis, 1996). Compulsive checking itself can undermine confidence in memory, causing more checking (Boschen & Vuksanovic, 2007). Whether worries and obsessions become problems depends partly on their perceived controllability (Borkovec et al., 1991; Grisham & Williams, 2009; Stapinski et al., 2010) and the person's self-efficacy to manage worrisome future possibilities (Cieslak, Benight, & Lehman, 2008). Lack of self-efficacy for remembering actions, for distinguishing imagined from real past actions, for maintaining control over thinking, and the like, have been called "cognitive confidence" or "thought control confidence," and this kind of self-efficacy has been implicated in obsessions and compulsions (Grisham & Williams, 2009; Hermans et al., 2008).

Theories of worry view it as reinforced by anxiety reduction, by a sense of control, or by perceived risk reduction (Borkovec et al., 1991; Craske, 1999; Stapinski et al., 2010). To the extent that a sense of control over scary thoughts is important, self-efficacy for exercising such control should be important as well (Fentz et al., 2013; Gallagher et al., 2014; Kent & Gibbons, 1987; cf. Goldin et al., 2012).

Treatment of Phobia and Compulsion

Psychological approaches to helping people to overcome avoidance and rituals, subjective fear and panic attacks, and worries, obsessions, and intrusive bothersome thoughts, have achieved notable successes in recent decades, although much room for improvement remains (Stewart & Chambless, 2009). Behavioral, social, and cognitive interventions can benefit these problems very much in many cases. I begin with reviewing treatments for phobia and compulsive behavior as they are important in their own right and gain additional importance because reducing such behavior robustly reduces the subjective fear, panic, obsessions, intrusive thoughts, and depressed mood that often come with it.

Performance-Based Exposure Treatments Historically pivotal to phobia treatment was systematic desensitization (Wolpe, 1958) and similar methods derived from it, that guide people to imagine performing their avoided activities. Also influential were modeling methods in which people watched others doing the phobia-related activities. The next major advance was performance-based treatment (sometimes called *in vivo* exposure), in which people directly perform their avoided activities (e.g., Agras, Leitenberg, & Barlow, 1968; Bandura, Blanchard, &

Ritter, 1969). In performance-based treatment of compulsive rituals, the person engages in ritual-provoking activities (e.g., touches the contaminated door handle or thinks "Damn God!") but then refrains from the rituals. This method is also called exposure and response (or ritual) prevention. Such performance-based methods are reliably more effective than methods based on only imagining or viewing the activities (Bandura et al., 1969; Steketee & Barlow, 2002).

Treatment focused entirely on guiding the person to engage in the phobic activity, with no explicit attention to thinking or feeling, can enduringly eliminate specific phobias and their negative cognitive and emotional accompaniments within a few hours in many cases, and with widely generalized benefits (Bandura et al., 1969; Ollendick et al., 2009). Even apparently nonbehavioral problems, such as worrying and catastrophizing, can be sustained in part by subtle avoidance behaviors that insulate people from corrective learning experiences (Rachman, Radomsky, & Shafran, 2008). Because avoidance behavior is a common element of otherwise disparate problems, and because changes in avoidance behavior can powerfully change the subjective anxiety, panic and stress, and intrusive thoughts that often go with it (e.g., Vögele et al., 2010; Williams & Falbo, 1996), therapies for diverse problems increasingly place emphasis on identifying and eliminating avoidance behavior. One recent approach is "transdiagnostic" treatment (e.g., Barlow et al., 2011), which seeks to correct dysfunctional avoidance patterns irrespective of diagnosis using mainly methods developed for phobias.

Exposure Concept of Treatment Treatments based on imagining, watching, or actually doing phobia-related activities are sometimes called collectively exposure treatment. The exposure concept likens treatment to Pavlovian extinction, in which unreinforced exposure to the conditioned fear stimulus classically deconditions the person's fear and avoidance (e.g., Foa & McNally, 1996). Contact with relevant stimuli is required to learn just about anything. But identical durations of contact with phobic stimuli routinely produce highly disparate degrees of change in phobic behavior, between treatment groups and between individuals within groups (e.g., Williams, Dooseman, & Kleifield, 1984; Williams & Zane, 1989). The exposure idea cannot explain such variability in benefit because it casts treatment in passive mechanistic terms and points to fixed outer stimuli instead of to flexible inner mental processes.

Guided Mastery Treatment Treatment based on self-efficacy theory or guided mastery treatment (Williams, 1990) seeks to build a strong sense of self-efficacy by fostering people's performance accomplishments. Although the belief that one can do an activity is a cognition, the best way to acquire such a cognition is from firsthand success at doing the activity (Bandura, 1997). Therefore, the guided mastery therapist assists people to succeed at tasks that otherwise would be too difficult, while guiding them to do the tasks proficiently, free of embedded defensive maneuvers and self-restrictions that limit their perceptions of success and thereby limit the benefits they gain from success (Williams et al., 1989; Williams, 1990). Assisting people to do what they fear usually produces better results than does simply encouraging them to expose themselves to scary stimuli (Feske & Chambless, 1995; Gloster et al., 2011; Öst et al., 1997; Williams et al., 1984; Williams & Zane, 1989). Nevertheless, a small but not trivial minority of people with phobias fail to benefit much or at all from current performance-based *in vivo* exposure treatments, including even some people who are motivated, diligent, resourceful, cooperative, and free of other serious problems.

Cognitive Therapies Investigators have sought to boost the power of performance-based exposure treatments by engaging people's mediating cognitive processes by verbal means. Cognitive therapies designed to help people to change their overestimations of danger and to adopt alternative, less self-undermining ways of construing events, are widely used in combination with exposure methods to treat phobic avoidance and rituals (Beck et al., 1985). It is not always easy to show that cognitive therapies add to the benefits of exposure treatments (Feske & Chambless, 1995; Fischer & Wells, 2005; Meuret, Wolitzky-Taylor, Twohig, & Craske, 2012; Olatunji et al., 2013; Williams & Falbo, 1996). Nevertheless, verbal-cognitive interventions appear able to contribute benefits beyond the benefits of exposure alone (Clark, 1999).

Treatment of Anxiety

Treatment of Subjective Anxiety People usually feel anxious about something in particular, and often avoid it as well. Mastering the avoidance behavior alone often eliminates any accompanying subjective anxiety as a byproduct (e.g., Bandura et al., 1969; Öst et al., 1997). When people's anxiety fails come to an end despite having performed the activity for sufficient time, social cognitive theory suggests that the therapist needs to guide the person to abandon subtle avoidance maneuvers, defensive activities, and self-protective rituals that circumscribe their sense of mastery and thereby prolong their fear (Williams, 1985, 1990; Williams & Zane, 1989).

That phobic people engage in such defensive behaviors, now widely called "safety behaviors," when they perform phobia-related tasks, and the importance of such behaviors for the treatment of subjective anxiety, were first described by me (Williams, 1985, pp. 123–4). I called these behaviors "embedded defensive activities" and pointed out that they include rituals (e.g., carrying a bottle of water in case one's mouth dries out) and subtle self-restrictions on performance, such as driving an

automobile on the freeway but staying only in the slow lane. I concluded,

> Embedded defensive activities may in part explain why some people perform feared activities for extended periods with little or no decline in subjective distress. If the evaluator of anxious clients focuses only on gross avoidance (i.e., not going to work, not flying in airplanes) he or she may overlook these embedded avoidance behaviors and erroneously conclude that the client "doesn't avoid anything." It is especially important to identify embedded performance restrictions when formulating treatment plans because such maneuvers can provide a basis for direct behavioral intervention to help clients learn to function more effectively and with less discomfort.
>
> (Williams, 1985, p. 124)

We tested the impact that embedded defensive behaviors have on anxiety reduction during treatment with agoraphobic people by treating them with either guided mastery therapy or *in vivo* exposure alone (Williams & Zane, 1989). Both treatment groups received individual therapist-supervised *in vivo* exposure to the same exposure settings for the same duration of time. The groups differed only in that guided mastery therapists engaged and corrected participants' defensive behaviors, whereas exposure-only therapists urged participants to fully expose themselvcs but did not attended to their embedded defensive behaviors. The results showed that guided mastery treatment produced significantly greater anxiety reductions during post-treatment behavioral tests than did exposure alone, and that this treatment advantage increased significantly more during the follow-up period.

Although embedded defensive activities (embedded in the performance of a feared activity) were renamed and reinterpreted by Salkovskis (1991) as "safety behaviors," the label and the interpretation of the behaviors in terms of safety/danger are open to challenge. Such behaviors and their therapeutic importance were discovered in the light of self-efficacy theory, which sees them not as safety behaviors but as *confidence* behaviors (i.e., behaviors that affect self-efficacy), a fact that can be put to good therapeutic use as explained in detail by Williams and Zane (1989) and Williams (1990). To illustrate, when an agoraphobic woman first attempts to enter a supermarket, she might be more able and confident if she carries a bottle of water with her. The therapist can recommend that she carry water to help her succeed when she first tries to enter the market. But thereafter the confidence behavior can *undermine* her confidence if she thinks, "I can go in there only because I take water with me." The confidence behavior thus can switch from a confidence-boosting aid to a confidence-undermining dependency. The guided mastery therapist uses confidence behaviors but quickly withdraws them so people learn to function without them (Williams, 1990). Scientists ponder whether "safety behaviors" help or hurt; the answer is that they can do both, depending on how and when the person uses them.

Subjective anxiety has long been treated by guiding people to imagine themselves encountering stressors and coping effectively with them, and by a variety of relaxation and cognitive reappraisal treatment methods under the broad label cognitive-behavioral therapy (Clark, 1999; Craske, 1999; Barlow, 2002; Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012). Some recent trends have explored the combination of conventional cognitive-behavioral methods with techniques based on acceptance and commitment therapy and mindfulness therapy, with promising results (Hayes-Skelton, Roemer, & Orsillo, 2013; Hofmann, Sawyer, Witt, & Oh, 2010).

Treatment of Panic Attacks Most people with panic attacks display some avoidance, gross or subtle (Williams, 1985; Williams & Falbo, 1996), so performance-based therapies for avoidance can have a notable impact on panic attacks (Barlow, 2002). Treatment methods focused specifically on panic include cognitive therapies designed to increase rational appraisal and decrease catastrophic thoughts, somatic interventions to induce autonomic arousal or other bodily responses in panic (sometimes called interoceptive exposure), and breathing and relaxation techniques designed to normalize and calm physiological arousal (Craske, 1999).

A meta-analysis of 13 studies of panic treatment with cognitive-behavioral therapies, with diverse selection criteria, found rates of panic-free participants after treatment ranging from 53% to 85%, with a weighted mean of 71% (Chambless & Peterman, 2004). Many studies have preselected panickers with few phobias and few other problems, who are easier to treat than panickers with other problems (Sanchez-Meca, Rosa-Alcazar, Martin-Martinez, & Gomez-Conesa, 2010; Williams & Falbo, 1996).

Treatment of Troubling Thoughts

Helping people with troubling thoughts often involves a combination of elements administered together. Worries and obsessions are directed toward specific activities or stimuli, and are often accompanied by mental or behavioral avoidance reactions or neutralizing rituals, reassurance seeking, and other actions that paradoxically maintain and increase intrusions (Craske, 1999; Rachman, 1998; Salkovskis, 1996). Therefore, the most widely used technique, regardless of whether the thoughts are worries, obsessions or visualized future calamities, is with enactive or imaginal performance of avoided activities, with response prevention as appropriate for rituals (e.g., de Silva, Menzies, & Shafran, 2003). Thus, a person can deliberately think an intrusive thought then practice not mentally reacting to it. Cognitive treatments (Clark, 1999; Craske, 1999; Rachman & Shafran, 1998) contain a variety of verbal elements, including dialogue aimed at conveying that obsessions are normal, challenging excessive responsibility beliefs and catastrophic meanings, increasing tolerance of uncertainty, and methods to alter

problematical imagery, but without trying to decrease the number of intrusive thoughts directly (Salkovskis, 1996). Cognitive therapy generally helps (e.g., Hanrahan, Field, Jones, & Davey, 2013).

Innovative Treatments

Recent years have witnessed an efflorescence of innovative "third wave" treatments for the problems under our consideration here (Schmidt, 2012). Space and scope do not permit more than a simple mention of some of these as promising possibilities.

Treatments designed to alter people's information-processing biases away from fear-related information towards more benign information can have a beneficial effect on anxiety (Amir & Taylor, 2012; Mathews & MacLeod, 2005; See, MacLeod, & Bridle, 2009) although the size of the effect appears relatively small (Hallion & Ruscio, 2011) and bias retraining failed to alter maladaptive social behavior (Bunnel, Beidel & Mesa, 2013) or to integrate profitably into a standard cognitive-behavioral treatment program (Rapee et al., 2013).

Other new therapies include acceptance and commitment therapy (Arch et al., 2012; Swain, Hancock, Hainsworth & Bowman, 2013), mindfulness-based therapies (Boettcher et al., 2014; Hofmann et al., 2010; Khoury et al., 2013), and "technology-assisted" e.g., Internet-based self-help and minimal therapist contact methods (Newman, Szkodny, Llera, & Przeworski, 2011; Anderson et al., 2013; Haug, Nordgreen, Öst & Havik, 2012). All of these are early in their testing but have shown some promising results (Khoury et al., 2013; Hayes-Skelton et al., 2013; Swain et al., 2013; Hofmann et al., 2010).

Prevalence of Disorders or Distribution of Psychosocial Problems?

The extent to which psychological problems occur in the community has usually been measured as prevalence rates of mental disorders rather than as frequency distributions of dimensional scores. Probably just about everybody has experienced needless avoidance, fears, and unpleasant thoughts now and again, so their prevalence as mental disorders is largely in the eye of the beholder, dependent on which responses and cutoffs one chooses to define disorder. For example, in one analysis the prevalence of DSM-III generalized anxiety disorder dropped from 45% to 9% of the population when the required duration of anxiety increased from 1 month to 6 months and the number of defining anxiety responses increased from three to six (Breslau & Davis, 1985).

The prevalence of social anxiety disorder ranges from about 2% to 19% of the population, depending on the cut-off scores one chooses and on how interviewers pose their questions (Stein, Walker, & Forde, 1994). The prevalence of obsessive-compulsive disorder using the DSM-IV definition is about one-half of that using the DSM-III definition (Crino, Slade, & Andrews, 2005). A reanalysis of epidemiological findings applying the DSM-IV clinical significance criterion lowered the U.S. national prevalence of mental illnesses by 19.2 million people at a single stroke (Narrow, Rae, Robins, & Regier, 2002). Plainly, the extent of mental problems in the population would be characterized far better by distributions of problem severity dimension scores than by frequency counts of mental disorders. But epidemiological findings (e.g., Kessler et al., 2005) nevertheless show that psychological problems of the kinds we have been considering are common.

Conclusion

I have encouraged readers to approach anxiety disorders and obsessive-compulsive disorders by questioning the meanings of both "anxiety" and "disorders". Calling psychological phenomena by their own proper names without a pseudo-unifying anxiety label removes a comforting illusion of understanding, but it helps us to see psychosocial problems more clearly on their own terms. Although we say glibly that people with phobias avoid because they are anxious, the evidence favors the far different conclusion that people with phobias avoid and feel anxious because they have maladaptive thoughts.

Perhaps more important is to question mental disorder theory. Most critics of DSM conclude with a wish to use mental dimensions to develop better measures of mental disorders. I disagree wholeheartedly. Here and now, psychosocial dimensions serve our research and treatment needs in every way, and without the distortions of mental disorders. The study of anxiety, phobia, compulsion, and troubling thoughts is only undermined by conceiving these problems as psychopathology. Neither medicine nor psychiatry is wedded to a wrong theory of behavior. Far from subordinating the science of mental measurement to the medical diagnosis model, which would be like the victorious General Grant surrendering his army to the defeated General Lee in Mark Twain's tall tale, scientists can celebrate the ascendance of variability-based psychosocial problems and the retirement of pathology-based mental disorders, and never look back.

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10

Trauma- and Stressor-Related Disorders

Lori A. Zoellner, Belinda Graham, and Michele A. Bedard-Gilligan

Trauma- and stressor-related disorders are a new grouping of disorders introduced in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), whose etiology pairs the onset of the mental disorder with the presence of an external event. These events range from extreme high-magnitude stressors such as torture, terror attacks, rape, childhood sexual abuse, natural disasters, or war, to more commonly experienced stressors such as the loss of a romantic relationship, persistent physical illness, or business difficulties. Ascribing the etiology of a mental disorder to an external event as a causal mechanism is difficult at best. A large number of mental disorders (e.g., major depression, panic disorder, schizophrenia, social phobia, specific phobia, generalized anxiety disorder, substance use disorders) are likely to begin, or are exacerbated, after a stressor (e.g., de Graaf, Bijl, Ravelli, Smit, & Vollenbergh, 2002; Klauke, Deckert, Reif, Pauli, & Domschke, 2010). Yet, the presence or the nature of the stressor often leaves a large portion of the variance of who develops a mental disorder unexplained (e.g., Kendler & Gardner, 2010). This is even the case with disorders such as posttraumatic stress disorder (PTSD; Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003; Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012), which argues for the need for multifactorial gene and environment etiologic models.

Stressors and Natural Recovery

A much larger debate is the pathologizing of normal human suffering and the overdiagnosis of disorders such as PTSD. Traumatic stressors are actually quite common (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), with lifetime prevalence of trauma being 60.7% for men and 51.2% for women in a representative U.S. sample. After a traumatic event of a significant magnitude, the majority of individuals will have psychological reactions such as insomnia, irritability, reactivity to reminders, and avoidance of related thoughts or feelings. These reactions usually abate within the first 3 months after the event and continue to decline over time, even for high-magnitude stressors (e.g., Foa & Riggs, 1995; Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992; Riggs, Rothbaum, & Foa, 1995). The presence of early reactions is not considered pathological, does not consistently predict the development of long-term psychopathology, and instead represents a "natural recovery curve" (Figure 10.1). Accordingly, disorder is characterized by the persistence of these reactions over time and not by their initial presence. Conversely, if the threat of the trauma persists, it is logical to expect that reactions will persist and are not pathological.

Not all stressors confer the same degree of conditional risk for the development of disorder. Events such as being a prisoner of war, combat, and rape confer a much higher risk of PTSD than witnessing an event or experiencing a natural disaster (e.g., Breslau, Chilcoat, Kessler, & Davis, 1999; Kessler et al., 1995). For example, in the Breslau study, assaultive violence conferred more than 10 times the risk for developing PTSD than learning about a trauma to a loved one, the reference type with the lowest risk of PTSD. Notably, the idea that not all traumatic events or types of exposure (e.g., direct versus witnessing) confer the same risk is sometimes counterintuitive. For events like natural disasters and shootings on college campuses, the scale of the event heightens public perception of elevated risk and need for psychiatric intervention although rates of PTSD following these types of events is relatively low. Meta-analytic studies (Brewin et al., 2000; Ozer et al., 2003; Trickey et al., 2012) have further identified predictive factors associated with PTSD. For example, as reported by Brewin et al. (2000), pre-existing factors such history of psychopathology ($r = .11$), previous child abuse ($r = .14$), being female ($r = .13$),

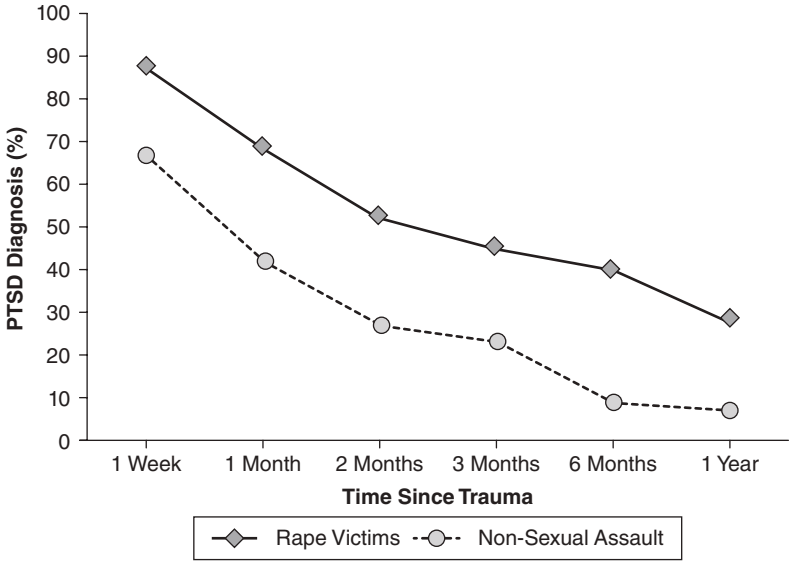

Figure 10.1 Patterns of natural recovery for individuals experiencing either sexual or non-sexual assault up to 1 year following trauma exposure. Posttraumatic stress disorder (PTSD) diagnosis was made using interviewer-rated PTSD based on DSM-IV. At 1 week, PTSD diagnosis was based on symptom but not duration criteria. Data obtained from the original datasets used in publications from Rothbaum, Foa, Riggs, Murdock, and Walsh (1992); Riggs, Rothbaum, and Foa (1995); and Foa and Riggs (1995).

and lower intelligence (r = .18) are weaker predictors of PTSD. Trauma-specific and post-event factors, however, such as severity of the event (r = .23), lack of social support (r = .40), and ongoing post-event stress (r = .32) tend to be stronger predictors for of PTSD. Similarly, military models emphasize post-conflict factors as conferring increased risk (e.g., King, King, Fairbank, Keane, & Adams, 1998); and, among refugees who have experienced trauma, post-migration problems including difficulties with adaptation, distress, loss of culture, and lack of social support increase the risk of PTSD (e.g., Carswell, Blackburn, & Barker, 2011).

Thus, traumatic stressor exposure is common and, for the majority of individuals exposed to stressors, normal psychological reactions that occur in the immediate aftermath will abate within days or months of the event. Psychopathology does not reflect the presence of these reactions but rather the persistence of these reactions, which occurs for only a minority of individuals. Pre-event factors confer some conditional risk to the development of long-term pathology, but event and post-event factors tend to be stronger predictors and a large portion of variance remains unexplained. Any etiological model for trauma- and stressor-related disorders must explain these consistent findings.

Prevalence and Co-occurrence

In a national U.S. sample, the lifetime prevalence of PTSD in women is 10.4% and in men is 6.8% (Kessler et al., 1995). World prevalence is difficult to estimate, although anxiety disorders, including PTSD, are the most commonly occurring disorders in the Americas, Europe, Africa, Middle East, and Asia (Goldney, Fisher, & Hawthorne, 2004). Global rates of PTSD vary widely, most likely due to diagnostic methodology and culturally sensitive translation. The actual incidence of trauma across regions differs hugely. For example, one study in Sierra Leone, which has been in a long civil war, reported that 99% of individuals met PTSD criteria (de Jong, Mulhem, Ford, van der Kam, & Kleber, 2000). Although PTSD is diagnosed worldwide, it remains unknown how well the disorder in its current form is substantiated across cultures.

As might be expected, rates of PTSD vary across specific samples, with higher rates in veterans, active duty military service members, and refugees. Rates of PTSD from the National Vietnam Veterans Readjustment Survey range from 18% to 31% depending on methodology (Dohrenwend et al., 2006). In recent U.S. wars, 12–13% of veterans report experiencing PTSD (e.g., Hoge et al., 2004). Among refugees resettled in Western countries, a systematic review found that almost 1 in 10 had symptoms consistent with PTSD and 1 in 20 had symptoms consistent with depression (Fazel, Wheeler, & Danesh, 2005).

PTSD shows a high degree of co-occurrence with a variety of other mental disorders. In the National Comorbidity Study (Kessler et al., 1995), 88% of men and 79% of women with a lifetime diagnosis of PTSD had at least one other lifetime mental disorder. In both men and women, nearly half of all individuals with lifetime PTSD also experienced a major depressive episode in their lifetime. In men, co-occurring alcohol abuse or dependence (51.9%) and conduct disorders (43.3%) were common. More recently, PTSD was strongly associated with both the anxiety disorders (panic, agoraphobia, simple phobia, social anxiety disorder, general anxiety disorder) and mood disorders (major depressive episodes, dysthymia, manic/hypomanic episodes; Kessler et al., 2005). This simply argues that, as conceptualized, there is considerable overlap in PTSD with both anxiety and depressive disorders (e.g., Zoellner, Pruitt, Farach, & Jun, 2014).

Diagnostic Considerations

The DSM-5 trauma- and stressor-related category (American Psychiatric Association, 2013) includes seven disorders: PTSD, acute stress disorder, adjustment disorders, other specified trauma- and stressor-related disorders, unspecified trauma- and stressor-related disorder, reactive attachment disorder, and disinhibited social engagement disorder. The latter two are disorders of childhood and will not be discussed.

PTSD and acute stress disorder share the same traumatic stressor criteria involving "exposure to a threatened death, serious injury, or sexual violation" (pp. 271, 280) and include direct experiencing, witnessing, or learning about a stressor. DSM-5 PTSD includes the presence of four clusters of 20 symptoms: intrusions; avoidance; negative alteration in cognitions and mood; and arousal and reactivity. These symptoms must last for at least 1 month, cause clinically significant distress or impairment in functioning, and be not attributable to the effects of a substance or other medical condition (Box 10.1). In contrast, acute stress disorder can only be diagnosed in the first month following trauma exposure and symptoms must persist for at least 3 days. Nine of 14 symptoms regardless of cluster are required. These symptoms are a subset of the PTSD symptoms and include the additional dissociative symptoms of depersonalization or derealization.

Box 10.1 Summary of DSM-5 Posttraumatic Stress Disorder Diagnostic Criteria

A. Actual or threatened death, serious injury, or sexual violence. One or more of the following ways:
   1. Direct experience
   2. In-person witnessing
   3. Learning about such exposure to a close family member or friend
   4. Repeated or extreme exposure to details of the traumatic experience(s)

B. Intrusion symptoms associated with the traumatic event(s). One or more of the following:
   1. Intrusive memories of the traumatic event(s)
   2. Distressing dreams related to the traumatic event(s)
   3. Feeling or acting as if the event(s) was happening again
   4. Psychological distress to trauma reminders
   5. Physiological reactions to trauma reminders

C. Persistent avoidance of trauma-related stimuli. One or both of the following:
   1. Avoidance of trauma-related memories, thoughts, or feelings
   2. Avoidance of trauma-related external reminders

D. Negative cognitions and mood associated with the traumatic event. Two or more of the following:
   1. Lack of memory for important aspects of the traumatic event(s)
   2. Negative beliefs about self, others, or the world
   3. Distorted cognitions about the cause or consequences of the trauma(s)
   4. Persistent emotional distress
   5. Diminished interest in important activities
   6. Detachment from others
   7. Inability to feel positive emotions

E. Arousal or reactivity associated with the traumatic event. Two or more of the following:
   1. Irritability or angry outburst
   2. Reckless behavior
   3. Hypervigilance
   4. Exaggerated startle response
   5. Concentration problems
   6. Sleep problems

F. Duration of Criteria B, C, D, and E for more than 1 month

G. Clinically significant distress or impairment in important areas of functioning

H. Disturbance not due to physiological effects of a substance or medical condition

Specify whether:

With dissociative symptoms: Either depersonalization or derealization

Specify if:

With delayed expression: Criteria must not be met until 6 months after the event(s)

Adapted from the *Diagnostic and Statistical Manual of Mental Disorder*, 5th edn. (American Psychological Association, 2013)

The diagnosis of acute stress disorder has long been one of considerable controversy, owing to its lack of predictive power for the development of PTSD and its conceptual problem of separating a continuous phenomenon into two separate disorders of acute stress disorder and PTSD (e.g., Marshall, Spitzer, & Liebowitz, 1999). The DSM-5 concedes, "approximately half of individuals who eventually develop PTSD initially present with acute stress disorder" (American Psychiatric Association, 2013, p. 284). Notably, the *International Classification of Diseases* 11th revision (ICD-11) working group (Maercker et al., 2013) regards acute reactions as normal and recommends that acute stress be transferred from the stressor-related disorder category into a section representing reasons for clinical encounters that are not themselves a disorder or disease.

Adjustment disorder, other specified trauma- and stressor-related disorder, and unspecified trauma- and stressor-related disorders are generally considered "catch all" or "residual" diagnoses (i.e., clinically significant psychiatric impairments that do not meet threshold criteria for PTSD or other mental disorders such as depression or anxiety disorders, such as culturally specific syndromes or persistent complex grief reactions). These additional diagnoses allow for capturing the wide range of reactions seen to stressors. In particular, in adjustment disorder, there is marked distress that is out of proportion to the severity or intensity of the stressor and emotional or behavioral symptoms that develop within 3 months and dissipate within 6 months of the stressor. Indeed, PTSD symptoms commonly occur in those adjusting to various life stressors, even when the stressor does not meet the definition of a traumatic event (e.g., Gold, Marx, Soler-Baillo, & Sloan, 2005). In surveys of clinicians, adjustment disorders are some of the most common diagnoses identified (Maercker et al., 2013). Yet, there are concerns that this disorder is too broad, insufficiently researched, and may reflect the presence of somatic symptoms, particularly for those with medical disorders (e.g., Bisson & Sakhuja, 2006). Finally, a diagnosis of adjustment disorder is no longer considered for those with major depression within 6 months after the death of a loved one. This is based on little or no systematic differences between individuals who develop a major depression in response to bereavement or in response to other severe stressors (e.g., Kendler, Myers, & Zisook, 2008).

DSM-5 and ICD-11 Definitions of PTSD The changes for PTSD in DSM-5, compared with DSM-IV, appear relatively minor and should not have a substantial impact on prevalence or conceptualization of the diagnosis. The diagnosis has retained the vast majority of symptoms (e.g., intrusions, avoidance, hyperarousal). The changes include shifting PTSD from an anxiety disorder to a newly created category of trauma- and stressor-related disorders, a redefinition of a traumatic event, a shifting of the clusters of symptoms, including adding four more symptoms (negative beliefs/expectations, distorted blame, persistent negative emotions, reckless or self-destructive behavior), and the creation of a dissociative subtype. Similarly, in the proposed ICD-11 (Maercker et al., 2013), stressor-related disorders are grouped together, ranging from common negative life events to extreme traumatic stressors. Particular attention was made to eliminate non-specific symptoms; and, as such, the proposed ICD-11, for PTSD, focuses specifically on three core elements: reexperiencing, avoidance, and the perception of heightened current threat (arousal symptoms). In addition, a diagnosis of complex PTSD is being considered for ICD-11. This diagnosis focuses on severe, prolonged or repeated stressors and includes the three core components of PTSD and additional components of enduring disturbances of affect, self, and interpersonal relationships. Although some evidence is emerging for this distinction (e.g., Cloitre, Garvert, Brewin, Bryant, & Maercker; 2013), the evidence for a distinct disorder is not strong to date (e.g., Resick et al., 2012) and, as such, was not included in DSM-5.

As noted previously, in DSM-5, PTSD moved from the anxiety disorders to a new "trauma- and stressor-related disorder" category. The evidence base for this shift is relatively weak (Craske et al., 2009; Zoellner et al. 2014; Zoellner, Rothbaum, & Feeny, 2011), although argued for by the DSM-5 committee (Friedman et al. 2011). Refocusing attention on the "trauma" itself, rather than on anxiety, fear, and threat, may make clinicians more inclined to consider PTSD diagnosis over other disorders commonly associated with reactions to stressors, such as depression, borderline personality disorder, and generalized anxiety disorder. Clinicians also run the risk of making an "attribution bias" error, attributing the presence of symptoms to an external source. This may be particularly problematic with depression, given its high overlap of symptoms with PTSD.

DSM-5 revised the parameters for what constitutes a traumatic stressor for acute stress disorder and PTSD. The objective component of the stressor has been further clarified. This clarification helps address "bracket creep," referring to the expansion of the definition of trauma beyond its intended boundaries. Indirect exposure is now defined as, "learning that the traumatic event occurred to a close family member or close friend" in which the "actual or threatened death must have been violent or accidental" (American Psychiatric Association, 2013, p. 271). The definition also includes "experiencing repeated or extreme exposure to aversive details of the traumatic event," such as with responders collecting human remains (p. 271). The DSM-5 definition now explicitly excludes witnessing traumatic events through electronic media, unless in a vocational role (e.g., television journalists). However, the boundary between what is and is not a trauma is still murky. The veracity of the event is not addressed, allowing for PTSD following a hypnagogic episode or other hallucinations or false memories (e.g., alien abduction, implanted memories). Further, some events (e.g., sexual harassment, cancer, heart attack, complicated childbirth) do not universally fit the spirit of the definition of a trauma. In these circumstances, the unique characteristics of the specific event need to be considered before assuming that

the criterion A stressor definition has been met. Finally, in DSM-5, the requirement that an event involve a subjective response of intense fear, helplessness, or horror has been eliminated, reducing conceptual risk of confounding the stimulus and response and following empirical data showing a lack of additional predictive ability of the subjective criteria (e.g., Bedard-Gilligan & Zoellner, 2008).

Owing to lack of empirical support, DSM-5 moved away from a three-cluster model (reexperiencing, avoidance, hyperarousal) of PTSD. In factor analytic studies, a fourth factor of either "dysphoria" or "emotional numbing" commonly emerges (e.g., King, D. W., Leskin, King, L. A., & Weathers, 1998; Simms, Watson, & Doebbelling, 2002), largely differing based on the placement of irritability/anger and sleep problems. DSM-5 did not adopt either of these four-factor models but created a new "negative alterations in cognitions and mood" symptom cluster. Based on U.S. veteran (N = 345) and U.S. national (N = 2,953) samples, DSM-5 field trial data (Miller et al., 2013) showed that the DSM-5 factor structure (i.e., reexperiencing, avoidance, negative alterations in cognitions and mood, hyperarousal) provided an adequate fit for the data. However, a version of the four-factor dysphoria model slightly improved the model fit. Further, neither psychogenic amnesia nor the new symptom of risky behavior fit well with other PTSD symptoms. Despite this, and the body of literature consistently finding poor fit for psychogenic amnesia (e.g., King et al., 1998), these symptoms were included in DSM-5. As discussed by Miller and colleagues, the psychogenic amnesia item may reflect greater symptom severity or a dissociative subtype of PTSD and the risky behavior item was retained to reflect an important symptom often found among adolescents.

DSM-5 now includes a diagnostic specifier of "with dissociative symptoms." This requires the presence of either depersonalization described as "out-of-body" experiences, such as observing oneself from the outside, or derealization described as the perception of unreality or being in a dreamlike state. Research with both veteran and civilian samples (e.g., Wolf et al., 2012) have found that individuals with dissociative presentations represent distinct patient classes. Other studies, however, argue that dissociative presentations reflect a continuous construct (e.g., Ruscio, Ruscio, & Keane, 2002). This calls into question whether or not dissociative reactions seen in PTSD are indicative of a qualitatively distinct subtype.

Finally, with DSM-5's expansion of PTSD symptoms from 17 to 20 and the inclusion of non-specific general distress-related symptoms, concerns about the heterogeneity of the diagnosis have been raised (e.g., Galatzer-Levy & Bryant, 2013; Zoellner et al., 2014). Galatzer-Levy et al. (2013) point out that, although DSM-IV criteria suffered from considerable symptom profile heterogeneity (79,794 combinations), DSM-5 changes result in an eightfold increase (636,120 combinations). This argues that care needs to be taken when examining PTSD as a unified, distinct phenotype, recognizing the vast variability within the diagnosis.

Etiology

Biological and Genetic Factors

Genetic Factors Approximately one-third of the variance in PTSD may be attributed to genetic factors (e.g., Stein, Jang, Taylor, Vernon, & Livesley, 2002). Large-scale genome-wide association studies of PTSD are in their infancy. They either have not yielded conclusive associations with PTSD (e.g., Shen et al. 2013) or have yielded associations broadly associated with other psychiatric disorders (e.g., Solovieff et al., 2014), arguing for a general rather than a specific vulnerability as suggested by a genetic psychopathology "p" factor (Caspi et al., 2013).

The vast majority of PTSD research to date has used a candidate-gene approach, where allele or genotype frequencies based on some underlying hypothesized mechanism are compared. The most studied candidate gene to date is 5-HTTLPR polymorphism, which is related to serotonin transport and reuptake and has been implicated in anxiety and depressive disorders. It involves both a long (L) and a short (S) allele, with the S allele reducing serotonergic expression and uptake. In a meta-analysis, Gressier and colleagues (2013) found no strong association between 5-HTTLPR and PTSD but noted potential differences between men and women and the presence of an association for highly trauma-exposed individuals.

Among other candidate genes potentially associated with PTSD, two additional genetic associations in PTSD that are noteworthy are FK506-binding protein 5 (FKBP5), a marker of the hypothalamic–pituitary–adrenal (HPA) axis (e.g., Mehta et al., 2011), and a brain-derived neurotropic factor (BDNF) Val66Met SNP (e.g., Zhang et al., 2006). FKBP5 is involved in glucocorticoid functioning and may contribute to increased sensitivity of the amygdala/HPA axis response to stress. BDNF Val66Met SNP carriers show impaired extinction learning, which is associated with altered activation of the amygdala, prefrontal cortex, and hippocampus (e.g., Soliman et al., 2010). Other candidate genes in PTSD have focused on the serotonergic, noradregeneric, and dopaminergic systems and other markers of the HPA axis. As seen in the 5-HTTLPR polymorphism above, across candidate-gene studies, to date, replication has been an issue, with most studies being underpowered and having low rates of PTSD.

Both 5-HTTLPR and FKBP5 SNPs have also been implicated in gene and environment interactions in the development of PTSD. These studies examine whether the effect of a genotype differs by the presence or the absence of an environmental pathogen or vice versa. The presence of these interactions, which alter main effects, may account for the lack of candidate-gene replication discussed above. In these studies, the environmental pathogens are not typically considered the trauma

exposure itself but instead some form of developmental adversity (e.g., childhood abuse sexual abuse, maltreatment) or adult adversity (e.g., living in a high crime area, low socioeconomic status). The S allele is associated with an increased risk of PTSD in high-risk environments (e.g., Grabe et al., 2009). Similarly, FKBP5 SNPs may interact with childhood abuse or adversity to confer a risk for adult PTSD (e.g., Binder et al., 2008).

In the coming years, research on epigenetic regulation in PTSD is going to increase. Epigenetic mechanisms focusing on regulation of the chromatin structure and DNA methylation are potent regulators for gene transcription in the central nervous system. Notably, epigenetic molecular mechanisms may underlie the formation and stabilization of both context- and cue-triggered conditioning based in the hippocampus and amygdala, consistent with the focus on long-term potentiation and related synaptic plasticity (Zovkic & Sweatt, 2013). As evidenced in the emerging research literature, Mehta et al. (2013) reported that in PTSD changes in DNA methylation have a greater impact in those with exposure to childhood maltreatment and that these changes may mediate the gene-expression changes.

Brain Regions and Systems of Interest Neuroimaging in PTSD has primarily focused on brain regions most strongly implicated in fear conditioning and extinction: the sensory cortex, dorsal thalamus, lateral and central nucleus of the amygdala, hippocampus, and structures in the medial prefrontal cortex, including the anterior cingulate cortex. Two of the most recurrent findings in individuals with PTSD are decreased medial prefrontal cortex activation and increased amygdala activation (e.g., Francati, Vermetten, & Bremner, 2007). This is broadly consistent with a view of a car "accelerator" and "brake" model of fear activation (VanElzakker, Dahlgren, Davis, Dubois, & Shin, 2013), whereby the prelimbic cortex acts as a fear response accelerator during conditioning and the ventral infralimbic cortex acts as brakes during extinction. There are consistent gray matter reductions in the anterior cingulate cortex and ventromedial prefrontal cortex, the left temporal pole/middle temporal gyrus, and the left hippocampus in patients with PTSD compared with trauma-exposed individuals without PTSD (Kühn & Gallinat, 2013). This is consistent with a view of reduced top-down control of the amygdala, resulting in its hyper-responsiveness to fearful stimuli.

Both the prefrontal cortex and amygdala have connections to the hippocampus. The hippocampus, which is associated with conditioned fear and associative learning, has also been implicated in PTSD. In particular, it is associated with explicit memories of the traumatic event and learned responses to contextual cues. In PTSD, there is increased hippocampal activation to trauma and emotional stimuli (e.g., Gilbertson et al., 2002). Lower hippocampal volume also may be a vulnerability factor for developing PTSD, though data on early stress, trauma, or PTSD "damaging the brain" by reducing hippocampal volume are mixed (Gilbertson et al., 2002).

The HPA axis, which includes the hypothalamus, the pituitary gland, and adrenal gland, is also involved with stress and the regulation of emotions. It is logical that impairments in the functioning of this system in PTSD, as evidenced by the stress hormone cortisol, would be present, yet the field has been plagued with inconsistent findings. A large meta-analysis (Klaassens, Giltay, Cuijpers, van Veen, & Zitman, 2012) showed no differences in basal cortisol between individuals who were exposed to trauma in adulthood and non-trauma-exposed controls. However, in a smaller subset of studies, using a low-dose dexamethasone suppression test, cortisol suppression was higher in individuals with adult trauma exposure than controls, suggesting that adult trauma exposure, but not PTSD *per se*, is associated with a stronger HPA axis feedback response. This is consistent with results following child trauma exposure (e.g., Carpenter et al., 2007). Lack of consistent findings in PTSD may reflect the multitude of factors that influence cortisol functioning, the presence of other psychopathology such as depression, and considerable heterogeneity in the type of trauma exposure.

Psychosocial Models of Etiology

Classical Conditioning and Extinction Models The proximal cause in PTSD is considered to be exposure to a traumatic event. One of the most obvious explanations for the development of subsequent fear and PTSD symptoms is classical (Pavlovian) conditioning. In a conditioning model, the experience of danger or perceived danger (an unconditioned stimulus) leads to the development of learned danger signals (conditioned stimuli). Previously neutral stimuli become paired with the traumatic event (e.g., time of day, sounds, people, conditioned stimulus) and lead to the development of persistent anxiety-based reactions such as reexperiencing of the trauma memory, physiological reactivity upon exposure to non-dangerous but trauma-related reminders, and avoidance of these reminders.

In PTSD, there is evidence for increased sensitization to conditioning. Relative to trauma-exposed controls, individuals with PTSD show an enhanced conditioned fear response (e.g., Glover et al., 2011) and elevated physiological responding to personally relevant trauma scripts (e.g., Pole, 2007). Further, individuals with PTSD consistently show an attentional bias to threat, characterized by increased attentional capture of potentially threatening information and difficulty disengaging from these cues (e.g., Olatunji, Armstrong, McHugo, & Zald, 2013). They also show greater fear generalization, that is, broadened responding beyond the conditioned stimuli alone, greater difficulty inhibiting fear in response to safety cues (e.g., Jovanovic et al., 2010), and enhanced startle reactivity particularly in stressful or aversive contexts (e.g., Grillon & Baas, 2003). Similarly, avoidance of trauma reminders, a hallmark symptom of PTSD, develops naturally and rapidly as an innate species-specific defense response to protect from danger (e.g., Bolles, 1970) and is guided by classical conditioning about environmental cues (Bouton, 2004).

Yet, the sole focus on classical conditioning fails to capture the broad range of etiologic factors underlying PTSD. The primary criticism is that, if a traumatic event is conceptualized as a central etiologic event, then exposure should almost inevitably result in disorder. However, only a minority of individuals exposed to traumatic events develop persistent PTSD symptoms. To better account for the range of responding following trauma exposure, one intuitively appealing variant of the classical conditioning model is a dose-dependent response or stressor dose model. In this model, the severity, duration, and proximity to a traumatic event, or "dose" of trauma exposure, determine who will and who will not develop PTSD. The model has a solid empirical basis derived from the animal stress research (e.g., Bowman & Yehuda, 2003), from meta-analytic studies in humans showing the predictive value of event-related characteristics such as life threat (e.g., Brewin et al. 2000), and from the predictive value of heightened physiological arousal immediately after trauma exposure (e.g., Coronas et al., 2011). The dose-dependent model provides an easy and practical rubric for conceptualizing initial PTSD risk based on trauma severity.

Enhanced conditioning may also be linked with impairments in extinction learning consistently shown in individuals with PTSD (e.g., Milad et al., 2008). An impaired fear extinction hypothesis posits that individuals who develop chronic PTSD have impairments in learning new inhibitory associations to trauma-related reminders (conditioned stimuli). Repeated presentation of the conditioned stimulus in the absence of the unconditioned stimulus continues to evoke the conditioned response rather than no longer evoking the conditioned response. That is, these reminders continue to signal potential danger for the trauma survivor with PTSD, even when danger is no longer present. This is consistent with the observed pattern after trauma exposure of the initial presence of psychological reactions and then a general decline of these reactions over time, marking adaptive extinction processes, instead of continued reaction over time in those with PTSD, marking pathological extinction processes. Extinction processes are thought to involve learning of a new inhibitory association, which is contextually gated. Extinction serves to make the meaning of the conditioned stimulus ambiguous, potentially signaling threat or not, and internal and environmental contexts disambiguate the meaning of the conditioned stimulus (e.g., Bouton & Swartzentruber, 1991). Accordingly, although learning to be afraid is relatively easy, representing an evolutionarily adaptive excitatory association, learning to not be afraid is a difficult inhibitory association dependent on contextual factors. This is consistent with the evidence reviewed previously showing greater amygdala activation and dorsal anterior cingulate cortex activation, which are associated with fear activation, and less activation in the hippocampus and ventral medial prefrontal cortex which are associated with slowing down or inhibiting fear activation in individuals with PTSD (e.g., Milad et al., 2009). In a study of monozygotic twins discordant for trauma exposure, this extinction deficit was linked specifically to trauma exposure resulting in PTSD (Milad et al., 2008). Thus, extinction deficits seen in PTSD may reflect not a pre-existing vulnerability factor for developing PTSD but instead a mechanism associated with trauma exposure or post-event processing of the traumatic event. Figure 10.2 provides a simplified view of the role of conditioning and extinction processes in PTSD.

Memory Processing Models Original dual memory models (e.g., van der Kolk, 1987; Brewin 1996) explain the development of PTSD by theorizing that impaired memory encoding, due to factors such as stress response and dissociation, leads to poorly integrated and elaborated trauma memories that are difficult to recall verbally and intentionally. These abnormal memory processes are considered causal, particularly for reexperiencing symptoms, and provide a theoretical basis for the debated construct of repressed and later recovered traumatic memories (Lynn, Lilienfeld, Merckelbach, Giesbrecht, & van der Kloet, 2012).

More recently, a revised dual representation model of memory in PTSD provides an account of intrusive memories that makes explicit links to underlying neural processes (Brewin, Gregory, Lipton, & Burgess, 2010). It proposes two separate memory systems, operating in tandem, that become disconnected in PTSD. *Contextual representations* are flexible representations that are consciously accessible, context-dependent, and connected to the inferior temporal cortex, hippocampus, and parahippocampus brain structures. *Sensory representations*, in contrast, are inflexible, involuntary, sensation bound, disintegrated from the autobiographical memory base, and are connected to the superior parietal areas, amygdala, and insula areas of the brain. Owing to the stress associated with a traumatic experience and dissociation, memories of the trauma may be overrepresented as isolated sensory representations. As such, they are thought to be recalled in a fragmented and disorganized way. Recovery involves reactivation of sensory and contextual representations together and integration of them into a complete memory.

Although a meta-analysis suggested that peri-traumatic dissociation, dissociation at the time of the traumatic event, was a strong predictor of PTSD (Ozer et al., 2003), the relationship is strongest in cross-sectional studies and prospective studies are decidedly more mixed (e.g., Murray, Ehlers, & Mayou, 2002). Empirical support for a relationship between dissociation and narrative memory fragmentation is also weak (Bedard-Gilligan & Zoellner, 2012). Although some studies of memory quality have found trauma narratives to be rated either by the trauma survivor or by outside raters coding narratives as more disorganized and dominated by somatosensory elements than other memory types (e.g., Koss, Figueredo, Bell, Tharan, & Tromp, 1996), other research contradicts this (e.g., Porter & Birt, 2001). Furthermore, degree of

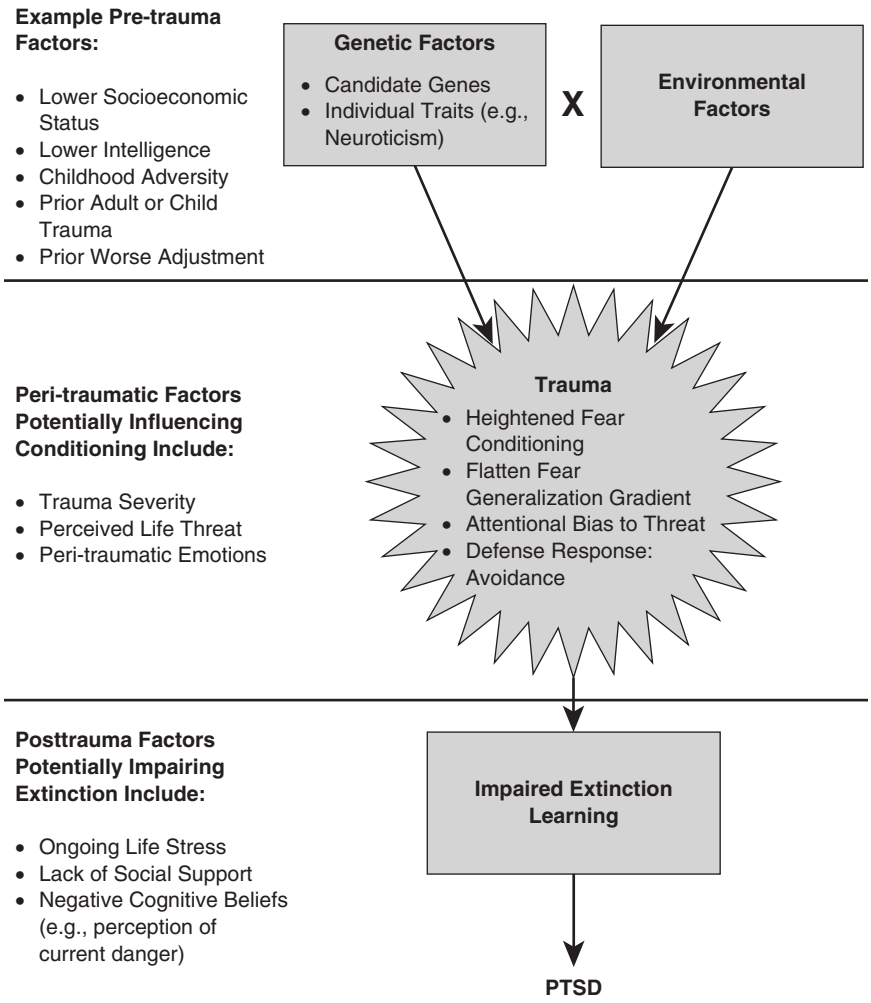

Figure 10.2 Conditioning and extinction model of posttraumatic stress disorder (PTSD). A conditioning and extinction model of PTSD argues for genetic and environmental factors contributing to an increased likelihood of traumatic conditioning and resistance to posttrauma extinction learning. It should be noted that peri-traumatic and posttrauma factors in both adult (Brewin et al., 2000; Ozer et al., 2003) and child meta-analyses (Trickey et al., 2012) are associated with a higher likelihood of developing PTSD than pre-trauma factors.

fragmentation does not reliably distinguish those with and without PTSD (e.g., Zoellner & Bittinger, 2004). However, a recent review suggests that the empirical support for separate perceptual (i.e., sensory) and episodic (i.e., verbal) memory systems in PTSD is stronger than previously believed (Brewin, 2014). Thus, the contention that separate memory systems exist and that trauma memories are different from other autobiographical memories is still a matter of considerable debate.

Cognitive Models Cognitive or attribution-based models focus on the individual's maladaptive beliefs about the traumatic event, the world, and the self as causal to PTSD. One of the first attributional models was proposed by Janoff-Bulman (e.g., 1992). It posits that shock from the trauma shatters previously held beliefs about safety and the worthiness of self, leading to PTSD symptoms. In an extension of this type of model, Ehlers and Clark (2000) suggest that PTSD results when poor elaboration of the trauma memory, excessive association of the memory with external cues, and negative appraisals of the trauma and posttrauma reactions lead to an enhanced sense of threat and an inability to process the event. A normal response to trauma involves conceptually driven processing in which the personal meaning of the event is strongly elaborated within the wider autobiographical memory base. In contrast, excessive data-driven processing, in which external and sensory components are prioritized over self-referential meaning, leads to an unelaborated trauma memory that can be easily triggered by matching sensory cues. Unhelpful appraisals of the event and its consequences as dangerous and incompatible with adaptive beliefs about the self, world, and others, are also critical to the maintenance of PTSD. Both fragmented trauma memories that can be triggered as intrusions and nightmares (similar to dual process models) and unhelpful meanings around the trauma and personal reactions to it contribute to an ongoing sense of current threat. Strategies to manage this threat, such as rumination, thought suppression, and avoidance are helpful in reducing short-term

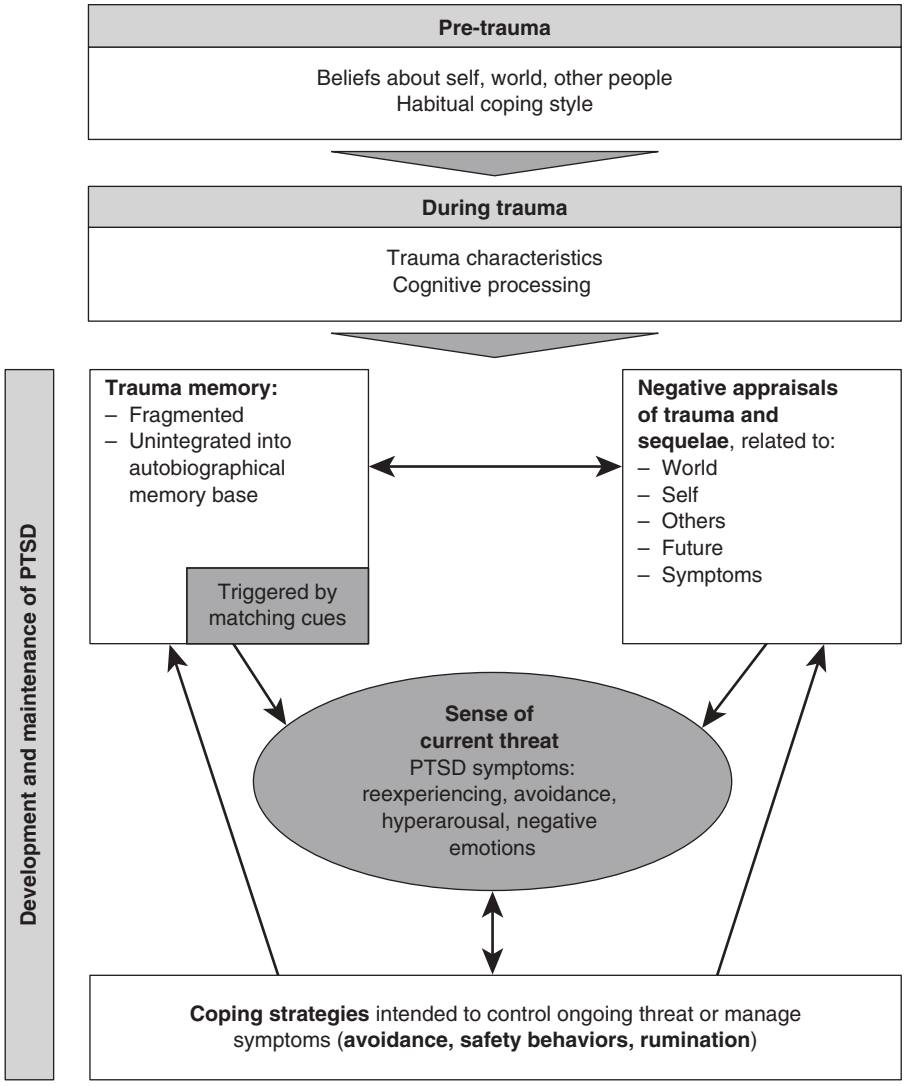

Figure 10.3 Cognitive model of posttraumatic stress disorder (PTSD). PTSD is characterized by an ongoing sense of current threat that is maintained by pathological memory encoding, negative appraisals of the trauma and its consequences and coping strategies that minimize negative affect in the short-term but inhibit recovery overall (adapted from A. Ehlers and D. M. Clark, A cognitive model of posttraumatic stress disorder, *Behaviour Research and Therapy*, *38*, 319–45. © 2000, with permission from Elsevier).

distress and negative affect, but prevent adaptive learning, inhibit recovery, and maintain symptoms of PTSD (see Figure 10.3 for the hypothesized feedback loop).

Research exploring this cognitive processing model has focused on exploring both peri-traumatic processing (i.e., conceptual- and data-driven processing) and on cognitive strategies used following an event. In experimental (e.g., Kindt, van den Hout, Arntz, & Drost, 2008) and prospective studies (e.g., Ehring, Ehlers, & Gluckman, 2008), processing an event in a manner consistent with data-driven rather than conceptual processing predicts PTSD symptoms. Furthermore, maladaptive cognitive appraisals following a traumatic event, such as mental defeat (e.g., Kleim, Ehlers, & Gluckman, 2007), defined as a loss of personal autonomy, and rumination (e.g., Kleim et al., 2007) also predict development of PTSD, lending further evidence to the notion that cognitive processing styles are important in explaining the development of PTSD.

Emotional Processing Theories Emotional processing theories emphasize the processing of the emotional experience of trauma exposure. Horowitz (1993) posits a psychodynamic processing model in which PTSD symptoms are the result of an inability to integrate the traumatic event into existing cognitive schemas. Emotional processing theory (Foa & Kozak, 1986) similarly focuses on the development of adaptive or pathological fear structures. Fear structures are associative networks for escaping danger and include information about the feared stimulus, fear responses, and the meaning of the stimulus and responses. These structures become pathological when they contain excessive response elements that are resistant to modification, including when avoidance or physiological reactivity is present or when these structures include unrealistic elements. In therapy, within- and between-session fear reduction (clinically termed habituation but more likely reflecting extinction processes) is considered

an indicator of emotional processing. Subsequent elaboration of emotional processing theory for PTSD emphasizes the role of unrealistic, pathological cognition elements, namely that the world is extremely dangerous and the victim is extremely incompetent (e.g., Foa, Huppert, & Cahill, 2006). Avoidance behavior characteristic of PTSD prevents the individual from accessing the fear structure and learning corrective information that would correct the abnormal, pathological elements of the network (Foa et al., 2006). Successful recovery, whether natural or therapeutic, depends on accessing the fear network and pairing it with corrective information to alter the pathological structure and reprocess the memory.

Consistent with emotional processing theory, indicators of engagement such as greater fear expression (e.g., Foa, Riggs, Massie, & Yarczower, 1995) and higher distress during retelling of the traumatic memory during exposure therapy (e.g., Rauch, Foa, Furr, & Filip, 2004) are predictors of better PTSD recovery. In addition, between- but not within-session fear reduction during imaginal exposure to the traumatic memory predicts decreases in PTSD symptoms (e.g., Rauch et al., 2004; Bluett, Zoellner, & Feeny, 2014). Yet, not all individuals who show PTSD improvement show between-session fear reduction (Bluett et al., 2014), arguing against this as necessary for PTSD recovery.

Cultural-and Gender-Specific Considerations in Etiology In epidemiological studies, female gender (e.g., Kessler et al., 1995) and identifying as non-White (e.g., Roberts, Gilman, Breslau, J., Breslau, N., & Koenen, 2011) are associated with an increased risk for PTSD. Associated factors such as lower education, decreased social support, or increased trauma exposure may help account for this (e.g., Pole, Best, Metzler, & Marmar, 2005; Tolin & Foa, 2006). Alternatively, building on above theories, conceptualization of "self" as independent versus interdependent may vary as a function of culture, resulting in alterations in trauma-relevant processes such as the formation of the traumatic memory and the integration of the experience into the individuals' conceptualization of their identity (Jobson, 2009).

Cross-cultural research is broadly consistent with cultural differences in beliefs about the self and identity relevant to coping with trauma. Emotional, self-focused retellings of personal events (e.g., Wang & Ross, 2005) are more prevalent in individualistic cultures compared with collectivist cultures. Similarly, the association between appraisals of personal responsibility and control in emotional situations and positive emotion is more likely to be seen in individuals from individualistic rather than collectivist cultures (e.g., Mesquita & Walker, 2003). Furthermore, in collectivist cultures, identity of self is more likely to be related to others and social roles.

From a much more radical perspective, social construction theories argue that culture is critical for the genesis of PTSD itself and that PTSD pathologizes normal human suffering. This perspective argues that PTSD does not exist in nature but has developed over the course of recent history, shifting from a historical view of trauma triggering underlying psychopathology (i.e., the trauma alone is insufficient to produce lasting psychological damage) to the trauma itself causing disorder (e.g., Jones & Wessely, 2007). These theorists point to historically inconsistent symptoms, a striking increase in historical occurrence, the lack of diagnostic specificity, the political agenda of veteran and anti-war activists to validate suffering, and the related lobby for the diagnosis being included in the DSM as evidence. For example, from recent wars, prevalence of PTSD in the United Kingdom tends to be lower than in the United States, potentially due to cultural differences in handling emotion such as "keeping a stiff upper lip" (Hoge et al., 2004; Ramchand et al., 2010). Other historical analyses point to the emergence of the concepts of "traumatic memories," "flashbacks," and "re-experiencing" symptoms and the association between type of symptoms reported and era of service for military members (e.g., Jones et al., 2003). Although some have suggested that overreporting of symptoms may be explained by secondary gains such as disability claims or legitimizing posttrauma behavior, evidence is lacking (e.g., Frueh, Hamner, Cahill, Gold, & Hamlin, 2000). Regardless of social causation or construction, definitions of illnesses or disorders are influenced by social and cultural factors, and the clinical utility of the construct across cultures must be evaluated.

Summary of Etiological Models Prominent biopsychosocial theories of PTSD focus on fear learning, memory disruptions, and information processing deficits as crucial to the development and maintenance of the disorder and incorporate salient factors experienced during the event (i.e., peri-traumatic) and post-event to explain the persistence of symptoms. Conditioning and extinction models provide a solid translational basis to link the animal and human research and good convergent evidence with the developing neuroscience of PTSD. Subsequent memory and information processing models provide an elaborated account of the memory disturbances that characterize PTSD as well as insight into ongoing excessive and inappropriate emotional responses. Cognitive and emotional processing models prioritize the relationship of trauma and trauma memories with pre-existing belief systems and their subsequent meaning and conceptualize ongoing traumatic distress at an intra- and interpersonal level.

Interventions

Intervention approaches can roughly be divided into those that attempt to prevent the development of chronic psychopathology after trauma exposure and those that treat the chronic psychopathology. Prevention approaches typically include all trauma-exposed or highly symptomatic trauma-exposed individuals within the first month after

trauma exposure using brief (typically one to five sessions) intervention strategies, with the idea that full treatment interventions may not be necessary given the high likelihood of natural recovery. Treatment approaches usually also use time-limited interventions (9–12 sessions), though some continue for months or years. Brief early intervention approaches have consistently suffered from an inability to beat natural recovery, small effect sizes, lack of well-controlled research, or even iatrogenic effects. To date, brief prevention efforts have met most success when focusing only on those who are highly symptomatic, using multiple sessions, and using intervention strategies derived from successful treatment strategies for chronic PTSD (Kearns, Ressler, Zatzick, & Rothbaum, 2012). Treatment efforts have been much more successful, showing relatively large effect sizes that are maintained over time.

Biological and Pharmacological Interventions

Selective Serotonin and Norepinephrine Reuptake Inhibitors Selective serotonin reuptake inhibitors (SSRIs) and serotonin–norepinephrine reuptake inhibitors (SNRIs) work by blocking the reuptake of serotonin or norepinephrine, respectively, which increases synaptic levels of 5-hydroxytryptamine (5-HT) receptors (i.e., serotonin) in the synapse. This starts a cascade of downstream effects on other neurotransmitters, second messengers, and immediate early genes (i.e., gene that are activated transiently and rapidly), producing long-term neurochemical changes in the brain. The SSRIs are considered the first-line pharmacological treatments for PTSD, because of the largest psychotropic evidence base (Stein, Ipser, & Seedat, 2006). Several large-scale, placebo-controlled clinical trials support their efficacy in PTSD, albeit with generally moderate effect sizes (e.g., Brady et al., 2000; Davidson, Rothbaum, van der Kolk, Sikes, & Farfel, 2001; Marshall, Beebe, Oldham, & Zaninelli, 2001). Pointing to a potential underlying mechanism, in a recent meta-analysis, hippocampal volume increased from pre- to posttreatment, though this increase was not associated with PTSD symptom improvement (Thomaes et al., 2013). There does not appear to be a benefit for one SSRI over another, nor does there appear to be differences between high and low therapeutic fixed doses. SNRIs have been less studied in the treatment of PTSD, although they show comparable benefits to SSRIs and show maintenance of gains through short-term follow-up. Data on older tricyclic antidepressants, which inhibit both serotonin and norepinephrine reuptake and generally have worse adverse-effect profiles than SSRIs, typically show low response rates or no clear advantage over SSRIs. Longer-term follow-up with and without SSRI/SNRI continuation have not typically been examined, although clinical recommendations suggest medication maintenance for at least one year (Davidson, 2006). In general, the SSRIs have the strongest evidence base of pharmacological treatment for PTSD and are considered a frontline pharmacological treatment option because they provide moderate symptom reduction across symptom constellations and address the common co-occurrence of depression.

Other Common Psychotropic Medications Benzodiazepines bind to a specific receptor site on the gamma-aminobutyric acid-A receptor (GABA-A) complex and facilitate GABA inhibitory effects via effects on a chloride ion channel. In small studies examining frequently prescribed benzodiazepines, neither alprazolam (Braun, Greenberg, Dasberg, & Lerer, 1990) nor clonazepam for PTSD-sleep related problems (Cates, Bishop, Davis, Lowe, & Woolley, 2004) have demonstrated efficacy. Further, benzodiazepine administration after trauma exposure does not appear to prevent the development of later PTSD (e.g., Gelpin, Bonne, Peri, Brandes, & Shalev, 1996). Azapirones bind to the 5-HT1A receptor and are thought to alter control of the firing rate of serotonin neurons. They typically take 2–4 weeks to take effect, are generally well tolerated, and lack the dependence issues of the benzodiazepines. Only one open trial shows a potential benefit of buspirone (Duffy & Malloy, 1994) and no large-scale randomized control trials have yet been conducted. Evidence for anticonvulsants and antipsychotics, either alone or as an augmentation, is largely negative (e.g., Davidson, Brady, Mellman, Stein, & Pollack, 2007) or at best mixed (e.g., Hamner et al., 2009). For the antipsychotics, risky side effect profiles (e.g., lipid abnormalities, glucose intolerance) and need for careful monitoring often outweigh their questionable efficacy.

Novel Pharmacological Targets Given the limitations of our current medications targeting monoamine or GABA neurotransmitter systems, investigators are also pursuing novel molecular targets including modulatory agents, such as neuropeptides, which are short-chain amino acid neurotransmitters and neuromodulators implicated in anxiety, pain, and stress regulation. Their activity is more discretely localized than antidepressants, suggesting fewer adverse effects, but few have been successfully translated from animal models to humans, as they cannot easily cross the blood–brain barrier. Neuropeptides that are being pursued include brain tachykinin neurokinin-1 (NK_1) antagonists, corticotropin-releasing factor antagonists, neuropeptide Y, and vasopressin. A small randomized controlled trial of NK_1 for PTSD was negative (Mathew et al., 2011). Other molecular targets, including agents that block the effects of glutamate or that promote compensatory neurogenesis such as riluzole and ketamine, are also being pursued.

Disrupting Posttraumatic Memory Reconsolidation Instead of directly reducing PTSD symptoms, a novel treatment approach is to either weaken the initial consolidation of trauma memories or target pathological trauma memories and alter their reconsolidation. The consolidation

and reconsolidation process is thought to make a memory temporarily susceptible or labile to new information and has received considerable recent attention (e.g., Schiller et al., 2010). This raises the intriguing question of whether a trauma memory could actually be erased. Yet, this seems unlikely; an associative memory network is complex and any consolidation or reconsolidation would have to access all aspects of the memory.

Beta blockers and glucocorticoid receptor antagonists may disrupt the consolidation or reconsolidation of potentially pathological fear memories in animals, particularly newly formed memories. Initial evidence for the efficacy of disrupting consolidation or reconsolidation in humans is sparse and equivocal. Although several open trials initially increased excitement for propranolol in preventing PTSD (e.g., Brunet et al., 2011), these trials suffered from methodological problems and failed to show PTSD symptom benefit at follow-up. Results of a randomized control trial (Hoge et al., 2012) do not support the preventive use of propranolol in the acute aftermath of a traumatic event. It is also unclear whether glucocorticoids such as cortisol, which is released during emotionally arousing experiences, disrupt or promote memory consolidation. Whereas human studies suggest that low doses of cortisol may disrupt reconsolidation (e.g., de Quervain & Margraf, 2008), animal studies suggest that blocking glucocorticoid receptors may have the same effect (e.g., de Quervain, Aerni, Schelling, & Roozendaal, 2009). At present, only case reports suggest the utility of using glucocorticoid augmentation of exposure therapy (Yehuda, Bierer, Pratchett, & Malowney, 2010).

Cognitive Enhancers An alternative approach to disrupting the trauma memory is instead to pharmacologically enhance new therapeutic learning, particularly the learning that occurs during exposure therapy. Drugs that promote extinction learning may yield greater, faster, or longer-lasting fear reduction. The most studied of these memory-enhancing drugs is d-cycloserine (DCS). DCS may strengthen the consolidation of new extinction learning by increasing activity in amygdalar N-methyl-D-aspartate receptors, which are central to the neural circuitry of extinction. Across the anxiety disorders, DCS produces moderate to large effect sizes in augmenting exposure therapy at posttreatment, relative to placebo plus exposure (Norberg, Krystal, & Tolin, 2008). Yet, the research to date on DCS exposure augmentation in PTSD is not strong. A small randomized trial found that exposure therapy augmented with DCS showed *less symptom reduction* than those with placebo (Litz et al., 2012). Two larger PTSD randomized trials found that DCS did not enhance overall treatment effects (de Kleine, Hendriks, Kusters, Broekman, & van Minnen, 2012; Rothbaum et al., 2014), but may be associated with a stronger treatment response for those who were more severe and needed more sessions (de Kleine et al., 2012). Other compounds, such as yohimbine and methylene blue, are also being examined for their cognitive enhancing effects. In sum, the benefit of cognitive enhancing drugs may be largely for quickening response, for those with more severe symptoms, or for those with prior cognitive behavioral treatment (CBT) failures.

Psychosocial Interventions Interventions for PTSD that include an explicit focus on trauma memories and reminders are strongly supported in stringent meta-analyses of large-scale randomized controlled trials, with large pre- to posttreatment effect sizes and strong maintenance of gains. These treatments will be reviewed below. Brief versions of these treatments such as four to five sessions of cognitive behavioral therapy (e.g., exposure and cognitive restructuring) also have been used to prevent the development of PTSD for highly symptomatic individuals (see Kearns et al., 2012). In contrast, treatments without a trauma focus (e.g., supportive counseling, relaxation) tend to achieve only comparable or slightly better outcomes than waitlist or supportive counseling (e.g., Bradley, Greene, Russ, Dutra, & Westen, 2005; Bisson, Roberts, Andrew, Cooper, & Lewis, 2013).

Exposure-Based Treatments Exposure therapies focus on approaching the trauma memory and trauma-related reminders and help reduce trauma-related distress by facilitating new learning about the meaning of the trauma and altering maladaptive beliefs about oneself, others, and the world. *Prolonged exposure* (Foa, Hembree & Rothbaum, 2007), one of the most commonly used variants, is based on emotional processing theory. *In vivo* exercises (i.e., real-life exposure to avoided situations) and *imaginal exposure* (i.e., revisiting of the traumatic memories and discussion of the revisiting) are considered the main therapeutic elements. Exposure therapy is empirically supported in large-scale controlled studies for PTSD following sexual violence (e.g., Foa et al., 1999; Resick, Nishith, Weaver, Astin, & Feuer, 2002), military experience (e.g., Keane, Fairbank, Caddell, & Zimering, 1989; Schnurr et al., 2007), and heterogeneous samples (e.g., Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998), with very large effect sizes and gains maintained at up to 5–10 years after treatment (Resick, Williams, Suvak, Monson, & Gradus, 2012). In addition, prolonged exposure has been shown to be efficacious not only in reducing PTSD symptoms but also in reducing symptoms of depression (e.g., Foa et al., 2005), improving social functioning (e.g., Foa et al., 2005), and reducing associated symptoms such as guilt (e.g., Resick et al., 2002) and anger (e.g., Cahill, Rauch, Hembree, & Foa, 2003).

Several variants of exposure-based therapies exist. Some consider *eye movement desensitization and reprocessing*, which includes visual or auditory tracking during activation of the trauma memory, a variant of exposure therapy; whereas, others do not. The additive benefit of the eye movement bilateral stimulation is not clear, nor

is the underlying mechanism of the eye movements (e.g., Davidson & Parker, 2001; Seidler & Wagner, 2006). Eye movement desensitization and reprocessing, in large-scale randomized controlled trials, shows large pre- to post-treatment improvement on PTSD, depression, and anxiety comparable to other exposure therapies (Rothbaum, Astin, & Marsteller, 2005; Taylor et al., 2003; van der Kolk et al., 2007), although some concerns exist about maintenance of gains over time (e.g., Rothbaum et al., 2005; Taylor et al., 2003).

Another exposure variant, *narrative exposure therapy* (Schauer, Neuner & Elbert, 2011) was developed with refugees from diverse cultures. It involves repeated exposure to memories of traumatic events across the lifespan and the creation of a written narrative that may be shared for public information. Moderate to large reductions in PTSD severity have been observed and maintained up to a 1-year follow-up, but studies frequently suffer from small sample size, inconsistent controls, variable administration, and the impact on indices of psychopathology and functioning are attenuated when benchmarked against other treatments.

Cognitive Treatments Cognitive treatments for PTSD focus on changes in the person's understanding of the trauma and its meaning in their life. In *trauma-focused cognitive therapy*, new, adaptive information is introduced via cognitive restructuring and behavioral experiments to update the trauma memory to be more useful and to help incorporate it into the wider autobiographical memory base. Imaginal or written exposure to traumatic memories is often included but primarily intended to uncover trauma-related beliefs for subsequent exploration. *Cognitive processing therapy* (Resick & Schnicke, 1993) involves discussion of key themes, including challenges to safety, trust, power, and self-esteem and exposure to the memory through written accounts. Although randomized controlled trials are less plentiful than for exposure therapies, large and consistent effects sizes can be seen across mixed trauma samples (e.g. Ehlers et al., 2003, 2014), rape survivors (e.g., Resick et al., 2002), survivors of childhood sexual abuse (Chard, 2005), combat veterans (e.g., Monson et al., 2006), and refugees (Schulz, Resick, Huber, & Griffin, 2006).

Skills-Based Treatments In contrast to trauma-focused therapies, skills-based therapies target such skills as enhancing coping, reducing emotional dysregulation, and reducing difficulties with interpersonal skills. *Stress inoculation training* teaches anxiety reduction strategies including relaxation and breathing retraining, thought stopping, and guided self-dialogue, and addresses anxiety-related avoidance via *in vivo* exposures. It has been found to be superior to supportive counseling but the combination of prolonged exposure and stress inoculation training does not enhance trauma-related outcomes (Foa et al., 1999). Notably, stress inoculation training is largely a "dead therapy" in that very few researchers are currently investigating it or training therapists in its conduct.

Alternative treatments that teach participants to experience distressing thoughts and feelings without judgment and to make adaptive behavioral changes without explicitly addressing trauma memories are gaining momentum. Variants on *acceptance and commitment therapy* show promise in small-scale or open reports (e.g., Orsillo & Batten, 2005). Similarly, protocols derived from *dialectical behavior therapy* and *mindfulness-based interventions* also show promise in small, randomized controlled trials (e.g., Kearney, McDermott, Malte, Martinez, & Simpson, 2013). However, the observed effects through skills training are lower than exposure or cognitive intervention effect sizes, large-scale randomized trials need to be conducted, the hypothesized mechanisms sometimes do not change with the intervention, and state-of-the-art treatment comparators (e.g., cognitive processing therapy, prolonged exposure) have not been included in trials to date.

Combined Treatments Given substantial overlap across established treatment protocols, questions remain over the contributions of individual components to outcomes and potential cumulative benefits of multiple approaches. Cognitive shifts are thought to drive symptom reduction in cognitive therapy (Kleim et al., 2013), and evidence suggests that cognitive changes may underlie PTSD symptom change in primarily exposure-based treatments (Zalta et al., 2014). Typically, combination studies fail to show additive benefits of combined treatment approaches (e.g., Foa et al., 1999, Foa et al., 2005), though there are notable exceptions, particularly when components are tightly controlled (Marks et al., 1998; Bryant et al., 2008). Phased treatment approaches, beginning with emotion regulations skills training, may help reduce dropout and improve outcomes by enhancing engagement and alliance prior to starting exposure therapy (e.g., Cloitre et al., 2010), but evidence for this additional benefit in earlier trials has been questioned (Cahill et al., 2003). Taken together, there is very little evidence across a number of well-done randomized control trials that "more" therapeutic techniques produce better outcomes.

Adaptations to PTSD Treatments

Web-Based or Application-Based Delivery Computerized and web-based interventions may be cost-effective, flexible ways of delivering treatment that help to overcome barriers, including geographical remoteness, transportation difficulties, and stigma. Several randomized controlled trials of web protocols based in trauma-focused CBT principles (e.g., Lange et al., 2003; Litz, Engel, Bryant, & Papa, 2007) achieved moderate reductions of PTSD, depression, and anxiety symptoms. Outcomes typically do not match in-person individualized exposure therapies, but scalability makes them an attractive alternative treatment.

Virtual Reality Augmentation to exposure therapy has been attempted using virtual-reality environments generated through a head-mounted device that includes images, smells, sounds, or movements similar to traumatic events. Including virtual reality may help to address stigma related to seeking help, assist with emotional engagement, and provide a proxy for *in vivo* exposure to environments, like combat scenes, that are dangerous or inaccessible. However, concerns remain about the burden of introducing specialized equipment. Treatment studies conducted to date are equivocal and lack sufficient controls to determine the additive benefit of virtual reality over standard exposure (Motraghi, Seim, Meyer, & Morisette, 2014; Rothbaum et al., 2014).

Intensive Delivery of Psychotherapy Adapting standard protocols to reduce number or duration of sessions may better suit some participants and reduce attrition. Compared with standard protocols, reduced dose of cognitive processing therapy (Galovski, Blain, Mott, Elwood, & Houle, 2012) and intensive trauma-focused CBT held daily over 7 days (Ehlers et al., 2014) led to similar reductions in PTSD and depression symptoms without adverse effects. In small randomized control trials, single or limited sessions of exposure led to large reductions in PTSD symptoms and fear one to two years later (e.g., Başoğlu, Şalcioğlu, & Livanou, 2007). These studies argue that intensive delivery and, maybe even, fewer sessions may benefit a number of patients.

Group Interventions for PTSD Debate continues as to the potential for successful trauma-focused work in groups, particularly because of the challenges of managing traumatic disclosure and confidentiality. Reliable research comparing group and individual administration is lacking (Bisson et al., 2013). CBT, described above, often has group elements. However, several randomized controlled trials indicate limited benefit on PTSD or depression symptoms from group interventions such as mindfulness (e.g., Kearney et al., 2013) or CBT (Schnurr et al., 2003). Well-done comparison trials between individual and group modalities are needed.

Contraindicated Interventions A primary dictum of treatment provision is to avoid inflicting harm on those who are seeking help. However, by virtue of intuition, habit or the pursuit of innovation, interventions that are not supported by reliable research continue to be implemented.

Single Incident Debriefing With its roots in World War I battle debriefing, encouraging emotional expression in a single session immediately after trauma reemerged as a first-line intervention in the 1980s. Although often viewed as helpful at the time, rigorous systematic reviews have been unequivocal that this type of intervention is at best ineffective and, at worst, harmful in the long-term for preventing PTSD, particularly for those at higher risk (e.g., Mayou, Ehlers, & Hobbs, 2000) and should not be used (Rose, Bisson, Churchill, & Wessely, 2002). It is believed that early emotional expression may impede the process of natural recovery after trauma, particularly with highly aroused survivors, for whom it might maintain overactivation of the sympathetic nervous system, disrupt useful encoding of the traumatic memory, and reinforce negative appraisals of the event and personal reactions to it. As an alternative, other one session interventions that focus on things such as psychoeducation and skills building such as *psychological first aid* (e.g., Ruzek et al., 2007) or the military "Battlemind" approach (e.g., Adler, Bliese, McGurk, Hoge, & Castro, 2009), at present, have limited randomized control trials, are sometimes given months after the traumatic events (i.e., not prevention *per se*), and point to only small effects for the prevention of PTSD.

Cannabinoids and Hallucinogenics Limited trials of the psychotherapeutic use of cannabinoids and psychedelic drugs in PTSD treatment to date are not persuasive and the risk of misuse and adverse effects are of concern (Farach et al., 2012). Although active cannabinoid components may be useful therapeutically for trauma-related nightmares (Fraser, 2009), they may also exacerbate anxiety (e.g., Crippa et al. 2011). Combined 3,4-methylenedioxymethamphetamine, commonly known as MDMA or "ecstasy," has not had adverse effects when paired with adapted exposure therapies in poorly conducted, small sample pilot studies (e.g., Mithoefer et al., 2013). Significant concerns remain about the adverse-effect profile of MDMA, including neurotoxic effects, serotonin depletion, and lasting memory alterations. Greatly improved scientific rigor and larger samples would be required to draw reliable conclusions on the additive or subtractive effects of these drugs, but ongoing research is controversial and limited by the regulatory environment.

Recovered Memory Therapy Professional guidelines, including those of the British Psychological Society, warn against the use of techniques including hypnotherapy and guided imagery to uncover 'repressed' memories, owing to the potential for generating false memories alone or in combination with recollections of actual events (Wright, Öst, & French, 2006). There is evidence that memories of bizarre events, including alien abduction, can be recovered (e.g., McNally, 2012). False memories of childhood atrocities, including sexual abuse, have a potentially devastating impact on both clients and others. Recovered memory techniques should be avoided and extreme caution taken in determining the veracity of newly acquired memories for early experiences, particularly within the window of normal childhood amnesia.

Summary of Biological and Psychosocial Interventions Trauma-focused psychotherapy interventions for PTSD have proven effectiveness across many large-scale,

well-controlled clinical trials. SSRIs or SNRIs are also effective interventions, although comparisons with psychotherapy and psychotherapy augmentation studies are largely missing. Although other approaches show promise, effect sizes are often lower than or comparable to established protocols and do not yet have substantive well-controlled empirical support. Current emphasis is on isolating specific mechanisms of change and tailoring treatments to improve efficacy and reach. Adapted treatments do not have better outcomes than standard approaches, so clinicians should be wary of deviating from established protocols on a case-by-case basis. That said, for those for whom these interventions are ineffective or for those who do not have clinical access, alternative treatments or novel modes of delivery should be considered.

Conclusion

Trauma and stressor exposure is ubiquitous, but only a minority of individuals who experience traumatic events develop chronic psychopathology. Pre-trauma factors are typically not strong predictors of who is going to develop psychopathology. Even trauma-specific and posttrauma factors, which are better predictors of who is going to develop chronic psychopathology, do not account for a large portion of the variance. The mechanisms that underlie healthy or unhealthy reactions to trauma will only be discovered through longitudinal studies that incorporate pre-trauma assessments and different cultural and societal contexts that incorporate biological, social, and psychological factors. Vulnerability to developing long-term problems likely has genetic markers and contributing environmental factors and is characterized by deficit functioning in key brain areas such as the prefrontal cortex, amygdala, and hippocampus. Fear conditioning and extinction processes are also likely at play. This vulnerability is also likely influenced by social support, interpersonal connections, day-to-day engagement, and perceived safety. Pre-existing beliefs, reactions during trauma, and personal appraisals may also impact the consolidation of traumatic memories in adaptive or maladaptive forms. Thus, scientific study of reactions following trauma exposure must incorporate multiple levels of analysis that include environmental, personal, and biological factors.

Consistent with our limitations in understanding the etiology of psychopathology following trauma, existing short-term interventions immediately after trauma exposure for preventing the development of long-term psychopathology following traumatic events produce, at best, modest long-term benefits beyond natural recovery. For chronic pathology, effective trauma-focused psychosocial and also psychotropic interventions exist, with a large majority of individuals making reliable and lasting gains (Jayawickreme et al., 2014). Questions remain about key mechanisms promoting therapeutic change, as well as necessary and sufficient therapeutic components. Similar questions remain about efficacy and efficiency of interventions that move beyond traditional models of in-person one-on-one psychotherapy.

Whether considering epigenetic factors in the context of resilience after child abuse, determining policies for first response after mass disasters, or guiding those providing care to individuals who have experienced personal calamity, bringing these domains together is now crucial. Only through an integration of understanding of etiological factors, natural recovery, and therapeutic recovery across biopsychosocial levels of analysis will the field move forward and ultimately improve the lives of trauma survivors. This multi-level approach will guide the next generation of researchers seeking to identify at risk individuals, mitigate long-term impact, guide our early intervention development, and develop more effective and targeted treatments. The field of trauma and stressor-related disorders has multiple grand challenges that will guide research and clinical endeavors for years to come.

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11

Depressive and Bipolar Disorders

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Mood Disorders

Consider the case of David, a 24-year-old male who sought neuropsychological testing to assess for attention deficit hyperactivity disorder (ADHD).

David had been taking classes at a local university for the past 4 years to complete his degree. He wanted to be assessed for ADHD because he was experiencing significant difficulties sustaining and paying attention in his classes, and his grades began to suffer. He failed his classes one semester and was placed on academic probation.

Additionally, David was running his own Internet advertising business while attending school. He reported that he was able to keep the business running, but that he felt it was suffering due to his current state of disorganization. He was unable to arrive to meetings on time, took poor notes, and had considerable difficulty keeping the business organized. As a result, he was in debt and could not properly manage his finances.

Although many elements of David's story are common in individuals with ADHD, they are also familiar to many people who suffer from severe depression. During David's assessment, he was given a diagnostic interview as well as the Beck Depression Inventory (BDI), a 21-item self-report of depressive symptoms. David reported depressed mood, feelings of hopelessness about his future, irritability, guilt about his failing business and poor academic performance, marked fatigue and sluggishness, anhedonia, and severe sleep disturbance (marked insomnia, sleep continuity disturbance, and early morning awakenings). His symptoms were persistent and severe enough to meet the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5; American Psychological Association, 2013) criteria for a major depressive episode, and his high score on the BDI (43) indicated that this episode was particularly severe. Moreover, David also reported substantial symptoms of anxiety and worry, such as feelings of dread, numbness, hot and cold sweats, shortness of breath, and heart palpitations. Anxiety symptoms are often comorbid in an episode of major depressive disorder, and can contribute to the overall impairment of the episode.

Although David had been aware that his mood was lower than he would like it to be, it was not his mood, but rather his inability to keep himself organized, make everyday decisions, or focus on his work that was driving him to seek an assessment for ADHD, which he suspected was the root of his day-to-day difficulties. David was given a battery of neuropsychological tests, in addition to diagnostic interviews. Although the results of these assessments were indicative of impairments in attention and concentration, the severity of David's depression and anxiety made it nearly impossible to assign an unequivocal diagnosis of ADHD. Instead, the evaluator recommended that David first seek treatment for his depression and anxiety, as she hypothesized that his attention and concentration symptoms would improve as a result.

David's story is not an uncommon one in clinical settings. Many individuals with moderate to severe depression experience marked impairments in their ability to do their jobs, perform well in school, and stay on top of household chores. However, major depressive disorder is often responsive to treatment either with psychotherapy, pharmacotherapy, or their combination, and many see a restoration of their general functioning following a targeted treatment regimen.

We are all familiar, at least casually, with what depression is like. We have all experienced occasional periods of sadness, where nothing seems worth doing, we become tired and slowed down, and our lives are no longer fun.

Some have also experienced the opposite state, when we feel on top of the world, excited and reckless, and we become hyperactive and think that we can accomplish anything. In other words, *depression* and *mania*, in mild and temporary forms, are part of everyday existence. For some people, however, these mood swings become so prolonged and extreme that the person's life is seriously disrupted. These conditions are known as *mood disorders* or *affective disorders*.

Mood disorders have been recognized as clinical entities for over 2000 years. Indeed, Hippocrates described both depression and mania in detail in the fourth century B.C.E. As early as the first century C.E., the Greek physician Aretaeus observed that manic and depressive behaviors sometimes occurred in the same person and seemed to stem from a single disorder. Although they have been studied for centuries, as yet, there are no completely satisfactory explanations for the puzzling features of mood disorders. In this chapter, we first describe the mood disorders as well as their diagnosis, symptoms, dimensions, and risk factors. Then, we review what is known about the causes and treatment of mood disorders from various theoretical perspectives.

Depressive and Manic Episodes

Typically, mood disorders are episodic. Within days, weeks, or months, a person who has been functioning normally is plunged into despair or becomes euphoric. Once the episode runs its course, the person may return to normal or near-normal functioning, though he or she is likely to have recurrences of mood disturbance. The severity, duration, and nature of the episode (whether depressive or manic) determine the diagnosis. Below are the typical features of depressive and manic episodes.

Major Depressive Episode Onset of a *major depressive episode* is usually gradual, occurring over a period of several weeks or months, and the episode typically lasts several months and then ends, as it began, gradually (Coryell et al., 1994). Major depressive episodes affect mood, motivation, thinking, somatic, and motor functioning. The characteristic features of a major depressive episode are as follows (see Table 11.1 for the DSM-5 diagnostic criteria):

1. *Depressed mood:* Almost all depressed adults report some sadness, ranging from mild melancholy to total hopelessness. Mildly or moderately depressed people may have crying spells; severely depressed individuals often say they feel like crying but cannot. Deeply depressed people see no way that they or anyone else can help them—the helplessness–hopelessness syndrome.
2. *Loss of pleasure or interest in usual activities:* Anhedonia, the loss of pleasure or interest in one's usual activities, is the other most common characteristic of a major depressive episode. Even emotional responses to pleasant stimuli are diminished during a major depressive episode (Sloan, Strauss, & Wisner, 2001).
3. *Disturbance of appetite:* Most depressed people have poor appetite and lose weight; however, a minority react by eating more and putting on weight. Whichever type of weight change, the same change tends to occur with each depressive episode (Kendler et al., 1996).
4. *Sleep disturbances:* Insomnia is an extremely common feature of depression and can take one or more of three forms. Depressed people may have trouble falling asleep initially, or may awaken repeatedly throughout the night, or may wake up too early in the morning and be unable to fall back to sleep. However, like eating, sleep may increase rather than decrease, with the depressed person sleeping 15 hours a day or more. Depressed individuals who exhibit excessive sleeping are usually the same ones who eat excessively (Kendler et al., 1996).
5. *Psychomotor retardation or agitation:* In retarded depression, the most common psychomotor pattern, the depressed person is fatigued, movement is slow and deliberate, posture is stooped, and speech is low and halting, with long pauses before answering. In severe cases, individuals may fall into a mute stupor. Some evidence suggests that the symptom of psychomotor retardation is related to low presynaptic dopamine levels (Paillere et al., 2001). Less frequently, in agitated depression, the person shows incessant activity and restlessness—hand wringing and pacing.
6. *Loss of energy:* The depressed person usually exhibits a sharply reduced energy level and may feel exhausted all the time.
7. *Feelings of worthlessness and guilt:* Depressed people see themselves as deficient in whatever attributes they value most: intelligence, beauty, popularity. These feelings of worthlessness are often accompanied by a profound sense of guilt. Depressed individuals believe that any problem is their fault.
8. *Difficulties in thinking:* As in David's case, depressed people have trouble concentrating, remembering, and making decisions, even about everyday matters (what to wear, etc.). The harder a mental task, the more difficulty they have (Hartlage, Alloy, Vasquez, & Dykman, 1993).
9. *Recurring thoughts of death or suicide:* Many depressed people have recurrent thoughts of death and suicide. Often, they say that they (and everyone else) would be better off if they were dead.

Manic Episode A typical *manic episode* begins relatively suddenly, over a few days, and is usually shorter than a

TABLE 11.1
Changes from DSM-IV-TR to DSM-5 Criteria for Manic, Hypomanic, Depressive, and Mixed Episodes

Condition	Criteria Description
Manic Episode	A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood, **and abnormally and persistently increased goal-directed activity or energy**, lasting at least 1 week (or any duration if hospitalization is necessary). B. During the period of mood disturbance **and increased energy or activity**, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree **and represent a noticeable change from usual behavior**: 1. inflated self-esteem or grandiosity 2. decreased need for sleep 3. more talkative than usual or pressure to keep talking 4. flight of ideas or subjective experience that thoughts are racing 5. distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli) 6. increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation 7. excessive involvement in activities that a high potential for painful consequences C. The symptoms do not meet criteria for a Mixed Episode D. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning, or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features. E. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or a general medical condition (e.g., hyperthyroidism). Manic-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, light therapy) should not count toward a diagnosis of Bipolar I Disorder.
Hypomanic Episode	A. A distinct period of persistently elevated, expansive, or irritable mood **and abnormally and persistently increased activity or energy**, lasting throughout at least 4 days, that is clearly different from the usual non-depressed mood **and present most of the day, nearly every day**. B. During the period of mood disturbance **and increased energy and activity**, three (or more) of the following symptoms have persisted (four if the mood is only irritable), **represent a noticeable change from usual behavior**, and have been present to a significant degree: 1. inflated self-esteem or grandiosity 2. decreased need for sleep 3. more talkative than usual or pressure to keep talking 4. flight of ideas or subjective experience that thoughts are racing 5. distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli) 6. increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation 7. excessive involvement in pleasurable activities that have a high potential for painful consequences C. The episode is associated with an unequivocal change in functioning that is characteristic of the person when not symptomatic D. The disturbance in mood and the change in functioning are observable by others E. The episode is not severe enough to cause marked impairment in social or occupational functioning, or to necessitate hospitalization, and there are no psychotic features. F. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or a general medical condition (e.g., hyperthyroidism). Hypomanic-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, light therapy) should not count toward a diagnosis of Bipolar II Disorder. **Note: A full hypomanic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a hypomanic episode diagnosis. However, caution is indicated so that one or two symptoms (particularly increased irritability, edginess, or agitation following antidepressant use) are not taken as sufficient for diagnosis of a hypomanic episode, nor necessarily indicative of a bipolar diathesis.**
Major Depressive Episode	A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure. 1. depressed mood, most of the day, nearly every day, as indicated by subjective report 2. markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day. 3. significant weight loss when not dieting, or weight gain, or decrease or increase in appetite nearly every day. 4. insomnia or hypersomnia nearly every day 5. psychomotor agitation or retardation nearly every day (observable by others) 6. fatigue or loss of energy nearly every day 7. feelings of worthlessness or excessive or inappropriate guilt 8. diminished ability to think or concentrate, or indecisiveness, nearly every day 9. recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or plan

	B.	The symptoms do not meet criteria for a Mixed Episode
	C.	The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning
	D.	The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).
	E.	The symptoms are not better accounted for by bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.
Mixed Episode	A.	The criteria are met for both a Manic Episode and for a Major Depressive Episode (except for duration) every day during at least a 1-week period.
	B.	The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.
	C.	The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or a general medical condition (e.g., hyperthyroidism).

Note: Bold text indicates additions to diagnostic criteria in the DSM 5. Strikethrough text indicates diagnostic criteria that have been deleted in the DSM 5.

depressive episode, lasting from several days to several months. The features of a manic episode are as follows (see Table 11.1 for the DSM-5 diagnostic criteria):

1. *Elevated, expansive, or irritable mood accompanied by abnormally and persistently increased goal-directed activity or energy*: Typically, people in a manic episode feel "high" and on top of the world and have limitless enthusiasm for whatever they are doing. This expansiveness is often mixed with irritability when someone tries to interfere with their behavior. In some cases, irritability is the manic person's dominant mood, with euphoria either intermittent or simply absent. The elevated or irritable mood is accompanied by super-charged energy and increased goal-directed activity across multiple domains.
2. *Inflated self-esteem*: People with mania believe they have highly important plans, and special talents and abilities. They tend to see themselves as extremely attractive, powerful, and capable of great achievements even if they have no such aptitude. They may begin composing symphonies, designing nuclear weapons, or calling the White House with ideas on how to run the country.
3. *Sleeplessness*: Manic episodes are almost always marked by a decreased need for sleep. Manic individuals may sleep only 2 or 3 hours a night, but have twice as much energy as others.
4. *Talkativeness*: People with mania tend to talk loudly, rapidly, and constantly. Their speech is often full of puns and jokes that they alone find funny.
5. *Flight of ideas*: Manic individuals' speech often shifts rapidly from topic to topic. They often have racing thoughts; this may be why they speak so rapidly—to keep up with their ideas.
6. *Distractibility*: Manic individuals are easily distracted. Their attention is frequently pulled by irrelevant and unimportant aspects of the environment.
7. *Hyperactivity*: The expansive mood is usually accompanied by restlessness and increased goal-directed activity—physical, social, occupational, and often sexual.
8. *Reckless behavior*: The euphoria and grandiose self-image of manic people often lead them to impulsive, reckless actions: shopping sprees, reckless driving, careless business investments, sexual indiscretions, and so on. They may impulsively call friends in the middle of the night or spend the family savings on a new Porsche.

A hypomanic episode is briefer and less severe than a manic episode, and does not require impairment, but has similar symptoms as a manic episode. A hypomanic episode does, however, require a change in functioning and must be observable by others. When a person meets criteria for major depression and manic or hypomanic episodes simultaneously (e.g., exhibits manic hyperactivity and grandiosity, but also cries and is suicidal), it's called a mixed episode. Mixed episodes are not uncommon (Vieta & Morralla, 2010) and are proposed as a specifier of mood disorders in DSM-5.

Mood Disorder Syndromes

Major Depressive Disorder People who experience one or more major depressive episodes, with no mania or hypomania, have, according to the DSM, major depressive disorder (MDD). The prevalence of this disorder during any given month is close to 4% of men, and 6% of women. The lifetime risk is about 20% (Kessler & Wong, 2009). Major depression is predicted to be the leading cause of disability and premature death worldwide in the 21st century (World Health Organization, 2003). Depression leads to more office visits than any other medical problem except hypertension and greater impairment—more workdays lost, more time in bed—than chronic medical conditions, such as diabetes or heart disease (Druss, Rosenheck, & Sledge, 2000; Wells & Sherbourne, 1999). Further, although there are effective treatments for depression, most people with major depression do not receive adequate treatment (Young, Klap, Sherbourne, & Wells, 2001). Moreover, each successive generation born since World War II has shown higher rates of depression

(Burke, Burke, Rae, & Regier, 1991; Compton, Conway, Stinson, & Grant, 2006; Klerman, 1988). According to some experts, we are in an "age of depression."

Course Major depression is a highly recurrent disorder, with about 80% of all people with first onset of major depression experiencing at least one recurrence (Boland & Keller, 2009). The more previous episodes, the younger the person was at first onset, female gender, family history of depression, the more stressful life events endured recently, the less social support the person received, and the more negative cognitions the individual has, the greater the likelihood of recurrence (Belsher & Costello, 1988; Burcusa & Iacono, 2007). Over a lifetime, the median number of episodes per patient is four, with a median duration of 4½ months per episode (Judd, 1997; Solomon et al., 1997).

The course of recurrent depression varies considerably. Following a depressive episode, some people return to their level of functioning prior to the onset of the disorder, whereas others still show serious impairment in job status, income, marital adjustment, social relationships, and recreational activities 10 years after the episode (Judd et al., 2000). Depression also affects the immune system, leaving people more susceptible to illness and death (Raison & Miller, 2012). Thus, it is difficult for people recovering from a depressive episode to resume their former lives. Indeed, research on "stress generation" indicates that the symptoms and behaviors characteristic of a depressive episode actually generate stressful life events, which in turn can maintain the depression and produce a cycle of chronic stress and impairment (Daley et al., 1997; Liu & Alloy, 2010).

The early course or prodrome of depression (early symptoms or signs of an impending episode) has also garnered attention (Jackson, Cavanaugh, & Scott, 2003; Iacoviello, Alloy, Abramson, & Choi, 2010). Certain symptoms, such as sad mood, anhedonia, hopelessness, difficulty concentrating, worrying/brooding, decreased self-esteem, and irritability, are more likely to be present among individuals entering a depressive episode than among demographically similar individuals who did not develop an episode. In addition, the durations of prodromal and residual (symptoms still present as an episode is remitting) phases are correlated, the prodromal and residual symptom profiles are quite similar, and the order of symptom onset during the prodrome is negatively correlated with the order of symptom remission (Iacoviello et al., 2010).

Groups at Risk of Depression Although depression can strike anyone at any time, some groups are especially vulnerable to depression. Research has shown that Hispanic youth, and especially Hispanic girls, tend to have higher rates of depressive symptoms than white or African American youth (Twenge & Nolen-Hoeksema, 2002; McLaughlin, Hilt, & Nolen-Hoeksema, 2007). Some of these differences, however, can be accounted for by controlling for parental education level as well as differences in poverty and perceived support at home and at school (Kennard, Mahtani, Hughes, Patel, & Emslie, 2006; Mikolajczyk, Bredehorst, Khelaifat, Maier, & Maxwell, 2007). Never married and formerly married individuals have higher depressive symptoms than those who are married (Mirowsky & Ross, 2003; Turner & Lloyd, 1999). In addition, later-born cohorts have a higher lifetime prevalence of depression (Kessler et al., 2003), which has led to the popular belief that there is currently a depression "epidemic" among today's youth. A large meta-analysis has shown, however, that when concurrent assessment rather than retrospective recall is used, there is no evidence for an increased prevalence of child or adolescent depression over the past 30 years (Costello, Erkanli, & Angold, 2006). At the other end of the age spectrum, comorbid conditions associated with aging rather than aging itself may be responsible for the increase in depressive symptoms seen with age (Snowdon, 2001). According to Nguyen and Zonderman (2006), rates of depression remain relatively stable across most of the adult lifespan (ages 25–70) and increase thereafter.

One of the most consistent findings in depression research is the gender difference in rates of depression. Epidemiological studies conducted in the United States and some European countries have shown that women are twice as likely to develop depression as men (Angst et al., 2002; Kuehner, 2003). In addition, the dramatic gender difference in depression rates in women first emerges during adolescence (Hankin & Abramson, 2001; Nolen-Hoeksema, 2001) and lasts through adulthood. Prior to adolescence, boys and girls tend to have similar rates of depression and some research has shown that boys might be at an even higher risk for developing depression (Twenge & Nolen-Hoeksema, 2002). These differences in prevalence rates of depression are due to women having a higher risk of first onset, not a difference in the persistence or recurrence of the depressive episodes (Kessler, 2003). There are some cultural groups, such as the Old Order Amish and Orthodox Jews, that serve as intriguing exceptions to these widespread findings (Angst et al., 2002; Piccinelli & Wilkinson, 2000). Depression rates for women in these groups may be lower because these groups' cultural norms reduce the sexual objectification of girls in adolescence (Hyde, Mezulis, & Abramson, 2008).

Many factors have been implicated in the gender difference in depression rates across the lifespan. Adolescent girls might not welcome the physical changes that accompany puberty as much as boys do, and research has shown that this "body dissatisfaction" is often associated with depressive symptoms (Siegel, Yancey, Aneshensel, & Schuler, 1999; Hankin & Abramson, 2001). Researchers also have identified several psychological factors that contribute to women's vulnerability to developing depression. One is women's interpersonal orientation. It has been suggested

that women tend to derive more of their self-worth from their relationships than men (Cyranowski, Frank, Young, & Shear, 2000). Adolescent girls generally score higher than boys on measures of need for social approval and reassurance seeking, which puts them at higher risk for developing depressive symptoms (Rudolph & Conley, 2005). Women's greater interpersonal orientation makes them more susceptible to experiencing more interpersonal stressors, which have been associated with depression (Hammen, 2003; Hamilton, Stange et al., 2013).

Although individuals can respond to sad mood in a variety of ways, rumination is especially harmful. Rumination involves responding to one's sad mood in a passive manner by repeatedly dwelling on the causes and symptoms of one's depressive mood rather than taking active steps to change one's mood (Nolen-Hoeksema, 1991). Women tend to ruminate more than men in response to sad mood, and controlling for the gender difference in rumination makes the gender difference in depression disappear (Nolen-Hoeksema & Jackson, 2001; Nolen-Hoeksema, Larson, & Grayson, 1999).

Along with biological and psychological factors, social factors also play a role in explaining the higher rates of depression in women. Women experience higher levels of sexual and physical abuse than men, which put them at a higher risk for developing depression across their lifetime (Weiss, Longhurst, & Mazure, 1999; Kendler, Gardner, & Prescott, 2002). Childhood sexual abuse has been found to be an especially strong predictor of development of future psychopathology, including depression (Kendler, Kuhn, & Prescott, 2004).

No single factor is implicated in the disparity in rates of depression among men and women. Hyde, Mezulis, and Abramson (2008) proposed a model that suggests that women have a variety of vulnerability factors that interact with new stressors that emerge in adolescence to produce the gender gap in depression.

Bipolar Disorder Whereas major depression is confined to depressive episodes, bipolar disorder typically involves both manic or hypomanic and depressive episodes. Some individuals have a manic episode—or a series of such episodes—with no depressive episodes. Such cases, although they involve only one "pole," are still classified as bipolar disorder, because, aside from the absence of depressive episodes, they resemble the classic bipolar disorder. Some researchers suspect that they are simply cases of insufficient follow-up for depressive episodes. Alternatively, some individuals have both depressive and hypomanic, rather than fully manic, episodes. Thus, DSM-5 has divided bipolar disorder into two types. In *bipolar I disorder*, the person has had at least one manic episode and usually, but not necessarily, at least one major depressive episode as well. In *bipolar II disorder*, the person has had at least one major depressive episode and at least one hypomanic episode, but has never met the diagnostic criteria for manic episode. In a less common pattern, called the *rapid-cycling type*, the person (usually a woman) switches back and forth between depressive and manic or hypomanic episodes (with at least four mood episodes per year), with little or no "normal" functioning between (Leibenluft, 2000). This pattern, which tends to have a poor prognosis (Leibenluft, 2000), occurs naturally in some bipolar patients, but also occurs in about 25% of bipolar patients in response to antidepressant medication (Suppes, Dennehy, & Gibbons, 2000).

The occurrence of manic or hypomanic episodes is not all that differentiates bipolar disorder from major depression (Table 11.2; Rehm, Wagner, & Ivens-Tyndal, 2001). First, bipolar disorder is much less common than major depression, affecting an estimated 0.5–3.5% of the world population (Miklowitz & Johnson, 2006). Second, unlike major depression, bipolar disorder occurs in the two sexes with approximately equal frequency, and bipolar disorder is more prevalent among higher socioeconomic groups. Third, whereas people who are married or have intimate relationships are less prone to major depression, they are not at decreased risk for bipolar disorder. Fourth, people with major depression tend to have histories of low self-esteem, dependency, and obsessional thinking, whereas people with bipolar disorder are more likely to have a history of hyperactivity or ADHD (Sachs et al., 2000; Walshaw, Alloy & Sabb, 2010). Fifth, the depressive episodes in bipolar disorder are more likely to involve psychomotor retardation, excess sleep, weight/appetite increase than those in major depression (Benazzi, 2000, 2001). Sixth, bipolar disorder mood episodes are generally briefer and more frequent than are those in major depression (Cusin, Serretti, Lattuada, Mandelli, & Smeraldi, 2000). Seventh, bipolar disorder is associated

TABLE 11.2
Differences Between Bipolar Disorder and Major Depression

	Bipolar Disorder	Major Depression
Sex ratio	Equal	2 : 1 (women: men)
Course	More frequent, brief episodes	Less frequent, longer episodes
Prognosis	Greater impairment; but some outcomes of high accomplishment	Less impairment
Prevalence	0.5–4.4% of the US population	17% of the US population
Marital status	No difference in rates for married vs. unmarried people	Lower rates in married people
Depressive episodes	Psychomotor retardation common	Psychomotor retardation less common
Genetics	Strong heritability	Weaker heritability
Personality features	Hyperactivity, ADHD, impulsivity	Dependency, low self-esteem, and obsessional thinking

Source: Adapted from Alloy, Riskind, & Manos (2004). *Abnormal Psychology: Current Perspectives* (9th ed.)

with greater impairment in marital and work functioning, substance use, as well as heightened risk of suicide, and has a worse long-term outcome than major depression (Goodwin & Jamison, 2007; Judd et al., 2005; Kessler et al., 2006; Miklowitz & Johnson, 2006). Finally, bipolar disorder has a stronger genetic predisposition and therefore is more likely to run in families than major depression (Goodwin & Jamison, 2007). Although it was previously believed that major depression had an earlier age of onset than bipolar disorder, it is now common to see bipolar disorder diagnosed in children and adolescents as well, most likely because of greater awareness of the disorder in youth (Youngstrom, Birmaher, & Findling, 2008).

Depression and Bipolar Disorder Spectra Many people are chronically depressed or experience depressed and expansive mood periods that are not severe enough to merit a major depressive or manic episode diagnosis. Owing to the variation in mood disorders, from more mild and chronic to more severe and episodic, many researchers support the notion that both depression and bipolar disorder should be considered as continua or spectra as opposed to discrete disorders (Hankin, Fraley, Lahey, & Waldman, 2005; Merikangas et al., 2007).

Unipolar Depression Spectrum Depressions can manifest in many different ways and differ in severity and duration. DSM-5 recently changed the name of the prior diagnosis of dysthymic disorder to *persistent depressive disorder (dysthymia)*, which involves a mild, persistent depression that may occur for 2 or more years. Dysthymic individuals are typically morose, pessimistic, introverted, overconscientious, and incapable of fun (Akiskal & Cassano, 1997). In addition, these individuals often have lower energy, low self-esteem, and disturbances of eating, sleeping and thinking that are associated with MDD, but their functioning is worse (Klein, Schwartz, Rose, & Leader, 2000). Whereas dysthymia is chronic, MDD is usually episodic, although major depressive episodes can last months or longer. Dysthymia, like MDD, is 1.5–3 times more common in women than men and it has the same neurophysiological abnormalities and responses to antidepressant medication as MDD (Akiskal, Djenderedjian, Rosenthal, & Khani, 1997). In DSM-5, clinicians can make specifications of the severity of an episode of mild, moderate, or severe. Researchers have termed it *double depression* when an major depressive episode is superimposed on dysthymia. Double depression is considered to be on the more severe end of the unipolar depression spectrum. In essence, an individual with dysthymia may sink into a major depressive episode (about 77% of dysthymic individuals develop MDD; Klein et al., 2000) and then recover from the major depression but continue their mild, persistent dysthymia. Kessing (2007) examined the subtypes of depression in a large-scale epidemiological study and found no clear demarcation between mild, moderate, and severe depression, supporting a continuum. In addition, depressive symptoms may change over time; so that individuals change diagnosis from meeting criteria for MDD, to minor depression (a subthreshold episode), to dysthymia, and subsyndromal states (Kessing, 2007). Although all three disorders have high rates of recovery, people with double depression and dysthmia have more impaired social and physical functioning than those with MDD (Rhebergen et al., 2009). In addition, the more chronic dysthymia is characterized by a worse quality of life, inadequate social support, and slower rates of improvement compared to MDD (Klein, Shankman, & Rose, 2006; Subodh, Avasthi, & Chakrabarti, 2008). DSM-5 included a new diagnosis of *disruptive mood dysregulation disorder* to capture chronic and severe persistence of irritability that is often associated with emotional dysregulation and often leads to frequent temper outbursts.

Bipolar Spectrum Similar to the unipolar spectrum, bipolar disorder can manifest itself in many different ways. Like dysthymia, *cyclothymic disorder* is a chronic condition that may last for years and may never go a few months without a hypomanic or depressive phase. Cyclothymic disorder is mild and persistent and becomes a way of life. For instance, individuals with cyclothymic disorder come to depend on their hypomanic phase in order to work long hours and catch up on previously delayed tasks before slipping back into a normal or depressed state. Family members often describe cyclothymic individuals as "moody," "high-strung," "hyperactive," and "explosive" (Akiskal et al., 1977). Bipolar disorders appear to form a spectrum of severity from the milder, subsyndromal cyclothymic disorder, to bipolar II disorder, to full-blown bipolar I disorder at the most severe end (Cassano et al., 1999; Goodwin & Jamison, 2007). Three lines of evidence support this spectrum model. First, equivalent rates of bipolar disorder have been reported in the first- and second-degree relatives of cyclothymic disorder and bipolar I individuals (Akiskal et al., 1977; Depue et al., 1981), and increased rates of cyclothymic disorder are found in the first-degree relatives of bipolar patients (Chiaroni, Hantouche, Gouvernet, Azorin, & Akiskal, 2005). In addition, among monozygotic twins, when one twin had bipolar disorder, the co-twin had elevated rates of both bipolar and cyclothymic disorders (Edvardsen et al., 2008). These findings suggest that cyclothymic disorder shares a common genetic diathesis with bipolar disorder. Second, individuals with cyclothymic disorder, like bipolar I patients, often experience an induction of hypomanic episodes when treated with tricyclic antidepressants (Akiskal et al., 1977). Finally, individuals with cyclothymic disorder are at increased risk of developing bipolar I or II disorder when followed over time (Alloy, Urosevic et al., 2012; Birmaher et al., 2009; Kochman et al., 2005; Shen, Alloy, Abramson, & Grandin, 2008). Similar to unipolar depression, bipolar disorder also can carry a specifier of severity of mild, moderate, and severe.

Dimensions of Mood Disorder

In addition to the important distinctions between bipolar and depressive disorders, there are certain dimensions that researchers and clinicians have found useful in classifying mood disorders. We discuss three dimensions: psychotic versus non-psychotic, early versus late onset, and endogenous-reactive. In addition, we discuss the importance of life events in the onset of mood disorders, because this was the original basis of the endogenous-reactive distinction.

Psychotic Versus Non-psychotic Some individuals who have episodes of major depression or mania may also experience associated symptoms of psychosis. In fact, the diagnosis of MDD or bipolar disorder in DSM-5 can have the additional distinction of "with mood-congruent psychotic features" (American Psychiatric Association, 2013, pp. 152, 186). To get this qualifier for either a major depressive or a manic episode, the psychotic symptoms must occur during mood episodes. Whereas prior issues of the DSM differentiated psychotic mood disorders from the diagnosis of schizoaffective disorder by this mood qualifier, the DSM-5 can have a specifier of "with mood-incongruent psychotic features' that suggests the psychotic symptoms can be outside of a mood episode. Historically, the psychotic characterization was used to describe the severity of the episode, but more recent research suggests that there may be other factors at work (Forty et al., 2009; Bora, Yucel, Fornito, Berk, & Pantelis, 2008).

Major depressive disorder with psychotic features is not uncommon. Roughly 14% of individuals with MDD had a history of episodes with psychotic features (Johnson, Howarth, & Weissman, 1991). Crebbin, Mitford, Paxton and Turkington (2008) found that the diagnosis of psychotic depression was more prevalent than schizophrenia in first episodes of psychosis. In psychotic depression, hallucinations, delusions, and extreme withdrawal are usually congruent with the depressed mood. For instance, the content of these delusions or hallucinations is generally themed around personal inadequacy, guilt, or deserved punishment. Individuals with psychotic features have generally more severe depressive episodes and greater hormonal disturbances (Contreras et al., 2007). Importantly, the diagnosis of MDD with psychosis is relative unstable over time. Over 50% of individuals initially diagnosed with MDD with psychotic features switched diagnosis to bipolar disorder, schizophrenia, or schizoaffective disorder within 10 years (Ruggero et al., 2011).

Episodes of mania with psychotic features are more prevalent than depressive episodes with such features. Estimates of lifetime prevalence of psychotic features occurring during at least one manic episode range from 50–75% of those diagnosed with bipolar disorder (Ozyildrim, Cakir, & Yaziki, 2010; Canuso, Bossi, Zhu, Youssef, & Dunner, 2008). Although present to a lesser degree in many manic episodes, thoughts of grandiosity, lack of judgment/insight, and suspiciousness/persecution were at more delusional levels in those with psychotic features. One study found that working memory deficits may be a marker for differences between those with a diagnosis of bipolar disorder with psychosis and those without this specifier (Allen et al., 2010).

Researchers continue to debate whether mood disorders with psychotic features are distinct disorders or simply on the more severe end of a spectrum. Kraepelin (1921), in his original classification system, listed all incapacitating mood disorders under the heading "manic-depressive psychosis," which he considered distinct from those episodes at the non-psychotic-level. Some researchers still hold this position. For instance, Bora and colleagues (2008) suggest that a mood episode with psychotic features may be associated with different genetic and neurobiological markers compared with those without psychotic features. Other researchers argue that psychotic features are present only at the severe levels of a disorder. For instance, Ozyldirim and colleagues (2010) describe psychotic episodes of mania as more severe, more likely to lead to hospitalization, and less responsive to some medications.

Early Versus Late Onset Evidence over the last few decades suggests that age at onset is an important factor in the trajectory of mood disorders. In general, people who develop a mood disorder earlier have poorer outcomes. For example, individuals with recurrent MDD before 15 years of age had more clinical features, poorer social outcomes and psychosocial adjustment, and greater anxiety comorbidity than those without recurrent episodes (Hammen, Brennan, Keenan-Miller, & Herr, 2008). In addition, an earlier age of onset of depression is associated with a higher risk of suicide intent (Thompson, 2008) and with neural abnormalities in different brain regions (Chen et al., 2012). Further, those who developed an earlier onset of depression are more likely to misuse alcohol, harm themselves, and have higher rates of comorbidity (Voshaar, Kapur, Bickley, Williams, & Purandare, 2011). Heritability may play a role, as the earlier the onset of a depressive disorder, the more likely it is the person has relatives with a mood disorder (Klein et al., 1999).

In recent years, research also has shown similar trajectories in cases of early onset bipolar disorder. Oedegaard, Syrstad, Morken, Akiskal, & Fasmer (2009) found that about 60% of people with bipolar disorder had an early onset (before the age of 20 years) and 13% occurred in childhood (before 13 years of age). Childhood onset has been associated with a more chronic, severe, and recurrent course of disorder, with poorer functioning and quality of life than adult onset (Perlis et al., 2009; Birmaher et al., 2009). In addition, early onset was associated with a higher percentage of first-degree relatives with a history of mental illness (Baldessarini et al., 2012). Finally, research suggests a genetic difference for those who develop early onset bipolar disorder (Priebe et al., 2012).

Investigators have begun to examine the early onset of bipolar disorder in children and many researchers still

question the validity of this diagnosis. Bipolar disorder was recognized only recently in children and a lack of consensus on its features remains (Luby & Navsaria, 2010). Even though this issue remains controversial, the prevalence of new cases of bipolar disorder in children is increasing (Danner et al., 2009). Some suggest that the increased prevalence may be due to an increase in awareness of childhood onset (e.g., Moreno et al., 2007), whereas others argue that there is an increase in misdiagnosis (e.g., Danner et al., 2009).

Endogenous Versus Reactive Originally, the terms endogenous and reactive were used to identify whether or not a depressive episode was preceded by a precipitating event. Those linked to these stressful events were considered reactive, whereas those that were not linked to an event were called endogenous (literally, "born from within") and considered to have a more biological basis. Proponents of Kraepelin's tradition make a distinction between nonpsychotic depressions as generally reactive, whereas psychotic depressions are endogenous (Rehm et al., 2001).

Research has subsequently shown that most depressive episodes, including those in bipolar disorder, are preceded by stressful life events (Alloy, Abramson, Urozevic, Bender, & Wagner, 2009; Johnson & Kizer, 2002), and stressful events are a major cause of depressive episodes (Kendler, Karkowski & Prescott, 1999). In some cases, there is a precipitating event for the first episode, but life events become progressively less important for subsequent recurrences, an effect known as "kindling" (Morris, Ciesla, & Garber, 2010; Monroe & Harkness, 2005). Generally speaking, despite the definition of endogenous and reactive, these terms are no longer used to indicate whether there was a precipitating event, but to describe different patterns of symptoms (Rehm et al., 2001). Individuals who show marked anhedonia with associated physical symptoms, such as early morning awakening, weight loss, and psychomotor changes, are described as having a depression that is qualitatively different from those that occur after a death or loss of a loved one, and are thus characterized as endogenous. In DSM-5, this endogenous characteristic is referred to as melancholic features. Indeed, recent research confirms that those with melancholic features are more likely to report episodes coming "out of the blue" and to be more severe than those with depression without melancholic features (Parker, Fletcher, & Hadzi-Pavlovic, 2012).

The distinction between endogenous and reactive depression based on symptoms seems to have greater validity. Individuals with endogenous depression differ from those with reactive depression in their sleep patterns. Individuals who prefer evening activities have reported more severe depressive symptoms compared with others based on this biological characteristic (Hidalgo et al., 2009). People with endogenous depression also are more likely to show neurobiological abnormalities and respond to biological treatments, such as electroconvulsive therapy (Rush & Weissenburger, 1994). Accordingly, some researchers still suspect that endogenous depression is more biological, although there is some research to the contrary. For example, if endogenous depression were more biologically based, research would show that individuals with these features have greater family histories of depression, but numerous studies have shown that they do not (Rush & Weissenburger, 1994). In sum, the distinction between reactive and endogenous depression is generally based on symptom presentation, because most mood episodes, whether mania or depression, are preceded by life events.

Life Events in Depression and Bipolar Disorders Depression is usually precipitated by events that involve failures or uncontrollable interpersonal loss such as death, divorce, or separation (Cronkite & Moos, 1995, Kendler et al., 1999; Monroe Rohde, Seeley, & Lewinsohn, 1999). By the same token, if a person has social support in the form of close personal relationships, he or she is less likely to succumb to depression in the face of stressful life events (e.g., Panzarella, Alloy, & Whitehouse, 2006). Further, stressors that are in part dependent on the individual's behavior (such as a fight) are more likely to lead to depression than events that are independent of their behavior (such as a death in the family; Hammen, 2006; Liu & Alloy, 2010). In addition, some researchers postulate that if an individual holds a specific personality predisposition, events that are specifically congruent with that style predict mood symptoms (e.g., Francis-Raniere, Alloy, & Abramson, 2006). For instance, Francis-Raniere and colleagues (2006) showed that for personalities characterized by self-criticism and concern about performance evaluation, congruent negative and positive events predicted depressive and hypomanic symptoms, respectively.

The association between stressful life events and depressive or manic episodes differs for first onset of depression or mania and future recurrences. The kindling hypothesis, first postulated by Post (1992), suggests that stressful life events are important for the first onset, but that the association between events and episodes becomes weaker as the number of episodes increases (Bender & Alloy, 2011; Kendler, Thornton, & Gardner, 2000; Monroe & Harkness, 2005). Monroe and Harkness (2005) suggest two interpretations of these findings. The first interpretation is that subsequent episodes are less reliant on stressful events and eventually become autonomous; therefore, depressive episodes may occur without a precipitating event (Monroe & Harkness, 2005). Another interpretation is that an event may still be required to precipitate subsequent depressive episodes, but events can be of decreasing severity as individuals experience more episodes, wherein eventually minor events, daily hassles, or expectation of a stressful event could trigger an episode (Monroe & Harkness, 2005).

Similar to depressive disorders, stressful life events also have been shown to predict symptoms and episodes of mania and depression in bipolar disorder. In depression, negative life events generally precede an episode, whereas both negative and positive life events can precede a hypomanic/manic episode (Alloy, Abramson, Urosevic, Bender et al., 2009; Johnson, 2005). However, particular types of negative and positive events have been found to trigger bipolar mood episodes. For example, events that disrupt daily social routines (e.g., sleep/wake times, meal times), predict both depressive and hypomanic/manic symptoms and episodes (Malkoff-Schwartz et al., 1998, 2000; Sylvia et al., 2009). In addition, life events involving attainment of or striving toward a desired goal predict increases in manic symptoms or onsets of hypomanic episodes among individuals with bipolar spectrum disorders (Johnson et al., 2008; Nusslock et al., 2007). The psychological and neurobiological mechanisms by which life events trigger mood episodes are of considerable importance in ultimately understanding the causes of mood disorders.

Suicide

Suicide is defined as death from a self-inflicted injury committed with the intent to die. In addition to completed suicide, other suicidal behaviors include suicide attempts, which are defined as self-injurious behaviors committed with certain or ambivalent intent to die, and suicidal ideation, which consists of thoughts about being dead or killing oneself (Miller, Rathus, & Linehan, 2007). Of the many reasons why people take their own lives, depression and bipolar disorder are among the most common. MDD is the psychiatric diagnosis most commonly associated with suicide, with the risk of suicide among depressed individuals approximately 20 times greater than in the general population (Centers for Disease Control and Prevention, 2015). Individuals who have also had a manic episode are at even greater risk, with the rates of completed suicide among individuals with a bipolar disorder nearly 60 times greater than the general population (Fountoulakis, Gonda, Siamouli, & Rhimer, 2009).

Prevalence In 2011, the last year for which statistics are available, over 38,000 people died by suicide in the United States, making suicide the tenth leading cause of death. At this rate, approximately one suicide occurs every 14 minutes in the United States (Hoyert & Xu, 2012). These rates are even higher among younger populations, with suicide being the second leading cause of death for 15- to 24-year-olds and the fourth leading cause of death for 5- to 14-year-olds in the United States (Hoyert & Xu, 2012). These statistics do not account for those who engage in the wide range of suicidal behaviors, including thoughts, plans, gestures, and attempts, which are far more prevalent than completed suicide. Among adults in the United States between the years 2008 and 2009, approximately 8.3 million (3.7% of the adult U.S. population) reported having suicidal thoughts, 2.2 million (1.0% of the U.S. adult population) reported having suicidal plans, and 1 million (0.5% of the U.S. adult population) reported making a suicide attempt (Crosby, Han, Ortega, Parks, & Gfoerer, 2011).

Despite these high rates, suicide may still be underreported, for a variety of reasons. First, the deaths of many people who commit suicide may actually appear accidental. For example, the leading methods of suicide include firearms (50.5% of completed suicides), suffocation (24.7% of completed suicides), and poisoning (17.2% of suicides), and it is frequently difficult to determine whether death by these causes is accidental or purposeful (Earton, 2012). Furthermore, it has been estimated that approximately 15% of fatal automobile accidents are actually suicides (Finch, Smith, & Pokorney, 1970). Another obstacle to obtaining accurate statistics on the prevalence of suicide is the variability in the definition of suicide (Sainsbury & Jenkins, 1982). Although completed suicide is the most severe end of the suicidal spectrum, many other thoughts and behaviors may be considered suicidal. For example, individuals may experience suicidal thoughts, develop plans to commit suicide, or engage in non-fatal suicidal behaviors, such as cutting or burning themselves, without ever actually attempting or committing suicide. Because many behaviors characterize suicide, a consistent definition of suicide is needed to most effectively monitor the incidence of suicide and examine trends over time and across studies (Goldsmith, Pellmar, Kleinman, & Bunny, 2002).

Along these same lines, research suggests that an individual's intent to die must be taken into account when determining whether behaviors are suicide attempts or not (O'Carroll, Berman, Maris, & Moscicki, 1996). Research suggests that only 39% of people who attempt suicide are truly determined to die and that another 13% are ambivalent about dying (Kessler, Borges, & Walters, 1999). Furthermore, research within the first decade of the 21st century highlights the importance of differentiating suicidal behavior from non-suicidal self-injury, which is characterized by the direct destruction of bodily tissue without the intent to die (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006). In sum, more accurate classification of suicidal behaviors is needed, as engagement in prior suicidal behavior, such as ideation and attempts, is among the best, if not the best, predictor of eventual completed suicide (e.g. Joiner et al., 2003; Kessler et al., 1999; Nock et al., 2008).

Risk Factors Given the costs of suicide to both society and individuals, a better understanding of the risk factors associated with suicide is critical (Hoyert & Xu, 2012). Risk factors may be stable or transitory, characteristic of the individual or of the environment, and they may have occurred in the past (distal) or be occurring in the present (proximal). Suicide is a complex phenomenon with many causes and is typically a culmination of a

variety of biological, psychiatric, and environmental factors (Rihmer, 2007; for a review of risk factors, see the U.S. Department of Health and Human Services' National Strategy for Suicide Prevention, 2001.)

Gender Although women are more likely than men to think about and attempt suicide, particularly during adolescence, males commit suicide at nearly four times the rate of females and represent approximately 79% of all completed suicides in the United States (Centers for Disease Control and Prevention, 2015; Lewinsohn, Rohde, Seeley, & Baldwin, 2001). A common explanation for this difference in completed suicide is that males tend to choose more lethal methods. Firearms are the most commonly used method of completed suicide among males, whereas poisoning is the most common method of completed suicide among females (Centers for Disease Control and Prevention, 2015).

Race/Ethnicity The highest rates of suicide are found among American Indian/Alaska Native adolescents and young adults (31 suicide deaths per 100,000) and among non-Hispanic White Americans (13.5 suicide deaths per 100,000). The lowest rates of suicide are seen among Hispanics (6 suicide deaths per 100,000) and non-Hispanic African Americans (5.1 suicide deaths per 100,000). These lower rates among African Americans and Hispanics, however, may be indicative of underreporting and misclassification rather than actual differences due to race/ethnicity (Centers for Disease Control and Prevention, 2015; Rockett, Samora, & Cohen, 2006).

Age In general, the risk of committing suicide increases as a function of age, particularly among males. Older adults are disproportionately likely to die by suicide, with 9 of every 100,000 females aged 45–54 years dying by suicide and 31 of every 100,000 males 75 and older dying by suicide (Centers for Disease Control and Prevention, 2015). Although rates are highest among older adults, suicide rates for adolescents and young adults between ages 15 and 24 have quadrupled among males and doubled among females over the past 60 years. Although suicide rates in this age group have declined almost 30% since 1994, suicide remains the second leading cause of death among 15- to 24-year olds (Hoyert & Xu, 2012). Approximately 16–17% of high-school students consider suicide, between 13% and 16.5% make plans for an attempt, and approximately 4–8% make an attempt each year (Grunbaum et al., 2002). Furthermore, for every completed youth suicide, it is estimated that 100–200 attempts are made (Centers for Disease Control and Prevention, 2015).

Sexual Orientation Research over the 1990 and 2000s suggests that lesbian, gay, and bisexual individuals are more likely than heterosexual individuals to attempt and commit suicide. This may be particularly true among youth (see Ploderl et al., 2013, for a review). Researchers hypothesize that increased social stressors among these populations, including peer victimization, "anti-gay" social climates, and physical abuse by parents contribute to increased rates of depression and substance use, thus conferring additional risk for suicide (Hatzenbuehler, 2011).

Biology/Genetics Twin and adoption studies show that the predisposition to engage in suicidal behavior may be at least partly inherited. Relatives of individuals who have died by suicide are two to six times as likely to commit suicide themselves (Mann et al., 2009), and children of parents who suffer from mental health disorders are more likely to engage in suicidal behavior (Gureje et al., 2011). Although the specific genes responsible for increased suicide risk are unknown, genes related to the neurotransmitter serotonin may be potential candidates for suicidal behavior, as altered brain serotonin functioning has been associated with attempted and completed suicide among individuals with depression (Galfalvy, Huang, Oquendo, Currier, & Mann, 2009). In addition, hyperactivity of the hypothalamic–pituitary–adrenal (HPA) axis has been implicated as a risk factor for suicide in major depression (Coryell, Young, & Carroll, 2006).

Mental Disorders Attempted and completed suicides are very rare in the absence of a major mental disorder (Beautrais, Joyce, Mulder, & Fergusson, 1996), and psychiatric risk factors are often the most powerful and clinically useful predictors of suicide. Approximately 90% of people who attempt or commit suicide have at least one (usually untreated) major mental disorder. The most common disorders associated with suicide are major depression (56–87% of suicide attempts and completions), substance use disorders (26–55%), and schizophrenia (6–13%). In addition, individuals with depression or bipolar disorder who commit or attempt suicide most often do so during a major depressive episode and very rarely during mania (Rihmer, 2007). Specific risk factors within major depressive episodes, in order of significance, include: (1) current suicidal ideation, plan, or wish to die; (2) prior suicide attempt; (3) severe depression characterized by hopelessness and/or guilt; (4) current or recently released psychiatric inpatient status; (5) diagnosis of bipolar II, followed by bipolar I, followed by unipolar depression; 6) depressive mixed states; (7) cyclothymia; (8) psychotic symptoms; and (9) concurrent anxiety disorders, substance use, and serious medical illness (Coryell & Young, 2005; Fiedorowicz et al., 2009; Rihmer, 2007; Oquendo et al., 2004).

Protective Factors Certain factors may actually decrease the risk for suicide. These protective factors include effective treatment for mental disorders and substance abuse, easy access to treatment and support for help seeking,

family and adult/teacher/community support, cultural and religious beliefs that discourage suicide, and skills in problem solving and active coping (Centers for Disease Control and Prevention, 2015; Eisenberg, Ackard, & Resnick, 2007; Meadows, Kaslow, Thompson, & Jurkovic, 2005).

Predicting and Preventing Suicide The friends and family members of individuals who commit suicide are often shocked, indicating that they saw no signs that their loved one was at risk. Despite this fact, most suicidal people clearly communicate their intent prior to their death, often to their close relatives. Also, most individuals who commit suicide have visited a psychiatrist or general practitioner within weeks or months of their fatal attempt (Isometsä, Henriksson, Aro, & Heikkinen, 1994). Research suggests, however, that upon these visits to professionals, suicidal individuals were either prescribed vitamins or improper psychotropic medications (Rutz, 1996). Clearly, the recognition and management of pre-suicidal individuals are poor (Oquendo, Malone, Ellis, Sackeim, & Mann, 1999). Solutions to this problem include better recognition of the signs of suicide, greater awareness of treatment possibilities, and improved training for non-psychiatric health care professionals and other key personnel who are in positions to recognize the signs of suicide (Gonda, Fountoulakis, Kaprinis, & Rihmer, 2007).

If suicide can be predicted, then perhaps it can be prevented. Researchers have identified several key areas of suicide prevention, including 1) providing education and awareness about the signs of and effective treatments for suicide directed both to the general public and to healthcare providers and school personnel; 2) implementing screening tools for identifying individuals most at risk of suicide so that these individuals might be directed to appropriate treatment; 3) increasing access to mental health services and effective treatments for psychiatric disorders, as individuals who receive appropriate treatment for an underlying psychiatric disorder have the highest likelihood of recovery (Rudd & Joiner, 1998); 4) implementing effective suicide prevention programs; 5) restricting access to lethal means such as firearms, as this may delay a suicide attempt and allow time for the individual to seek help; and 6) managing how suicide is portrayed and reported in the media, as research supports a connection between media portrayals and subsequent increases in suicide rates, particularly among youth (Gould, Jamieson, & Romer, 2003). For reviews, see Mann et al., (2005) and U.S. Department of Health and Human Services (2001).

Mood Disorders: Causes and Treatments

Most theories of the causes of mood disorders, as well as treatments for mood disorders, have focused on depression because it is far more common than hypomania and mania. However, some theoretical perspectives have addressed bipolar disorder as well, and we discuss some of these. Among the general theoretical approaches to mood disorders, behavioral/interpersonal, cognitive, and neuroscience perspectives have had the greatest influence on understanding the causes of and generating treatments for mood disorders. Thus, we present these three perspectives in greater detail and then discuss the psychodynamic approach more briefly.

Interpersonal Perspective Interpersonal theories of depression (e.g., Coyne, 1976; Joiner, 2000; Giesler, Josephs, & Swann, 1996) focus on the social context of depression. According to interpersonal formulations, depressed individuals engage in various maladaptive interpersonal strategies in an attempt to improve or regulate mood. But, these strategies are typically unsuccessful.

Excessive Reassurance Seeking Excessive reassurance seeking is an aversive behavioral style in which depressed individuals persistently seek reassurance that others care about and value them (Coyne, 1976). Coyne posits that whereas the act of reassurance seeking is an individual's behavioral attempt at changing or improving their depressed state, the behavior actually serves to maintain the depression and worsens the individual's interpersonal environment. The excessive reassurance seeking irritates and drives away friends and family members, leaving the individual rejected (Joiner, Metalsky, Katz, & Beach, 1999) and with a weakened social support network (Potthoff, Holahan, & Joiner, 1995). The depressed individual uses this withdrawal as evidence for depressive cognitions (e.g., "I'm unlovable"). The generated stress and further entrenchment of these depressive beliefs serves to deepen the depression in a self-perpetuating cycle (Joiner, 2000). A meta-analytic review of 38 (N = 6,973) cross-sectional studies examining the relationship between depression and excessive reassurance seeking (Starr & Davila, 2008) revealed a moderate effect size (r = .32) between excessive reassurance seeking and depression. Indeed, excessive reassurance seeking has been found to predict subsequent increases in depressive symptoms in both adults and youth (e.g., Abela, 2005; Davilla, 2001; Joiner, Metalsky, Gencoz & Gencoz, 2001; Joiner et al., 1999).

Negative Feedback Seeking Giesler, and colleagues (1996) suggest that depressed individuals actually seek out rejection and negative feedback from others. They propose that depressed individuals find rejection and negative feedback more predictable and in line with their negative self views. However, the presence of these negative stressors may serve to deepen the depression rather than relieve it. Researchers have found support for the transactional relationship between negative feedback, rejection, and depression (Borelli & Prinstein, 2006; Pettit & Joiner, 2001; Joiner et al., 1999).

Behavioral Perspective

Extinction Early behavioral theories focused on the relationship between external reinforcers and behavior. Many

behaviorists regard depression as a result of behavioral extinction (Fester, 1973; Lewinsohn, 1974; Jacobson, Martell, & Dimidjian, 2001). Generally, the behavioral theory of depression suggests that once behaviors are no longer rewarded (reinforced), individuals cease to perform these behaviors that were previously rewarded (extinction). Through the resulting withdrawal and inactivity, individuals become depressed. The behavioral theorists propose several reasons for the reduction in positive reinforcement that leads to the extinction of behaviors and subsequent depression. For example, Lewinsohn (1974) proposed that positive reinforcement is influenced by the number of available reinforcers in the environment, the range of stimuli that the individual finds reinforcing, and the ability of the individual to obtain reinforcement from the environment. In addition, Ferster (1973) proposed that changes in the environment and the increased passivity of the individual could influence the frequency of reinforcement of behaviors.

A number of studies have supported the extinction perspective on depression. Lewinsohn and colleagues conducted several studies supporting a direct correlation between the number of pleasurable activities engaged in and depression (e.g. Lewinsohn & Graf, 1973; MacPhillamy & Lewinsohn, 1974). In fact, one of the most prominent objections to this model of depression has been that depressed individuals may lack the ability to have a pleasurable response to positively reinforcing stimuli (anhedonia), rather than just lacking positively reinforcing stimuli themselves.

Behavioral Activation Therapy In keeping with the extinction theory, behavioral activation treatment for depression is designed to increase activity and, in turn, develop more positively reinforcing behavior patterns in depressed individuals (Lewinsohn, Biglan, & Zeiss, 1976; Martell, Addis, & Jacobson, 2001). Jacobson and colleagues (2001) found that even severely depressed people show an elevation in mood if they are more behaviorally active and, thus, experience increased positive reinforcement. The authors describe that in behavioral activation, therapists help clients engage in planned activities, creating positive reinforcement and alleviating depressed mood. In behavioral activation, the client and therapist identify specific activities (positively reinforcing behaviors) that the client deems to be most helpful. Clients are then encouraged to engage in the activity based on a predetermined schedule, whether they feel like it or not in the moment. Depending on the success of the behavior in alleviating mood or improving quality of life, clients are asked to continue engaging in the behavior. Over time, clients are asked to engage in progressively more difficult activities, from getting out of bed at a regularly scheduled time to engaging in positive interpersonal activities. These changes break and reverse the cycle of sad mood, decreased activity, and withdrawal. More recent variants of behavioral activation include a values component early in therapy to identify pleasurable activities that may be more consistent with the client's core values (e.g., Lejuez, Hopko, & Hopko, 2003).

Substantial research lends support for behavioral activation as a successful treatment for depression. Behavioral activation therapy has been found to both reduce acute depression and prevent relapse over a 2-year follow-up (Jacobson et al., 1996; Gortner, Gollan, Dobson, & Jacobson, 1998). Several recent meta-analyses supported the utility of behavioral activation in treating depression (Cuijpers, van Straten, & Warmerdam, 2007; Ekers, Richards & Gilbody, 2008; Mazzucchelli, Kane, Rees, 2009). In general, the reviews found that activity scheduling/behavioral activation treatments for depression were as effective as other interventions (e.g. cognitive therapy) and more effective than control conditions in alleviating depression. For example, Cuijpers and colleagues' (2007) review of 16 randomized trials (N = 780 clients) of behavioral activation found a large pooled effect size of 0.87 (95% confidence interval: 0.60–1.15) with similar levels of depressive symptom reduction in individuals receiving behavioral activation as in those receiving comparable treatments (e.g., cognitive therapy). Their analysis also found that overall behavioral activation gains were maintained over time.

A review of the literature assessed the different specific treatment components of behavioral activation and their effectiveness (Kanter et al., 2010). The identified components include activity monitoring, assessment of life goals and values, activity scheduling, skills training, relaxation training, contingency management, procedures targeting verbal behavior, and procedures targeting avoidance. The authors found that activity scheduling, relaxation, and skills training interventions received empirical support on their own. All other techniques were effective, but only within larger treatment packages. Thus, they found that the most important components of behavioral activation were activity monitoring and scheduling in line with the main extinction theory of depression.

Behavioral treatments for depression also have included social skills training, aimed at ameliorating the aversive interpersonal behavior such as reassurance seeking and negative feedback seeking. Social skills training teaches depressed clients basic techniques to engage in satisfying social interactions through modeling of positive interpersonal behaviors by the therapist and role-playing by the client. Many behavioral treatments for depression are multifaceted, involving both behavioral activation and social skills training.

Cognitive Perspective Depression and mania involve changes in emotional, motivational, cognitive, and physical functioning. Cognitive theories of mood disorders hold that the cognitive changes are the crucial factor. According to cognitive formulations, the way people think about themselves, the world, and the future gives rise to the other phenomena in depression and mania.

Helplessness and Hopelessness The hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989) was derived from earlier work on learned helplessness (Seligman, 1975). Learned helplessness was first demonstrated with laboratory dogs. Seligman and his colleagues found that when dogs were exposed to inescapable electric shocks and then later were subjected to escapable shocks, they either did not try to escape or were slow and inept at escaping. The investigators concluded that when the shocks were inescapable, the dogs had learned that they were *uncontrollable*—a lesson they continued to act upon later even when it was possible to escape the shocks (Maier, Seligman & Solomon, 1969; Peterson, Maier, & Seligman, 1993). Seligman (1975) noted that this phenomenon closely resembles depression—that depression is a reaction to seemingly inescapable stressors in which the person learns that he or she lacks control over reinforcement and as a result gives up. The learned helplessness model is consistent with the finding that uncontrollable loss events typically precipitate depressive episodes. Note the difference between the learned helplessness and extinction theories. The crucial factor in extinction theory is an objective environmental condition—the lack of positive reinforcement—whereas the crucial factor in learned helplessness is a subjective cognitive process, the *expectation* of lack of control over reinforcement.

Exposure to uncontrollable (versus controllable) stress results in neurobiological changes consistent with depression (Minor & Saade, 1997; Maier & Watkins, 2005), and depressed patients who see themselves as helpless tend to show higher levels of 3-methoxy-4-hydroxyphenylglycol (MHPG), a product of norepinephrine metabolism (Samson, Mirin, Hauser, Fenton, & Schildkraut, 1992). Norepinephrine abnormalities are often found in depressed people. In addition, positron emission scans of people doing unsolvable problems, which tend to produce learned helplessness, show that learned helplessness is associated with increased limbic system brain activity. The limbic system is also implicated in the processing of negative emotions such as depression (Schneider et al., 1996).

The original learned helplessness model had certain weaknesses. Although it explained the passivity characteristic of depression, it did not explain the equally characteristic sadness, guilt, and suicidal thoughts, or that different cases of depression vary considerably in severity and duration. Consequently, Abramson and colleagues (1989) revised the helplessness model to a hopelessness theory. According to the hopelessness theory, depression depends not just on the belief that there is a lack of control over reinforcement (a *helplessness expectancy*), but also on the belief that negative events will persist or recur (a *negative outcome expectancy*). When a person holds both of these expectations—that bad things will happen and that there is nothing one can do about it—he or she develops hopelessness, and it is this hopelessness that is the immediate cause of the depression (Abramson et al., 1989).

Hopelessness, in turn, stems from the inferences people make regarding stressful life events, that is, the perceived causes and consequences of such events. People who see negative life events as due to causes that are (1) stable (permanent rather than temporary); (2) global (generalized over many areas of their life rather than specific to one area of their functioning); and (3) internal (part of their personalities, rather than external, or part of the environment) are at greatest risk for developing hopelessness and, in turn, severe and persistent depression. Similarly, people who infer that stressful events will have negative consequences for themselves or who infer that the occurrence of stressful events means that they are incompetent and unworthy are more likely to become hopeless and depressed. In fact, Abramson et al. (1989) proposed that *hopelessness depression* constitutes a distinct subtype of depression, with its own set of causes (negative inferential styles combined with stress), symptoms (passivity, sadness, suicidal tendencies, low self-esteem), and appropriate treatments. This theory also applies to suicide. Hopelessness is the best single predictor of suicide, even better than depression (Beck, Brown, Berchick, Stewart, & Steer, 2006; Abramson et al., 2000).

The hopelessness theory has been extensively tested with mostly positive results. Depressed individuals are more likely than non-depressed individuals to attribute negative events to internal, stable, and global attributions (Joiner & Wagner, 1995; Sweeney, Anderson, & Bailey, 1986) and to exhibit expectations of low control or helplessness (Weisz, Southham-Gerow, & McCarty, 2001). Moreover, a negative inferential style is relatively stable over many years (Romens, Abramson, & Alloy, 2009) and predicts who has been depressed in the past (Alloy, Abramson, Hogan et al., 2000), who among never depressed individuals will develop a first onset of major depression and hopelessness depression (Alloy, Abramson, Whitehouse et al., 2006), who will become suicidal in the future (Abramson, Alloy, Hogan et al., 1998), and who, having recovered from depression, will relapse or have a recurrence (Illardi, Craighead, & Evans, 1997; Alloy, Abramson, Whitehouse et al., 2006). It also predicts who will have a worse course of depression (Iacoviello, Alloy, Abramson et al., 2006) and who, in a group of depressed people, will recover when exposed to positive events (Needles & Abramson, 2003).

Moreover, consistent with the vulnerability stress hypothesis of the hopelessness theory, many studies have found that the combination of a negative inferential style (the vulnerability) and exposure to negative life events (the stress) predicts subsequent increases in depressive symptoms (e.g., Abela, Stolow, Mineka, Yao, & Zhu, 2011; Gibb, Beevers, Andover & Holleran, 2006; Hankin, Abramson, Miller, & Haeffel, 2004). Other studies have shown that the reason a combination of stress and negative inferential style predicts depression is that this combination predicts hopelessness. It is hopelessness that, in turn, predicts depression (Alloy & Clements, 1998; Iacoviello, Alloy, Abramson, & Choi, 2010; Hamilton, Shapero et al.,

2013). Finally, people who show this combination also exhibit many of the symptoms hypothesized to be part of the hopelessness depression subtype (Alloy, Just, & Panzarella, 1997; Alloy & Clements, 1998; Iacoviello et al., 2013), and these symptoms cluster to form a distinct dimension of depression (Joiner, Steer et al., 2001). However, there is also conflicting evidence. For example, some researchers have found that the negative attributional style and stress combination does not necessarily lead to depression (Cole & Turner, 1993; Lewinsohn, Joiner, & Rohde, 2001).

Given that much evidence indicates that a negative inferential style does make people vulnerable to depression, it is important to discover the developmental origins of this cognitive vulnerability. Both social learning factors and a history of maltreatment may contribute to the development of negative inferential styles and depression. Individuals whose parents had negative cognitive styles, provided negative inferential feedback about the causes and consequences of stressful events in the individual's life (e.g., told their child, "You weren't invited to that party because you're unpopular, and now you'll be seen as a social outcast at school"), and whose parenting was low in warmth and affection are more likely to have negative cognitive styles as adults (Alloy, Abramson, Tashman et al., 2001; Garber & Flynn, 2001; Ingram & Ritter, 2000). In addition, people with childhood histories of emotional abuse from either parents or nonrelatives (peers, teachers, etc.) are also more likely to have negative cognitive styles as adolescents or adults (Gibb, Abramson, & Alloy, 2004; Gibb et al., 2001; Hamilton, Shapero et al., 2013). Emotional maltreatment also predicts onsets of depressive episodes (Liu et al., 2009). Thus, a history of negative emotional feedback and abuse may lead to the development of later cognitive vulnerability to depression. However, prospective studies beginning in childhood are needed to test this hypothesis.

Negative Self-Schema A second major cognitive theory of depression, Beck's (1967, 1987) negative self-schema model, evolved from his findings that the thoughts and dreams of depressed patients often contain themes of self-punishment, loss, and deprivation. Self-schemata are memory representations about the self that guide the way individuals process information from the environment such that individuals' attention is directed toward information that is congruent with the content of their self-schemata. Beck hypothesized that individuals who have negative self-schemata involving themes of inadequacy, failure, loss, and worthlessness are vulnerable to depression. Such negative self-schemata are often represented as a set of dysfunctional attitudes, such as "I am nothing if I do not succeed at this job," in which the person believes that his or her self-worth is dependent on being perfect or on others' approval. Further, when confronted with a negative life event, individuals with this type of cognitive style are hypothesized to develop negatively biased perceptions of themselves (low self-esteem), their personal world, and their future (hopelessness). According to Beck, this negative bias—the tendency to see oneself as a "loser"—is the fundamental cause of depression. If childhood experiences lead someone to develop a cognitive schema in which the self, the world, and the future are viewed in a negative light, that person is then predisposed to depression. Stress can easily activate the negative schema, and the consequent negative perceptions merely strengthen the schema (Beck, 1987; Clark, Beck, & Alford, 1999).

Research supports Beck's claim that depressed individuals have unusually negative self-schemata (Dozois & Dobson, 2001; Mathews & MacLeod, 2005) and that these schemata can be activated by negative cues. Negative self-schemata can also be activated by sad mood in people who have recovered from depression (Ingram, Miranda, & Siegel, 1998; Gemar, Segal, Sagrati, & Kennedy, 2001), and such reactivated negative schemata predict later relapse and recurrence of depression (Segal, Gemar, & Williams, 1999). There is also strong evidence that depressed individuals recall negative material more easily than positive material (Mathews & MacLeod, 2005) and recall autobiographical memories that are overly general rather than specific (Williams et al., 2007). The evidence that depressed individuals exhibit attentional biases toward negative stimuli is much more mixed; however, other findings provide stronger support for the notion that depressed individuals have difficulty inhibiting or disengaging their attention from negative material once they attend to it (Gotlib & Joorman, 2010; Koster, Lissnyder, Derakshan & De Raedt, 2011). This difficulty may underlie depressed individuals' tendency to persistently ruminate on their negative affect and the causes and consequences of their negative mood (Gotlib & Joorman, 2010; Koster et al., 2011). Rumination, in turn, has been found to predict subsequent onset of major depressive episodes (Nolen-Hoeksema, 2000; Spasojevic & Alloy, 2001; Robinson & Alloy, 2003) and more severe depressions (Nolen-Hoeksema, Wisco & Lyubomirsky, 2008).

Other studies indicate that people at high risk of depression, based on having negative cognitive styles, a past history of major depression, or parents who are depressed, selectively attend to and remember more negative than positive information about themselves (Alloy, Abramson, Murray et al., 1997; Ingram & Ritter, 2000; Taylor & Ingram, 1999). Still other research suggests that depressed individuals may have two distinct types of negative self-schemata, one centered on dependency, the other on self-criticism (Nietzel & Harris, 1990). For those with dependency self-schemata, stressful interpersonal events involving rejection or abandonment lead to depression. For those with self-criticism schemata, achievement failures should trigger depression. This hypothesis has been supported more strongly for dependency self-schemata and social events than for self-criticism schemata and failure (Coyne & Whiffen, 1995).

Although Beck's negative self-schema model of depression hypothesizes that depressed people exhibit

systematic biases in their processing of negative information, some evidence suggests that depressed individuals' pessimism is sometimes realistic, a phenomenon known as "depressive realism" or the "sadder but wiser" effect (Alloy & Abramson, 1988; Alloy, Wagner et al., 2010). For example, Lewinsohn, Mischel, Chaplin and Barton (1980) found that depressed individuals' evaluations of the impression they had made on others were more accurate than those of two non-depressed groups, both of whom thought they had made more positive impressions than they actually had. Similarly, Alloy and Abramson (1979) found that depressed people were far more accurate in judging how much control they had over outcomes than were non-depressed participants, who tended to overestimate their control when they were doing well and to underestimate it when they were doing poorly. Thus, it may be that non-depressed people are optimistically biased and that such biases are essential for psychological health (Alloy & Abramson, 1988; Haaga & Beck, 1995; Alloy, Wagner et al., 2010). Research supports this view. Alloy and Clements (1992), for example, found that individuals who were inaccurately optimistic about their personal control at baseline were less likely than more realistic participants to become depressed a month later in the face of stress.

Although most research on the cognitive theories of depression (both Beck's theory and the hopelessness theory) has focused on unipolar depression, findings suggest that cognitive models may be applicable to bipolar disorder as well. Individuals with bipolar disorders exhibit cognitive styles and self-schemata that are as negative as those with unipolar depression (Alloy, Abramson, Walshaw, Keyser et al., 2006; Alloy, Abramson, Walshaw, & Neeren, 2006). Moreover, negative cognitive styles combine with life events to predict subsequent increases in hypomanic/manic and depressive symptoms among people with bipolar disorder (Francis-Raniere et al., 2006; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999). However, evidence suggests that the cognitive styles of individuals with bipolar spectrum disorders have a distinct character; they are specific to themes of high incentive motivation, goal striving, and reward sensitivity (Alloy, Abramson, Walshaw, Gerstein et al., 2009; Lam, Wright, & Smith, 2004).

Behavioral Approach System Dysregulation The distinctive nature of cognitive styles among bipolar individuals is consistent with the behavioral approach system (BAS) hypersensitivity model of bipolar spectrum disorders. The BAS hypersensitivity model is a motivational cognitive theory of bipolar disorder, originally developed by Depue and Iacono (1989) and expanded by Alloy and Abramson (2010), Alloy, Abramson, Urošević, Bender et al. (2009) and Urošević, Abramson, Harmon-Jones, and Alloy (2008). The BAS regulates approach motivation and goal-directed behavior. It is activated by rewards or external or internal goal-relevant cues. BAS activation is implicated in the generation of positive goal-striving emotions such as happiness (Gray, 1994). In addition, the BAS has been linked to a reward-sensitive neural network involving dopamine neurons that project between several emotion- and reward-relevant limbic and cortical brain systems (Depue & Iacono, 1989). According to the BAS hypersensitivity model, an overly sensitive BAS that is hyperreactive to relevant cues increases a person's vulnerability to bipolar disorder. When vulnerable individuals experience events involving rewards or goal striving and attainment, their hypersensitive BAS becomes excessively activated, leading to hypomanic/manic symptoms, such as increased energy, optimism, euphoria, decreased need for sleep, grandiosity, and excessive goal-directed behavior (see also, Johnson, 2005; Johnson, Edge, Holmes, & Carver, 2012). Alternatively, in response to events involving irreconcilable failures, losses, or non-attainment of goals, their overly sensitive BAS becomes excessively deactivated, leading to a shutdown of behavioral approach and depressive symptoms, such as decreased energy, sadness, loss of interest, hopelessness, and decreased motivation and goal-directed activity.

Recent evidence supports the BAS hypersensitivity model. Individuals with bipolar spectrum disorders exhibit significantly higher levels of self-reported BAS sensitivity and reward responsiveness on behavioral tasks than do individuals without mood disorders (see Alloy & Abramson, 2010; Urošević et al., 2008 for reviews). As noted above, they also exhibit BAS-relevant cognitive styles (Alloy, Abramson, Walshaw, Gerstein et al., 2009) and overly ambitious goal striving and goal setting, as well as greater cognitive reactivity and positive generalization in response to success experiences (Johnson et al., 2012). Furthermore, they typically exhibit increased relative left frontal cortical activity on electroencephalography, a neurobiological indicator of BAS sensitivity and activation, both at rest and in response to rewards (Coan & Allen, 2004; Harmon-Jones et al., 2008). Moreover, high BAS sensitivity predicts first onset of bipolar spectrum disorders in individuals with no prior history of bipolar disorder (Alloy, Bender et al., 2012), faster time to onset of hypomanic and manic episodes (Alloy, Abramson, Walshaw, Cogswell et al., 2008), and a greater likelihood of progressing to a more severe bipolar diagnosis over follow-up among bipolar spectrum individuals (Alloy, Urošević et al., 2012). Finally, as noted previously, life events involving goal striving or attainment are especially likely to trigger hypomanic or manic episodes among bipolar spectrum individuals (Johnson et al., 2008; Nusslock et al., 2007), consistent with the BAS hypersensitivity model.

Cognitive Behavioral Therapy Beck and his colleagues developed a multifaceted therapy that includes behavioral assignments, modification of dysfunctional thinking, and attempts to change schemata (Beck, Rush, Shaw, & Emery, 1979). Cognitive behavioral therapy (CBT) identifies, challenges, and ultimately aims to modify cognitive

schemata to generate less negative information processing (Hollon, 2006). The alteration of schemata is considered most important and, according to Beck's theory, will immunize the patient against future depressions. First, however, the therapist attempts to remediate the current depression, through "behavioral activation"—that is, getting the patients to engage in pleasurable activities (see the discussion of behavioral activation therapy above)—and by teaching them ways of testing and challenging their dysfunctional thoughts. Depressed patients are asked to record their negative thoughts, together with the events that preceded them. They are then asked to counter such thoughts with rational responses and record the outcome. In addition, with the therapist's help, depressed patients are encouraged to conduct behavioral "experiments" that also allow them to test and challenge the veracity of their dysfunctional thoughts. CBT also includes *reattribution training*, which aims to correct negative attributional styles (Beck et al., 1979). Patients are taught to explain stressful events in more constructive ways ("It wasn't my fault—it was the circumstances," "It's not my whole personality that's wrong—it's just my way of reacting to strangers") and to seek out information consistent with these more hopeful attributions. A similar approach is used with suicidal patients. Beck and colleagues see this as a way of correcting negative bias and combating hopelessness.

In some encouraging evaluations, CBT has been shown to be at least as effective as medication (e.g., DeRubeis et al., 2005; DeRubeis, Siegle, & Hollon, 2008). A combination of CBT and pharmacotherapy may have a slight advantage when compared with either treatment by itself (DeRubeis et al., 2008; Kupfer & Frank, 2001). Moreover, some studies have supported less relapse and recurrence for CBT than for pharmacotherapy (e.g., Dobson et al., 2008; Hollon et al., 2005; Jarrett et al., 2001). There is also some debate as to whether CBT works as well as medication for severely depressed patients (DeRubeis, Gelfand, Tang, & Simons, 1999; Blackburn & Moorhead, 2000), but other studies suggest that it does (e.g., DeRubeis et al., 2005; Fournier et al., 2009).

Furthermore, there is controversy about the mechanisms by which CBT produces change. For example, there is evidence that CBT produces changes both in negative cognitions, as it is hypothesized to work, as well as in abnormal neurobiological processes (Blackburn & Moorhead, 2000; DeRubeis et al., 2008). So whether it works through the proposed cognitive mechanism or by changing biological processes in unclear. However, some studies indicate that much of the depression symptom improvement in CBT occurs in one between-session interval, called "sudden gains," and that these sudden gains appear to be associated with the correction of patients' negative beliefs in the immediately preceding therapy session, consistent with a cognitive mechanism of treatment efficacy (Tang et al., 2005, 2007). Also, as noted, Beck's CBT is multifaceted, including behavioral activation, together with cognitive restructuring. Some studies found that the behavioral activation component of CBT worked as well as the entire treatment package, both at alleviating depression and at preventing relapse (Dobson et al., 2008; Gortner, Gollan, Dobson, & Jacobson, 1998). Thus, it could be that CBT is just as effective without its cognitive components. Regardless of how it works, CBT does indeed work. Thus, recent efforts have extended this approach successfully to the prevention of depression in children and adolescents (e.g., Brunwasser, Gillham, & Kim, 2009; Garber et al., 2009). For example, the Penn Resiliency Program (Brunwasser et al., 2009) is a group intervention designed for late elementary and middle-school students that teaches cognitive behavioral and social problem-solving skills and is based on the Beck and hopelessness theories of depression. The Penn Resiliency Program has demonstrated some success in preventing symptoms of anxiety and depression among youth and its effects can be long-lasting (Gillham, Hamilton, Freres, Patton, & Gallop, 2006). CBT also has been extended to the treatment of patients with bipolar disorder as an adjunct to mood-stabilizing medications (Basco, 2000; Newman, Leahy, Beck, Reilly-Harrington, & Gyulai, 2002), with some success (see Nusslock, Abramson, Harmon-Jones, Alloy, & Coan, 2009, for review). Nusslock and colleagues (2009) suggested that considering the implications of BAS hypersensitivity in bipolar disorder might further improve the effectiveness of CBT for bipolar conditions.

CBT has also been modified further to incorporate mindfulness techniques. Mindfulness was adopted from eastern traditions such as Buddhism and refers to a mental state during which the individual aims to harness his or her attention and focus on momentary somatic, sensory, environmental, and cognitive experience in an open and accepting manner (Bishop et al., 2004; Kabat-Zinn, 1994; Marlatt & Kristeller, 1999). Mindfulness involves the self-regulation of attention and the development of an open/accepting orientation to the present moment upon which that attention is focused. In mindfulness practice, meditation exercises are used to help individuals develop this skill to focus on bodily, cognitive, and emotional experiences. Segal, Williams and Teasdale (2002) developed mindfulness-based cognitive therapy to help depressed individuals develop a decentered view of their thoughts, emotions, and sensations. They suggest that viewing these occurrences as mental events rather than reflections of reality changes the way one relates to thoughts, thereby breaking down the process through which maladaptive thoughts and mental processes (rumination) maintain depression. Recent reviews suggest that mindfulness-based CBT is effective in treating depression and reducing relapse rates (Coelho, Canter, & Ernst, 2007; Hofmann, Sawyer, Witt, & Oh, 2010) and as effective as pharmacotherapies in preventing relapse (Kuyken et al., 2008; Segal, 2010).

Acceptance and Commitment Therapy Acceptance and commitment therapy (ACT; Hayes, Strosahl, & Wilson, 1999) is often referred to as a 'third wave' of CBT. ACT,

like CBT, combines cognitive and behavioral components, but is based on relational frame theory (Hayes, Barnes-Holmes, & Roche, 2001), a contextual theory of language and cognition. ACT consists of six core components including acceptance, defusion, contact with the present moment, self as context, values, and committed action. Unlike CBT, the primary aim of ACT is not symptom reduction but rather increased psychological flexibility, or the ability to contact one's present moment experiences more fully and to engage in values-consistent behavior. Thus, the treatment aims to help individuals increase their acceptance of their subjective experiences. These experiences include distressing thoughts, beliefs, sensations and feelings. Orienting behaviors toward living life in line with one's values (e.g., moving toward desired relationships or career goals) is thought to improve the individual's quality of life. A central feature of ACT is learning to view attempts to control unwanted experiences such as negative emotions or thoughts as ineffective. In fact, the model suggests that these attempts at controlling experiences actually serve to increase distress. Clients are encouraged to encounter their thoughts and feelings without the aim of controlling them. Instead, they are guided to focus on leading a values-oriented life. Thus, ACT promotes experiential acceptance and a commitment to one's actions to move toward this more valued life. A recent meta-analytic review documented growing evidence for the efficacy of ACT interventions (Hayes, Luoma, Bond, Masuda, & Lillis, 2006). More recent (Forman, Herbert, Moitra, Yeomans, & Geller, 2007) examinations of the efficacy of ACT suggest that it is at least as efficacious as well-established cognitive therapies in treating depression. In their study, Forman and colleagues reported that similar to those in the CBT group, clients in the ACT intervention group exhibited large gains in clinician rated functioning, quality of life, life satisfaction, and significant reductions in depressive symptoms.

Psychodynamic Perspective

Loss and Attachment Psychodynamic theorists have proposed several causes of depression. In his classic essay of 1917, "Mourning and Melancholia," Freud (1994) likened the experience of depression to that of mourning for a lost object, either real or imagined. Although sadness is a natural response to a loss, Freud posited that depression is an overreaction to such an event. The individual is filled with conflicting feelings—love for this lost object, but simultaneously anger and resentment toward it. The anger soon turns into feelings of guilt because the individual feels responsible for the loss of the loved object and failing to live up to his or her own ideals. The depressive episode becomes a cry for love brought on by feelings of emotional insecurity (Rado, 1951). The finding that depression is frequently precipitated by life events involving interpersonal loss is consistent with this psychodynamic theory.

Another psychodynamic perspective on the etiology of depression comes from attachment theory, which offers an expansion of the idea of loss triggering depression. Bowlby (1982) and other attachment theorists proposed that the loss of an attachment figure would increase the likelihood of other stressors for the child and simultaneously decrease his or her resiliency to future adversity, which predisposes him or her to adult depression. It is also postulated that the child will have unresolved mourning due to his or her young age and inability to understand the experience of loss (Bowlby, 1980). In fact, research has shown that a strong predictor of adult depression is history of a loss between age 5 and second grade (Coffino, 2009).

Attachment theory posits that an infant's experiences with his or her caregiver will influence how he or she relates to others in the future, especially in intimate relationships (Bowlby, 1972; Hazan & Shaver, 1987). Children who view their caregiver as a secure base who will consistently respond to their distress will have better psychological outcomes such as higher self-esteem and lower levels of depression than those who have an unreliable, rejecting caregiver (Ainsworth, Blehar, Waters, & Wall, 1978). Both children and adults with this insecure attachment style tend to have higher levels of depressive symptoms than their securely attached counterparts (Kobak, Sudler, & Gamble, 1991; Muris, Meesters, van Melick, & Zwambag, 2001; Bifulco, Mahon, Kwon, Moran, & Jacobs, 2003).

Along with attachment, researchers also have focused on another important aspect of the parent–child relationship as a risk factor for depression—parenting itself. Studies have found that maternal depression is a strong predictor of childhood and adolescent depression (Hammen, Shih, & Brennan, 2004). Maternal depression decreases the quality of parenting and creates stressful life events for the family, both of which are risk factors for depression. One style of parenting, labeled "affectionless control" is characterized by low parental warmth and high psychological control, and has been shown to be an especially strong predictor of depression in children (Parker, 1983; Alloy, Abramson, Smith, Gibb, & Neeren, 2006).

Interpersonal Therapy A common theme in these psychodynamic theories is how well an individual is able to relate to others. In the 1980s, Klerman and Weissman developed interpersonal therapy based in part on this assumption (Weissman, 2006). This therapy was inspired by the work of Harry Stack Sullivan, who believed that interpersonal behaviors are of central importance to adaptive functioning as well as by the attachment theories of Bowlby. Unlike traditional psychodynamic therapy, which often is quite lengthy, interpersonal therapy is a brief therapy (lasting 12–16 sessions) that focuses on the interpersonal issues that arise in depression. It seeks to address the issues surrounding grief, interpersonal disputes, role transitions, and a lack of social skills. Studies

have shown that interpersonal therapy is an efficacious treatment for depression across the lifespan and performs as well as medication (e.g., Mufson, Weissman, Moreau, & Garfinkel, 1999; Hinrichsen, 2008).

Neuroscience Perspective Although mood disorders have a number of putative causes, including behavioral, cognitive, and environmental factors, an individual's underlying biology also significantly impacts whether he or she develops a mood disorder. In this section, we discuss genetic, neurophysiological, neuroimaging, hormonal, and neurotransmitter research on mood disorders, as well as biologically based treatments for these disorders.

Family and Genetic Studies Some of the clearest evidence for the biological contribution to mood disorders comes from research examining genetics. For example, family studies have shown that an individual's risk of developing a mood disorder increases if there is a family history of mood disorder, and that the amount of genetic material in common relates to the elevation in the risk (i.e. higher risk for those with full siblings/parents with the disorder, lower but still elevated risk for those with grandparents/aunts/uncles with the disorder). When compared with individuals with no family history of mood disorder, individuals with a first-degree relative with MDD are almost three times more likely to develop the disorder, whereas those who have a first-degree relative with bipolar disorder are almost ten times more likely to develop bipolar disorder, and the risk becomes even higher with early onset in the index case (Rasic, Hajek, Alda, & Uher, 2014; Smoller & Finn, 2003: Sullivan, Neale, & Kendler, 2000). Although it is not uncommon to find a family history of unipolar depression among those with bipolar disorder, individuals with unipolar depression typically do not have a family history of bipolar disorder (Wilde et al., 2014). This supports a conceptualization of mood disorders that specifies a unique etiology for bipolar disorder. More recent research exploring the heritability of bipolar I disorder compared with unipolar depression has shown that upwards of 71% of the genetic liability to mania is independent of the liability to depression, although bipolar II disorder may have more shared genetic factors with depression (McGuffin et al., 2003).

Twin studies and adoption studies have been used to parse the contribution of genetics and shared environment to the etiology of the disorders. Twin studies have found that concordance rates of mood disorders are higher in monozygotic twins when compared with dizygotic twins, with bipolar disorder once again evidencing a higher contribution of genetic risk. Twin studies of bipolar disorder have found heritability of bipolar diagnosis or hypomanic/manic episodes to be upwards of 75% (Kendler, 1993; Cardno et al., 1999). Adoption studies have explored the relative incidence of mood disorder onset in the biological compared with adoptive parents of children who were adopted at an early age. An early study found that 31% of adoptees with mood disorders had biological parents with a mood disorder, as compared to only 2% of adoptees who did not develop depression or mania/hypomania (Mendlewicz & Rainer, 1977). Wender and colleagues (1986) conducted a more comprehensive investigation of adoptees, their adoptive parents, and their biological parents, siblings and half siblings, and found that the prevalence of MDD was eight times greater in the biological families of depressed individuals. The same study also found that the prevalence of suicide was over 15 times greater in the biological relatives of adoptees with depression.

Genetic linkage and association studies, although not new to the field of mood disorders, have become much more prevalent, owing to the completion of the Human Genome Project and the use of genome-wide association data. These studies rely on the use of genetic sequencing to detect commonalities in populations with and without mood disorders. One of the earliest association studies examined an 81-member Amish clan with an atypically high incidence of mood pathology, primarily bipolar disorder, and found genetic abnormalities on chromosome 11 (Egeland et al., 1987). A more recent and comprehensive analysis of this population found striking diversity in genetic profiles, with no convergence of evidence implicating a common genetic pathway associated with bipolar disorder (Georgi et al., 2014). Genetic studies of mood disorders in the general population echo this finding. Meta-analyses of genetic studies have observed polymorphisms on regions on chromosomes 2, 13, and 18 associated with the transcription of a wide variety of functional proteins, including neurotransmitters, neuronal ion channels and structural components of cells (Craddock & Forty, 2006: Liu et al., 2010). It is clear that the biological contribution to mood disorders cannot be represented as a single genetic abnormality. Rather, depression and mania are most likely the result of a complex integration of multiple genetic variations with multiple psychological and environmental factors.

Many of the other putative biological variables, such as alterations in neurotransmitter function and hormonal regulation, described below, are also seen in the children of those with mood disorders, even those who have never suffered from the disorder themselves. For example, a twin study examining the stress hormone cortisol, which has been shown to be elevated in individuals with mood disorders, found that "high risk" twins (a psychopathology-free individual whose monozygotic twin had been diagnosed with a mood disorder) had higher levels of evening cortisol relative to low risk (psychopathology-free monozygotic) twins (Vinberg, Bennike, Kyvit, Andersen, & Kessing, 2008). Finally, research also has found that functional polymorphisms of neurotransmitter systems implicated in mood regulation interact with other well-established risk factors, specifically significant life stressors. Alterations in the transcription of the serotonin

transporter (responsible for recycling serotonin after its release into a synapse), the serotonin 2A receptor, tryptophan hydroxylase (an enzyme important in serotonin creation), as well as the enzyme catechol-O-methyltansferase, a chemical important in the regulation of dopamine levels within the synapse (Levinson, 2006). The functional polymorphism involving the serotonin transporter has garnered a great deal of attention after the "short" allele (the genotype that leads to the production of a less efficient serotonin transporter) was found to interact with stressful life events to precipitate depression (Caspi et al., 2003). A meta-analysis further supported the role of the 5-HTTLPR gene as a moderator of the relationship between stress and depression (Karg, Burmeister, Shedden & Sen, 2011).

Neurophysiological Research Of particular interest to neurophysiological researchers is the role that biological rhythms play in the onset and course of mood disorders. Sleep disruption is one of the most common symptoms of depression and bipolar disorder. Depressed individuals also consistently show abnormalities in their progression through the various stages of sleep (Hasler, Buysse, Kupfre, & Germain, 2010; Palagani, Baglioni, Ciapparelli, Gemignani, & Riemann, 2013; Steiger et al., 2009). One of the most documented abnormalities is shortened rapid eye movement (REM) latency. In depressed individuals, the time between sleep onset and REM onset, the stage of sleep in which dreaming occurs, is much shorter than in non-depressed individuals (Kupfer & Foster, 1972; Kupfer, 1976). Shortened REM latency is predictive of differential response to antidepressant treatment over psychotherapy, persistence of shortened REM latency beyond episode recovery, familial history of shortened REM latency and higher likelihood of relapse (Buysse & Kupfer, 1993; Giles, Kupfer, Rush, & Roffwarg, 1998). However, shortened REM latency may not be specific to depression, having also been observed in schizophrenia, panic disorder, obsessive-compulsive disorder, mania, and eating disorders (Steiger & Kimura, 2009). Sleep disruption is also a prominent feature of bipolar disorder (Harvey et al., 2005; Harvey 2008; Mehl et al., 2006).

The disruptions observed in the sleep cycles of individuals with mood disorders are suggestive of a flaw in the body's circadian system, or "biological clock." This circadian center is thought to reside in the suprachiasmatic nucleus in the brain (Cermakian & Boivin, 2003). Many believe that abnormalities in this circadian pacemaker stem from genetic variations. Shi et al. (2008) reported a significant association between the interaction of three circadian genes and bipolar disorder, suggesting that this interaction contributes to genetic vulnerability to the disorder. Moreover, Benedetti and colleagues (2007) found that individual variations in circadian genes can influence sleep and activity symptoms in mood disorders, though one study suggests that a particular gene, 3111T/C CLOCK, may only have influence over these symptoms in bipolar disorder, not unipolar depression (Serretti et al., 2010).

Circadian disruption plays a central role in the social *zeitgeber* theory of mood disorders, a biopsychosocial theory developed by Ehlers, Frank, and Kupfer (1988). Social *zeitgebers* (translated from German as "time givers") are abundant in our everyday lives, routines, relationships, jobs, etc. These social "timing cues" are purported to possess the ability to entrain internal circadian rhythms, suggesting that disruption in social rhythms will lead to disruption in biological rhythms, resulting in affective symptoms (Ehlers et al., 1988). Life events play a crucial part in the social *zeitgeber* theory as they are thought to be the initiators of this chain of events leading to episode onset. The social *zeitgeber* theory has been most frequently applied to bipolar disorder. Shen and colleagues (2008) found significant irregularity of social rhythms among individuals with bipolar spectrum disorders, and evidence suggests individuals with bipolar disorder are more susceptible to the social rhythm-disrupting effects of life events than individuals without bipolar disorder (Boland, Bender, Alloy, Connor, & Labelle, 2012). Social rhythm irregularity has also predicted shorter time to onset of major depressive and manic/hypomanic episodes (Shen et al., 2008). Substantial research also points to disruption in circadian rhythms in depression and bipolar disorder (Leibenluft, Albert, Rosenthal, & Wehr, 1996; Kennedy, Kutcher, Ralevski, & Brown, 1996; Shi et al., 2008). Jones, Hare, and Evershed (2005) found that circadian rhythm and sleep loss patterns were less stable among participants with bipolar disorder than among those without bipolar disorder, even when the participants with bipolar disorder were not in a mood episode. Also consistent with the model, researchers have shown that stressful life events that disrupt social rhythms predict both manic and depressive episodes in bipolar disorder (Malkoff-Schwartz et al., 2000; Sylvia et al., 2009).

Treatment approaches aimed at stabilizing social and circadian rhythms have gained empirical support. Interpersonal and social rhythm therapy (IPSRT), a psychotherapy that combines elements of interpersonal therapy (described above) with a sleep and social rhythm stabilizing regimen, has been shown to increase time to relapse in individuals with bipolar I disorder (Frank et al., 2005). Additionally, participants randomized to IPSRT had higher regularity of social rhythms than those in intensive clinical management, and this regularity was significantly associated with reduced likelihood of relapse during maintenance treatment (Frank et al., 2005). IPSRT is also showing promise as a treatment for individuals with bipolar II disorder (Swartz, Frank, Frankel, Novick, & Houck, 2009), as well as adolescent samples (Hlastala, Kotler, McClellan, & McCauley, 2010) and youths at high risk for developing bipolar disorder (Goldstein et al., 2013).

Another variant of depression with strong links to biological rhythm disturbance is seasonal affective disorder, or SAD. Individuals with seasonal affective disorder experience depressive symptoms solely in the winter months. DSM-5 does not list SAD as its own separate disorder,

but rather as a "course specifier" of depression. To meet criteria for this specification, the individual has to experience this pattern of seasonality for at least 2 years, experience full remission of mood symptoms at a regular time of year, and report a history of seasonal mood episodes that outnumber non-seasonal episodes.

The predominant theory of the pathogenesis of SAD is that the disorder is precipitated by a lag in circadian rhythms, such that the individual experiences the kind of physical retardation during the day that should be taking place at night (Teicher et al., 1997; Nurnberger et al., 2000). Wehr and colleagues (2001) suggested that the circadian pacemaker, which regulates seasonal changes in behavior via the transmission of a "day length" signal to other sites in the body, may function differently in those with SAD compared with healthy individuals. Moreover, genetic studies have revealed circadian clock-related polymorphisms in SAD, with these genetic influences impacting both susceptibility to SAD, as well as diurnal preference (Johansson et al., 2003).

Roughly 75% of individuals with SAD report clinical improvements when treated with morning exposure to bright, artificial light (Oren & Rosenthal, 1992), with one study demonstrating significant effects after only 1 hour of exposure (Reeves et al., 2012) Additionally, light therapy, when applied at the first sign of seasonal symptoms, may prevent progression to a full-blown episode (Meesters et al., 1993). This preference for morning exposure has support in the literature (Lewey et al., 1998; Terman, J. S., Terman, M., Lo, & Cooper, 2001) and one study has shown a positive correlation between circadian phase advance and improvement in depressive symptoms (Terman et al., 2001). Although light therapy remains the gold standard for treatment of SAD, recent pharmacological advances have resulted in a novel antidepressant called agomelatine, which serves as both a melatonergic receptor agonist and a serotonin 2-C receptor antagonist, and has been shown to restore disrupted circadian rhythms (Koesters, Guaiana, Cipriani, Becker, & Barbui, 2013; Pirek et al., 2007; Quera Salva et al., 2007; Zupanic, & Guilleminault, 2006).

Neuroimaging Research There has been a surge in studies investigating the neural substrates of mood disorders over the past 10 years. Taken together, results from these studies implicate key brain regions thought to underlie mood disorders and their respective core domains of psychopathology. These regions include areas associated with emotion processes and mood regulation such as amygdala, cingulate gyrus, ventral striatum, and ventromedial prefrontal cortex as well as areas that play a role in memory and the encoding of affect-related information, including the hippocampus (Scoville & Milner, 1957). For example, magnetic resonance imaging (MRI) studies report amygdala abnormalities in MDD and bipolar disorder (Grotegerd et al., 2013; see Savitz & Drevets, 2009 for a review). Although reduced hippocampal volume has been consistently reported in elderly, middle-aged, and chronically ill individuals with MDD, few studies show volume reduction of this brain region in bipolar disorder. With regard to frontal and cingulate regions, volumetric decreases and functional abnormalities in orbital frontal cortex, dorsolateral prefrontal cortex, and anterior cingulate cortex have been detected in individuals with MDD and bipolar disorder. Additionally, because cortical and subcortical gray matter regions have been implicated in both MDD and bipolar disorder, recent work has also investigated white matter regions linking these areas. Frontal and subcortical white matter hyperintensities have been detected in major depression (Disabato & Sheline, 2012; Nobuhara et al., 2006) and bipolar disorder (de Asis et al., 2006; Tighe et al., 2012). Diffusion tensor imaging, an MRI technique, provides evidence of altered white matter integrity in frontal, parietal, occipitotemporal regions in MDD (Bessette, Nave, Caprihan, & Stevens, 2014; Ma et al., 2007; Yang, Huang, Hong, & Yu, 2007) as well as frontal, frontal-subcortical, corpus callosum regions in bipolar disorder (Barysheva, Jahanshad, Foland-Ross, Altshuler & Thompson, 2013; Lin, Weng, Xie, Wu, & Lei, 2011; Mahon et al., 2009; Sussman et al., 2009).

Hormone Imbalance Hormonal abnormalities also have been associated with mood disorders, specifically with bipolar and unipolar depression. Hypersecretion of cortisol is one of the most consistent and robust findings in the depression literature. Individuals with severe depression evidence hypersecretion of corticotropin-releasing hormone (CRH), exaggerated responding to adrenocoticotropic hormone, enlarged pituitary and adrenal glands, and elevated cortisol concentrations in plasma, urine, saliva, and cerebrospinal fluid (Young, 2004). One of the most frequently used assessments for HPA axis dysregulation involves the administration of dexamethasone, a glucocorticoid agonist which should inhibit cortisol production, and CRH, which should promote cortisol production. Individuals with severe or chronic depression experience what is called dexamethasone "non-suppression," meaning that after being given even very high doses of dexamethasone, their bodies fail to inhibit cortisol secretion, and their levels of circulating and salivary cortisol remain very high. Additionally, individuals with severe depression show reduced responding to CRH, suggesting that their bodies' ability to react appropriately (to promote or inhibit) to hormonal cues is severely impaired. Hypercorteoslemia, once thought to be a trait-like characteristic of depression, has subsequently been shown to abate as mood symptoms remit (Deshauer, Duffy, Meany, Sharma, & Grof, 2006), and persists only in those who are at high risk of depressive episode relapse in the immediate future (Daban, Vieta, Mackin, & Young, 2005). Cortisol hypersecretion among healthy individuals has also been shown to be a risk factor for the first onset of MDD in prospective studies (Goodyer, Herbert, Tamplin, & Altham, 2000; Harris et al., 2000).

Hypercortesolemia has been associated with impairments in learning and memory, marked hippocampal atrophy, and reductions in frontal lobe volume and impairments in functions associated with these areas (Young, 2004; Daban et al., 2005). Many of the brain areas that are negatively impacted by elevated cortisol are those which contain high concentrations of glucocorticoid receptors, lending credence to the theory that elevated cortisol is the result of impaired production/inhibition and may be the cause of many of the cognitive deficits seen in depression. There is mixed evidence as to whether hormonal abnormalities are also present during mania and hypomania, although some work has shown that irritable mania and mixed mood episodes do demonstrate cortisol abnormalities similar to those seen during a major depressive episode (Daban et al., 2005).

Other hormones, such as thyroid hormone and gonadal/ovarian hormones, have also demonstrated dysregulation in mood disorders. Research has shown that individuals with unipolar and bipolar depression have both lower basal thyrotropin and thyroid-stimulating hormone levels relative to euthymic individuals (Sassi et al., 2001; Sullivan et al., 2001). Individuals with particularly low thyroid levels have also been shown to be at greater risk of relapse (Joffe & Marriott, 2000). Additionally, a number of other studies have demonstrated a greatly increased risk for depression, mania, suicide, and even affective psychosis and psychiatric hospitalization during periods of drastic shifts in ovarian hormones (estrogen, progesterone), such as menarche, childbirth, and menopause in those individuals with a history of mood disorders (Rasgon, Bauer, Glenn, Elman, & Whybrow, 2003; Teatero, Mazmanian, & Sharma, 2014). The established influence of estrogen on important neurotransmitters such as serotonin and dopamine (Joffe, & Cohen, 1998; Kenna, Jiang, & Rasgon, 2009) could help explain its influence in mood disorders.

Neurotransmitter Dysfunction A critical element in the neurobiology of mood disorders is the manner in which the creation, release, and metabolism of neurotransmitters, particularly serotonin (5-hydroxytryptamine, or 5-HT), norepinephrine (NE) and dopamine (DA), is dysregulated. Direct studies of neurotransmitter action within a living individual are prohibitively invasive and disruptive to the individual's biochemistry; thus, evidence for dysfunction in these systems is gathered from secondary sources, such as measures of neurotransmitter metabolites in blood, alterations of the genetics regulating neurotransmitter and receptor production and functioning, and studies of the efficacy of drugs that act on certain neurotransmitter systems.

The efficacy of selective serotonin reuptake inhibitors (SSRIs), such as fluoxetine (Prozac®), sertraline (Zoloft®), and paroxetine (Paxil®) among others, have made these drugs the most common pharmacological treatment for depression, and has resulted in a great deal of research exploring the role of serotonin in mood. Research has shown that a reduction in 5-HT activity is associated with mood disorders and suicide. For example, a depletion study, in which the chemical precursor for serotonin, L-tryptophan, was experimentally reduced, resulted in depressed mood (Ruhe, Mason & Schene, 2007). Other research has shown that individuals with mood disorders have reductions in cerebrospinal fluid levels of 5-HT metabolites, suggesting they have lower levels of 5-HT in their brains (Firk & Marcus, 2007). As mentioned above, genetic research has also demonstrated that reuptake of 5-HT after release into the synapse may also be altered in those with depression, and that this alteration interacts with life stress to precipitate the onset of the disorder (Caspi et al., 2003; Kuzelova, Ptacek, & Macek, 2010). Investigations using PET found that depressed individuals show lower 5-HT receptor binding when compared with controls (Yaltham et al., 2000). Additionally, suicide completers have demonstrated alterations in 5-HT activity (Bach et al., 2014; Mann et al., 1992), including subnormal 5-HT levels and impaired 5-HT receptors in the brain stem and frontal cortex (Arango & Underwood, 1997; Meyer et al., 1999).

Catecholamines, including DA and NE, derived from the amino acid tyrosine, are thought to be critical to the phenomenology of mood disorders, particularly bipolar disorder. The DA neurons in the ventral tegmental area of the brainstem project to reward and cognition centers, such as the nucleus accumbens and the prefrontal cortex. Reductions in DA in these brain areas have been associated with blunted reward response, anhedonia, decreased motivation, and difficulties in concentration, all of which are symptoms of both unipolar and bipolar depression (Naranjo, Tremblay, & Busto, 2001). In bipolar disorder, there is evidence that DA signaling is elevated during periods of mania and reduced during periods of depression (Berk et al., 2007), and such findings are consistent with the BAS hypersensitivity model of bipolar disorder discussed above. Other evidence for the role of DA in bipolar disorder includes the phenomenological similarities between mania/hypomania and amphetamine use (amphetamines increase dopamine in the synapse), high rates of comorbidities with bipolar disorder and other disorders impacted by DA (such as ADHD and substance abuse), structural abnormalities in dopaminergic brain structures in those with bipolar disorder, reduced CSF levels of DA metabolites (suggesting reduced DA volume), increased activity of enzymes responsible for DA breakdown (such as catechol-O-methyltransferase) and decreased DA receptor binding in individuals with bipolar disorder (Cousins, Butts, & Young, 2009). All of these findings suggest that individuals with bipolar disorder may possess physical alterations to dopaminergic brain structures, reduced DA release, increased rates of DA breakdown when it is released, and impaired signaling when DA reaches the post-synaptic receptor.

Early studies posited a central role of NE, due in large part to the findings that experimentally increased levels of NE produce mania, whereas decreased levels produce

depression (Schildkraut, 1965; Delgado & Moreno, 2000). Additionally, many of today's novel antidepressants, such as buproprion (Wellbutrin®) and venlafaxine (Effexor®), target NE signaling and disrupt reuptake of released neurotransmitter into the presynaptic neuron (Nutt et al., 2007). There is also evidence that other effective treatments, such as electroconvulsive therapy (ECT) may act by increasing the bioavailability of DA and NE (Nutt, 2006). A meta-analysis of monoamine depletion studies found that depletion of 5-HT, DA and NE results in slightly lowered mood in those individuals with a family or personal history of depression, but not in normal controls (Ruhe et al., 2007). The authors of this analysis suggest that reductions in the bioavailability of these monoamines probably represents a vulnerability to depression which then interacts with other environmental, social, and biological risk factors to actually lead to depression.

In addition to traditional neurotransmitter models of major depressive and bipolar disorder, there has also been investigation into other neurochemicals, such as glutamate, gamma aminobutyric acid, a variety of neuropeptides, and cell-signaling molecules, which may play a role in dysregulated affect (Kugaya & Sanacora, 2005). Pharmaceutical interventions targeting these novel neurobiological candidates are currently being explored (Matthew, Manji, & Charney, 2008). Finally, changes in plasticity (a neuron's ability to grow or shrink) and neuroarchitecture can result from treatment with antidepressants and mood stabilizers, and are also theorized to play an important role in mood disorders (Sanacora, 2008; Frey et al., 2007).

Psychopharmacology Whatever the neuroscience perspective ultimately explains about the causes of mood disorders, it has contributed importantly to their treatment in the form of antidepressant and antimanic medications. The primary classes of antidepressant medication include the monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants, tetracyclic antidepressants, SSRIs, and serotonin-norepinephrine reuptake inhibitors (SNRIs). Newer types of antidepressants include noradrenergic and specific serotonergic antidepressants, norepinephrine reuptake inhibitors, norepinephrine–dopamine reuptake inhibitors, selective serotonin reuptake enhancers, and norepinephrine–dopamine disinhibitors. Each of these classes seems to work by acting on certain neurotransmitters. For example, MAOIs interfere with an enzyme that breaks down 5-HT, DA and NE; tricyclics block the reuptake of 5-HT and NE; tetracyclics increase levels of NE and 5-HT; SSRIs block the reuptake of 5-HT; and the SNRIs inhibit the reuptake of 5-HT and NE.

The choice of antidepressant depends on which drug provides the greatest symptom relief with the fewest adverse effects (Zimmerman et al., 2004). Additionally, treatment history, co-occurring psychiatric disorders, and particular clinical symptoms also should be taken into account. MAOIs, one of the older classes of antidepressant drugs, demonstrate higher levels of adverse effects, and are thus typically only prescribed for select manifestations of depression (i.e., "atypical depression") or when other medications prove ineffective (McGrath et al., 2000). Tricyclics, the oldest class of antidepressant medication, can also have negative side effects, including drowsiness, sexual dysfunction, blurred vision and increased heart rate. Moreover, given their toxicity (ten times normal dosages), it is easy to overdose on tricyclics and thus, they must be prescribed with caution to suicidal patients. Nonetheless, they have been shown to be significantly more effective than placebo (Arroll et al., 2005).

Clinicians perceive the newer medications (e.g. SSRIs and SNRIs) to be more effective than older classes of antidepressants (Petersen et al., 2002); thus, they have gradually displaced most other antidepressants. Importantly, however, efficacy of SSRIs for the treatment of depression remains controversial, as some studies report superior efficacy over placebo (Hollon, DeRubeis, Shelton, & Weiss, 2002; Thase, 2002), whereas others report that they may not be much more effective than placebos (Kirsch, moore, Scoboria, & Nicholls, 2002). Two meta-analyses of clinical trials reported a small effect in mild and moderate depression, but a more robust effect in severe depression (Fournier et al., 2010; Kirsch et al., 2008). Further, SSRIs have the added advantage of fewer adverse effects than tricyclics, and they act more quickly. Emerging research into novel antidepressant treatments have demonstrated that administration of ketamine, an anesthetic neurochemical that mimics glutamate and acts on N-methyl-D-aspartate receptors, is associated with a rapid and sustained antidepressant effect on treatment-resistant and refractory depression (Machado-Viera, Salvadore, Diazgranados & Zarate, 2009). Further studies are needed to explore whether ketamine, or other glutamatergic agonists, could represent viable medical interventions for depression.

There is also a long-standing debate about whether pharmacotherapy or psychotherapy is a more effective approach to treating depression. Recent work comparing the two types of treatment found that antidepressants are as effective as psychotherapy for MDD, whereas medication yields better results for dysthymic disorder (Cuijpers, van Straten, van Oppen, & Andersson, 2008; Imel, Malterer, McKay, & Wampold, 2008). Further, SSRIs may be more efficacious than psychotherapy treatment initially, but more patients have been shown to discontinue antidepressants, as compared with those in psychotherapy, perhaps due to adverse medication effects. Moreover, as discussed earlier, psychotherapies such as behavioral activation and CBT have been found to reduce relapses and recurrences of depression.

For bipolar disorder, mood stabilizer medications, particularly lithium, are typically the first choice of pharmacological treatment. Antidepressants have also been used to treat bipolar disorder, but some work reports potential triggering of rapid cycling and, in some cases, initiation of manic, hypomanic, or mixed episodes (Harel & Levkovitz,

2007). Individuals with bipolar disorder who take antidepressants are typically prescribed a mood stabilizer as well (Sachs et al., 2007).

Aside from lithium, most of these medications are anticonvulsants, such as sodium valproate (Macritchie et al., 2002). For patients with bipolar disorder experiencing more severe manic or mixed episodes, either lithium plus an antipsychotic is typically prescribed, or valproate plus an antipsychotic. For patients with less severe symptoms, lithium, valproate or an antipsychotic are prescribed. Lithium is among the most commonly prescribed mood-stabilizing medications for bipolar disorder. It works to control manic symptoms and prevent relapses of manic and depressive episodes. Nonetheless, it is important to note the difficulty in determining the maintenance dose, primarily because, often, the effective dose is close to the toxic dose, which may lead to convulsions, delirium and, in rare instances, death. Typically, an individual who has overdosed will experience warning signs, such as nausea, which tell them they should discontinue use. One additional complication with lithium is that patients who stop taking it after approximately 2 years may experience a new depressive or manic episode and increased risk of suicide (Baldessarini, Tondo, & Viguera, 1999). As a result of these potential dangers associated with lithium, alternative treatment options have been introduced, including use of anticonvulsants, such as valproate, lamotrigine, gabapentin, topiramate, carbamazepine, and oxcarbazepine. Various psychotherapies are often used as an adjunctive treatment along with pharmacotherapy (Nusslock et al., 2009).

Electroconvulsive Therapy Another long-standing biological treatment for mood disorders is ECT. Electric shock, when applied to the brain under controlled circumstances, has been shown to ameliorate symptoms of severe, refractory depression. ECT is conducted by administering a shock of approximately 70–130 volts directly to the patient's brain, resulting in a convulsion similar to a seizure. The treatment is typically administered approximately nine or ten times, spaced over the period of several weeks. The patient is generally hospitalized prior to treatment and is anesthetized throughout the procedure.

ECT was first developed in the 1930s, and although it is clear that ECT does work (Bailine et al., 2000; Cohen et al., 2000), it is still relatively unclear how it works. Yatham and colleagues (2010) found that ECT functions to downregulate 5-HT receptors, much in the same way as antidepressants. However, ECT is capable of further downregulating the 5-HT receptors in individuals with treatment-resistant depression, thus perhaps shedding some light on why it is often efficacious in this difficult to treat subset of patients.

ECT is not without complications, however. The most common adverse effect is memory dysfunction, both anterograde (learning new material after treatment) and retrograde (recalling material from before treatment) amnesia. Anterograde memory deficits are temporary (Hay & Hay, 1990; Criado, Fernandez, & Ortiz, 2007), though retrograde memory deficits take longer to resolve, with most experiencing a marked loss 1 week following treatment, and a nearly complete recovery within 7 months post treatment. Longer-term studies of up to 12 years of follow-up suggest that few cognitive impairments remain (Elias, Chathanchirayil, Bhat, & Prudic, 2014). In many cases, however, some subtle memory losses persist beyond the typical 7-month post-treatment period, particularly for events that occurred in the 1-year period prior to treatment, and particularly for interpersonal events (Lisanby, Maddox, Prudic, Devanand, & Sakeim, 2000). ECT confined to one hemisphere of the brain (ideally the right hemisphere), or ECT focused on the frontal rather than the temporal lobes, is associated with decreased risk for cognitive impairment following treatment (Sackeim et al., 2000; Bailine et al., 2000).

A newer biological treatment for depression uses powerful magnetic fields to restore dysfunctional brain activity in depressed individuals. Transcranial magnetic stimulation (TMS) involves placing an electromagnetic coil on the scalp through which high-intensity current produces a brief magnetic field that induces electrical current in neurons, thus resulting in neural depolarization (George, Lisanby, & Sackeim, 1999). This is the same depolarization effect seen in ECT. An advantage of TMS lies in its ability to apply more finely tuned stimulation than ECT. Although occasional mild headache and discomfort at the stimulation site has been reported, patients receiving TMS generally report no other adverse side effects (George et al., 1999). The method has been approved for treatment-resistant depression in several countries, but it has yet to gain approval in the United States, largely due to the Food and Drug Administration's concerns about its efficacy (Marangell, Martinez, Jurdi, & Zboyan, 2007). Tests of TMS using a sham control have yielded conflicting results and have been difficult to compare, owing to non-standardized stimulation methods, thus making the true efficacy of the treatment difficult to determine (Burt, Lisanby, & Sackeim, 2002; Marangell et al., 2007). Moreover, a recent meta-analysis has shown ECT to be more effective than TMS in treating depression (Berlim, Van den Eynde, & Daskalakis, 2013).

Conclusion

Although they have been recognized and studied for centuries, mood disorders still remain something of a mystery. Although major advances in the understanding and treatment of mood disorders have been made over the last several decades, there are still gaps in our knowledge of these conditions. As discussed in this chapter, many different potential causes and treatments for the mood disorders have been suggested from a variety of theoretical perspectives. More recent theoretical models, such as the BAS hypersensitivity model or social *zeitgeber* theory,

for example, have attempted to integrate research across multiple perspectives. It is likely that a full understanding of the causes of mood disorders will require even further integration of cognitive, behavioral, psychosocial, and neurobiological mechanisms in the future.

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12

Personality Disorders

Cristina Crego and Thomas A. Widiger

In 1980, the American Psychiatric Association (APA) published the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III; American Psychiatric Association, 1980), which introduced the innovative multiaxial classification system. Axis II was devoted primarily to personality dysfunction, owing to the prevalence of maladaptive personality traits in general clinical practice, the substantial impact that these traits can have on the course and treatment of other mental disorders, and the tendency of clinicians to miss comorbid maladaptive personality functioning as their attention is drawn to concerns regarding anxiety, depression, substance usage, or other form of psychopathology (Frances, 1980). This multiaxial system has been instrumental in the increased recognition of personality disorders within clinical practice (Loranger, 1990) and research (Blashfield & Intoccia, 2000). The multiaxial system, however, has been deleted in DSM-5 (American Psychiatric Association, 2013). No explanation for this decision was provided by the APA. However, a commonly expressed concern with respect to the multiaxial system was that few clinicians were actually using it, at least the components for indicating presence of a medical disorder, level of functioning, and social stressors (Frances, First, & Pincus, 1995).

DSM-5 includes 10 personality disorders, organized into three clusters: (a) paranoid, schizoid, and schizotypal (the odd-eccentric cluster); (b) antisocial, borderline, histrionic, and narcissistic (dramatic-emotional-erratic cluster); and (c) avoidant, dependent, and obsessive-compulsive (anxious-fearful cluster). These diagnoses and their criterion sets are identical to those included in the prior editions, DSM-IV-TR (American Psychiatric Association, 2000) and DSM-IV (American Psychiatric Association, 1994). Proposed for DSM-5 were major revisions to the personality disorders such as use of prototype narratives, self-interpersonal impairment criteria for each personality disorder, deletion of half of the disorders, and a shift from categorical to dimensional classification. However, because of the magnitude of the proposed changes, vocal opposition to them, and the inadequate documentation of their empirical support, none of them was approved (Skodol, Morey, Bender, & Morey, 2013; Widiger, 2013). In this chapter, we begin with a discussion of personality disorders in general, followed by a discussion of the proposals that were made for DSM-5, and the five-factor model (FFM) of personality disorder (Widiger & Costa, 2013) that is closely aligned with one of the proposals. We then discuss five specific personality disorders (antisocial, narcissistic, borderline, schizotypal, and dependent) including how they are understood from the perspective of the FFM.

Personality Disorder in General

Virtually all persons, including everyone with psychological problems, will have a characteristic manner of thinking, feeling, behaving, and relating to others that would have been present prior to the onset of their anxiety, mood, substance use or other mental disorder. For many of these persons, these personality traits will be so maladaptive that they would constitute a personality disorder, defined in DSM-5 as "an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment" (American Psychiatric Association, 2013, p. 645).

It is estimated that 10–15% of the general population would meet criteria for one of the 10 DSM-5 personality

disorders (Torgersen, 2012). The prevalence of personality disorders within clinical settings is estimated to be well above 50% (Zimmerman & Mattia, 2001). As many as 60% of inpatients within some clinical settings are diagnosed with borderline personality disorder (American Psychiatric Association, 2013; Hooley, Cole, & Gironde, 2012). The prevalence of personality disorders, however, is generally underestimated in clinical practice, owing to a lack of time to provide sufficiently systematic or comprehensive evaluations of personality functioning (Miller, Few, & Widiger, 2012) and perhaps also to a reluctance to diagnose personality disorders because insurance companies may consider them to be untreatable (Zimmerman & Mattia, 1999).

Personality disorders are among the most difficult of disorders to treat because they involve well-established behaviors that can be integral to a client's self-image (Millon, 2011). Nevertheless, much has been written on the treatment of personality disorder (e.g., Beck, Freeman, Davis, & Associates, 1990; Clarkin, Fonagy, & Gabbard, 2010; Critchfield & Benjamin, 2006; Gunderson & Gabbard, 2000; Livesley, 2003; Magnavita, 2010; Young, Klosko, & Weishaar, 2003) and there is empirical support for clinically and socially meaningful changes in response to psychosocial and pharmacologic treatments (Magnavita, 2010). The development of an ideal or fully healthy personality structure is unlikely to occur through the course of treatment, but given the considerable social, public health, and personal costs associated with some of the personality disorders, such as antisocial and borderline, even moderate adjustments to personality functioning can represent substantial social and clinical benefits.

DSM-5 Personality Disorder Proposals

The major innovation of DSM-III was the inclusion of specific and explicit criterion sets to facilitate the obtainment of reliable diagnoses (Spitzer, Williams, & Skodol, 1980). However, proposed for DSM-5 was a return to the method used in DSM-II (American Psychiatric Association, 1968), in which clinicians would match their global perception of a patient to a paragraph description, considering the narrative "as a whole rather than counting individual symptoms" (Westen, Shedler, & Bradley, 2006, p. 847). Gestalt matching to paragraph narratives is preferred by clinicians, probably because it is much easier and quicker than having to systematically assess each individual diagnostic criterion (Spitzer, First, Shedler, Westen & Skodol, 2008). However, this proposal met with considerable objection (Pilkonis, Hallquist, Morse, & Stepp, 2011; Widiger, 2011; Zimmerman, 2011), due largely to the weak empirical support for its reliability and validity. The proposal was subsequently abandoned by the Personality and Personality Disorders Work Group (PPDWG).

An additional DSM-5 proposal was to revise the definition and diagnostic criteria for each respective personality disorder to incorporate psychodynamic clinical theory concerning impairments in the perception of the self (identity and self-direction) and interpersonal relatedness (empathy and intimacy) (Skodol, 2012). These features were derived from psychoanalytic theory concerning pathology of the self (Livesley & Jang, 2000). Empirical support for this proposal, however, was limited and largely indirect (see Bender, Morey, & Skodol, 2011). In addition, the APA is shifting toward a neurobiological model of psychopathology (Kupfer & Regier, 2011) and it is likely that this proposal was not well received.

A further proposal for DSM-5 was to delete half of the diagnostic categories, largely to address the problematic diagnostic co-occurrence (Skodol, 2012). Originally slated for deletion were the narcissistic, dependent, paranoid, schizoid, and histrionic personality disorders. Narcissistic, however, was saved from the chopping block, due in large part to critical reviews that documented its empirical support (Pincus, 2011; Miller, Widiger, & Campbell, 2010; Ronningstam, 2011). The empirical support for the deletion of the others, however, was also questioned (Mullins-Sweatt, Bernstein, & Widiger, 2012). All of the DSM-IV personality disorders were eventually retained in DSM-5. Questions remain, however, concerning the extent of empirical support for histrionic (Blashfield, Reynolds, & Stennett, 2012), paranoid and schizoid personality disorder (Hopwood & Thomas, 2012).

The Chair and Vice Chair of the DSM-5 Task Force indicated that the primary contribution of DSM-5 would be a shift toward a dimensional model of classification (e.g., Regier, 2008; Regier, Narrow, Kuhl, & Kupfer, 2010). The Nomenclature Work Group of a DSM-5 research planning conference, charged with addressing fundamental assumptions of the diagnostic system, concluded that it will be "important that consideration be given to advantages and disadvantages of basing part or all of DSM-V on dimensions rather than categories" (Rounsaville et al., 2002, p. 12). They suggested that a dimensional model be developed in particular for the personality disorders. "If a dimensional system of personality performs well and is acceptable to clinicians, it might then be appropriate to explore dimensional approaches in other domains" (Rounsaville et al. 2002, p. 13). A subsequent DSM-5 research planning conference was devoted to documenting research supporting this shift for the personality disorders section (Widiger & Simonsen, 2005). This was followed by a third DSM-5 research planning conference that was devoted to proposals to shift the entire manual to a dimensional model, including the personality disorders (Krueger, Skodol, Livesley, Shrout, & Huang, 2008).

The DSM-5 dimensional trait model for personality disorders consisted initially of 37 traits. The list of 37 traits was first reduced to 25, on the basis of a factor analysis, while maintaining the original trait assignments (American

Psychiatric Association, 2011). The trait assignments were then subsequently revised again. The list of 25 traits was retained, but there were shifts in how they were assigned (American Psychiatric Association, 2012; Skodol, 2012). The final proposals by the PPDWG included a five-domain, 25-maladaptive trait model, that could be used by itself to describe a patient and was also part of the diagnostic criterion sets for the traditional personality disorder categories (Krueger et al., 2011). The five broad domains were negative affectivity, detachment, psychoticism, antagonism, and disinhibition. The proposal was approved by the DSM-5 Task Force but rejected by a DSM-5 scientific oversight committee and the APA Board of Trustees. The rationale for this rejection is unclear. No report by the oversight committee has been provided.

The dimensional trait proposal was embedded within other major proposals, including the addition of psychodynamically oriented deficits in self and interpersonal relatedness to the diagnostic criterion sets (Skodol, 2012). This aspect of the proposal did not appear to have much empirical support. There is a considerable body of research to support the dimensional trait proposal. However, very little of this research was actually presented to the oversight committee. The report to the oversight committee was confined largely to studies authored by work group members (Blashfield & Reynolds, 2012), failing to cite a considerable body of additional research (Widiger, Samuel, Mullins-Sweatt, Gore, & Crego, 2012). For example, it was important for the dimensional trait proposal to be "acceptable to clinicians" (Rounsaville et al., 2002, p. 13). There have been a number of studies documenting empirically that clinicians prefer the dimensional trait model over the existing diagnostic categories (e.g., Glover, Crego et al., 2012; Lowe & Widiger, 2009; Samuel & Widiger, 2006), but this research was not included within the PPDWG literature review (American Psychiatric Association, 2012). Included instead were the studies that suggested a lack of support by clinicians for such a shift (i.e., Rottman, Ahn, Sanislow, & Kim, 2009; Spitzer et al., 2008). In any case, it was clear that there was considerable opposition to the proposal by well-known and well-regarded personality disorder clinicians (e.g., Gunderson, 2010; Shedler et al., 2010).

The dimensional trait proposal, however, is included within Section 3 of DSM-5, for emerging models and measures. The introduction to DSM-5 explicitly acknowledges the failure of the categorical model: "the once plausible goal of identifying homogeneous populations for treatment and research resulted in narrow diagnostic categories that did not capture clinical reality, symptom heterogeneity within disorders, and significant sharing of symptoms across multiple disorders" (p. 12). It is further asserted that dimensional approaches will "supersede current categorical approaches in coming years" (p. 13).

Five-Factor Model of Personality Disorder

This chapter describes a dimensional trait model conceptualization of the DSM-5 personality disorders that is conceptually and empirically aligned with the DSM-5 dimensional trait proposal included within Section 3 of the DSM-5. The model provided in this chapter is the FFM of general personality structure developed within psychology (Widiger & Costa, 2013). Five broad domains of personality functioning have been identified empirically through the study of the languages of a number of different cultures (John, Naumann, & Soto, 2008). Language can be understood as a sedimentary deposit of the observations of persons over the thousands of years of the language's development and transformation. The most important domains of personality functioning would be those with the greatest number of terms to describe and differentiate their various manifestations and nuances, and the structure of personality would be evident in the empirical relationship among the trait terms (Goldberg, 1993). Such lexical analyses of languages have typically identified five fundamental dimensions of personality: neuroticism (or negative affectivity) versus emotional stability, introversion versus extraversion, closedness versus openness to experience, antagonism versus agreeableness, and conscientiousness (constraint) versus disinhibition. The five domains of the FFM align well with the five included in Section 3 of DSM-5. As stated in DSM-5, the "five broad domains [of DSM-5] are maladaptive variants of the five domains of the extensively validated and replicated personality model known as the 'Big Five,' or the Five Factor Model of personality" (American Psychiatric Association, 2013, p. 773). FFM neuroticism aligns with DSM-5 negative affectivity, FFM introversion aligns with DSM-5 detachment, FFM openness (or unconventionality) aligns with DSM-5 psychoticism; FFM antagonism aligns with DSM-5 antagonism, and FFM low conscientiousness aligns with DSM-5 disinhibition.

Each of the five broad domains of the FFM can be differentiated further in terms of underlying facets. For example, the facets of antagonism versus agreeableness include suspiciousness versus trusting gullibility, callous tough-mindedness versus tender-mindedness, confidence and arrogance versus modesty and meekness, exploitation versus altruism and sacrifice, oppositionalism and aggression versus compliance, and deception and manipulation versus straightforwardness and honesty (Costa & McCrae, 1992). Figure 12.1 indicates how each of the 25 maladaptive traits (as well as the 12 additional traits that were deleted from the proposal) included in Section 3 of DSM-5 would be organized with respect to the FFM.

Each of the DSM-5 personality disorders can be readily understood as maladaptive and/or extreme variants of the FFM domains and facets (Lynam & Widiger, 2001; Samuel & Widiger, 2004). For example, DSM-5 obsessive-compulsive personality disorder (OCPD) is primarily a disorder of maladaptively extreme conscientiousness, including the FFM facets of deliberation (OCPD rumination), self-discipline, achievement-striving (OCDP workaholism), dutifulness (OCPD over-conscientiousness, scrupulousness about matters of ethics and morality), order (OCPD preoccupation with details), and competence

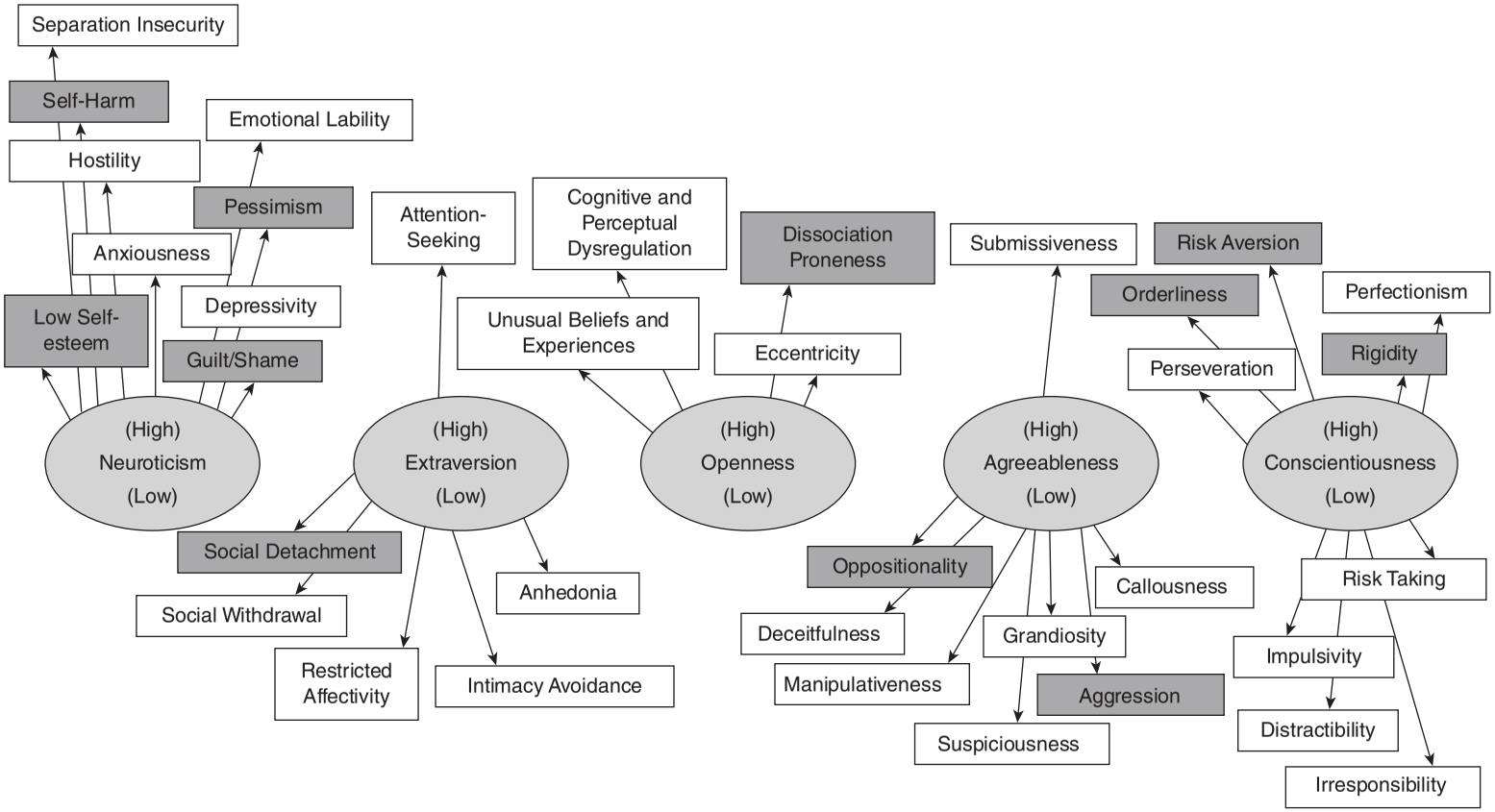

Figure 12.1 DSM-5 traits arranged with respect to the five-factor model of personality. Note: Traits in blue are from the original 37 trait proposal but have since been removed from the DSM-5 Section 3 trait model (a 37th trait, unusual perceptions, does not appear in the figure because it was folded into cognitive-perceptual dysregulation).

(OCPD perfectionism). The FFM description also goes beyond the DSM-IV-TR, now DSM-5, diagnostic criteria by including high anxiousness (excessive worry), low excitement-seeking (risk aversion), and closed mindedness to feelings (constricted), values (dogmatism), and actions (inflexibility; Samuel, Riddell, Lynam, Miller, & Widiger, 2012). Table 12.1 indicates how each of the DSM-5 personality disorders can be understood from the perspective of the FFM. Existing measures of the FFM can in fact be used to provide valid assessments for most of the DSM-5 personality disorders (Miller, 2012). However, measures to assess the DSM-5 personality disorders from the perspective of the FFM, such as the Five-Factor Borderline Inventory (Mullins-Sweatt et al., 2012) have also been developed (Widiger, Lynam, Miller, & Oltmanns, 2012).

There are a number of advantages to a FFM of personality disorder approach (Crego & Widiger, 2015; Widiger & Trull, 2007). The dimensional trait model addresses the many fundamental limitations of the categorical system (e.g., heterogeneity within diagnoses, inadequate coverage, lack of consistent diagnostic thresholds, and excessive diagnostic co-occurrence). It provides a description of abnormal personality functioning within the same model and language used to describe general personality structure, allowing for a more comprehensive system that would enable clinicians to identify personality strengths as well as deficits. It would transfer to the psychiatric nomenclature a wealth of knowledge concerning the origins, development, universality, and stability of personality structure (Widiger & Trull, 2007). Finally, it would represent a significant step toward a rapprochement and integration of psychiatry with psychology. Empirical support for the integration of the DSM-IV-TR (and DSM-5) personality nomenclature with the FFM is summarized by Clark (2007), Widiger, Samuel et al. (2012), and Widiger and Costa (2012).

Five Personality Disorders and Their Five-Factor Formulations

Space limitations prohibit a detailed coverage of all 10 DSM-5 personality disorders; neither would that seem worthwhile because some of them have an uncertain future (Skodol, 2012). However, the five discussed herein are not entirely the same as the five originally proposed for retention in DSM-5. We discuss the five personality disorders for which there has been the most research: antisocial, borderline, narcissistic, schizotypal, and dependent. The narcissistic and dependent were slated for deletion, whereas the avoidant and obsessive-compulsive were to be retained. However, there is arguably more research to support the validity and clinical utility of the narcissistic and dependent than for OCPD or avoidant (Mullins-Sweatt, Bernstein et al., 2012). For each of the five diagnoses chosen for this chapter, we describe what is known about their etiology, pathology, differential diagnosis, comorbidity, course, and treatment. We also indicate how each of them can be understood from the perspective of the FFM and how this conceptualization can help to address one or more issues that have been problematic for that personality disorder.

Antisocial Personality Disorder Antisocial personality disorder (ASPD) has been included within every edition of the DSM. One might even characterize ASPD as the prototypic personality disorder as the term "psychopath" originally referred to all cases of personality disorder (Schneider, 1923). The term "psychopathy" now refers to a particularly severe variant of ASPD (Derefinko & Widiger, 2013). Much of the current research on ASPD is being conducted with regard to this more severe variant, as assessed, for instance, by the *Psychopathy Checklist-Revised* (PCL-R; Hare, 2003; Hare, Neumann, & Widiger, 2012).

Definition DSM-5 defines ASPD as a pervasive pattern of disregard for and violation of the rights of others. Its primary diagnostic criteria include criminal activity, deceitfulness, impulsivity, recklessness, aggressiveness, irresponsibility, and indifference to the mistreatment of others. DSM-5 ASPD overlaps substantially with PCL-R psychopathy. The primary differences are the inclusion of glib charm, arrogance, lack of empathy, and shallow affect within the PCL-R, and the requirement within DSM-5 for the evidence of conduct disorder within childhood (Widiger, 2006). More recent formulations of psychopathy have extended its description to include such traits as fearlessness (Malterer, Lilienfeld, Neumann, & Newman, 2010), boldness (Patrick, Fowles, & Krueger, 2009), dominance, and invulnerability (Lynam et al., 2011).

Etiology and Pathology Twin, family, and adoption studies have provided substantial support for a genetic contribution to the etiology of the criminal, delinquent tendencies of persons meeting criteria for ASPD, accounting for approximately 50% of the variance in antisocial behavior (Waldman & Rhee, 2006). Exactly what is inherited in ASPD, however, is not known. It could be an impulsivity, an antagonistic callousness, or an abnormally low anxiousness, or all of these dispositions combined.

Numerous environmental factors have also been implicated. Shared, or common, environmental influences account for 15–20% of variation in antisocial behavior (Rhee & Waldman, 2002). Not surprisingly, shared environmental factors such as low family income, inner-city residence, poor parental supervision, single-parent households, rearing by antisocial parents, delinquent siblings, parental conflict, harsh discipline, neglect, large family size, and young mother have all been implicated (Farrington, 2006). Nonshared environmental influences (30%) comprise the remaining variance not accounted for by the genetic (50%) or shared environmental (20%)

TABLE 12.1
DSM-5 Personality Disorders from the Perspective of the Five-Factor Model of General Personality Functioning

	Antisocial	Narcissistic	Borderline	Schizotypal	Dependent	Paranoid	Schizoid	Histrionic	Avoidant	Obsessive-compulsive
Neuroticism (vs. emotional stability)										
Anxiousness (vs. unconcerned)	*Fearlessness*			Social anxiousness	Separation insecurity				Evaluation apprehension	Excessive worry
Angry hostility (vs. dispassionate)	**Anger**	**Reactive anger**	**Dysregulated rage**			**Vengeful**				
Depressiveness (vs. optimistic)			**Despondence**		Pessimism					
Self-consciousness (vs. shameless)	*Glib charm*	**Shame** *Indifference*	**Self-disturbance**	**Social discomfort**	**Shamefulness**				**Mortified**	
Impulsivity (vs. restrained)			Behavioral dysregulation							
Vulnerability (vs. fearless)	*Invincibility*	**Need for admiration**	**Affective dysregulation**		**Helplessness**			**Neediness for attention, rapidly shifting emotions**		
Extraversion (vs. introversion)										
Warmth (vs. coldness)					**Intimacy needs**	*Reserved*	*Social anhedonia*	**Intimacy seeking**		*Detached coldness*
Gregariousness (vs. withdrawal)		**Exhibitionism**		*Social withdrawal*		*Aloof*	*Social withdrawal*	**Attention seeking**	*Social withdrawal*	
Dominance (vs. submissiveness)	**Domineering**	**Authoritative**			*Timidity*				*Timidity*	
Activity (vs. passivity)							*Inactive*			
Excitement-seeking (vs. lifeless)	**Thrill-seeking**						*Lifeless*	**Flirtatious**		*Risk aversion*
Positive emotionality (vs. anhedonia)							*Physical anhedonia*	**Melodramatic emotionality**	*Joylessness*	
Openness (vs. closedness)										
Fantasy (vs. concrete)		**Grandiose fantasies**	**Dissociative tendencies**	*Aberrant perceptions*				**Romantic fantasies**		
Aesthetics (vs. disinterest)										
Feelings (vs. alexithymia)							*Alexithymic*	**Touchy feely**		*Constricted*
Actions (vs. predictable)				*Odd-eccentric*						*Inflexibility*
Ideas (vs. closed-minded)				*Aberrant ideas*						
Values (vs. dogmatic)						*Dogmatic*				*Dogmatism*
Agreeableness (vs. antagonism)										
Trust (vs. mistrust)	*Cynicism*			*Suspiciousness*	**Gullibility**	*Suspicious*		*Suggestibility*		
Straightforwardness (vs. deception)	*Manipulation*	*Manipulation*	*Manipulative*			*Circumspect*				
Altruism (vs. exploitative)	*Exploitative*	*Entitlement*			**Selflessness**					
Compliance (vs. aggression)	*Aggressive*		*Oppositional*		**Subservience**	*Combative*				
Modesty (vs. arrogance)	*Arrogance*	*Arrogance*			**Self-effacing**			*Vain*	**Timorous**	
Tender-mindedness (vs. tough-minded)	*Callous*	*Lack of empathy*								
Conscientiousness (vs. disinhibition)										
Competence (vs. laxness)					*Ineptitude*					**Perfectionism**
Order (vs. disorderly)								*Disorderliness*		**Fastidious**
Dutifulness (vs. irresponsibility)	*Lax*									**Punctilious**
Achievement striving (vs. lackadaisical)		**Acclaim-seeking**								**Workaholism**
Self-discipline (vs. negligence)	*Unreliable*				*Negligence*					**Doggedness**
Deliberation (vs. rashness)	*Rash*		*Rashness*					*Impressionistic thinking*		**Ruminative deliberation**

influences. Nonshared environmental factors may include delinquent peers, individual social and academic experiences, or sexual, physical abuse (Moffitt, 2005).

The interactive effects of genetic and environmental influences are difficult to tease apart though, and create confusion about what these estimates mean in terms of causation. For example, the individual who is genetically predisposed to antisocial behavior will elicit experiences that can in turn contribute to the development of antisocial behavior, such as peer problems, academic difficulty, and harsh discipline from parents. In other words, the person genetically disposed to antisocial behavior can help create an environment that would reinforce antisocial behavior. In addition, antisocial individuals receive their genes from antisocial parents who had also likely modeled delinquent and irresponsible behavior. In sum, it can be difficult to disentangle what is really genetic and what is really environmental. Studies that explicitly address these issues have found that environmental factors continue to play an important part in the etiology of antisocial behavior beyond genetic factors alone. For instance, after controlling for the genetic component of physical maltreatment, Jaffee, Caspi, Moffitt, and Taylor (2004) found that the environmental etiological effect of physical maltreatment remained.

Considerable research effort has been focused on the pathology of psychopathy. This extensive research base indicates that many deficits can be involved, leading to a very complex picture. Historically, the psychopathic individual was said to suffer from "superego lacunae" or a "semantic dementia" (Cleckley, 1941) that involved a deficit of conscience or, more generally, a deficient processing of feelings and emotion. Laboratory research is now providing support for this theoretical model in studies assessing the psychopath's autonomic reaction to emotional words and fearful images (Blackburn, 2006).

Many psychophysiological deficits have also been associated with psychopathy, including for example a low level of physiological arousal and/or fear response (Fowles & Dindo, 2006). Support for this hypothesis has included abnormally low physiological responses (reduced skin conductance) to a conditioned stimulus paired with electric shock, indicating that the psychopath may not develop the expected anticipatory arousal from threat of physical punishment. Additional autonomic arousal assessments include low resting heart rate levels and startle response deficits (Derefinko & Widiger, 2015). There is also the suggestion that persons with ASPD may have abnormally low levels of anxiousness. Some distress-proneness (FFM anxiousness or neuroticism) and attentional self-regulation (FFM constraint or conscientiousness) may be necessary in order to develop an adequate sense of guilt or conscience. Normal levels of neuroticism will promote the internalization of a conscience by associating wrongdoing or misbehavior with distress and anxiety, and the temperament of self-regulation will help modulate impulses into socially acceptable channels (Fowles & Kochanska, 2000).

Cognitive functioning deficits have also been implicated. The psychopath's notorious failure to accurately anticipate negative consequences suggests a cognitive deficit (Hiatt & Newman, 2006). Existing research indicates that the psychopath experiences stable deficits in the cognitive domains of attention and response modulation (Gao, Glenn, Schug, Yang, & Raine, 2009). According to this research, psychopaths continue to engage in behaviors that are initially interpreted as positively reinforcing, even when additional information is presented indicating substantial overall costs (Newman & Lorenz, 2003).

Differential Diagnosis ASPD is most closely associated with narcissistic personality traits, the differentiation of which will be discussed later in this chapter. At times, ASPD may be difficult to differentiate from a substance dependence disorder because many persons with ASPD develop a substance-related disorder, and many persons with substance dependence engage in antisocial acts. However, the requirement that conduct disorder be present prior to the age of 15 will usually assure the onset of ASPD prior to the onset of a substance-related disorder. If both were evident prior to the age of 15, then it is likely that both disorders are now present and both diagnoses should be given (Widiger, 2006). Often, ASPD and substance-dependence will interact, exacerbating each other's development.

Epidemiology The National Institute of Mental Health Epidemiologic Catchment Area study estimated that 3% of males and 1% of females meet criteria for ASPD. Subsequent studies have replicated this rate, but it has also been suggested that this finding may have underestimated the prevalence in males, owing to a failure to consider the full range of ASPD features. Other estimates have been as high as 6% in males (Kessler et al., 1994). Within prison and forensic settings, the rate of ASPD has been estimated to be as high as 50% in males (Hare et al., 2012). However, the ASPD criteria may inflate the prevalence within such settings, owing to the emphasis on overt acts of criminality, delinquency, and irresponsibility. More specific criteria for psychopathy provided by the PCL-R (Hare, 2003) obtain a more conservative estimate of 20–30% of male prisoners by placing relatively less emphasis on the history of criminal behavior and more emphasis on personality traits associated with this criminal history (e.g., callousness and arrogance).

ASPD is much more common in men than in women (Verona & Vitale, 2006). A sociobiological explanation for the differential sex prevalence is the presence of a genetic advantage for social irresponsibility, infidelity, superficial charm, and deceit in men but not women. These traits may be related to reproductive success for men (having more offspring) but also contribute to a higher likelihood of developing features of ASPD (Derefinko & Widiger, in press).

Course ASPD is the only personality disorder for which much is known about childhood antecedents. Approximately 40% of the children diagnosed with DSM-5 conduct disorder grow up to meet criteria for ASPD, particularly those with childhood onset of conduct disorder and/or have callous-unemotional traits. The presence of conduct disorder is in fact essentially required for the diagnosis of ASPD (American Psychiatric Association, 2013). There are also compelling data to indicate that ASPD is a relatively chronic disorder which persists in adulthood, although research suggests that as the person reaches middle to older age, the frequency of criminal acts decreases. Nevertheless, the core personality traits may remain largely stable (Hare et al., 2012).

Five Factor Model Reformulation ASPD can be understood primarily as excessive, maladaptively low conscientiousness and high antagonism (see Table 12.1). Specifically, these individuals would be described as aimless, unreliable, lax, negligent, and hedonistic (low in the facets of self-discipline and deliberation), as well as manipulative, exploitative, aggressive, and ruthless (low in straightforwardness, altruism, compliance, and tender-mindedness). Additional facets of the FFM would be seen in prototypic cases of ASPD-psychopathy, including low anxiousness (fearlessness), low self-consciousness (glib charm), low vulnerability (invincibility), high impulsivity, high angry hostility, high dominance, and high thrill-seeking (Derefinko & Lynam, 2013).

An ongoing issue surrounding the diagnosis of ASPD has been the failure to include all of the personality traits of psychopathy identified originally by Cleckley (1941), emphasizing instead those traits that could most easily be identified by objectively observed behaviors (e.g., irresponsible and/or illegal acts). An advantage of the FFM conceptualization is that it includes all of the traits that are common to both ASPD and the PCL-R, including deception, exploitation, aggression, irresponsibility, negligence, rashness, angry hostility, impulsivity, excitement-seeking, and assertiveness (see Table 12.1) as well as the traits that are unique to the PCL-R, including glib charm (low self-consciousness), arrogance, and lack of empathy (tough-minded callousness). In addition, the FFM includes those traits of psychopathy that were emphasized originally by Cleckely (1941) but were not included in either the DSM-5 or the PCL-R (i.e., low anxiousness or fearlessness). Considerable evidence supports the FFM conceptualization of ASPD (Lynam & Widiger, 2007a). Miller (2012) has shown that the extent to which an individual's FFM profile matches the FFM profile for a prototypic case of psychopathy can even be used as a quantitative indication of the likelihood that a person would be diagnosed as psychopathic. A self-report measure of ASPD-psychopathy from the perspective of the FFM (the Elemental Psychopathy Assessment) was developed by Lynam et al. (2011).

An FFM conceptualization of ASPD also provides some clarity in regards to the often discussed but poorly understood concept of the "successful psychopath" (Hall & Benning, 2006). Systematic research has been confined largely to the study of the "unsuccessful" psychopath, which typically means the incarcerated criminal. However, there is considerable social and theoretical interest in understanding the psychopath who is indeed exploitative, callous, and ruthless, but either manages never to get arrested or convicted, or who pursues a white-collar career that only flirts with the edges of the legal system (Hall & Benning, 2006). From the perspective of the FFM, these persons share many of the traits of the prototypic psychopath (i.e., high fearlessness, high in assertiveness and gregariousness, and high in the exploitativeness, deceptiveness, and callousness of antagonism), but are high rather than low in the facets of conscientiousness (Mullins-Sweatt, Glover, Derefinko, Miller, & Widiger, 2010). Such persons would be even more dangerous than most of the incarcerated psychopaths because they share the disposition to engage in behavior harmful to others, but also possess the traits (deliberation, competence, and self-discipline) that contribute to a more "successful" criminal career. A potential case illustration of such a "successful" psychopath is provided by the infamous serial killer, Theodore Bundy. As stated in Samuel and Widiger's paper regarding Bundy's FFM ratings:

> Perhaps the most noteworthy finding from the FFM ratings was his generally high ratings on the domain of conscientiousness. In contrast with the impulsive, under-controlled behavior that one would typically expect from an antisocial criminal, Bundy was described as being competent, orderly, achievement oriented and deliberate. Perhaps it was his characteristic style of careful planning and deliberate execution that enabled Bundy to avoid capture and arrest for so many years.
>
> (Samuel & Widiger, 2007)

Researchers have been attempting to identify the single, core pathology of psychopathy for many years, and a variety of compelling but inconsistent models have been proposed. These alternative conceptualizations can be integrated and their inconsistencies addressed by the FFM (Derefinko & Lynam, 2013). Their apparent inconsistency may reflect that each alternative model of pathology is focusing on a different facet or even a different domain of the FFM. For example, poor fear conditioning and electrodermal hypoarousal (Fowles & Dindo, 2006) would be placing particular emphasis on the low neuroticism (i.e., low anxiousness or low vulnerability). Lack of response modulation, or an inability to refrain from acting on first impulse (Hiatt & Newman, 2006) would involve the disinhibition of low conscientiousness. A deficit in empathy or the processing of affective language (Blackburn, 2006) can be understood in terms of the antagonism facet of callous tough-mindedness. In sum, the personality profile for the prototypic psychopath involves a constellation

of personality traits that together provides a virulent and at times even lethal mix (i.e., high antagonism, low conscientiousness, low vulnerability, low anxiousness, high assertiveness, high gregariousness, and high excitement-seeking). Researchers though are approaching this constellation like the blind men from Indostan, each interpreting the location of the elephant upon which they are laying their hands in a much different manner, depending upon which component of the FFM is the focus of their attention (Lynam & Widiger, 2007a).

Treatment ASPD is considered to be the most difficult personality disorder to treat (Gunderson & Gabbard, 2000; Hare et al., 2012). Individuals with ASPD can be seductively charming and may declare a commitment to change, but they often lack sufficient motivation. Their declarations of desire to change might even be dishonest. They will also fail to appreciate the future costs associated with antisocial acts (e.g., imprisonment and lack of meaningful interpersonal relationships), and may stay in treatment only as required by an external source, such as a parole. Residential programs that provide a carefully controlled environment of structure and supervision, combined with peer confrontation, have been recommended (Gunderson & Gabbard, 2000). However, it is unknown what benefits may be sustained after the ASPD individual leaves this environment. During inpatient treatment individuals with ASPD may manipulate and exploit staff and fellow patients. Studies have indicated that outpatient therapy is not likely to be successful, although the extent to which persons with ASPD are entirely unresponsive to treatment may have been somewhat exaggerated (Salekin, 2002). Rather than attempting to develop a sense of conscience in these individuals, therapeutic techniques should perhaps be focused on rational and utilitarian arguments against repeating past mistakes. These approaches would focus on the tangible, material value of prosocial behavior (Young et al., 2003).

Narcissistic Personality Disorder Narcissistic personality disorder (NPD) was first included within the APA diagnostic manual in DSM-III. Its inclusion "was suggested by an increasing psychoanalytic literature and by the isolation of narcissism as a personality factor in a variety of psychological studies" (Frances, 1980, p. 1053). However, it was not included in the 10th edition of the World Health Organization's *International Classification of Diseases* (ICD-10; World Health Organization, 1992) despite its presence in the DSM since 1980, as it has been perceived internationally as largely an American concept. It was at one point slated for deletion in DSM-5 (Skodol, 2012).

Definition NPD is defined in DSM-5 as a pervasive pattern of grandiosity (in fantasy or behavior), need for admiration or adulation, and lack of empathy. Its primary diagnostic criteria include a grandiose sense of self-importance, preoccupation with success, power, brilliance, or beauty, the belief that one is special and can only be understood by high status individuals, a demand for excessive admiration, a strong sense of entitlement, an exploitation of others, a lack of empathy, and arrogance (American Psychiatric Association, 2000).

There has been some concern, however, that the DSM-5 criterion set may place too much emphasis on a grandiose narcissism, which can be associated with success in work and career, failing to adequately recognize a vulnerable narcissism indicated by a need for admiration, self-devaluation, and feelings of vulnerability, humiliation or rage in response to criticism or rebuke (Miller et al., 2010; Pincus & Lukowitsky, 2010). It is suggested that narcissistic persons fluctuate between states of grandiosity and vulnerability, and when in the latter state will not appear at all arrogant, grandiose, or conceited.

Etiology and Pathology There has been little systematic research on the etiology of NPD. Twin studies have supported heritability for narcissistic personality traits (South, Reichborn-Kjennerud, Eaton, & Krueger, 2012), although given the trait complexity of narcissism (Glover, Miller, Lynam, Crego, & Widiger, 2012; Pincus & Lukowitsky, 2010; Ronningstam, 2005), it is not entirely clear what precisely is being inherited. The predominant models for the etiology of NPD have been largely social learning or psychodynamic (Ronningstam, 2005). One model proposes that NPD develops through an excessive idealization by parental figures, which is then incorporated by the child into his or her self-image (Horton, 2011). NPD may also develop through unempathic, neglectful, inconsistent, or even devaluing parental figures who have failed to adequately mirror a child's natural need for idealization (Millon, 2011). The child may find that the attention, interest, and perceived love of a parent are contingent largely on achievements or successes. They might then develop the belief that their own feelings of self-worth are dependent upon a continued recognition of such achievements, status, or success by others. The character armor of arrogant self-confidence would mask a vulnerability and at times rage over the feeling of having been so neglected, and perhaps even mistreated and devalued, as a child.

Conflicts and deficits with respect to self-esteem are central to the pathology of NPD (Ronninstam, 2005), and there is considerable empirical support for this model in studies published within the general personality literature (Miller et al., 2010; Morf & Rhodewalt, 2000; Pincus & Lukowitsky, 2010). Individuals with NPD must continually seek and obtain signs and symbols of recognition to compensate for conscious or perhaps even unconscious feelings of inadequacy. Narcissism is not simply arrogant self-confidence as it is more highly correlated with an instability in self-confidence rather than a consistently high self-confidence. Individuals with NPD may at times claim that it is not narcissism if they are in fact brilliant, talented, and successful. However, the pathology would

still be evident by an excessive need for the recognition and acknowledgment of their achievements.

Differential Diagnosis Narcissistic personality disorder overlaps substantially with ASPD (Widiger, 2006). Prototypic cases of NPD and ASPD will share the features of lack of empathy, exploitation of others, and arrogance. However, persons with NPD are generally seeking recognition, fame, and success, whereas antisocial persons are generally seeking material benefits. Persons with ASPD will also be generally more impulsive and disinhibited than individuals with NPD. Individuals with ASPD will also lack the vulnerability that can be evident in some persons with NPD.

Epidemiology NPD is among the least frequently diagnosed personality disorders within clinical settings, with estimates of prevalence as low as 2% (American Psychiatric Association, 2013). A curious finding is that many community studies that have used a semistructured interview have not even been able to identify one single case, despite the substantial amount of research on maladaptive narcissistic personality traits within community and college samples (Miller et al., 2010; Morf & Rhodewalt, 2001; Ronningstam, 2005). NPD is perhaps one of the more difficult personality disorders to assess, as persons are reluctant to acknowledge arrogance, sense of entitlement, lack of empathy, and conceit (Cooper, Balsis, & Oltmanns, 2012). DSM-5 NPD is diagnosed more frequently in males (American Psychiatric Association, 2013), consistent with the finding within general personality research that men tend to be, on average, more arrogant than women (Lynam & Widiger, 2007b).

Course This disorder does not generally abate with age and may even become more evident into middle or older age. Persons with this disorder might be seemingly well adjusted and even successful as a young adult, having experienced substantial achievements in education, career, and perhaps even within relationships (Ronningstam, 2005). However, narcissistic personality traits are associated with relationship failure (Miller et al., 2010). Relationships with colleagues, peers, and intimates can become strained over time as the lack of consideration for and even exploitative use of others becomes cumulatively evident. Successes might also become more infrequent with age as the inability to accurately perceive or address criticism and setback contributes to a mounting number of defeats. Persons with NPD may at times not recognize their pathology until they have had a substantial number of setbacks, or they have finally recognized that the excessive importance they have given to achievement, success, and status has led to an emptiness and loneliness in their older age (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2005).

Five Factor Model Reformulation One of the facets of FFM antagonism is arrogance, the central trait of NPD (American Psychiatric Association, 2013; Millon, 2011). However, as indicated in Table 12.1, additional traits of narcissism can also be described in terms of the FFM. An advantage of the FFM dimensional classification is the ability to distinguish between arrogant persons who are high versus low in neuroticism (Campbell & Miller, 2013; Glover, Miller et al., 2012). A longstanding concern within the clinical literature on NPD is the distinction between individuals with NPD who are consistently self-confident, arrogant, and conceited (described as the arrogant or grandiose narcissist; Ronningstam, 2005; Pincus & Lukowitsky, 2010) versus the person with NPD who is quite insecure and self-conscious (referred to as the vulnerable narcissist). The dimensional perspective of the FFM would not suggest the creation of artifactual subtypes of this diagnostic category to distinguish such persons but would simply describe the extent to which a person who is high in arrogance is also low in the anxiousness, self-consciousness, and vulnerability facets of neuroticism. Further distinctions would be provided by the extent to which the person is low in extraversion (the shy narcissist) versus high in extraversion (the outgoing, interpersonally engaging narcissist), or high in conscientiousness (the narcissist who is relatively successful in school, college, and career). Glover, Miller et al. (2012) developed a measure of NPD from the perspective of the FFM titled the Five Factor Narcissism Inventory (FFNI).

Treatment Persons rarely seek treatment for their narcissistic personality traits. Individuals with NPD enter treatment seeking assistance for another mental disorder, such as substance abuse (secondary to career stress), mood disorder (secondary to career setback), or even something quite specific, such as test anxiety. One may at times have persons with NPD seeking treatment for a growing sense of discontent and futility with their lives (Ronningstam, 2005). Once an individual with NPD is in treatment, he or she will have difficulty perceiving the relationship as collaborative and will likely attempt to dominate, impress or devalue the therapist. They can idealize their therapists (to affirm that the therapist is indeed of sufficient status or quality to be treating them) but they may also devalue the therapist to affirm to themselves a sense of superiority . How best to respond is often unclear as the establishment and maintenance of rapport will be an immediate and ongoing issue. It may at times be preferable to simply accept the praise or criticism, whereas at other times it is preferable to confront and discuss the motivation for the devaluation (or the idealization). Therapists must be careful not to become embroiled within intellectual conflicts and competitions. Individuals with NPD can be acutely aware of the self-esteem conflicts of their therapist, and it is best for the therapist to model a comfortable indifference to losing disputes or conflicts.

Borderline Personality Disorder Borderline personality disorder (BPD) was a new addition to DSM-III (American Psychiatric Association, 1980; Spitzer, Endicott, & Gibbon, 1979). However, it has since become the single

most frequently diagnosed (Gunderson, 2001) and studied (Blashfield & Intoccia, 2000) personality disorder.

Definition BPD is a pervasive pattern of impulsivity and instability in interpersonal relationships, affect, and self-image (American Psychiatric Association, 2013). Its primary diagnostic criteria include frantic efforts to avoid abandonment, unstable and intense relationships, impulsivity (e.g., substance abuse, binge eating, or sexual promiscuity), recurrent suicidal thoughts and gestures, self-mutilation, and episodes of rage and anger.

Etiology and Pathology There are studies supportive of BPD as a disorder with a distinct genetic disposition but many studies have also suggested a shared genetic association with mood and impulse control disorders (South et al., 2012). There is also substantial empirical support for a childhood history of physical and/or sexual abuse, parental conflict, loss, and neglect (Silk, Wolf, Ben-Ami, & Poortinga, 2005). Past traumatic events are present in many (if not most) cases of BPD, contributing to the comorbidity with post-traumatic stress and dissociative disorders (Gunderson, 2001; Hefferman & Cloitre, 2000). BPD is perhaps best understood as an interaction of an emotionally unstable temperament with a cumulative and evolving series of intensely pathogenic relationships (Gunderson, 2001; Widiger, 2005).

The pathogenic mechanisms of BPD are addressed in numerous theories. Most concern issues of abandonment, separation, and/or exploitative abuse. Persons with BPD will often describe quite intense, disturbed, and/or abusive relationships with the significant persons of their past (Gunderson, 2001). The development of malevolent perceptions and expectations of others is not surprising (Ornduff, 2000). These malevolent expectations and lingering feelings of bitterness and rage, along with an impairment in the ability to regulate affect (Linehan, 1993), may contribute to the perpetuation of intense, hostile, and unstable relationships.

Differential Diagnosis Most persons with BPD develop quite a number of other mental disorders, including mood, dissociative, eating, substance use, and anxiety disorders (Hooley et al., 2012). It can be difficult to differentiate BPD from these disorders if the assessment is confined to current symptomatology (Gunderson, 2001). The diagnostic criteria for BPD require that the symptomatology be evident since adolescence, which should differentiate BPD from other mental disorders in most cases. If a chronic mood disorder is present, then the additional features of transient, stress-related paranoid ideation, dissociative experiences, impulsivity, and anger dyscontrol of BPD should be emphasized in the diagnosis (Gunderson, 2001).

Epidemiology It is estimated that 1–2% of the general population would meet the DSM-5 criteria for BPD (Torgersen, 2012). BPD is the most prevalent personality disorder within most clinical settings (Hooley et al., 2012). Approximately 15% of all inpatients (51% of inpatients with a personality disorder) and 8% of all outpatients (27% of outpatients with a personality disorder) will meet criteria for BPD. Approximately 75% of persons diagnosed with BPD are female (Lynam & Widiger, 2007b).

Course Individuals with BPD are likely to report having been emotionally unstable, impulsive, and angry as children, however there is little longitudinal research on the childhood antecedents of BPD (De Fruyt & De Clercq, 2012). As adolescents, their intense affectivity and impulsivity may contribute to involvement with rebellious groups, along with the development of a variety of other psychological disorders, including eating, substance, and mood disorders. BPD is at times diagnosed in children and adolescents but considerable caution should be used when doing so, as some of the symptoms of BPD (e.g., identity disturbance, hostility, and unstable relationships) could be confused with a normal adolescent rebellion or identity crisis (Gunderson, 2001).

As adults, persons with BPD may be repeatedly hospitalized, owing to their affect and impulse dyscontrol, psychotic-like and dissociative symptoms, suicidal behavior, and non-suicidal self-injurious behavior (Gunderson, 2001). Persons with BPD are said to be manipulative with respect to their suicidal gestures, threats, and attempts, but the risk of death from suicide in people who suffer from BPD is actually high (Hooley et al., 2012).The risk of suicide is increased with a comorbid mood disorder and substance-related disorder. It is estimated that 3–10% of persons with BPD will have committed suicide by the age of 30 (Gunderson, 2001). Intimate relationships tend to be unstable and explosive, and employment history is generally poor. As the person reaches the age of 30, affective lability and impulsivity may begin to diminish (Skodol et al., 2005; Zanarini et al., 2005). These symptoms may lessen earlier if the person becomes involved with a supportive and patient sexual partner. Some, however, may obtain stability by abandoning the effort to obtain a relationship, opting instead for a lonelier but less volatile life.

Five Factor Model Reformulation BPD is primarily composed of excessively high negative affectivity. In particular, these individuals are at the very highest range of anxiousness, angry hostility, depressiveness, impulsiveness, and vulnerability (see Table 12.1). Clients who have BPD will also likely be low in the agreeableness facets of trust and compliance, and low on the conscientiousness facets of order and deliberation (Trull & Brown, 2013).

The FFM conceptualization of BPD is helpful in explaining its substantial prevalence within clinical settings and its excessive diagnostic comorbidity. A diagnostic category defined primarily by all of the facets of neuroticism (i.e., vulnerability to stress, impulse

dyscontrol, despondence, rage, and other components of negative affectivity) will be highly prevalent within clinical settings. In addition, most of the other DSM-5 personality disorders include at least some components of neuroticism. Lynam and Widiger (2001) demonstrated that much of the diagnostic co-occurrence of BPD with other personality disorders is explained by common facets of neuroticism. Mullins-Sweatt, Edmundson et al., (2012) developed a measure of BPD from the perspective of the FFM titled the Five Factor Borderline Inventory.

Treatment Clients with BPD form relationships with therapists that are similar to their other significant relationships; that is, the therapeutic relationship can often be tremendously unstable, intense, and volatile. Ongoing consultation with colleagues is recommended to address potential negative reactions toward the client (e.g., distancing, rejecting, or abandoning the patient in response to feelings of anger or frustration) as well as positive reactions (e.g., fantasies of being the therapist who in fact rescues or cures the patient, or romantic, sexual feelings in response to a seductive patient). Sessions should emphasize the building of a strong therapeutic alliance, monitoring self-destructive and suicidal behaviors, validation of suffering and abusive experience (but also helping the client take responsibility for actions), promotion of self-reflection rather than impulsive action, and setting limits on self-destructive behavior (Gunderson, 2001). The tendency of borderline patients to engage in "splitting" (polarization of an emotional response) should also be carefully monitored and addressed (e.g., devaluation of prior therapists, coupled with idealization of current therapist).

The APA (2001) has published practice guidelines for the psychotherapeutic and pharmacologic treatment of persons with BPD. Because patients with BPD can present with significant suicide risk, a thorough evaluation of the potential for suicidal ideation and activity should have the initial priority (Hooley et al., 2012). There is empirical support for both psychodynamic and cognitive-behavioral treatments of BPD (American Psychiatric Association, 2001). The most common version of the former approach is mentalization-based treatment (Bateman & Fonagy, 2012). This treatment uses many structured techniques to help persons with BPD to "mentalize," or stand outside their feelings and more accurately observe the feelings within themselves and others. The most common form of cognitive-behavioral BPD treatment is dialectical behavior therapy (DBT; Lynch, Trost, Salsman, & Linehan, 2007). The dialectical component of DBT was derived largely from Zen Buddhist principles of overcoming suffering through acceptance (Linehan, 1993). Mastery of conflict is achieved in part through no longer struggling or fighting adversity; pain is overcome when it is accepted as an inevitable, fundamental part of life. This principle is taught in part through the meditative technique of mindfulness, in which one attempts to empty one's mind of all thoughts, but accepts whenever and wherever the mind naturally travels. DBT initially focuses on reducing self-harm and para-suicidal behaviors that are disruptive to treatment. Contracts may be implemented, wherein time with the therapist is limited secondary to treatment disruptive behavior. This can even go so far as to include suspension of treatment secondary to suicidal behavior. After mastery of treatment disruptive behavior, DBT teaches coping skills focused on emotional control and interpersonal relatedness. Individuals in DBT attend regular sessions with an individual therapist and discuss problems in applying the new skills. These sessions are augmented with a didactic skills-training group.

Schizotypal Personality Disorder Schizotypal personality disorder (STPD) was a new addition to DSM-III. Its diagnostic criterion set was developed originally from studies of biological relatives of persons diagnosed with schizophrenia (Spitzer et al., 1979).

Definition STPD is characterized by a pervasive pattern of social and interpersonal deficits marked by an acute discomfort with close relationships, eccentricities of behavior, and cognitive-perceptual aberrations. Diagnostic criteria for STPD include odd beliefs, magical thinking, social withdrawal, unusual perceptual experiences, odd speech, inappropriate or constricted affect, social anxiety, and social withdrawal (American Psychiatric Association, 2013).

Etiology and Pathology STPD is not included within the personality disorder section of the ICD-10; it is classified instead as a form of schizophrenia (World Health Organization, 1992). There is compelling empirical support for a genetic association of STPD with schizophrenia (Kwapil & Barrantes-Vidal, 2012) which is not surprising given that the diagnostic criteria were obtained from the observations of biological relatives of persons with schizophrenia (Miller, Useda, Trull, Burr, & Minks-Brown, 2001).

A predominant model for the psychopathology of STPD is deficits or defects in the attention and selection processes that organize a person's cognitive-perceptual evaluation of and relatedness to his or her environment (Raine, 2006). These deficits may lead to discomfort within social situations, misperceptions and suspicions, and a coping strategy of social isolation. Correlates of central nervous system dysfunction seen in persons with schizophrenia have been observed in STPD laboratory studies, including performance on tests of visual and auditory attention (e.g., backward masking and sensory gating tests) and smooth pursuit eye movement (Parnas, Licht, & Bovet, 2005). This dysfunction may be the result of dysregulation along dopaminergic pathways, which could be serving to modulate the expression of an underlying schizotypal genotype.

Differential Diagnosis An initial concern for many clinicians when confronted with a person with STPD is whether the more appropriate diagnosis might be schizophrenia. Persons with severe variants of STPD can closely resemble persons within the prodromal (or residual) phase of schizophrenia (American Psychiatric Association, 2013). A differentiation of the two conditions can be guided in part by age of onset and whether there is a recent deterioration in functioning. Persons with STPD will have evidenced their social anxiety, social withdrawal, magical thinking, odd behavior, and perceptual aberrations since childhood, and will not typically be characterized by any recent deterioration in functioning. Longitudinal follow-up would provide the conclusive differentiation, as a prodromal phase of schizophrenia will eventually be followed by an active psychotic episode.

The close relationship of STPD to the phenomenology, genetics, and pathology of schizophrenia has suggested that perhaps it should in fact be classified in future editions of the *Diagnostic Manual* as a variant of schizophrenia rather than a personality disorder (Skodol, 2012). In DSM-5, STPD is included in both the personality disorders and the schizophrenia spectrum sections. However, contrary to conceptualizing STPD as a form of schizophrenia is that STPD is much more comorbid with other personality disorders than with psychotic disorders, persons with STPD very rarely develop schizophrenia, and schizotypal symptomatology is evident within the general population in persons with no apparent genetic relationship to schizophrenia (Raine, 2006).

Epidemiology STPD may occur in as much as 3% of the general population although most studies with semi-structured interviews have suggested a somewhat lower percent (Torgersen, 2012). STPD might occur somewhat more often in males (Parnas et al., 2005; Raine, 2006).

Course There is insufficient research to describe the childhood precursors of adult STPD (De Fruyt & De Clercq, 2012). There are, however, retrospective reports of neurodevelopmental abnormalities in infancy and childhood for persons diagnosed with STPD (Raine, 2006; Parnas et al., 2005). During childhood they would be expected to have appeared peculiar and odd to their peers, and may have been teased or ostracized. Achievement in school might be impaired, and they may have been heavily involved in esoteric fantasies and peculiar interests. As adults, they may drift toward esoteric, fringe groups that support their magical thinking and aberrant beliefs. These activities can provide structure for some persons with STPD, but they can also contribute to a further loosening and deterioration if there is an encouragement of aberrant experiences. The symptoms of STPD do not appear to remit with age (Miller et al., 2001; Raine, 2006). The course appears to be relatively stable, with some proportion of schizotypal persons remaining marginally employed, withdrawn, and transient throughout their lives. Schizotypal symptoms, as measured in scales assessing cognitive-perceptual aberrations, magical thinking, and social and physical anhedonia, have been studied longitudinally in a number of community samples, and the findings to date do not suggest any meaningful likelihood of the development of schizophrenia (Gooding, Tallent, & Matts, 2005).

Five Factor Model Reformulation The FFM conceptualization of STPD views it as a disorder of introversion (social withdrawal), neuroticism (anxiousness), and maladaptive openess. Key to STPD are the magical ideation, cognitive-perceptual aberrations, and eccentric behaviors, which are considered within the FFM of personality disorder to be maladaptive variants of openness to ideas, fantasies, and actions (Edmundson & Kwapil, 2013).

FFM openness has obtained weak relationships with schizotypal thinking in some studies (Krueger et al., 2011; Watson, Clark, & Chmielewski, 2008). Recent studies, however, have reported a convergence of FFM openness with the cognitive and perceptual aberrations of schizotypy (De Fruyt et al., 2013; Gore & Widiger, 2013; Thomas et al., 2013). The existence of the relationship appears to depend on how both schizotypal thinking and FFM openness are conceptualized and assessed (Chmielewski, Bagby, & Markon, 2014; Gore & Widiger, 2013). Traditional measures of FFM openness emphasize a healthy psychological functioning, with items concerning aspects of self-realization and self-actualization. However, other measures of openness include as well aspects of unconventionality and peculiarity. Edmundson et al. (2011) developed a measure of STPD from the perspective of the FFM titled the Five Factor Schizotypal Inventory.

Treatment Persons with STPD may seek treatment for anxiousness, perceptual disturbances, or depression. Treatment of persons with STPD should be cognitive, behavioral, supportive, and/or pharmacologic, as they will often find the intimacy and emotionality of reflective, exploratory psychotherapy to be too stressful and they have the potential for psychotic decompensation.

Persons with STPD will often fail to consider their social isolation and aberrant cognitions and perceptions to be particularly problematic or maladaptive. They may consider themselves to be simply eccentric, creative, or nonconformist. Rapport can be difficult to develop, as increasing familiarity and intimacy may only increase their level of discomfort and anxiety (Millon, 2011). They are unlikely to be responsive to informality or playful humor. The sessions should be well structured, to curb loose and tangential ideation.

Practical advice is usually helpful and often necessary (Kwapil & Barrantes-Vidal, 2012). The therapist should serve as the patient's counselor or guide to more adaptive decisions with respect to everyday problems (e.g., finding an apartment, interviewing for a job, and personal appearance). Persons with STPD should also receive social skills

training directed at their awkward and odd behavior, mannerisms, dress, and speech. Specific, concrete discussions on what to expect and do in various social situations (e.g., formal meetings, casual encounters, and dates) should be provided.

Most of the systematic empirical research on the treatment of STPD has been confined to pharmacologic interventions. Low doses of neuroleptic medications (e.g., thiothixene) have shown some effectiveness in the treatment of schizotypal symptoms, particularly the perceptual aberrations and social anxiousness (Silk & Feurino, 2012)). Group therapy has also been recommended for persons with STPD but only when the group is highly structured and supportive (Millon, 2011). The emotional intensity and intimacy of unstructured groups will usually be too stressful. Schizotypal patients with predominant paranoid symptoms may even have difficulty in highly structured groups.

Dependent Personality Disorder A diagnosis of dependent personality disorder (DPD) was, technically speaking, a new addition to DSM-III, in that it had not been included within the prior edition of the *Diagnostic Manual*. However, a diagnosis of passive-dependent personality trait disturbance was included within the first edition. The diagnosis, however, was slated for deletion in DSM-5 (Skodol, 2012), despite there being a considerable body of research to support the validity and clinical importance of dependent personality traits (Bornstein, 2012b; Gore & Pincus, 2013; Mullins-Sweatt, Bernstein et al., 2012). Fortunately, this disorder was retained because all of the proposals for DSM-5 were rejected, including the proposal to delete half of the personality disorders.

Definition DPD involves a pervasive and excessive need to be taken care of that leads to submissiveness, clinging, and fears of separation (American Psychiatric Association, 2013; Bornstein, 2005). Its primary diagnostic criteria include extreme difficulty making decisions without others' input, need for others to assume responsibility for most aspects of daily life, difficulty disagreeing with others, inability to initiate projects due to lack of self-confidence, and going to excessive lengths to obtain the approval of others.

Etiology and Pathology Insecure interpersonal attachment is considered to be central to the etiology and pathology of DPD (Bornstein, 2005; Luyten & Blatt, 2013). Insecure attachment and helplessness may be generated through a parent–child relationship, perhaps by a clinging parent or by continued infantilization during a time in which individuation and separation normally occurs (Bornstein, 2012a). However, the combination of an anxious and/or inhibited temperament with inconsistent or overprotective parenting may also generate and exacerbate dependent personality traits (Bornstein, 2005; Luyten & Blatt, 2013). Unable to generate feelings of security and confidence for themselves, dependent persons may rely on a parental figure for constant reassurance of their worth. Eventually, persons with DPD may come to believe that their self-worth is defined by their importance to another person.

Differential Diagnosis Excessively dependent behavior may be seen in persons who have developed debilitating mental and physical conditions, such as agoraphobia, schizophrenia, severe injuries, or dementia. However, a diagnosis of DPD requires the presence of the dependent traits since late childhood or adolescence (American Psychiatric Association, 2013). One can diagnose the presence of a personality disorder at any age during a person's lifetime, but if (for example) a DPD diagnosis is given to a person at the age of 75, this presumes that the dependent behavior was evident since the age of approximately 18 (i.e., predates the onset of a comorbid physical impairment or disability secondary to aging; Oltmanns & Balsis, 2011).

Epidemiology DPD is one of the more prevalent personality disorders and is estimated to occur in 5–30% of patients and 2–4% of the general community (Torgersen, 2012). Longitudinal studies have indicated that dependent personality traits are a risk factor for the development of depression in response to interpersonal loss (Gore & Pincus, 2013; Hammen, 2005).

Course To the extent that independent responsibility and initiative are required, job functioning will be impaired or unsatisfactory. Individuals with DPD are prone to mood disorders throughout life, particularly major depression and dysthymia, and to anxiety disorders, particularly agoraphobia, social phobia, and panic disorder (Bornstein, 2005). There is also a considerable body of research to indicate significant social and personal costs for dependent personality traits, including increased risk for suicide, victimization, and excessive health care use (Bornstein, 2012b).

The self-esteem of a person with DPD is said to depend substantially on the maintenance of a supportive and nurturant relationship (Bornstein, 2005; Luyten & Blatt, 2013), yet these intense needs for reassurance can have the paradoxical effect of driving the needed person away. The dependent person's worst fears are then realized (i.e., he or she is abandoned and alone), and his or her sense of self-worth, meaning, or value is then furthered injured, perhaps even crushed by the rejection (Shahar, Joiner, Zuroff, & Blatt, 2004). The dependent person might then indiscriminately select a readily available but unreliable (and perhaps even abusive) person simply to be with someone (Widiger & Presnall, 2012). This partner would again reaffirm the worst fears through the abuse, derogation, and denigration (i.e., conveying to the dependent person that he or she is indeed undesirable and unlovable and that the relationship is again tenuous).

Research on the relationship of dependency to depression, however, is not without fundamental concerns (Coyne, Thompson, & Whiffen, 2004). In theory, dependent personality traits contribute to the instability of intimate and supportive relationships through the expression of excessive needs for reassurance and/or a premorbid emotional instability and pathogenic cognitions that contribute to intense feelings of helplessness and neediness. However, it is also possible that the emotional instability and pathologic attitudes are themselves the result of unstable interpersonal relationships. Dependency is a personality disposition that is seen much more often in women than in men (Lynam & Widiger, 2007b). A provocative reformulation of dependency in women is that the apparent feelings of insecurity may say less about the women than the persons with whom the women are involved. "Men and women may differ in what they seek from relationships, but they may also differ in what they provide to each other" (Coyne & Whiffen, 1995, p. 368). In other words, "women might appear (and be) less dependent if they weren't involved with such undependable men" (Widiger & Anderson, 2003, p. 63).

Five Factor Model Reformulation Dependent personality is characterized in terms of the FFM by maladaptively high levels of agreeableness (meek, gullible, & compliant) and the neuroticism facets of anxiousness, self-consciousness, and vulnerability (see Table 12.1). Researchers have indeed verified an association between the FFM domain of agreeableness and dependent personality disorder symptomatology (Gore & Pincus, 2013; Lowe, Edmundson, & Widiger, 2009).

A controversial issue in the diagnosis of DPD has been its differential sex prevalence (Oltmanns & Powers, 2012). DPD is diagnosed much more frequently in females (American Psychiatric Association, 2013) and some have argued that this may reflect a masculine bias toward what constitutes a personality disorder and/or a failure of males to acknowledge the presence of dependency needs (Bornstein, 2005). One difficulty in resolving this issue is the absence of any gold standard or even theoretical basis for hypothesizing, let alone determining, whether there should in fact be a differential sex prevalence rate. Understanding the DSM-5 personality disorders from the perspective of the FFM, however, does provide a theoretical basis for gender difference expectations (Lynam & Widiger, 2007b). Researchers have consistently found that women tend to score higher than men on the domains of agreeableness and neuroticism (Costa & McCrae, 1992). Costa, Terracciano, and McCrae (2001) found these differences to be consistent across 26 cultures, ranging from very traditional (Pakistan) to modern (Netherlands). Thus, to the extent that DPD is a maladaptive variant of FFM agreeableness and neuroticism, one should then expect to obtain a differential sex prevalence rate.

This is not to suggest, however, that no gender bias operates in clinical assessments. Studies have indicated that some self-report inventories are providing gender biased assessments of DPD (Lindsay, Sankis, & Widiger, 2000). The Millon Clinical Multiaxial Inventory-III (MCMI-III; Millon, Millon, Davis, & Grossman, 2009) and the Minnesota Multiphasic Personality Inventory-2 (Colligan, Morey, & Offord, 1994) are two of the more commonly used measures of personality disorder, and these measures include gender-related items that are keyed in the direction of adaptive rather than maladaptive functioning. An item need not assess for dysfunction to contribute to a valid assessment of personality disorders. For example, items assessing for gregariousness can identify histrionic persons, items assessing for confidence can identify narcissistic persons, and items assessing conscientiousness can identify obsessive-compulsive persons (Millon, 2011). Items keyed in the direction of adaptive rather than maladaptive functioning can also be helpful in countering the tendency of some respondents to deny or minimize personality disorder symptomatology. However, these items will not be useful in differentiating abnormal from normal personality functioning and they will contribute to an over-diagnosis of personality disorders in normal or minimally dysfunctional populations, as seen, for example, in MCMI-III assessments in student counseling centers, child custody disputes, and personnel selection (Widiger & Boyd, 2009). Gore, Presnall, Miller, Lynam, & Widiger (2012) developed a measure of DPD from the perspective of the FFM titled the Five Factor Dependency Inventory.

Treatment There are no empirically validated treatments for DPD. Treatment recommendations are based essentially on anecdotal clinical experience. Persons with DPD will often be in treatment for one or more other mental disorders, particularly a mood (depressive) or an anxiety disorder. These individuals will tend to be very agreeable, compliant, and grateful, at times to excess (Bornstein, 2005, 2012b). Many individuals with DPD will find that the therapeutic relationship itself satisfies their need for support and concern and may then desist from seeking a partner. The client may be compliant and agreeable in order to be a patient that the therapist would continue to treat. Therapists should be careful to neither unwittingly encourage a compliant submissiveness, nor to reject the client in order to be rid of their clinging dependency.

An important component of treatment can be a thorough exploration of the need for support and its root causes. Cognitive-behavioral techniques can be useful to address feelings of inadequacy and helplessness, and to provide training in assertiveness and problem-solving techniques (Leahy & McGinn, 2012). Group therapy may be useful for persons with DPD, providing interpersonal feedback and modeling autonomous behavior. DPD is not known to respond to pharmacotherapy.

Conclusions

Maladaptive personality traits will often impair or impede the treatment of other mental disorders and should often be the focus of clinical treatment. Reviews of practitioners'

clinical records suggest, however, that personality disorders are not being diagnosed as frequently as they in fact occur, perhaps because the clinician's attention is being drawn to a mood, anxiety, substance use, or other form of psychopathology that has captured his or her immediate attention. It can be difficult to obtain insurance coverage for the treatment of a personality disorder due to the inaccurate assumption that they are not in fact treatable. This is regrettable because some maladaptive personality traits (e.g., borderline and antisocial) have substantial social and public health care costs.

Section 3 of DSM-5, for emerging models and measures, includes a five-domain, 25-trait dimensional model of personality disorder that is closely aligned, both conceptually and empirically with the FFM of general personality structure. The FFM offers a compelling alternative to the categorical diagnosis of personality disorders provided in DSM-5. Advantages of understanding personality disorders in terms of this dimensional model are the provision of more specific descriptions of individual patients (including adaptive as well as maladaptive personality functioning), the avoidance of arbitrary categorical distinctions, and the ability to bring to bear the extensive amount of research on the heritability, temperament, development, and course of general personality functioning to an understanding of personality disorders. It is unclear what role the FFM, or dimensional personality trait models in general, will play in future editions of the DSM, It is evident that the classification of psychopathology is shifting toward dimensional models (Regier, 2008), but there remains considerable opposition to this shift, including even for the personality disorders (Gunderson, 2010; Shedler et al., 2010).

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13

Sexual Dysfunctions and Paraphilic Disorders

Jennifer T. Gosselin

Sexual behavior has a long history of emotionally charged social and cultural expectations, rules, taboos, myths, and misconceptions. "Normal" versus "dysfunctional" or "deviant" sexual behaviors are socially constructed, determined arbitrarily, and are tied to the social norms of a particular time and place (Marecek, Crawford, & Popp, 2004). In ancient Greece, for example, a male mentor might have sex with his male adolescent student as part of their relationship, which was relatively accepted at that time as a way of demonstrating social standing or hierarchy (King, 1996; Plante, 2006). Today, this behavior is generally considered unethical, abusive and/or illegal, depending on the age of the student and mentor. In the Sambian tribe in New Guinea, an important rite involves younger boys performing oral sex on older boys to ingest semen, which is believed to provide strength and masculinity; when they get older, they transition into heterosexual adults (Stoller, 1985). In contrast, the Victorian era in England (during most of the 1800s) was a time of sexual conservatism, although like the Sambians, beliefs about sex were rigid and based on misconceptions about semen. The purpose of sex during this era was to produce children, not to experience pleasure. Masturbation was believed to be a cause of mental and/or physical illness, including blindness (King, 1996). In some cases, treatment for masturbation included clitoridectomy (removal of the clitoris), male circumcision, or castration (King, 1996; Lips, 2006). Throughout history, we find that today's deviance was yesterday's normalcy, and vice versa.

Important advances in describing sexual abnormalities for the scientific community came about in 1886 with Richard von Krafft-Ebing's book, *Psychopathia Sexualis*, a comprehensive, biomedical account of sexual deviance with clinical case reports (De Block & Adriaens, 2013), and in the early 1900s, when German psychiatrists created the journal *Zeitschrift für Sexualwissenschaft* (*Journal for Sexual Research*) and founded the Institut für Sexualwissenschaft (Institute for Sexual Research) in order to study and treat sexual dysfunction (Waldinger, 2008). The rise of the Nazis in Germany resulted in a swift halt to these research endeavors, however (Waldinger, 2008). In the US after World War II, researchers challenged social norms by studying the taboo topic of sex (Irvine, 1990; Kimmel & Plante, 2004). For example, Kinsey's survey research and Masters and Johnson's laboratory research have dispelled many myths about common sexual practices and sexual function (Kinsey, Pomeroy, & Martin, 1948; Masters, Johnson, & Kolodny, 1985).

In many nations around the globe, major developments and changes have occurred around sex, identity, and reproduction, such as improved methods of contraception and movements for the rights of women and individuals who identify as lesbian, gay, bisexual, transgender, or who are questioning their sexual orientation (LGBTQ). Homosexuality was included as a disorder in older editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), but was downgraded to "ego dystonic homosexuality" in 1980 and was removed as a disorder in the DSM-III-R (American Psychiatric Association, 1978), owing to the lack of empirical evidence that homosexuality is a psychological disorder, and political pressure (Ault & Brzuzy, 2009; Silverstein, 2009). A more likely explanation for distress among LGBTQ individuals is sociocultural, familial, and/or religious pressure to conform to a heterosexual majority and/or gender norms (Dickinson, Cook, Playle, & Hallett, 2012).

Despite social changes over time, a major thread that runs through the history of the conceptualization of sexual behaviors and dysfunctions is the alternating emphasis on psychology and biology, as if they were mutually exclusive (Berry, 2013b; Waldinger, 2008). While the mid-1900s included a rise in behaviorism and a psychogenic explanation of sexual dysfunction and deviance, the past two to three decades have been characterized by a biological explanation of these disorders and a concomitant dominance of pharmaceutical treatments (Waldinger, 2008).

Categories of Sexual Dysfunctions and Paraphilic Disorders

The recently released fifth edition of the DSM (DSM-5; American Psychiatric Association, 2013) includes substantial modifications to its diagnostic categorical structure for sexual dysfunctions and paraphilic disorders. Whereas DSM-IV-TR (American Psychiatric Association, 2000) had a section titled "Sexual and Gender Identity Disorders," which included *sexual dysfunction, the paraphilias, and gender identity disorder*, DSM-5 includes separate chapters for *sexual dysfunctions*, *paraphilias*, and *gender dysphoria*. This revised trifurcation is consistent with the recognition that these three diagnostic categories reflect distinct phenomena (Zucker et al., 2013).

Although most people have had occasional experiences that fall into a sexual dysfunction category—such as the inability to reach orgasm when so desired—sexual difficulties that are more frequent and distressing than an incidental occurrence are considered *sexual dysfunctions*, according to DSM-5. Sexual dysfunctions generally include difficulties or issues with sexual desire, arousal, orgasm, ejaculation, and/or pain during sex. *Paraphilias* involve strong sexual preferences, such as the requirement of particular objects or cross-dressing for sexual gratification, which may reflect relatively normal deviations in sexual behavior and may not warrant treatment. *Paraphilic disorders*, however, involve significant disruption to one's personal or interpersonal functioning and/or victimization of others as a result of the sexual preference (American Psychiatric Association, 2013). Additionally, *hypersexual behavior* is presented in the current chapter, despite its omission from the DSM. Diagnostic descriptions, prevalence, etiological or causal factors, and treatment approaches are discussed for each dysfunction or disorder presented, although data on the newly released diagnostic categories are generally unavailable.

Sexual Dysfunctions

Defining and Describing Sexual Dysfunctions DSM-5 defines *sexual dysfunctions* as "a heterogeneous group of disorders that are typically characterized by a clinically significant disturbance in a person's ability to respond sexually or to experience sexual pleasure" (American Psychiatric Association, 2013, p. 423). The explanation of sexual dysfunctions in DSM-5 abandons the highly criticized sexual response cycle as a diagnostic framework and instead presents a more nuanced, holistic picture of sexual functioning. This new framework emphasizes the interaction between the person and his/her psychological functioning, cultural, religious, and social forces, and situational and relationship factors, in addition to biology, which is in keeping with the current literature (Carvalho & Nobre, 2010). The previous dichotomy in DSM-IV-TR of specifying whether the dysfunction is due to psychological or both psychological and medical (combined) factors has been omitted in DSM-5, underscoring the inextricable nature of psychological and biological factors.

In order to receive a diagnosis for a sexual dysfunction, the difficulty must cause clinically significant distress. Further, a sexual dysfunction is ruled out if a psychological disorder, relationship difficulty, stressor, medical condition, or normal aging better accounts for the sexual symptoms, or if sexual stimulation is not sufficient to produce the desired sexual response. Symptoms must persist for at least 6 months for a diagnosis, except for *substance/medication-induced sexual dysfunction* (American Psychiatric Association, 2013). Symptoms must be frequent, specified as "approximately 75–100%" of the time for erectile disorder, female orgasmic disorder, female sexual interest/arousal disorder, and premature and delayed ejaculation.

The DSM-5 also includes several subtypes of sexual dysfunction (APA, 2013). The clinician indicates whether the dysfunction has been *acquired* after a period of normal functioning or if it is *lifelong* (since the first sexual experience), and whether the dysfunction is situational, meaning specific to particular partners, types of stimulation, or situations, or if it is *generalized* across these factors. Sexual difficulties must be considered "persistent or recurrent" by the clinician to be considered a disorder, and the clinician should also indicate the severity level of the disorder as resulting in *mild, moderate,* or *severe* distress.

The types of sexual dysfunction, according to DSM-5, are:

- Delayed ejaculation: Either: 1) Marked delay in ejaculation; or 2) Marked infrequency or absence of ejaculation, without the individual desiring delay.
- Erectile disorder: At least one of the three following symptoms: 1) Marked difficulty in obtaining an erection during sexual activity; 2) Marked difficulty in maintaining an erection until the completion of sexual activity; or 3) Marked decrease in erectile rigidity.
- Female orgasmic disorder: Presence of either: 1) Marked delay in, marked infrequency of, or absence of orgasm; or 2) Markedly reduced intensity of orgasmic sensations.
- Female sexual interest/arousal disorder: Lack of, or significantly reduced, sexual interest/arousal, as manifested by at least three of the following: 1) Absent/reduced interest in sexual activity; 2) Absent/reduced

sexual/erotic thoughts or fantasies; 3) No/reduced initiation of sexual activity, and typically unreceptive to a partner's attempts to initiate; 4) Absent/reduced sexual excitement/pleasure during sexual activity; 5) Absent/reduced sexual interest/arousal in response to any internal or external sexual/erotic cues (e.g., written, verbal, visual); 6) Absent/reduced genital or nongenital sensations during sexual activity.

- Genito-pelvic pain/penetration disorder: Persistent or recurrent difficulties with one (or more) of the following: 1) Vaginal penetration during intercourse; 2) Marked vulvovaginal or pelvic pain during vaginal intercourse or penetration attempts; 3) Marked fear or anxiety about vulvovaginal or pelvic pain in anticipation of, during, or as a result of vaginal penetration; 4) Marked tensing or tightening of the pelvic floor muscles during attempted vaginal penetration.
- Male hypoactive sexual desire disorder: Persistently or recurrently deficient (or absent) sexual/erotic thoughts or fantasies and desire for sexual activity.
- Premature (early) ejaculation: A persistent or recurrent pattern of ejaculation occurring during partnered sexual activity within approximately 1 minute following vaginal penetration and before the individual wishes it.
- Substance/medication-induced sexual dysfunction: A clinically significant disturbance in sexual function is predominant in the clinical picture. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2): 1) The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to a medication; 2) The involved substance/medication is capable of producing the symptoms in Criterion A.

Specific Modifications From the DSM-IV-TR to the DSM-5 for Sexual Dysfunctions A major change to the sexual dysfunction diagnostic categories is the separation of male and female disorders and the streamlining and merging of similar disorders. Previously, *hypoactive sexual desire disorder* applied to both genders. In DSM-5, the respective disorders are *female sexual interest/arousal disorder* and *male hypoactive sexual desire disorder*, based on a general consensus that men and women are different in this regard (Sungur & Gunduz, 2013). While DSM-IV-TR had been criticized for failure to recognize gender differences in sexual desire and arousal (Brotto, Bitzer, Laan, Leiblum, & Luria, 2010), DSM-5 has recently been criticized for assuming that men's desire is a homogenous process and for omitting the criterion of lack of pleasurable sensations during sex for men, which is included in female sexual interest/arousal disorder (Duschinsky & Chachamu, 2013). In other words, female sexual interest/arousal disorder entails a variety of different possible symptoms, from disinterest in sex to lack of pleasure during sex, while male hypoactive sexual desire disorder is only a lack of sexual thoughts, fantasies, or desire.

Another controversial modification is that *female sexual arousal disorder* and *hypoactive sexual desire disorder* have been combined in DSM-5 for women as female sexual interest/arousal disorder, and the formerly used *sexual aversion disorder* has been omitted from DSM-5. While some researchers argue that these changes reflect the difficulty of clinically separating desire and arousal and the lack of applicability of "desire" definitions to all women (Binik, Brotto, Graham, & Segraves, 2010; Brotto et al., 2010), other researchers argue that this change will result in the under-diagnosis of women with difficulties that do not meet the new criteria (Clayton, DeRogatis, Rosen, & Pyke, 2012). Another merging of disorders resulted in the sexual pain disorders *dyspareunia* and *vaginismus* being combined and labeled *genito-pelvic pain/penetration disorder*. Also, *female orgasmic disorder* now includes the optional criterion of "markedly reduced intensity of orgasmic sensations" (not just delay in or absence of orgasm) and an updated criterion to emphasize relationship issues: "The sexual dysfunction is not better explained by . . . severe relationship distress (e.g., partner violence)" (American Psychiatric Association, 2013, p. 429). The statements that the diagnosis is based on clinical judgment about the woman's "orgasmic capacity" and that the difficulty with orgasm occurs after a "normal sexual excitement phase" (American Psychiatric Association, 2000, p. 549) were both removed. These changes are generally attuned to current data and views on female sexual functioning (Basson, 2005; Graham, 2010).

For men, *male orgasmic disorder* was removed, with the new category of *delayed ejaculation* added (American Psychiatric Association, 2013). *Premature (early) ejaculation* and *erectile disorder* (formerly *male erectile disorder*) have remained. Since some researchers find the term premature ejaculation derogatory and had hoped to change the name of the disorder to *early ejaculation*, a combination term has been created with the word "early" in parentheses (Binik et al., 2010). The primary criterion of premature (early) ejaculation is an ejaculation that occurs within "approximately 1 minute" after vaginal penetration (American Psychiatric Association, 2013, p. 443), which is consistent with the definition proposed by the International Society for Sexual Medicine (Althof et al., 2010). Controversy continues, however, regarding the use of vaginal penetration in the definition, as this excludes same-sex activities (Jern et al., 2010). To acknowledge this issue, DSM-5 includes a note that "Although the diagnosis of premature (early) ejaculation may be applied to individuals engaged in nonvaginal sexual activities, specific duration criteria have not been established for these activities" (American Psychiatric Association, 2013, p. 443). Sexual dysfunction due to a substance and sexual dysfunction due to a general medical condition have been incorporated into the single disorder called *substance/medication-induced sexual dysfunction*.

Notably, the diagnostic criteria for the sexual dysfunctions have improved in their level of specificity in the

DSM-5, potentially reducing diagnostic reliability error due to differences in clinical judgment. For example, the formerly used symptom occurrence of "almost all or all occasions of sexual activity" has been specified as "approximately 75–100%" of the time for several disorders, which has received some support by researchers, even though it is an arbitrary cutoff (Segraves, 2010; Shindel & Lue, 2010). When presented to clients as a question of whether they are experiencing the problem for at least three of four encounters, for example, the clinician has a clearer threshold for diagnosis, compared to previous, more ambiguous diagnostic criteria.

Conversely, other researchers argue that distressed, treatment-seeking clients should generally be diagnosable and treated, even if they fall short of the new diagnostic thresholds (Nelson, 2010). Further, it is unlikely that experiencing symptoms for two of four encounters is qualitatively different than meeting the criteria for diagnosis, which is a criticism that applies to the dichotomous (disordered versus non-disordered) classification system of the DSM in general. While the new criterion of at least 6 months' duration of symptoms minimizes some degree of overdiagnosis, some researchers argue that this criterion may delay treatment for clients who do not meet this criterion (Sand, 2010). An additional criticism is that disorders of sexual interest/arousal are virtually always the result of one partner having a stronger desire for sex than the other partner, and low desire is pathologized, whereas excessive desire is not (Gehring, 2003). While DSM-5 indicates that a "desire discrepancy" between partners is inadequate to constitute a diagnosis in the partner with less desire, this diagnostic discrepancy still implies that treatment should focus on increasing the desire of the diagnosable partner, who has "deficient" desire, while a partner with excessive desire is not diagnosable.

Epidemiology of Sexual Dysfunctions Rates of sexual dysfunctions vary greatly from study to study, owing to methodological differences, diagnostic reliability issues, and the use of different participant samples (Rosen & Laumann, 2003). Given the short duration of time between the release of DSM-5 and the publication of the current chapter, epidemiology research has not yet been published using the new diagnostic categories; therefore, findings based on the previous DSM categories for sexual dysfunctions are presented here. Research across the globe indicates that approximately 30–47% of women and 20–31% of men have one or more sexual dysfunctions (Bagherzadeh et al., 2010; Echeverry, Arango, Castro, & Raigosa, 2010; Kadri, Alami, & Tahiri, 2002; Laumann, Paik, & Rosen, 1999; Lewis et al., 2004, 2010; Ornat et al., 2013; Zhang & Yip, 2012). Rates of female sexual dysfunction vary widely, from 15.5% (Burri & Spector, 2011) to as high as 55% (Fisher, Boroditsky, & Morris, 2004). When researchers include both sexual problems and overall sexual dissatisfaction, however, only about 14% of women report both concerns (Lutfey, Link, Rosen, Wiegel, & McKinlay, 2009).

The most common sexual dysfunctions among women tend to be hypoactive sexual desire disorder (Burri & Spector, 2011), female sexual arousal disorder (Nicolosi et al., 2006; Utian et al., 2005), and female orgasmic disorder (Fisher et al., 2004; West, Vinikoor, & Zolnoun, 2004). The most common sexual dysfunctions among men are premature ejaculation and erectile dysfunction (Dunn, Croft, & Hackett, 1999; Nicolosi et al., 2004, 2006). Rates of premature ejaculation range from 4% to 26% and tend to decrease with age (Breyer et al., 2010; Laumann, Glasser, Neves, & Moreira, 2009; McMahon, Lee, Park, & Adaikan, 2012; Simons & Carey, 2001), while rates of erectile dysfunction tend to increase with age (Hyde et al., 2012). More specifically, approximately 10–11% of men under 40 years, 30% of men 40–60 years, 49% of men 40–88 years, 76% of men 50–75 years, and 49% of men 75–95 years old, in separate studies, report erectile dysfunction, with an overall rate of 18% for men approximately 20–75 years of age or older (Feldman, Goldstein, Hatzichristou, Krane, & McKinlay, 1994; Grover et al., 2006; Hyde et al., 2012; Lewis et al., 2004; Mialon, Berchtold, Michaud, Gmel, & Suris, 2012; Prins, Blanker, Bohnen, Thomas, & Bosch, 2002; Saigal, Wessells, Pace, Schonlau, & Wilt, 2006; Shiri et al., 2003; Simons & Carey, 2001; Wentzell & Salmeron, 2009).

The wide variation in prevalence estimates of sexual dysfunction is a source of ongoing debate (see DeRogatis & Burnett, 2007, for a review). Prevalence estimates may be inflated because many studies do not include diagnostic interviews, and they often do not assess clinical distress associated with the dysfunction. Similar to the large variability in prevalence based on measurement differences, incidence rates of sexual dysfunction for men and women within the past year is 11%, while 68% of men and 69% of women report subclinical sexual difficulties over the previous year (Christensen et al., 2010). Despite studies that suggest that most men and women have had a recent sexual difficulty, the majority of people around the world tend to report that they are satisfied with their sexual functioning, including approximately 80% or more of men and women in Western nations (Laumann et al., 2006; Ornat et al., 2013). Further, research repeatedly shows that among women with sexual difficulties, a majority of them are still satisfied with their sex lives or are not distressed by their sexual difficulties (Burri et al., 2012; Burri & Spector, 2011; Ferenidou et al., 2008; King, Holt, & Nazareth, 2007).

Etiology of Sexual Dysfunctions Sexual desire, arousal, and behavior can be explained using a biopsychosocial model, meaning that internal processes, such as one's physiological systems, thoughts, and feelings, interact with one's behaviors and with external factors, such as immediate contexts, interpersonal, and larger social forces (Althof et al., 2005; Fagan, 2004). This interactionist approach provides a multidimensional explanation for

problems that usually do not have a clear, singular causal factor (King et al., 2007).

Biological/Genetic Factors in Sexual Dysfunctions Sexual dysfunctions typically manifest anatomically, implicating a wide range of vascular (blood vessel) and related medical, neurological, and anatomical conditions, as well as biochemical factors (Pfaus, 2009; Reffelmann & Kloner, 2010). Brain regions involving motivation, emotion, and pleasure, such as the anterior cingulate cortex, hypothalamus, and amygdala have been implicated in sexual function through the use of brain imaging studies during sex and orgasm (see Georgiadis, 2011, for a review). These studies, however, are compromised by a number of issues, such as small sample sizes, equipment and measurement difficulties involved in the logistics of performing a brain scan during sexual activity, as well as difficulties excluding non-sexual brain activation that is casued by body movement or registering touch. Despite these limitations, brain imaging research suggests that orgasm is related to temporal and prefrontal cortex regions of the brain, which actually show decreased blood flow during orgasm, possibly reflecting decreased self-focused attention (Georgiadis, 2011) and enjoyment of the moment instead.

Sex hormones (produced by the ovaries and testes) play a role in sexual interest and functioning in men and women (Berman, Berman, & Goldstein, 1999). Low levels of androgens, particularly testosterone, have been linked in some studies to loss of sexual arousal and orgasm in women (see Shifren, 2004, for a review), although other studies contradict this finding (Davis, Davison, Donath, & Bell, 2005; Moghassemi, Ziaei, & Haidari, 2011; Nyunt et al., 2005). In men, testosterone has more consistently been associated with sexual desire and activity, with low testosterone being linked with either decreased desire only (and not dysfunction; Hyde et al., 2012) or with both decreased desire and function (Bradford, 2001; Goldstein, 2004; Rowland, 2006). Similarly, low levels of estrogens in women may be related to diminished sexual desire and sexual functioning, although it is likely that sexual functioning in women is the result of a combination of estrogens and androgens (Berman & Bassuk, 2002; Rowland, 2006).

Neurotransmitters, such as serotonin and dopamine, also play a role in sexual functioning, a finding that is supported by animal and human research as well as by the sexual side effects of relevant medications (Basson, 2005; Kafka, 1997a; La Torre, Giupponi, Duffy, & Conca, 2013; Pfaus, 2009; Rowland, 2006). Changes in serotonin influence a wide range of physiological, behavioral, and mood-related phenomena, including appetite, nausea, sleep, body temperature, anxiety, depression, suicidality, and impulsivity (Carlson, 2001; Pinel, 2003). Increased levels of serotonin (due to antidepressant medications that block reuptake of serotonin, for example) tend to inhibit sexual activity and delay orgasm in both humans and rats (McMahon, 2004). Further, researchers have identified genetic variants involving specific serotonin receptors that may make certain individuals more susceptible to sexual side effects of selective serotonin reuptake inhibitors (SSRIs) than others (Bishop, Moline, Ellingrod, Schultz, & Clayton, 2006; Bly, Bishop, Thomas, & Ellingrod, 2013).

Dopamine is involved in motivation, leading to the repetition of rewarding experiences, such as eating and having sex (Bressan & Crippa, 2005; dopaminergic pathways are thus also important in addictive behaviors). The connection between dopamine and sexual behavior has been established, in part, through research on individuals who suffer from conditions involving dopaminergic pathways, such as Parkinson's disease and schizophrenia. For example, on the one hand, Parkinson's disease (which involves a depletion of dopamine) commonly includes diminished sexual interest and/or functioning, and patients who are treated with medications that facilitate dopamine transmission also tend to report an increase in impulse control difficulties, including hypersexual behavior (Weintraub et al., 2010). Schizophrenia, on the other hand, seems to involve excessive dopaminergic transmission, and patients who are treated with medications that inhibit dopamine transmission often report diminished sexual desire and may experience sexual dysfunction (Dominguez & Hull, 2005; Miclutia, Popescu, & Macrea, 2008).

In addition to the monoamines, nitric oxide (a neurotransmitter in the form of a soluble gas) plays a role in physiological sexual responses, such as erection, by relaxing smooth muscle tissue and allowing blood flow to the genitals. Medications such as sildenafil (Viagra™) increase erectile functioning by influencing the signaling of nitric oxide (Trussell, Anastasiadis, Padma-Nathan, & Shabsigh, 2004). Also involved in producing a sexual arousal response are neuropeptides (a type of neurotransmitter) called vasoactive intestinal polypeptides (VIPs; Rahardjo et al., 2011). VIPs additionally assist in registering sexual sensations and in producing lubrication in women as well as erection in men (Lara et al., 2012). A treatment avenue for individuals who do not respond to sildenafil may therefore be penile injection of VIPs that dilate the blood vessels (Porst et al., 2013; see also "Biological/Pharmacological Interventions for Sexual Dysfunctions," later in this chapter, p. 245).

In men and women, a proposed cause of limited physiological arousal includes insufficient nervous system response and/or blood flow to the genitalia. This physiological problem, in turn, may have a number of causes and related factors, including hypertension, cardiovascular and heart disease, genitourinary disease (such as disease or dysfunction involving the kidneys, prostate, urethra, or bladder), spinal cord injuries, and traumatic brain injury (Kriston, Gunzler, Agyemang, Bengel, & Berner, 2010; Lewis et al., 2004, 2010; Moreno, Lasprilla, Gan, & McKerral, 2013; Sipski, 2002). In addition to the conditions already mentioned, sexual dysfunction in men is predicted by obesity, endocrine disorders (such as

hypogonadism or dysfunction of the testes and thyroid disorders), smoking, hyperlipidemia (elevated lipids in the blood), Peyronie's disease (extreme curvature of the penile shaft), bicycle riding, lower urinary tract symptoms, HIV seropositive status, radical prostatectomy (surgery for prostate cancer), insomnia, stroke, urological conditions, opiate abuse, and chronic prostatitis/chronic pelvic pain syndrome (Chekuri, Gerber, Brodie, & Krishnadas, 2012; Hyde et al., 2012; Larsen, Wagner, & Heitmann, 2007; Lewis et al., 2004, 2010; Maggi, Buvat, Corona, Guay, & Torres, 2013; Namiki et al., 2012; Reffelmann & Kloner, 2010; Shindel, Vittinghoff, & Breyer, 2012; Smith, Pukall, Tripp, & Nickel, 2007; Sommer, Goldstein, & Korda, 2010; Wylie & Kenney, 2010).

Among women, breast cancer and its treatment, urinary incontinence (and particularly *coital incontinence* or involuntary urination during intercourse), *interstitial cystitis/ painful bladder syndrome*, and cervical and endometrial cancer treatment have been linked with sexual dysfunction (Brotto et al., 2008; Carbone & Seftel, 2004; Gardella et al., 2011; Garrusi & Faezee, 2008). Research on the link between hysterectomies and sexual dysfunction is mixed, with some women experiencing decreased orgasm or sexual arousal due to scar tissue or nerve damage, although sexual difficulties tend to abate over time, particularly after the patient has fully recovered from surgery (Bayram & Beji, 2010; Meston, 2004). Spinal canal cysts, called Tarlov cysts, have been linked to unwanted, bothersome, unrelenting genital arousal in women, termed *persistent genital arousal disorder* (Komisaruk & Lee, 2012). In addition, women who have suffered pelvic trauma, surgical or obstetrical complications, who have endometriosis or similar health conditions, those with sexually transmitted infections, chronic inflammation, skin diseases, tightening of the pelvic floor muscles, or those who have undergone female circumcision or female genital mutilation (a cultural practice in certain regions, particularly in the Middle East, including areas in Northern Africa) are more likely to experience dyspareunia (Berman et al., 1999; Burrows, Creasey, & Goldstein, 2011; Committee on Bioethics, 1998; Elgaali, Strevens, & Mardh, 2005; Fugl-Meyer et al., 2013). Finally, although some research has shown a connection between diabetes and female sexual dysfunction, depression and relationship issues may be more important contributing factors (Nowosielski, Drosdzol, Sipinski, Kowalczyk, & Skrzypulec, 2010).

Medication-Induced Sexual Dysfunctions A wide range of medications can influence sexual functioning by affecting the release of neurotransmitters, their receptors, or by influencing the vascular system. Antidepressant medications, such as SSRIs, monoamine oxidase inhibitors, and tricyclic antidepressants are associated with sexual dysfunction (see La Torre et al., 2013, for a review). A potential confounding variable is that diminished sexual desire or arousal may be a symptom of depression, rather than a side effect. While research suggests that a substantial portion (about 40–50%) of non-medicated individuals with depression have decreased sexual desire or sexual difficulties (Kennedy, Dickens, Eisfeld, & Bagby, 1999), research comparing treatment and control groups indicates that antidepressants also contribute to sexual impairment, particularly delayed or absent orgasm (Montgomery, Baldwin, & Riley, 2002). When asked about changes in sexual function pre- to post-medication, approximately 37% to 62% of patients taking antidepressants reported increased sexual dysfunction (Williams, Edin, Hogue, Fehnel, & Baldwin, 2010). In a handful of cases in a separate study, discontinuation of SSRIs seems to have led to persistent genital arousal disorder in women (Leiblum & Goldmeier, 2008).

A host of other medications have been associated with sexual dysfunction, including: anxiolytics (anti-anxiety medications), such as benzodiazepines; beta-blockers, which lower blood pressure; antipsychotic medications that increase levels of prolactin; oral contraceptives; antiretroviral treatment for HIV; and chemotherapy and other treatments for cancer (Asboe et al., 2007; Bishop, Ellingrod, Akroush, & Moline, 2009; Gruszecki, Forchuk, & Fisher, 2005; Kao et al., 2003; Knegtering, van der Moolen, Castelein, Kluiter, & van den Bosch, 2003; Murthy & Wylie, 2007; Rowland, 2006; Segraves, 2002; Trotta et al., 2008).

The most common strategies used to minimize these effects are switching antidepressant medications, adding medications to the existing SSRI treatment regimen, or reducing the dose of the antidepressant (Balon & Segraves, 2008). Medications that have shown some success in minimizing sexual side effects include milnacipran, mirtazapine, and gepirone extended release, as well as the addition of medications, such as bupropion or sildenafil to the current SSRI treatment regimen (Baldwin, Moreno, & Briley, 2008; Dhillon, Yang, & Curran, 2008; Fabre, Clayton, Smith, Goldstein, & DeRogatis, 2012; Nurnberg, Hensley, & Lauriello, 2000; Osvath, Fekete, Voros, & Almasi, 2007; Safarinejad, 2011; Zisook, Rush, Haight, Clines, & Rockett, 2006).

The use of oral contraceptives has been related to sexual dysfunction in some research, but other studies have found no clear impairment caused by oral contraceptives. For example, hormonal contraceptives (oral contraceptives and vaginal rings) have been associated with diminished sexual functioning and desire in women compared with women who were not using hormonal contraceptives (Wallwiener et al., 2010). Also, the risk for vulvar vestibulitis (which causes pain during sex) was increased for women with an extensive history of oral contraception use (Bouchard, Brisson, Fortier, Morin, & Blanchette, 2002). In a separate study, however, women taking oral contraceptives were actually significantly less likely to have difficulty with orgasm compared to women who were not taking oral contraceptives, which was attributed to increased enjoyment of sex without the worry of pregnancy, and potential reduction in painful gynecological

issues, such as ovarian cysts (Goshtasebi, Vahdaninia, & Foroshani, 2009). Other research found that intrauterine contraceptives resulted in rates of sexual dysfunction comparable to those found in the general population (Enzlin et al., 2012). A review of findings concluded that decreased desire, no change in desire, and improvements in desire were demonstrated in different studies (Davis & Castano, 2004). The mixed findings about the use of contraceptives and sexual function may be partly explained by genetic variation in susceptibility to sexual dysfunction as a result of medication (Bishop et al., 2009). One possibility is that sexual dysfunction due to hormonal contraceptives could be alleviated by changing the contraceptive medication regimen. For example, estradiol valerate with a progestogen called dienogest may improve sexual functioning (Caruso et al., 2011).

Psychosocial and Environmental Factors in Sexual Dysfunctions Previous sexual experiences, beliefs and knowledge about sex, as well as stress, depression, anxiety, eating disorders, relationship issues, and many other factors can play a role in the development of sexual difficulties and dysfunction (Araujo, Durante, Feldman, Goldstein, & McKinlay, 1998; Bancroft, Loftus, & Long, 2003; Bodenmann, Ledermann, Blattner, & Galluzzo, 2006; Clayton & Balon, 2009; Fabre & Smith, 2012; Laumann et al., 1999; Michael & O'Keane, 2000; van Lankveld & Grotjohann, 2000). For example, an individual may learn to associate sex with pain or fear due to past, traumatic experiences. In some studies, male and female survivors of childhood sexual abuse and/or female sexual assault survivors were more likely to experience sexual dysfunction (Burri & Spector, 2011; Holla, Jezek, Weiss, Pastor, & Holly, 2012; Laumann et al., 1999; Oberg, Fugl-Meyer, K. S., & Fugl-Meyer, A. R., 2002; Postma, Bicanic, van der Vaart, & Laan, 2013; Reissing, Binik, Khalife, Cohen, & Amsel, 2003; Sanjuan, Langenbucher, & Labouvie, 2009), although other research did not demonstrate the same findings (Dennerstein, Guthrie, & Alford, 2004). A potential mechanism that explains female sexual dysfunction in adult survivors of childhood sexual abuse is diminished functioning in brain areas that register physical sexual sensations, which may be adaptive in childhood but problematic in adulthood (Heim, Mayberg, Mletzko, Nemeroff, & Pruessner, 2013). Interpersonal avoidance or emotional distancing may also play a role in female sexual dysfunction among survivors of childhood sexual abuse (Staples, Rellini, & Roberts, 2012).

From a social cognitive perspective, individuals have a cognitive framework that reflects their perception of themselves as sexual beings, termed *sexual self-schemas*, which guide attention and information-processing in sexual situations (Cyranowski, Aarestad, & Andersen, 1999; Reissing, Laliberte, & Davis, 2005). Individuals also hold *sexual self-efficacy* beliefs, meaning beliefs about their ability to perform certain sexual behaviors in a given situation (Cyranowski, Aarestad, & Andersen, 1999). Negative self-perceptions in terms of sexual schematic representations and low self-efficacy for sexual behaviors interact with particular situations (such as difficulty achieving an erection or vaginal dryness) to result in particular thoughts in the moment (such as "I'm a failure," or "My partner doesn't find me sexually appealing"). These thoughts influence and interact with feelings, behaviors, and physiology, including one's sexual response (Nobre & Pinto-Gouveia, 2009).

For example, research has demonstrated that individuals with erectile disorder, as compared with those without this disorder, are more likely to explain negative sexual events—such as loss of an erection—as the result of something that is wrong with them (internal attribution) and as something that is likely to continue (stable attribution; Scepkowski et al., 2004). Diminished erectile response can also be evoked among sexually functional males by providing them with prior, bogus, negative erectile feedback and an internal, stable attribution of their supposed diminished erectile response (Weisberg, Brown, Wincze, & Barlow, 2001). Similarly, low self-efficacy for control over one's sexual response plays a key role in premature ejaculation and the level of distress caused by premature ejaculation, above and beyond a short duration to ejaculation alone (Patrick, Rowland, & Rothman, 2007).

Not unlike sexual self-schemas, *sexual scripts* generally refer to expectations about sex, including with whom it is acceptable to have sex, the appropriate reasons for sex, and where, when, and how sexual activity can take place (Gagnon, Rosen, & Leiblum, 1982). Sexual scripts can more specifically refer to a particular couple's implicit rules, expectations, and sequences of sexual activity, including the roles each partner takes on during the sexual activity, such as the "initiator," the "resister," or the "passive participant" (Gagnon et al., 1982; Lips, 2006; Matlin, 2000; McCormick, 1987). These scripts can contribute to sexual problems, particularly if they are inflexible (McCormick, 2010).

Research has also highlighted the important role of attention as a mechanism that directly impacts sexual arousal and performance (de Jong, 2009). One can become distracted by one's thoughts about sexual performance, the appearance of one's own body, possible intrusion by others during sex, and many other concerns (Barlow, 1986). Experimental research shows that individuals can manipulate their physiological, sexual arousal by shifting their attentional focus (Beck & Baldwin, 1994; Koukounas & Over, 2001). For example, focusing on sexual fantasies can increase arousal, while focusing on sexually irrelevant thoughts can decrease arousal. Similarly, focusing on enjoyable sexual sensations creates a feedback loop of increasing sexual arousal, while focusing on one's body or sexual performance in a judgmental way creates a feedback loop of decreasing arousal (de Jong, 2009). The term *spectatoring* or the *spectator role* refers to heightened self-focused attention that interferes with arousal due to concomitant thoughts of performance failure and negative

self-evaluation (Masters et al., 1985). In women, cognitive distraction during sex (such as being distracted by negative thoughts about one's physical appearance and worries about pleasing one's partner) is related to lowered consistency of orgasms, sexual esteem, and sexual satisfaction (Dove & Wiederman, 2000). In a laboratory setting, watching an erotic film displayed behind a transparent, reflective surface decreased vaginal arousal in sexually functional women compared with watching the film without the distracting self-reflection (Meston, 2006). In summary, physiological sexual processes cannot be separated from psychosocial factors, including cognitive processes, beliefs about the self, previous sexual experiences, interpersonal relationship issues, and the context of the sexual behavior.

Age, Cultural, and Gender-Related Factors in Sexual Dysfunctions Cultural and religious beliefs, practices, and expectations may also play a role in sexual difficulties, such as views that women should be subordinate to men and should not enjoy sex, the discouragement of body exploration, and the prearranged marriage of young girls. In certain Asian regions, individuals may complain of a sudden fear that the penis (among men) or vulva and nipples (among women) will retreat into the body and potentially be fatal—a fear called *koro* (American Psychiatric Association, 2013). *Dhat* syndrome, which occurs primarily in India, involves fatigue and depression associated with an excessive concern about the loss of semen—considered a fluid of strength and vitality—through means such as nocturnal emissions, masturbation, or urination (Udina, Foulon, Valdes, Bhattacharyya, & Martin-Santos, 2013). Over 80% of Moroccan women experiencing sexual dysfunction reported that their dysfunction was due to sorcery by someone who wished them harm (Kadri et al., 2002). More broadly, individuals and their sexual beliefs may be influenced by cultural values conveyed explicitly or implicitly through the media. For example, greater exposure to sexual objectification of the body through television and magazines is related to greater body self-consciousness during physical intimacy (Aubrey, 2007). Satisfaction with one's body and perceived sexual attractiveness, in turn, predict sexual satisfaction among women (Pujols, Seal, & Meston, 2010).

Age is also an important factor in sexual functioning. Sexual interest or desire generally declines with age for both men and women (Bancroft et al., 2003; Bitzer, Platano, Tschudin, & Alder, 2008; Eplov, Giraldi, Davidsen, Garde, & Kamper-Jorgensen, 2007; Hyde et al., 2012; Laumann et al., 1999; Lewis et al., 2004; Nicolosi et al., 2004), and older age in Western and Eastern samples across the globe is often associated with sexual dysfunction (Hyde et al., 2012; Lewis et al., 2004; Nicolosi et al., 2003; Turkistani, 2004). Research findings are mixed, however, with respect to the specific sexual dysfunction and its relationship with age and satisfaction with sex. For example, some researchers have reported that among women, sexual difficulties actually decrease with age (Kadri et al., 2002; Laumann et al., 1999), although other research contradicts this finding (Lutfey et al., 2009). In cases in which sexual function improves with age, a possible explanation is that older women tend to have the same sexual partner for a longer period of time than do younger women, which may allow for more consistent sexual behavior and orgasm (Laumann et al., 1999), particularly since women do not report masturbating at the same rates as men do (Meston & Ahrold, 2010). In research that shows declining sexual function with age, sexual difficulties have similar prevalence rates among women aged 30–49 (approximately 32–35%), but by age 50, sexual difficulties increase to 53% and increase further to 63% among women aged 60–79 (Lutfey et al., 2009), with vaginal dryness in particular tending to increase with age (Laumann et al., 1999; Lo & Kok, 2013; Nicolosi et al., 2004, 2006). Although some women may experience more pain during sex as they enter menopause, sexual satisfaction was not related to menopause status in one study (Cain et al., 2003), but sexual satisfaction did decrease with older age in another study (Ornat et al., 2013). Additional research shows that while menopausal women reported more sexual difficulties than premenopausal women, the menopausal women were significantly less distressed by these symptoms than their younger counterparts (Berra et al., 2010).

Among men, the ability to maintain and sustain an erection and to fully ejaculate tends to decrease with age (Blanker et al., 2001; Helgason et al., 1996; Laumann et al., 1999; Nicolosi et al., 2003). Hypogonadism (diminished function of the testes) is relatively common as men reach older age, and its symptoms (such as fatigue, diminished strength, and diminished sexual desire) can be difficult to separate from normal aging (Stanworth & Jones, 2008). Since sexual functioning may naturally decline with age for men and women, along with reproductive capacity, some researchers advocate an acceptance of these changes, rather than an emphasis on artificial maintenance of abilities associated with youth (Potts, Grace, Vares, & Gavey, 2006).

Evidence-Based Interventions for Sexual Dysfunctions The use of medication to treat sexual dysfunctions is generally accepted for men, but remains controversial for the treatment of female sexual dysfunctions (Tiefer, 2001). Most researchers recommend addressing relationship concerns for both men and women before considering medication (Hatzichristou et al., 2004). The popularity of medications, however—particularly for erectile dysfunction—has overshadowed both the acceptance of sexual changes that occur with age and psychological explanations and treatments of sexual dysfunction. Patients therefore readily seek medication as a simple solution to their difficulties (Tiefer, 2006), and physicians—many of whom have received minimal training in assessing and treating sexual dysfunctions—are often willing to write a

prescription, rather than discuss psychosocial components of sexual functioning or to schedule an additional appointment with the patient and his/her partner, as indicated (McCarthy & McDonald, 2009; Wiggins, Wood, Granai, & Dizon, 2007). Research endeavors have also moved away from psychotherapeutic approaches to treating sexual dysfunction and instead toward medical approaches, which tend to be better funded (Baucom et al., 1998).

Biological/Pharmacological Interventions for Sexual Dysfunctions Anatomically, sexual arousal in men and women is a similar process overall, involving the nervous and vascular systems, such as the dilation of blood vessels and flow of blood to the genitals (Segraves, 2002). Treatment responses to the same medications, however, tend to be quite different for men and women.

Treatments for men Most medical treatments for men involve maximizing blood flow to the genitals and/or relaxing smooth muscle tissue. Popular treatments for erectile disorder include phosphodiesterase-5 inhibitors, such as sildenafil (Viagra®), tadalafil (Cialis®), and vardenafil (Levitra®), which are nitric oxide donor drugs (see Porst et al., 2013, for a review of medical treatments). These medications have contraindications, however, such as use with particular heart or cardiovascular medications and the conditions for which they are prescribed, especially if these medications are taken at the same time. These contraindicated medications include other nitric oxide donor drugs and alpha-blockers for high blood pressure (Chrysant & Chrysant, 2012). Rather than targeting blood flow to the penis, another strategy is to target areas of the brain believed to be involved in sexual functioning. Apomorphine, a medication that is often prescribed for Parkinson's disease, has been shown to be beneficial to men with erectile dysfunction because of its action on dopamine receptors (Wylie & MacInnes, 2005).

Other, less commonly used treatments include the use of a vacuum constriction device that increases blood flow to the penis, intracavernosal injections (injections of medications into the penis), urethral suppository (depositing medication into the urethra), and surgically implanted prostheses (Archer et al., 2005; Sadeghi-Nejad & Seftel, 2004). Phentolamine, a VIP, may be administered intracavernosally, often in combination with papaverine, to dilate the blood vessels and promote an erection (Porst et al., 2013; see also "Etiology of Sexual Dysfunctions," above). Phentolamine has also been studied as an oral medication, which is likely to be more desirable for patients than injections into the penis (Ugarte & Hurtado-Coll, 2002).

Low testosterone, which can lead to erectile dysfunction, has been successfully treated with subcutaneous (under the skin) injections of testosterone pellets into the back (Cavender & Fairall, 2009) and in separate research with injections of long-acting testosterone undecanoate in men with type 2 diabetes and hypogonadism (Hackett et al., 2013). Simply becoming healthier—such as exercising, losing extra weight, and quitting smoking—can also improve sexual functioning, including erectile function (Archer et al., 2005; Larsen et al., 2007).

The usually undesirable sexual side effect of delayed orgasm from antidepressants and other medications is the desired effect for men with premature ejaculation, who may be effectively treated with these medications, such as SSRIs (Heiman & Meston, 1997; Waldinger, Zwinderman, Schweitzer, & Olivier, 2004). Antidepressants are not necessarily a panacea for premature ejaculation, however, as they can also cause unwanted side effects, such as diminished sexual desire (Barnes & Eardley, 2007). More recently, dapoxetine—an SSRI approved in Europe specifically for treatment of premature ejaculation—has demonstrated success in clinical trials, with minimal side effects (McMahon et al., 2011). Although it may seem counterintuitive, research has also provided some support for treatment of premature ejaculation with sildenafil, partly because it may facilitate additional, consecutive sexual encounters and a therefore longer total sexual experience (Barnes & Eardley, 2007).

Treatments for women Many treatments have targeted hormones that are thought to be etiological factors in female sexual dysfunction, particularly for symptoms of low desire and arousal (Brotto et al., 2010; Clayton & Hamilton, 2010). Androgen therapy—such as treatment with testosterone creams and testosterone transdermal (absorbed through the skin) patches—has shown some success in increasing sexual desire and arousal among postmenopausal women and women who have had their ovaries and uterus removed (Berman et al., 1999; Braunstein et al., 2005; Kingsberg, 2007; Shifren, 2004; Shifren et al., 2000, 2006). More recently, researchers have suggested that women with different etiologies of sexual dysfunction may respond differently to the same medications, including testosterone (Bloemers et al., 2013). In order to enhance the effects of testosterone, different combinations of medications have been used with some success, including sublingual (under the tongue) testosterone with sildenafil (Poels et al., 2013) and testosterone with buspirone (a serotonin receptor agonist) (van Rooij et al., 2013).

While many studies use low-dose, temporary testosterone treatments for female sexual dysfunction, testosterone is not recommended as a long-term treatment at high doses, owing to its masculinizing effects (such as facial hair growth, deepening of one's voice, and acne), and it is not approved by the U.S. Food and Drug Administration (FDA) to treat female sexual dysfunction (Shifren, 2004). Hormone replacement therapy to increase estrogen levels has also been used, although there may be significant health risks associated with these medications, including blood clots, heart attack, stroke, and breast cancer, and findings about its sexual benefits are mixed (Archer et al., 2005; Gonzalez, Viafara, Caba, & Molina, 2004). Some findings suggest that hormone therapies such as tibolone (available in Europe) and a transdermal patch that releases

estradiol combined with norethisterone acetate may be effective in improving sexual function among postmenopausal women, although these medications also include the potential health risks that are associated with hormone therapies (Nijland et al., 2008).

Dehydroepiandrosterone (DHEA), a precursor that is converted by the body into estrogen and testosterone, has also received recent research attention for treatment of female sexual dysfunction in postmenopausal women (Panjari & Davis, 2010). DHEA is available as an over-the-counter supplement, although it is not approved by the FDA as a treatment for sexual dysfunction. Despite initial findings of some benefits of DHEA for sexual function, the most recent, most empirically sound clinical trials have not shown oral DHEA to be effective in treating female sexual dysfunction (Panjari & Davis, 2010), although the findings of vaginally administered DHEA have been more promising (Labrie et al., 2009).

Aside from the emphasis on hormone therapies, dopaminergic drugs, such as bupropion and apomorphine, have shown some benefit in treating female sexual dysfunction (Caruso et al., 2004; Segraves, Clayton, Croft, Wolf, & Warnock, 2004). Similarly, a medication involved in serotonin pathways called flibanserin resulted in improvement in sexual symptoms for premenopausal women with hypoactive sexual desire disorder (DeRogatis et al., 2012).

Given their general success with men, vasoactive drugs, such as sildenafil (taken orally) and alprostadil (applied as a topical cream to the genital area) have been tested among women, although findings are either nonsignificant (Basson, McInnes, Smith, Hodgson, & Koppiker, 2002), mixed, or should be interpreted cautiously because of a substantial placebo effect (Bradford & Meston, 2009; Heiman, 2002; Heiman et al., 2006). In addition, even when these medications do result in the desired physiological response, women's subjective sense of sexual desire tends to remain unchanged. For example, a study of treatment with sildenafil showed improvement in orgasm among women with sexual dysfunction due to SSRIs, although these women did not show improvements in sexual desire, arousal, or pain above placebo effects (Stulberg & Ewigman, 2008). Despite successful outcomes with some medications for some women, there does not seem to be a "magic pill" to treat female sexual dysfunction (Bradford & Meston, 2009; Snabes & Simes, 2009). Some researchers recommend investing more research in psychotherapeutic treatments for women with sexual dysfunctions, given the limited and inconsistent success of psychopharmacological approaches and the more consistently positive outcomes offered by psychotherapeutic approaches (Gunzler & Berner, 2012).

Medical approaches other than medication include surgery to correct existing physiological problems, such as urinary incontinence (Filocamo et al., 2011; Serati, Salvatore, Uccella, Nappi, & Bolis, 2009). Similar to devices created to assist male sexual arousal, there are also female sexual arousal devices, such as the FDA-approved clitoral vacuum device called the EROS Clitoral Therapy Device, which uses a gentle suction to increase blood flow to the clitoris (Billups et al., 2001). One final, simple option is the use of over-the-counter lubricants to assist with vaginal dryness (Clayton & Hamilton, 2010).

Psychosocial Interventions for Sexual Dysfunctions Sexual difficulties often go untreated because they are one of the most challenging problems for people to discuss with a doctor, therapist, or even with a sexual partner. Treatment of sexual dysfunctions involves a learning process; clients must learn not only about sexual functioning in general, but also about their own bodies and the connection between their own psychological and physiological processes. They also must learn about their partner's body and preferences. Without education about sex, individuals' anxiety and misconceptions about their difficulties may exacerbate the problem (Kadri et al., 2002; Turkistani, 2004).

Just as the etiology of sexual dysfunctions can be explained using a biopsychosocial approach, the same, integrative approach applies to the treatment of sexual dysfunctions (Gambescia, Sendak, & Weeks, 2009). Rather than adopting a model of focusing on the "disease" (called a "disease-centered" approach), physicians and clinicians may better serve their patients (or clients) by adopting a *patient-centered approach* (Hatzichristou et al., 2004). This approach involves an empathic understanding of the patient's perspective of the problem, as well as patient education and active inclusion of the patient (and his or her partner, if possible) in exploring treatment options. Treatment providers should obtain a thorough account of past and present psychosocial, romantic relationship, sexual, mental health, and physical health functioning, as well as a physical examination and relevant laboratory tests when indicated (Basson, Wierman, van Lankveld, & Brotto, 2010; Hatzichristou et al., 2004). Further, clinicians should assess other factors that may account for sexual symptoms, such as depression, anxiety, relationship issues, body image issues, substance abuse, or other concerns that should be addressed. This process of data gathering may involve collaboration between multiple specialists in different fields. Throughout assessment and treatment, open, reciprocal communication between the client or patient and treatment provider is paramount for effective treatment of sexual dysfunctions (Hatzichristou et al., 2004).

Couples approaches to therapy Although clients may conceptualize their concerns as simple sexual "mechanics" or sexual frequency, it may be helpful to reframe the couples' concerns and goals in terms of relationship satisfaction, rather than as sexual performance only (McCarthy & McDonald, 2009). The interpersonal nature of sexual dysfunctions calls for treatment that addresses interpersonal communication and a sense of trust and shared involvement in treatment, including shared consideration

of treatment options. Further, labeling the partner with the sexual difficulty as the "dysfunctional" partner discounts the shared contributions to sex and may lead to a self-fulfilling prophecy of sexual inadequacy (Betchen, 2009). Aside from sexual functioning, the health of the couple's overall relationship may be viewed as a foundation for therapeutic success, as this is an important predictive factor in terms of increasing sexual well-being, as demonstrated among women seeking treatment (Stephenson, Rellini, & Meston, 2013). Even when medication is prescribed for a sexual dysfunction, research supports the use of couples counseling in addition to medication for achieving the best outcomes (Aubin, Heiman, Berger, Murallo, & Yung-Wen, 2009).

Patterns of sexual behavior and the couple's thoughts and beliefs about sex may play a major role in their sexual difficulties. Both individuals may have personal, unspoken or unexplored meanings attributed to sex, as well as broader relationship issues that, when addressed, may alleviate sexual symptoms (Barker, 2011). Through education and communication, modifications to sexual scripts can be made to satisfy both partners and allow more flexibility of the couple's sexual roles and sexual activities (Foley, 1994; Gagnon et al., 1982). Further, the dichotomy of "perfect sex" versus "utter failure" should be challenged so that clients can be free to engage in a variety of pleasurable encounters with their partners, without a looming fear of failure. The "Good Enough Sex" model encourages realistic expectations, malleability of sexual scripts, building of sexual and relationship skills, and active partner collaboration, while abandoning the notion of perfect sexual performance (McCarthy & Metz, 2008).

Behavior therapy Sexual problems are also behavioral problems, and research suggests that addressing the specific sexual difficulty is a critical part of therapy, in addition to addressing the couple's relationship in general (Baucom et al., 1998; see also Gambescia & Weeks, 2006, for a review of behavioral therapy techniques). For women, effective behavioral techniques include learning about and practicing masturbation for treatment of orgasmic disorder (LoPiccolo & Lobitz, 1972), as well as progressive dilation using fingers or plastic dilators for *vaginismus*, a condition involving vaginal contraction that makes penetration difficult or impossible (Masters & Johnson, 1970). In some cases, vaginal muscle biofeedback for dyspareunia and vaginismus can be helpful (Basson et al., 2010; Lechtenberg & Ohl, 1994). Women who experience pain in their vaginal opening when touched (termed *provoked vestibulodynia*) were generally successfully treated with a combination of psychosexual education, psychological counseling, and progressive steps completed at home involving touching the sensitive area, pelvic floor contraction and relaxation, and penetration (Backman, Widenbrant, Bohm-Starke, & Dahlof, 2008; see also Stinson, 2009, for a review of treatments for female sexual dysfunction). Pelvic floor exercises have also been used to successfully treat coital incontinence, which is linked to sexual aversion, lack of desire/arousal, dyspareunia, and anorgasmia (Salonia et al., 2004; see also Serati et al., 2009 and Moore, 2010, for reviews). When prescribing homework assignments, however, clinicians must be flexible. Even well-intentioned homework assignments, such as masturbation, may work for some clients and not for others, and failure to agree to or to complete these homework assignments should not be interpreted as resistance (Barker, 2011; Ellison, 1984). Antiquated notions about "correct" and "incorrect" ways to achieve orgasm, particularly for women, can also be counterproductive to therapy (Ellison, 1984).

Sexual difficulties due to distraction or negative self-focused attention (e.g., spectatoring) can be treated through *sensate focus*, which involves removing the psychological pressure involved in sexual performance by instead engaging in relaxed, sensual touch without the goal of intercourse or orgasm (Masters & Johnson, 1970; Sarwer & Durlak, 1997). When spectatoring involves concern about one's physical appearance, however, massage techniques may need to be altered so that there is dim lighting or other strategies to alleviate self-conscious anxiety (Wiederman, 2001). Another couples technique is the treatment of premature ejaculation through starting and stopping sexual activity to allow the man to better control and delay ejaculation, as well as through the use of the *squeeze technique*, which involves repeatedly squeezing the penis for a few seconds either at the frenulum (near the ridge at the top of the penis) or at the base of the penis to deter ejaculation until desired (Masters & Johnson, 1970; Masters et al., 1985).

Individuals who have come to associate sex with trauma or anxiety can unlearn this association through gradual exposure to sexual activity at home with their partner, which is an extension of Wolpe's *systematic desensitization* (Janata & Kingsberg, 2005; Wolpe, 1958). The individual learns relaxation techniques and may start with sexual imagery before moving to *in vivo* sexual exercises. He or she should have a sense of control over what occurs (unlike past traumatic experiences, if that is the case), and the exposure should begin with less threatening situations, such as non-sexual touching, and progress to increasingly more potentially anxiety-provoking stimuli, as he or she feels comfortable. Systematic desensitization has generally shown positive results for erectile disorder and sexual anxiety, although women with orgasmic difficulties seem to be better treated by masturbation exercises (Heiman & Meston, 1997).

Aside from sexual behavioral techniques, much of the outcome of therapy rests on the couple's ability to communicate clearly, openly, and specifically. Miscommunication about sex is common among couples due to the use of confusing euphemisms and reluctance to be specific about anatomy and behavior (Gambescia et al., 2009). Couples need to talk to each other about what they find pleasurable; verbal as well as nonverbal techniques may be used to

guide one's partner during sexual activity (Lechtenberg & Ohl, 1994). Providing feedback to one's partner is also likely to increase his or her self-efficacy for sexual activity (Reissing, Laliberte, & Davis, 2005).

Cognitive-behavioral therapy Cognitive-behavioral therapy (CBT) has also received empirical support as an effective treatment for sexual dysfunctions (McCabe, 2001; see ter Kuile, Both, & van Lankveld, 2010, for a review). The cognitive-behavioral approach to sexual dysfunction treatment involves facilitating the couple's communication, practicing sexual skills, and identifying and modifying maladaptive beliefs about sex that may be exacerbating or even causing the difficulty. For example, men who believe that a man must be ready for sex at any moment and that failure to sustain an erection is indicative of personal failure, or that impotence is an affront to a man's masculinity, will likely experience more debilitating anxiety and self-doubt when faced with loss of an erection, compared to men who do not add special significance to their body's functioning (Nobre & Pinto-Gouveia, 2006). Unrealistic expectations can also impede women's sexual functioning, such as the belief that women should be able to have an orgasm through intercourse during every sexual encounter, as well as beliefs that virtuous women, mothers, and older women should not be sexual (Nobre & Pinto-Gouveia, 2006). Dysfunctional automatic thoughts, such as thoughts of sexual performance failure or negative thoughts about one's partner lead to decreases in arousal and sexual function for men and women (Carvalho & Nobre, 2010; Nobre & Pinto-Gouveia, 2006). Cognitive-behavioral therapists can help undermine these dysfunctional automatic thoughts and any related maladaptive beliefs through psychoeducation, cognitive restructuring, and homework assignments involving behavioral techniques, such as sensate focus (McCabe, 2001). Similarly, individuals may benefit from learning *mindfulness* techniques, which assist individuals in attending to and enjoying their moment-to-moment sexual experience while also learning to relax (Brotto, Basson, & Luria, 2008).

Paraphilic Disorders and Hypersexual Behavior

Defining and Describing Paraphilic Disorders Paraphilia, literally meaning "lover" (*phile*) of that which is "beyond" or "abnormal" (*para*), can include a wide range of sexual activities, objects, and situations (*Merriam-Webster's Collegiate Dictionary*, 2001). According to DSM-5, a *paraphilia* is "any intense and persistent sexual interest other than sexual interest in genital stimulation or preparatory fondling with phenotypically normal, physically mature, consenting human partners" (American Psychiatric Association, 2013, p. 685). A paraphilia may or may not meet the criteria for a clinical disorder, however. A *paraphilic disorder* is defined as "a paraphilia that is currently causing distress or impairment to the individual or a paraphilia whose satisfaction has entailed personal harm, or risk of harm, to others" (pp. 685–6). In other words, paraphilic disorders are not simply normal variations in sexual pursuits; rather, they are troubling or problematic to the individual or they include victimizing others, or both. As with most psychological phenomena, behaviors involved in the paraphilias and paraphilic disorders range from mild—such as men wearing women's underwear, consensual blindfolding, and role playing—to severe—such as sexually abusing children or sadistic beating or cutting during sex, and clinicians must determine the level of distress or harm that warrants a diagnosis.

One addition to DSM-5 is the classification of paraphilic disorders into *anomalous activity preferences*, which include *courtship disorders* (meaning abnormal activities that are reminiscent of courtship, as in exhibitionistic disorder) and *algolagnic disorders*, which pertain to sexual gratification involving pain or torment to oneself or others. Second to anomalous activity preferences are *anomalous target preferences*, which include pedophilic (targeting children), fetishistic (requiring non-sexual objects as part of sex), and transvestic disorders (sexual arousal through cross-dressing, typically among heterosexual men). Although the criteria for all of the paraphilic disorders state that symptoms should occur over a period of at least 6 months for diagnosis, the DSM also notes that individuals who clearly meet the other criteria could be diagnosed if the symptoms seem enduring and are present for less than 6 months (American Psychiatric Association, 2013).

Unlike the vast majority of disorders in the DSM, some of the paraphilic disorders—specifically those involving nonconsenting partners, including pedophilic, voyeuristic, exhibitionistic, frotteuristic, and sexual sadism disorders—do not require the criterion of significant personal distress or impairment in order to be diagnosed, as long as the person has acted on his/her sexual urges with a nonconsenting victim (American Psychiatric Association, 2013). If the individual has not acted on urges to victimize others but experiences clinically significant distress or functional impairment due to these urges, a diagnosis can also be given. The remaining (non-victimizing) paraphilias do include the required significant distress or impairment criterion, to avoid pathologizing consensual, non-problematic variations in sexual preferences (American Psychiatric Association, 2013). In addition to the primary categories of paraphilias, the DSM also includes an *other specified paraphilic disorder*, which encompasses a range of deviant behaviors that the clinician can specify, such as making obscene phone calls (*scatologia*), having sex with animals or corpses, and the sexual use of urine, feces, and enemas. A final category, *unspecified paraphilic disorder*, involves situations in which the clinician is unable to or decides not to indicate the reason for failure to meet the diagnostic criteria for one of the paraphilic disorders (American Psychiatric Association, 2013).

According to the DSM-5, the courtship disorders are:

- Voyeuristic disorder: Recurrent and intense sexual arousal from observing an unsuspected person who

is naked, in the process of disrobing, or engaging in sexual activity, as manifested by fantasies, urges, or behaviors.
- Exhibitionistic disorder: Recurrent and intense sexual arousal from the exposure of one's genitals to an unsuspecting person, as manifested by fantasies, urges, or behaviors.
- Frotteuristic disorder: Recurrent and intense sexual arousal from touching or rubbing against a nonconsenting person, as manifested by fantasies, urges, or behaviors.

The algolagnic disorders are:

- Sexual masochism disorder: Recurrent and intense sexual arousal from the act of being humiliated, beaten, bound, or otherwise made to suffer, as manifested by fantasies, urges, or behaviors.
- Sexual sadism disorder: Recurrent and intense sexual arousal from the physical or psychological suffering of another person, as manifested by fantasies, urges, or behaviors.
- The disorders involving anomalous target preferences are:

- Pedophilic disorder: Recurrent, intense sexually arousing fantasies, sexual urges, or behaviors involving sexual activity with a prepubescent child or children (generally age 13 years or younger) . . . [and] the individual is at least age 16 years and at least 5 years older than the child or children.
- Fetishistic disorder: Recurrent and intense sexual arousal from either the use of nonliving objects or a highly specific focus on nongenital body part(s), as manifested by fantasies, urges, or behaviors . . . The fetish objects are not limited to articles of clothing used in cross-dressing (as in transvestic disorder) or devices specifically designed for the purpose of tactile genital stimulation (e.g., vibrator).
- Transvestic disorder: Recurrent and intense sexual arousal from cross-dressing, as manifested by fantasies, urges, or behaviors.

Specific Modifications From the DSM-IV-TR to the DSM-5 for Paraphilic Disorders The major revision to the paraphilic disorders is the decoupling of this section with sexual dysfunctions and gender dysphoria, such that paraphilic disorders are now housed in a unique chapter. As noted previously, the DSM now makes a clear distinction between paraphilic interests and paraphilic disorders in order to avoid pathologizing sexual preferences that are not problematic, and each disorder is now termed "disorder" (e.g., sexual sadism is now sexual sadism disorder) in order to avoid confusion and in keeping with the nomenclature of the DSM (Langstrom, 2010). There are ongoing criticisms of the perceived lack of clarity of paraphilic disorders in the DSM, such as the vague language of "recurrent and intense" when describing the symptoms (see Balon, 2013, for a review of diagnostic issues). The proposal to specify the number of victims required for diagnosis as a more concrete operational definition (for disorders involving nonconsent), however, was met with great opposition, as some argue that even one victim certainly justifies a diagnosis, and the client may not be forthcoming about the actual number of victims anyway (Krueger & Kaplan, 2012; O'Donohue, 2010). Nevertheless, the "Diagnostic Features" section of DSM-5 for voyeuristic disorder, exhibitionistic disorder, frotteuristic disorder, and sexual sadism disorder (but not pedophilic disorder) now specifies that "recurrent" generally refers to three or more victims or one or two victims on several different occasions, with the caveat that multiple victims is not actually a requirement for diagnosis.

The major categories and diagnostic criteria of paraphilic disorders are relatively unchanged, however, with the exception of modifications to the *specifiers*. For example, transvestic disorder no longer has the specifier with *gender dysphoria*, given that cross-dressing due to discomfort with one's gender is a separate disorder (see Chapter 24 in this volume). The specifiers *with fetishism* and *with autogynephilia* (being sexually aroused by viewing or imagining oneself as a woman) have been added to transvestic disorder. Fetishistic disorder now includes *partialism*, which is the sexual focus on a body part, such as feet. Sexual masochistic disorder now includes the specifier *with asphyxiophilia*, meaning pleasure derived from suffocation, which involves choking oneself or depriving oneself of oxygen via a noose, plastic bag, or other means, usually while masturbating (Hucker, 2011; Uva, 1995). This behavior can lead to accidental suicide. Two specifiers that have been added to all of the paraphilic disorders except for pedophilic disorder are *in a controlled environment* (for individuals living in a restricted setting, such as a hospital, that does not allow for the behavior to occur), and *in full remission* (5 years or more without distress or impairment for the non-victimizing disorders and lack of victimization for disorders involving nonconsenting partners).

Epidemiology of Paraphilic Disorders Accurate estimates of the prevalence of paraphilic disorders are difficult to obtain, as some of these behaviors are illegal, many are viewed negatively by society, and they are rarely seen in clinical settings as the predominant reason for treatment (Bhugra, Popelyuk, & McMullen, 2010). Overall, paraphilic disorders seem to be predominantly diagnosed in men, and although paraphilic interests without distress (including fantasies or behaviors) are quite common (62% in one male sample), distressing (i.e., more diagnosable) paraphilias occurred in only 1–2% of German men (Ahlers et al., 2011). The following rates of paraphilic interests and behaviors (including non-distressing fantasies) among men were reported, in descending order: voyeurism (39%), fetishism (36%), sadism (25%), masochism (19%), frotteurism (15%), pedophilia (10%), transvestism

(7%), and exhibitionism (4%) (Ahlers et al., 2011). Acts that were actually performed with a victim included: voyeurism (18%), sadism (16%), frotteurism (7%), exhibitionism (2%), and pedophilia (4%). In pedophilia, female victimization is more commonly reported than is male victimization, although sex offenders who target boys tend to have more victims than those who target girls (see Cohen & Galynker, 2002, for a review). The vast majority of men who reported carrying out consensual or non-victimizing paraphilic behaviors were generally not distressed by their behavior, with only one in 367 men reporting distress tied to transvestism or masochism, and an absence of distress among all of the men for the other consensual behaviors (Ahlers et al., 2011).

In a large Brazilian sample of men and women, paraphilic behaviors included: fetishism (13%), voyeurism (13%), incest (11%), exhibitionism (9%), sadomasochism (9%), and sex involving animals (3%), with men reporting these behaviors more than women (Oliveira & Abdo, 2010). Lower estimates were found in large-scale Swedish research, which reported lifetime rates of exhibitionistic behavior at 3%, voyeuristic behavior at about 8% (Langstrom & Seto, 2006), and transvestic behavior at approximately 3% among 18–60 year-olds (Langstrom & Zucker, 2005). One-year incidence rates for sexual behaviors involving bondage, discipline, sadomasochism, or dominance and submission were approximately 2%, with higher rates for men than for women (Richters, de Visser, Rissel, Grulich, & Smith, 2008).

Etiology of Paraphilic Disorders

Biological/Genetic Factors in Paraphilic Disorders The neurotransmitters that are believed to be involved in impulse control, mood, and sexual functioning, namely the monoamines (including norepinephrine, dopamine, and serotonin) are implicated in paraphilic disorders and hypersexual behavior (Blum et al., 2012; Bradford, 2001; Kafka, 2003b). Medications for Parkinson's disease and other conditions—particularly dopamine agonists—have been shown to induce paraphilias and related behaviors in case reports, including transvestism, frotteurism and pornography addiction, symptoms which tend to subside after medication modification (Cannas et al., 2006; Shapiro, Chang, Munson, Okun, & Fernandez, 2006; Tajima-Pozo, Bardudo, Aguilar-Shea, & Papanti, 2011). Multiple sclerosis, temporal lobe epilepsy, as well as brain lesions and brain tumors in various areas have also been associated with paraphilic disorders and hypersexual behavior (Bradford, 2001). The frontal lobe (which is involved in impulse control) and the temporal lobe (believed to be involved in sexual arousal) have both been implicated in these sexual disorders (Bradford, 2001; Cohen & Galynker, 2002; Fedoroff, 2008; Jordan, Fromberger, Stolpmann, & Muller, 2011). Fronto-temporal pathways have also been linked to criminal activity in general, however, and may not be specific to paraphilic disorders or sexual offending (Joyal, Black, & Dassylva, 2007). In addition, levels of sex hormones, particularly androgens, are likely to play a role in sexual desire and activity (Bradford, 2001; Jordan et al., 2011), which has been used as one explanation as to why paraphilic disorders are more common among males than among females (Arndt, 1991; Fagan, Wise, Schmidt, & Berlin, 2002; Langstrom & Zucker, 2005).

Psychosocial and Environmental Factors in Paraphilic Disorders Classical or Pavlovian conditioning has been a widely used model to explain the development of paraphilic disorders (see Akins, 2004, for a review). The development of a sexual fetish, for example, might be traced to a prior sexual experience that involved simultaneous arousal and presence of the object. Once the association is acquired, the individual touches or smells the object (most commonly underwear, shoes, stockings, belts, or perfume) in order to experience or enhance arousal, and repeated pairings of the object with masturbation and orgasm strengthen the association. In addition to the development of sexual fetishes, the pairing of sexual arousal with a particular object, situation, or type of person is likely to be involved in the development of other paraphilias.

Operant conditioning may also reinforce the behavior, as people who expose themselves, make obscene phone calls, or rub up against unsuspecting victims may be rewarded by the strong reaction of the victim (such as disgust, anger, and/or surprise) or by the satisfaction of the act itself and the evasion of punishment if they are not caught. The types of sexual stimuli that elicit arousal are also likely to be learned to some extent through social and cultural norms. For example, shoes are more likely to be a fetish object than are tables. Important models, such as parents, may also teach the deviant behavior through varying degrees of subtle or blatant modeling of the behavior (Stoller, 1985).

In addition to behavioral conditioning and modeling, many other factors, including one's psychosocial history and comorbid problems and disorders are likely to play a role in paraphilic disorders. For example, a history of sexual abuse is prevalent among individuals who develop pedophilia, masochism, and voyeurism (Abrams & Stefan, 2012; Cohen & Galynker, 2002; Langstrom & Seto, 2006; Marshall, Serran & Cortoni, 2000; Oliveira & Abdo, 2010).

Substance abuse is often a comorbid condition, and being under the influence of a substance may lower sexual inhibitions (Kafka & Hennen, 2002; Raymond, Coleman, Ohlerking, Christenson, & Miner, 1999). Individuals with paraphilic disorders or a history of sexual offenses are also more likely to have substance-abusing parents, compared to the general population (Langevin, R., Langevin, M., Curnoe, & Bain, 2006). Childhood narratives among child sex offenders tend to involve early sexual experiences (ranging from relatively enjoyable to traumatizing), families filled with conflict, neglect, rejection, abuse, and/or abandonment, and social difficulties and bullying at

school, although a minority of sex offenders reported a relatively normal childhood (Thomas et al., 2013).

Paraphilic disorders have been associated with a variety of additional difficulties, including a higher rate of psychological difficulties and dissatisfaction with life (Langstrom & Seto, 2006), as well as mood disorders, social anxiety, attention-deficit/hyperactivity disorder, and impulse control difficulties (Kafka & Hennen, 2002). Sexual offenders' impaired social skills and social anxiety may contribute to their disordered behavior (Hoyer, Kunst, & Schmidt, 2001). For example, they may target vulnerable individuals, such as children, perhaps partly due to their difficulty forming relationships with adults, lack of empathy, difficulty recognizing social cues, and distorted thinking about their victims (Blake & Gannon, 2008; Emmers-Sommer et al., 2004). Individuals who have ever engaged in exhibitionism, voyeurism, or transvestism tend to report being easily sexually aroused in general and engaging in high rates of masturbation (Langstrom & Seto, 2006; Langstrom & Zucker, 2005). Therefore, one possibility is that these individuals have a high potential for arousal to a wide variety of situations, fantasies, or objects, thereby increasing the comorbidity of symptoms.

Age, Cultural, and Gender-Related Factors in Paraphilic Disorders Most paraphilic disorders begin during adolescence, when sexual interests and preferences are developing, although certain childhood experiences may predispose individuals to developing these disorders (Abel, Osborn, & Twigg, 1993). For example, retrospective research links boyhood experiences involving nudity—such as experiences in the bathtub with a much older sister who viewed his genitals or being nude often in front of one's mother—with an increased likelihood of developing symptoms of exhibitionistic disorder (Swindell et al., 2011).

Gender differences in paraphilic disorders have been explained by socially prescribed gender roles, in addition to biological factors. For example, cross-dressing among men is more likely to be noticed and pathologized than is cross-dressing among women; further, cases of women who derive sexual pleasure from wearing men's clothing may be underreported or may not be as interpersonally distressing (Devor, 1996). Exposure to media and cultural norms that sexualize women and their clothing and undergarments may also play a role in the predominance of men versus women with paraphilias (Bhugra et al., 2010).

In addition, sexual scripts in many cultures specify that (in heterosexual relationships) men should be the pursuers, and women should be pursued. Taken a step further, some men may sexually harass women or violate their rights, as in scatologia and frotteuristic, sexual sadism, voyeuristic, and exhibitionistic disorders, not just for sexual gratification, but in an attempt to exert dominance and control. Paraphilic behaviors among women, however, can have different motivations and outcomes than the same behaviors among men, as females tend to be more sexualized than males. For example, female exhibitionists report exposing themselves to men for positive attention and to experience the excitement of the situation (Hugh-Jones, Gough, & Littlewood, 2005).

Evidence-Based Interventions for Paraphilic Disorders

Individuals with paraphilias are often reluctant to seek treatment, either because: 1) they do not believe their behavior is problematic; 2) they are embarrassed about their unusual sexual activities; and/or 3) their sexual activities are illegal. Accordingly, much of the research on the treatment of paraphilias includes samples of individuals who have been arrested and found guilty of sexual crimes and are mandated to receive therapy. These individuals are usually highly motivated to report that the treatment has worked, but may not necessarily be motivated to change. Individuals whose paraphilic behaviors have caused relationship discord—such as transvestism with a disapproving partner—may be more motivated to change their behavior to preserve their relationship. Most of the research on treatment of paraphilic disorders, however, involves treatment of sexual offenders.

For paraphilic disorders that involve the victimization of others, treatment providers must be appropriately trained to provide support while not condoning behavior that is harmful to others. At the same time, authoritarian confrontation without empathy may lead to therapeutic resistance (Patel, Lambie, & Glover, 2008). Instead of using confrontational attacks, therapists may make more progress by helping the client evaluate advantages and disadvantages of their behavior. This can be done by using language that implies change, by encouraging acceptance and exploration of ambivalence to change, by pointing out discrepancies between the client's values and behaviors, and by building the client's self-efficacy for change in an empathic environment (Patel et al., 2008).

In addition to the issue of confrontation for sexual offenders is the opposite side of the spectrum, namely pathologizing normal variations in sexual functioning. Clients' partners may view normal sexual behaviors as deviant, seeking treatment for the "problematic" partner, or clients themselves may present for therapy with excessive guilt related to normal sexual behaviors (Cantor et al., 2013). Clinicians must negotiate these situations with psychoeducation, couples counseling, and other appropriate therapeutic strategies, rather than colluding with the desire to ascribe unnecessary diagnoses (Cantor et al., 2013).

Biological/Pharmacological Interventions for Paraphilic Disorders Researchers have reported some degree of success in treating paraphilic disorders with antidepressants that influence serotonergic pathways, such as SSRIs, as well as medications that reduce the effects of androgens, which are hormones believed to influence sexual desire and activity (see Thibaut et al., 2010, for a review of

treatment options). Antidepressants have resulted in mixed findings for treatment of paraphilic disorders and slightly more favorable findings for difficulties that include sexual compulsions, although these studies tend to include small sample sizes and inadequate methodological controls (Baratta, Javelot, Morali, Halleguen, & Weiner, 2012). The addition of a psychostimulant (methylphenidate slow release) to an existing SSRI regimen was beneficial to a small sample of men with symptoms of paraphilias and/or sexual compulsions, underscoring the connection between impulse control difficulties and paraphilias (Kafka & Hennen, 2000).

Antiandrogens (also called *androgen deprivation therapy*) are medications that reduce the effects of androgens by competing with them at their receptor sites (Bradford, 2001). One type of antiandrogen, cyproterone acetate, blocks androgen receptors and affects hormones that influence the release of testosterone. Medroxyprogesterone acetate (MPA; Depo-Provera®) also results in a decrease in testosterone levels, although through a different mechanism of action. These medications have demonstrated success in treating paraphilias, particularly those involving nonconsenting partners, although they can have serious adverse effects (Bradford, 2001; Maletzky, Tolan, & McFarland, 2006; Saleh & Guidry, 2003). For example, antiandrogens may delay puberty in adolescent boys and may negatively impact bone density, which should be checked yearly (Thibaut et al., 2010). Owing to serious potential adverse effects, such as breast cancer, cardiovascular, and blood clotting disorders and its limited effectiveness, treatment with estrogens is generally not recommended. Similarly, treatment with antipsychotic medications for patients without psychotic symptoms has not been shown to be effective and has resulted in serious side effects, particularly when administering the "conventional" (older class of) antipsychotic drugs (Thibaut et al., 2010).

Newer medications with fewer adverse effects have been tested, such as gonadotropin-releasing hormone agonists (GnRH agonists) and luteinizing hormone-releasing hormone agonists (LHRH agonists; Greenfield, 2006). These agonists reduce the effect of the hormones that signal the release of androgens by paradoxically stimulating the hypothalamus to release more hormones, until the downregulation of receptors occurs. In many patients, an initial boost in sexual desire is followed by a steady decrease in desire as feedback mechanisms adjust to this over-stimulation of the hypothalamus. For example, a GnRH agonist called triptorelin and an LHRH agonist called leuprolide acetate have shown promising results in terms of reducing paraphilic urges and behaviors in men (Rosler & Witztum, 1998; Saleh & Guidry, 2003). The term *chemical castration* has been used to describe medications that reduce the effects of androgens, particularly the drug MPA; however, these medications do not necessarily cause impotence and their effects are often reversible, depending on the type of medication used (Bradford, 2001). Mandatory treatment with these medications, however, has been an issue of serious debate (Miller, 1998). Surgical castration (removal of the testes) is still performed in some regions, although it is likely to be discontinued as a treatment method in the future due to human rights considerations (Balon, 2013).

Psychosocial Interventions for Paraphilic Disorders A predominant model in the treatment of sexual offenders (and criminal offenders in general) is the risk–need–responsivity (RNR) treatment model (Andrews, Bonta, & Hoge, 1990; see also Andrews & Bonta, 2010, for a review). Assessment of risk of reoffending is the first step, with higher risk individuals assigned to more intensive treatment than those who are considered lower risk. The second component of the model is to identify and match the *criminogenic* needs of the individual (meaning behavioral problems or characteristics that are related to offending) to appropriate treatment, to address targeted difficulties or behaviors, such as social skills training. Cognitive-behavioral and social cognitive approaches are often used in this model. The third component, responsivity, involves tailoring the treatment to the skills, background, motivation, and abilities of the individual so that the treatment modality will likely have the greatest impact. Treatment providers consider whether individual or group therapy (or both) would be most helpful, for example, or whether more or less structure in treatment would be useful for a given individual. Research has generally shown favorable outcomes of the RNR approach in terms of reducing recidivism (Andrews & Bonta, 2010). This model has been criticized, however, for potentially leading treatment providers to view the person from a deficit perspective (as a series of risk factors and areas of insufficient development), rather than promoting the view the offender as a whole person, with personal strengths and positive life goals (Ward & Marshall, 2004; Whitehead, Ward, & Collie, 2007).

An alternative model, termed the good lives model (GLM) of rehabilitation, has been developed more recently not only to identify risk factors for sexual offenders' relapse, but also to emphasize the individual's personal goals and strengths, and to use these factors in treatment to motivate positive change (Ward & Marshall, 2004; Willis, Yates, Gannon, & Ward, 2013). By identifying the fundamental human needs (termed *primary goods*) that are being met through the deviant behaviors (such as stress reduction or intimacy needs), replacement strategies for meeting these needs can be developed. This model's emphasis on self-regulation, sociocultural and social learning considerations, and positive mastery experiences makes it consistent with the social cognitive approach to psychotherapy. Additionally, research shows that when a sexual offender is released from prison, community reintegration planning is important so that these individuals can develop healthy, stable lives without reoffending (Willis & Grace, 2008). Research supports the effectiveness of the

GLM approach (Whitehead et al., 2007), although its overall outcomes are similar to the RNR approach (Harkins, Flak, Beech, & Woodhams, 2012).

Both the GLM and RNR models apply CBT techniques as part of their treatment, and CBT has been used to treat paraphilic disorders in general, including those involving sexual offenses (see Schaffer, Jeglic, Moster, & Wnuk, 2010, for a review). Behavioral techniques may include extinguishing the arousal response that is triggered by the conditioned stimulus, such as a pair of shoes or a picture of a child (see Beech & Harkins, 2012, for a review). *Aversion therapy* or *aversive conditioning* includes the presentation of an aversive stimulus with an imagined or real presentation of the paraphilic stimulus. For example, the individual may become aroused by a pedophilic scenario and is then presented with a noxious odor or receives an electric shock or nausea-inducing substance (Marshall, 1974). Covert sensitization involves creating a vivid, imagined scene involving the deviant behavior that results in a negative experience or consequence, such as embarrassment or apprehension (Akins, 2004). Reconditioning the individual to become aroused to acceptable sexual stimuli, such as consenting adults, may also be used (Johnston, Hudson, & Marshall, 1992). Treatment may additionally include techniques such as role playing to facilitate improved anger management and emotional regulation, to increase empathy for others, to enhance communication skills, and to improve identification and interpretation of social cues. Relapse prevention involves assisting the client in identifying patterns of behavior, as well as cues and situations that may increase the risk for reoffending (Emmers-Sommer et al., 2004; Saleh & Guidry, 2003).

Cognitive strategies are also an important component of CBT for individuals with paraphilic disorders (Schaffer et al., 2010). Psychoeducation is used to help clients better understand their problem, how this problem has developed over time, and how to develop strategies to change their thoughts in order to control their behavior. Clinicians also refute sexual myths and schemas that the client may hold, such as the concept that "no means yes," that all women are manipulative, that children should be taught about sex through experience, or that a victim who is frozen with fear is providing consent. Clinicians may also facilitate imagery that involves a new sequence of events, such as a scenario involving control of the unwanted impulses (Akins, 2004).

Additional approaches include mindfulness techniques, solution-focused therapy, and group therapy. Mindfulness techniques have also been shown to be effective in helping sex offenders to self-regulate negative emotions, which often precede offenses. Examples of these strategies include meditation and breathing techniques, nonjudgmental awareness of thoughts and feelings, attention to the present moment, and distraction from anger or aggression through focus on a non-sexual body part, such as the soles of one's feet (Gillespie, Mitchell, Fisher, & Beech, 2012). Another approach is solution-focused therapy, a goal-oriented, collaborative treatment method that focuses on the times when the problem did not occur and builds on successful attempts to change and the development of appropriate social skills (Guterman, Martin, & Rudes, 2011). In addition to individual therapy, group therapy is also often used, particularly for sexual offenders. The group format allows for engagement in the therapeutic process, social skills building, facilitation of empathy and a sense of connection with the group, and confrontation to address defensiveness and denial, in a non-threatening environment of individuals who understand each others' experiences well (Jennings & Sawyer, 2003; Levenson & Macgowan, 2004).

In sum, research has generally demonstrated that CBT for sexual offenders is a preferred psychotherapeutic treatment method (Losel & Schmucker, 2005; O'Reilly, Carr, Murphy, & Cotter, 2010), although long-term follow up research indicates that pedophiliacs who target boys and rapists have worse recidivism rates compared to other sexual offenders (Maletzky & Steinhauser, 2002). A combination of psychotherapy and medical treatment may also be effective, with a minimum of 3–5 years of treatment recommended, particularly for severe cases (Thibaut et al., 2010).

Hypersexual Behavior Although DSM-5 includes a diagnostic category for individuals who do not want to have sex often enough, wanting to have sex too often is not included in DSM-5 as a disorder. *Excessive sexual drive* is included, however, in the 10th edition of the *International Classifications of Diseases* (ICD-10), developed by the World Health Organization. Many researchers have suggested that hypersexuality be included in the DSM, to reflect a host of maladaptive sexual behaviors that interfere with healthy relationships and healthy daily functioning (Bradford, 2001; Kafka, 2010; Reid, 2013). Excessive sexual drive and behavior have been given a variety of names with varied meanings, including *sexual addiction*, *sexual preoccupation*, *sexual impulsivity disorder*, *compulsive sexual behavior*, *out of control sexual behavior*, and *hypersexual desire disorder* or simply *hypersexual disorder* (Marshall, L. E., Marshall, W. L. Moulden, & Serran, 2008; Reid, 2013; Samenow, 2010; Skegg, Nada-Raja, Dickson, & Paul, 2010).

Although I prefer the term *hypersexual disorder*, the terminology used in the current chapter varies to reflect the various labels used in specific research. Despite the fact that hypersexual disorder is not clearly defined and encompasses a wide range of experiences, most definitions include characteristics such as the lack of control over sexual thoughts or activities, the repetitive, time-consuming nature of these thoughts and activities, acts of high-risk behaviors, and the experience of personal distress, impairment, relationship damage, or other negative consequences that do not deter the behavior (Black, Kehrberg, Flumerfelt, & Schlosser, 1997; Kafka, 2003a; Wiederman, 2004). The term *sexual addiction* is

also widely used, but may be perceived as using a disease model, as in drug addiction, which is controversial. Nevertheless, when compared to existing disorders in the DSM, hypersexual disorder does share similar criteria with the *substance-related and addictive disorders* (e.g., *gambling disorder*, which is included in the DSM), in which the individual: engages in the behavior when feeling anxious or depressed; may use the behavior to experience a sense of numbness or disconnect from reality; experiences irritability or withdrawal-like symptoms when reducing the behavior; and the behavior causes negative outcomes and/or distress, but there is repeated failure to minimize or cease to engage in the behavior after repeated attempts (Bancroft & Vukadinovik, 2004; Garcia & Thibaut, 2010; Giugliano, 2009). Similarly, DSM-5 categories of *pyromania* (setting things on fire) and *kleptomania* (recurrent stealing) share the impulsive qualities of hypersexual disorder.

Like sexual addiction, the term *compulsive sexual behavior* may also be problematic in some ways. Excessive sexual behavior may include unwanted, intrusive thoughts, as in obsessions, and an almost compulsory need to engage in ritualized sexual behaviors, as in compulsions. Research has thus demonstrated a moderate connection between obsessive-compulsive disorder (OCD) symptoms and sex addiction (Egan & Parmar, 2013). Given that the sexual behavior is likely to be pleasurable and may lead to guilt, however, which is not necessarily found in OCD, the repetitive compulsions performed to mitigate the obsessions found in OCD do not necessarily fit with hypersexual behavior. Hence, a consensus on the diagnostic label for this difficulty has yet to be reached, although *hypersexual disorder* seems to be a strong candidate, given that it does not have an implied etiology, it is an objective descriptor, and it suggests a continuum of behavior from hyposexual to hypersexual, similar to hypoglycemia and hyperglycemia in medical literature (Kafka, 2010; Samenow, 2010).

The problematic behavior of hypersexuality may be paraphilic in nature, or the behavior may be common sexual practice that is excessive and that causes distress and/or negative outcomes (Kafka, 1997b). The sexual activity or preoccupations may include excessively high rates of masturbation, excessive use of pornography or phone sex, excessive sex with multiple partners, with prostitutes, with nonconsenting partners, or with one consenting partner that results in relationship difficulties (Kafka, 2003a; Raymond, Coleman, & Miner, 2003). Research suggests that hypersexuality is not qualitatively distinct from other sexual urges and behaviors, however; like most disorders, hypersexuality is at an extreme end of a continuum of experiences and behaviors (Walters, Knight, & Langstrom, 2011).

Operational definitions of hypersexual disorder have included Kinsey's (1948, as cited in Kafka, 1997b) recommendation of at least seven orgasms a week, through any and all means, for several consecutive months, termed total sexual outlet. An additional criterion is the average amount of time spent per week devoted to real or imagined sexual activity (Kafka, 1997b). Other researchers suggest that impersonal sex be used as a criterion, meaning that the person disregards the other person (if present) in favor of focus on the sex act itself (Langstrom & Hanson, 2006).

About 5% of the U.S. population engages in hypersexual behavior, according to some estimates (Coleman, Raymond, & McBean, 2003), although individuals may be reluctant to report this problem, and estimates vary by sub-populations studied and measures used. In a New Zealand sample, approximately 4% of men and 2% of women indicated that their sexual urges or behaviors were "out of control" and problematic (Skegg et al., 2010). In a college sample, 7% of men and 2% of women reported lifetime experiences of compulsive sexual behavior (Odlaug & Grant, 2010). Approximately 8% of men in a separate study reported levels of use of pornography that were considered at risk of online sexual addiction (Becerra, Robinson, & Balkin, 2011). Sexual addiction among men with a low socioeconomic background who were not sex offenders was approximately 18% (Marshall et al., 2008).

Most of the research on hypersexual disorder involves self-identified sex addicts who request treatment. Among Germans seeking treatment for hypersexual behaviors, the most prevalent symptoms (according to their therapists) were pornography dependence (40%), compulsive masturbation (31%), and ongoing promiscuity (24%), with men being more highly represented in the first two symptom categories and women being more highly represented in the third (Briken, Habermann, Berner, & Hill, 2007). Hypersexual behavior is believed to be an anxiety-mitigating strategy, an impulse control or self-regulation difficulty, or some combination of these factors (Raymond et al., 2003; Stinson, Becker, & Sales, 2008). For example, the majority of men in one study with problematic paraphilic behaviors also met the criteria for ADHD, retrospectively, suggesting impulse control and self-regulation difficulties (Kafka & Hennen, 2000). This result is also supported by behavioral tests demonstrating significant inattention and impulsivity among a small sample of men with compulsive sexual behavior without paraphilic behaviors (Miner, Raymond, Mueller, Lloyd, & Lim, 2009). Similar to substance addiction and binge eating, there may be an initial reduction in tension after the sexual act, often followed by feelings of guilt (Black et al., 1997; Wiederman, 2004). Research also shows relatively high rates of hypersexuality among substance abusers, further highlighting the link with addiction (Stavro, Rizkallah, Dinh-Williams, Chiasson, & Potvin, 2013).

The etiology of hypersexual behavior is complex, although research points to childhood sexual abuse and an unsupportive or emotionally abusive family environment as potential precursors (see Aaron, 2012, for a review). Genetic and biochemical factors are also likely to play a role, including the "pleasure" areas of the brain involved in the dopamine and serotonin pathways, such as the nucleus

accumbens and the ventral tegmental area (and other limbic brain areas), with a potential environment-gene interaction during brain development leading to hypersexual behavior (see Blum et al., 2012, for a review). Regardless of the specific etiology, hypersexuality is described in the sexual addiction literature as being motivated by the "desire to fill the inner emptiness, anesthetize pain, and avoid feelings" (Seegers, 2003, p. 248).

Although there are self-help groups for sexual addiction similar to Alcoholics Anonymous, treatment for sexual addiction using a cognitive-behavioral model tends to focus on active coping and taking personal responsibility for one's behaviors, rather than using a disease model and relinquishing control to a higher power, *per se* (Marshall et al., 2008; Plant & Plant, 2003). Components of individual therapy may include: an assessment of the client's sexual history and current baseline levels of unwanted behaviors; motivational interviewing, similar to that which is used for substance addiction; psychoeducation about sexual functioning and sexual health; counseling for any previous sexual abuse or trauma, substance abuse, and/or psychological disorders; addressing intimacy and relationship issues; undermining any sexual fantasies that perpetuate the problem; and replacing problematic sexual behaviors with healthy ones, all in keeping with the client's cultural and religious context (Edwards, 2012).

The focus of treatment typically includes cognitive and behavioral techniques to increase alternative behaviors and diminish sexual thoughts and activities. In therapy, individuals are encouraged to develop coping strategies to regulate stress and mood (Coleman et al., 2003). Couples counseling may also be helpful (Schneider, 1989). Medications used to treat impulse control disorders, antidepressants with sexual side effects, and medications that result in decreased testosterone levels have also been indicated as treatments for hypersexual behavior (Raymond, Grant, Kim, & Coleman, 2002; Safarinejad, 2009). Naltrexone, which blocks opioid receptors and is used primarily to treat substance abuse, has been shown to be beneficial in treating hypersexual behavior in small-scale studies (Raymond, Grant, & Coleman, 2010; Ryback, 2004). Although there is an extensive history of research on and treatment of hypersexuality, opposition to calling it a disorder is based on concerns that it legitimizes and medicalizes behaviors that are socially unacceptable, rather than psychopathological (Halpern, 2011).

Summary, Diagnostic Issues, and Conclusion

Sexual dysfunctions and paraphilic disorders reflect a vast array of behaviors, all of which lie along a continuum and have varying degrees of concomitant distress, ranging from no distress to severe distress. Biological, social, and psychological factors play a role in each of these disorders and one cannot separate the influence of these factors on a given individual. The sexual dysfunction literature, which used to emphasize Masters and Johnson's behavioral techniques, is now dominated by medical research that assesses and treats these problems from a biological perspective (Berry, 2013a). Erectile dysfunction has been a predominant area of research, and successful treatment with medications has made this problem profitable to drug manufacturers and relatively easily solved by some consumers (Levine, 2010). Medical interest in female sexual dysfunction, while historically ignored, is now increasing, largely as a result of the potential for a finding a popular drug that could be the female counterpart to drugs such as Viagra (Hartley, 2006). Future directions in research also include investigating molecular genetic factors and the conditions for gene expression that play a role in both etiology and response to treatment (Burri, 2013). Nevertheless, conceptualizations and treatments of sexual functioning that consider interpersonal, psychological, and sociocultural factors are still recommended and supported by research outcomes, despite their decreasing application in the "real world" of highly medicalized treatment (Berry, 2013a; Hatzichristou et al., 2004).

Paraphilias and paraphilic disorders include a variety of behaviors, some of which may simply reflect unusual sexual preferences, such as transvestism, while other behaviors involve harmful and illegal activities, such as acting on pedophilic impulses. Diagnosable disorders must include either significant distress or harm to others (or both; American Psychiatric Association, 2013). Behavioral explanations of how deviant sexual behavior is learned are prevalent in this area of research (Akins, 2004). Much of the research emphasis, however, is devoted to pedophiles and sexual offenders and their treatment, which is a much needed line of research, although more research is needed to address the other paraphilias as well. Behavioral techniques involving punishment or changing learned associations may be used to treat these problems, although particularly for sex offenders, medications to lower sexual desire are likely to be more prevalent in the future (Saleh & Guidry, 2003; Thibaut et al., 2010).

The DSM as an Evolving Document The modifications included in DSM-5 have yet to be applied and studied in clinical populations at the date of publication of the current chapter. Nevertheless, calls for changes to the DSM will likely endure and there are ongoing, contentious discussions about the changes to DSM-5. The sexual dysfunction categories have been criticized for promoting a dogmatic view of sex as being heterosexual, with the only goals being vaginal intercourse and orgasm, to the exclusion of other types of fulfilling sexual behavior, while discounting or pathologizing individuals such as those who are celibate, those with same-sex partners, asexual people (without interest in sex), individuals with disabilities, those without partners, and those with sexually transmitted diseases who restrict their sexual behavior (Barker, 2011).

One of the specific, ongoing diagnostic clashes includes the proposed removal of dysparuenia (now included in

the broader genito-pelvic pain/penetration disorder) as a sexual dysfunction, with the assumption that it is a pain disorder (Binik, 2005), although other researchers oppose this idea (Tiefer, 2005). Future editions of the DSM may also include new disorders, such as *persistent genital arousal disorder* (spontaneous, recurrent, unwanted genital arousal that can last for hours or days with little or no relief) and sexual pain in men (Goldmeier, Mears, Hiller, & Crowley, 2009; Sungur & Gunduz, 2013).

The inclusion of particular paraphilias in the DSM is also an issue of debate. For example, the distress caused by transvestism likely involves social or interpersonal pressure to not engage in this behavior, rather than reflecting an inherent pathology (Reiersol & Skeid, 2006; Shindel & Moser, 2011). The inclusion of other paraphilias in the DSM has also been questioned, but for different reasons. Frotteuristic, voyeuristic, and exhibitionistic disorders have been criticized for a lack of empirical evidence to support their continued inclusion in the DSM (Hinderliter, 2010). Another issue is the concern about explicitly (in the DSM criteria) ruling out other disorders or conditions that may have led to paraphilic behavior, such as a psychotic episode, substance abuse, Alzheimer's disease, and mental retardation (First, 2010).

Some argue that disorders that involve nonconsenting partners, such as pedophilic disorder and sexual sadism disorder, be excluded from the DSM because the behaviors leading to the diagnosis are criminal and inclusion of the diagnosis implies that the individuals committing the crime have a mental illness that causes or even excuses their behavior (Halpern, 2011). A similar argument has been used in the long-standing debate over the possible inclusion in the DSM of "paraphilic coercive disorder" or the compulsive achievement of sexual gratification through rape (Frances & First, 2011; Holden, 1986). Similar to other disorders involving harm to others, however (such as pedophilia), the intense urge to rape and committing rape are considered abnormal and destructive, they tend to occur in a unique sub-population, and they also warrant treatment (Thornton, 2010). However, in order for "paraphilic coercive disorder" to be included in the DSM as a paraphilia, research would have to differentiate between those who commit rape to inflict dominance and violence upon another person versus those who specifically require the circumstance of rape in order to be aroused, as in a paraphilic fetish (Frances & First, 2011).

According to Frances and First (2011), "a rapist is not someone who has a mental disorder and psychiatric commitment of rapists is not justified," arguing for longer prison sentences instead (p. 558). An additional concern is the potential misuse of a "mental illness" as a legal defense for rape (Halpern, 2011). Labeling illegal behaviors as disorders is also a slippery slope; Hinderliter (2010) rhetorically asks if "embezzlement disorder" and "insider trading disorder" will be included in a future DSM. A counterpoint is that if simply released into the community after a relatively short prison sentence based on inadequate sentencing laws, without any required ongoing psychiatric treatment, the rapist has a substantial risk of reoffending. A diagnosis may assist in civil commitment cases, and the *paraphilia not otherwise specified* category has already been used extensively in the courtroom for this purpose (Beech & Harkins, 2012; Frances & First, 2011). While the debate continues, the inclusion of "paraphilic coercive disorder" is unlikely to be accepted as a new disorder in future editions of the DSM, with legitimate reasons.

Another debate that may resurface for DSM-6 is the proposed disorder of "hypersexuality," which was rejected for DSM-5, despite a promising DSM field trial (Reid et al., 2012). In addition, given that *Internet gaming disorder* is listed under "Conditions for Further Study" in DSM-5, it is conceivable that excessive use of Internet pornography could be listed in this section in the future (Griffiths, 2012). The proposal to rename pedophilia *pedohebephilia* and divide it into *hebephilia* (attraction to pubescent children ages 11–14) and pedophilia (attraction to prepubescent children under age 11) was rejected for DSM-5, but this issue may come up again (Balon, 2013; Blanchard, 2013). One concern about this proposal is that it may be considered overly inclusive or as potentially increasing the false positive rate of diagnosis (First, 2010).

Concluding Remarks Sexual dysfunctions and paraphilic disorders reflect distinct phenomena, although they both share a degree of social stigma and social dogma. Sexual behavior—which serves the important, broader purpose of the continuation of our species—is often either held to an impossible standard of perfect performance and pleasure, or is stigmatized when considered deviant in our society. Historically and socioculturally, for men, sex has been viewed as an important sign of manhood, and as such, "physicians, academics, and charlatans have regularly offered purportedly revolutionary cures (e.g., rhinoceros horn, Spanish flies, mandrake root, and a host of herbological methods) to safeguard men's sexual performance, on the basis of the accepted knowledge of their era" (Berry, 2013a, p. 22). The current emphasis on sexual function in our society can be easily identified through ads for "male enhancement" in one's email spam folder or through the many erectile dysfunction ads on television. The scope of these media campaigns has already begun to broaden to include women. While some may argue that there is an over-emphasis on sex in our current society, it is unlikely that the profitable industries involving sex—from pornography to pharmaceuticals—will curb their endeavors. It follows that the issue of sexual behavior will continue to find itself at the crossroads of society, culture, social activism, religion, the law, medicine, and psychology.

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14

Substance-Related and Addictive Disorders

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Of the major public health concerns of the 21st century, alcoholism and drug addictions are among the most pervasive, with devastating social, legal, and economic consequences for individuals and families, not only in the United States, but across the globe. The World Health Organization estimates that 76.3 million individuals have an alcohol-use disorder and 5.3 million individuals have drug use disorders (World Health Organization, 2010). The National Survey on Drug Use and Health reports more than half of the U.S. population, aged 12 and older, uses alcohol. Of these, 23.3% engage in binge drinking (i.e., drinking five or more drinks on the same occasion at least once in the past month), and nearly 7% are heavy drinkers (Office of Applied Studies, 2009). Along these lines, 10.5% of children live with a parent who has experienced an alcohol-use disorder in the previous year (Substance Abuse and Mental Health Services Administration, 2012). Although the prevalence rates of other drug use disorders are much lower than for alcohol, they are sizable by nearly any standard. For illicit psychoactive substances, the lifetime prevalence of substance-use disorders is roughly 6% in the United States, with lifetime cannabis abuse (i.e., 4.6%) being the most common after alcohol (e.g., Haynes, 2002). Substance use in the United States claims 600,000 lives annually, including 440,000 attributable to nicotine use, 125,000 from alcohol use, and 10,000 from heroin and cocaine use (exclusive of deaths from HIV; McCrady & Epstein, 1999). Alcoholism is the third major cause of death in the United States (behind coronary heart disease and cancer), with the life span of individuals with alcoholism being about 12 years shorter than their nonalcoholic counterparts (Mokdad, Marks, Stroup, & Gerberding, 2004). In addition to the serious problems individuals with alcohol or drug problems create for themselves, victims of family violence, accidents, and violent crime add to the numbers of those adversely affected. On days when alcohol is used, intimate partner violence increases (e.g., Mignone, Klostermann, & Chen, 2009). Parental substance use is associated with a host of emotional, behavioral, and social problems for children (e.g., Boris, 2009; Osborne & Berger, 2009), which may evolve into problems with alcohol and drugs as these children progress through adolescence and into adulthood. Thus, the effects of alcoholism and substance-use disorders can be accurately described as being part of an intractable, multigenerational vicious cycle.

Aside from the toll in human suffering, estimates of yearly direct and indirect economic and social costs arising from substance-use disorders are substantial. Individuals who abuse alcohol and other drugs consume a disproportionately large share of social resources from a variety of sources, some of which include specialized drug abuse treatment (Anglin, Nosyk, Jaffe, Urada, & Evans, 2013; Knudsen & Roman, 2012), treatment of secondary health effects (Olmstead, Cohen, & Petry, 2012), use of social welfare programs (Center for Substance Abuse Treatment, 2009), and involvement of the criminal justice system (e.g., arrests, incarceration, parole, and probation; Werb, Elliot, Rischer et al. 2007). In the United States, the most recent estimate of the societal cost of drug abuse (focusing on the cost of health care, productivity losses, criminal justice system and crime victim costs) was $193 billion for the year 2007, (National Drug Intelligence Center, 2011). Similarly, the economic cost of alcoholism for 2006 (the last year for which figures are available) was $1,235 billion, which translates into approximately $746

for every man, woman, and child in the United States (Bouchery, Harwood, Sacks, Simon, & Brewer, 2011).

The size and scope of substance abuse and dependence in our society have drawn significant and increasing scientific and public attention. Although our understanding of addiction to alcohol and drugs is far from complete, there has been great progress in the understanding of the etiology, course, and treatment of alcoholism and drug abuse. However, progress is not to be confused with consensus. There remains much controversy, not only about the etiology and treatment of substance misuse, but also, on a far more fundamental level, about how to conceptualize and operationalize substance-use disorders.

Definition and Description of Substance-Use disorders

For most of U.S. history, chronic and excessive substance use has been viewed either as immoral conduct or as a disease. More recently, with the rising influence of the behavioral sciences in this dialogue, addiction has also been viewed as maladaptive behavior subject to reinforcement contingencies that govern all learned human behavior. In turn, the definition and description of addictive behavior can vary considerably, based on which view (i.e., moral, disease, or behavioral) is emphasized.

Defining Addictive Behavior The evolution in thinking about what defines addictive behavior is based in large part on the observation that a number of processes are common to excessive behaviors that have been characterized as addictive. Miller (1995) defines addiction as consisting of three primary components: 1) preoccupation; 2) compulsion; and 3) relapse. Using drug addiction as an example, in the preoccupation phase, individuals place a great deal of emphasis on acquiring drugs. As such, social relationships and employment are jeopardized in the continuous search for drugs, and also suffer as a consequence of using drugs. In the compulsion phase, the individual continues to use drugs, despite serious negative consequences. During relapse, the individual stops using drugs for a period of time, however, eventually resumes using drugs at an abnormal level. To capture the essence of addiction that spans these behaviors, a broad definition has evolved that can be generally applied to all addictive behavior, including addiction to psychoactive substances. From this vantage point, addiction is viewed as a complex, progressive pattern of behavior having biological, psychological, and sociological components. Thus, this pattern of behavior is characterized by the individual's overwhelming pathological involvement in or attachment to substance use, subjective compulsion to continue use, and inability to exert control over it. Moreover, this behavior pattern continues despite its negative impact on the physical, psychological, and social functioning of the individual.

Although this depiction provides an overarching description of addictive behavior, it provides little insight into how to discern when use of drugs or alcohol crosses the normal threshold into problematic use, and similarly, from problematic use to disorder. More specifically, because of widely divergent social attitudes toward and prejudices about alcohol and drug use, there have been few generally accepted and agreed-on operational criteria for what level of use constitutes social use, abuse, and dependence. Substance use can follow one of several patterns; addiction "is not an all-or-nothing" phenomenon, but a continuum from recreational use to severe compulsion (Peele, Brodsky, & Arnold, 1991, p. 133).

Labels and Diagnostic Systems Throughout history, a number of value-laden terminologies have been used to describe individuals who use alcohol and other drugs, as well as the problems such use can create (Carroll & Miller, 2006). Labels assigned to behaviors may have tremendous implications for the way the individual and problem are conceptualized; names used to describe a given phenomenon greatly influence the way one thinks about a problem and how one addresses it. Individuals with alcohol problems have been called, at various times, "drunks," "dipsomaniacs," "alcoholics," "alcohol abusers," and so on. Those who use illicit drugs have been called "addicts," "dope fiends," "freaks," and "criminals." Of course, these names have important implications for how we think about people that have these problems and what can be done about it. In response to these issues, more formal diagnostic systems have come to the fore in recognition of the importance of labels and to bring order to the labeling system.

Although use, abuse, and addiction are best viewed as a continuum of behavior, much effort has been put forth to delineate boundaries between critical points on this continuum. There are several different definitional frameworks used to categorize various levels of addictive behavior that are widely referenced in the scientific and lay press. The most widely used framework is the psychiatric diagnostic approach, exemplified in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) and the International Classification of Diseases (ICD-10; World Health Organization, 1992). Using the DSM-5 system as an example, the diagnosis of alcohol or psychoactive substance-use disorders are measured base upon severity level of the particular substance-use disorder. A mild diagnosis would include meeting two or three criteria, moderate meeting four to five, and severe meeting six or more:

1. Physical tolerance.
2. Withdrawal.
3. Unsuccessful attempts to stop or control substance use.
4. Use of larger amounts of the substance than intended.
5. Loss or reduction in important recreational, social, or occupational activities.

6. Continued use of the substance despite knowledge of physical or psychological problems that are likely to have been caused or exacerbated by the substance.
7. Excessive time spent using the substance or recovering from its effects.

However, given the complexity of addictive behavior, it appears that a binary, either–or view implicit in the psychiatric diagnostic approach is too simplistic and provides little understanding of the complexities of the addictive process and how to change it. As noted by Shaffer and Neuhaus (1985), addictive behavior is not easily categorized and, as such, is not easily defined by a set of consensually agreed-on criteria. Indeed, different diagnostic systems often place greater emphases on different aspects of behavioral and physiological functioning in their definitions. In turn, these differences lead to fairly divergent estimates about the prevalence of alcohol and drug use disorders in the general population (for a review of this issue, see Grant & Dawson, 1999). Moreover, O'Brien, Volkow, and Li (2006) argue that dependence is often confused with physical dependence, which may occur with therapeutic applications of a variety of medications, and as a result, may create apprehension among clinicians to prescribe medications out of fear of creating addiction (Kranzler & Li, 2010). In response to this concern, given its emphasis on behavioral aspects of substance use, the DSM-5 has removed the terms abuse and dependence, and replaced them with the single diagnosis of substance-use disorder.

A Biopsychosocial Perspective Behavioral scientists have proposed an alternative biopsychosocial approach to defining alcoholism and drug abuse. According to this framework, alcohol and drug use disorders are not defined as unitary diseases, nor is it implicitly assumed that the observed substance use symptoms are the manifestation of a disease state. Instead, symptoms are viewed as acquired habits that emerge from a combination of genetic, social, pharmacological, and behavioral factors. Addiction is viewed as involving physiological changes in individuals (many of whom may be genetically and/or psychologically predisposed) and the complex interaction of environmental stressors and individual aspects of the person (including his or her experiential history) that produce and maintain addictive behaviors.

A comprehensive understanding of the synergistic relationship among these factors is essential, not only to understanding the degree and severity of substance use, but also for recognizing when use becomes abuse. This approach largely de-emphasizes labels and places greater emphasis on understanding the interplay between multiple factors that have led to and maintained the observed behavior.

Cultural and Social Issues

Definitions of substance abuse do not develop in isolation; the point at which substance use is said to move from recreational to disordered is determined by the social and cultural context in which the behavior occurs (e.g., Thombs, 2006). Thus, societal norms and definitions of substance use and abuse are inextricably intertwined. How we determine what qualifies as an alcohol or drug problem is derived from the boundaries implicitly (e.g., social stigma associated with being labeled an alcoholic or drug addict) or explicitly (e.g., laws prohibiting use of certain substances) drawn by the society in which the behavior takes place. In many respects, the norms for acceptable drinking and other drug use are delineated and communicated (implicitly or explicitly) to members of the society by the way the culture defines addiction.

A Sociocultural View of Alcoholism and Drug Abuse From a sociological perspective, clinical diagnostic criteria for alcoholism and other drug abuse are derived from societal norms and thus vary depending on when and where the diagnosis is made. More specifically, drinking or drug-taking behaviors that are considered disordered are those that deviate from socially accepted standards. Perhaps the cultural foundations of alcoholism, which can also be applied to other addictive behaviors, are best captured by Vaillant (1990), who stated: "Normal drinking merges imperceptibly with pathological drinking. Culture and idiosyncratic viewpoints will always determine where the line is drawn" (p. 6).

Moreover, problems with alcoholism and drug abuse have become increasingly medicalized, and certain factions appear to have an agenda in viewing these behaviors as symptoms of a disease state and convincing others to view them in a similar manner. If addictive behavior is defined as a disease, the label itself gives credibility to physicians' efforts to control, manage, and supervise the care provided to individuals seeking treatment. Thus, the medical community may have a strong, vested interest in defining addictive behavior as a disease. At its core, treatment of substance abuse is a business; as with all business, economics play a critical role (Carlson, 2006). The label serves to make legitimate such financially lucrative efforts as hospital admissions, insurance billing, expansion of the pool of patients available for hospital admission, consulting fees, and so forth.

However, it would be short-sighted to assume that societal norms alone shape the parameters for defining alcoholism and drug addiction. In some respects, the process of labeling what constitutes substance abuse or dependence restricts drinking and drug-using behavior by members of a given culture. In our culture, public intoxication, drinking early in the morning, drinking while at work, and drinking and driving are considered signs of problem drinking, which are included as indicators in many of our widely used diagnostic instruments (e.g., Michigan Alcoholism Screening Test; Selzer, 1971; Alcohol Use Disorders Identification Test; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001). Clearly, these "symptoms"

are culturally derived and are based on a commonly held set of beliefs about acceptable substance use; the nature of the symptoms would likely be different in another culture that had a different set of social norms. Thus, understanding the cultural beliefs and practices of individuals from diverse backgrounds and how they relate to substance use will be critical in the development of successful prevention and intervention programs (Grant et al., 2006).

The Disease–Moral Model of Addictive Behavior The disease model of alcoholism and abuse of other drugs is not the only theoretical concep-tualization of these disorders and, in some contexts, not the predominant one. From a social policy perspective, the idea that addictive behavior is deviant and immoral is on at least an equal footing with the disease model. This disease–moral model is summed up by a passage from a turn-of-the-century temperance lecturer, John B. Gough (1881), who wrote that he considered "drunkenness as a sin, but I consider it also a disease. It is a physical as well as moral evil" (p. 443). The disease and moral models of substance abuse are, in many respects, strange bedfellows. These models hold divergent and, at times, inconsistent views about the etiology and maintenance of addictive behavior. While the moral view holds that drinking and drug use are freely chosen acts for which individuals are responsible, the disease model, in many respects, espouses the opposite position. Thus, the conclusions and outcomes of the disease–moral model are, at times, rife with contradiction. For example, it is difficult to reconcile the inherent contradiction in treating the "illness" of psychoactive substance abuse with punishment (e.g., incarceration). How many other illnesses are routinely punished?

At a social policy level, much energy and effort is expended in veering between these two models. Yet, it is the disease–moral model that is the guiding hand that drives alcohol and drug policies today, with neither perspective completely displacing the other. For instance, judges sentence those convicted of driving while intoxicated to attend Alcoholics Anonymous (AA) meetings or face jail. Peele (1996) describes this policy as the "disease law enforcement model," which perhaps manifests itself most clearly in the debate between those who believe drug abusers should be treated and those who believe they should be incarcerated.

Gender Issues

Although social norms provide a context in which to understand addictive behavior, it is also important to understand that science does not stand above or outside of its social context, but is immersed in and highly influenced by it (Kuhn, 1962). As with all areas of inquiry, social and cultural factors influence the way scientists study alcoholism and drug abuse. The relationship between addictive behavior and gender is an important case in point. Alcoholism and other addictions have traditionally been considered problems of men; as such, it has been the study of addictive behavior among men that has shaped our understanding of the nature and course of these disorders. Two of the most widely known and influential investigations of alcoholism, Jellinek's (1952) on the phases of alcoholism and Vaillant's (1995) 45-year prospective longitudinal study of alcohol abuse in inner-city and college cohorts, were limited to male participants. Treatment methods and programs were also initially designed to treat male substance abuse; it was not unusual for women who needed treatment for addictive disorders to be placed in psychiatric wards, whereas men were treated in specialized alcoholism or drug abuse treatment programs (Blume, 1998).

Because the majority of research conducted in the area of substance abuse has been based largely on studies with male participants, a significant concern is that conclusions drawn from these studies may not generalize to women with substance-use disorders (Straussner & Zelvin, 1997). More recently, women have become the focus of increased attention by the research and treatment communities. Comparisons of substance use by men and women have revealed several important differences in epidemiology, development, and treatment. Wilsnack and Wilsnack (2002) identified two important public health policy implications of women's drinking: 1) women with drinking problems may be reluctant to seek treatment or may be prevented by embarrassed family members; and 2) prevalence rates of women's drinking may be underestimated, which makes it difficult to accurately determine the amount of harm caused by female drinking.

Epidemiological and Etiological Comparisons Data from the 2012 National Survey on Drug Use and Health (Substance Abuse and Mental Health Services Administration, 2013) reveal that the rate of substance abuse or dependence for males aged 12 and older were nearly twice as high as for females (11.6% versus 6.9%). Specifically, males were more likely than females to be current (past 30-day) users of several different illicit drugs including marijuana (9.6% versus 5.0%), nonmedical users of psychotherapeutic drugs (2.8% vs. 2.4%), cocaine (1.0% versus 0.3%), and hallucinogens (0.6% versus 0.3%). However, important changes in use patterns have taken place as a function of gender. In 2012, the rates of current illicit drug use were similar among males and females aged 12–17 (9.6% and 9.5%, respectively). This pattern represents a change from 2011, when the prevalence of current illicit drug use was higher for males than for females in this age group (10.8% vs. 9.3%). In 2012, females aged 12–17 were more likely than males to be current nonmedical users of psychotherapeutic drugs (3.2% vs. 2.4%). Similarly, Johnson et al. (2008) found the gap in binge drinking rates among 8th to 12th graders, although the rate is still higher in boys, has declined over the last few decades such that girls'

binge drinking appears to be catching up to that of boys (Johnston, O'Malley, Bachman, & Schulenberg, 2008). Moreover, a cross-national epidemiological study from 17 countries participating in the World Health Organization's Mental Health Survey Initiative concluded that there was consistent evidence across countries suggesting a general shift may be occurring with respect to traditional sex differences in drug use (Degenhardt et al., 2008).

Another area of difference between men and women are the factors that appear associated with alcohol and drug use. Although studies have consistently shown that the presence of psychiatric disorders significantly increases the risk of comorbid substance abuse (Xu et al., 2013), mental health appears to play an especially strong role in women's substance-use disorders. In part, this may reflect the fact that female substance users have higher rates of comorbid (i.e., co-occurring) psychiatric disorders, especially depressive and anxiety disorders, than do men (Brady & Randall, 1999).

These findings may also reflect that psychiatric disorders and substance-use disorders may in part stem from the separate or cumulative experiences of violence and trauma that may be more common among women versus men with substance-use disorders. For instance, in a community sample of 676 drug users, Medrano, Hatch, Zule, and Desmond (2002) found that 43% of women and 23% of men had been sexually abused, 58% of women and 39% of men had been emotionally abused, 52% of women and 50% of men had been physically neglected, and 65% of women and 52% of men had been emotionally neglected during childhood.

Although trauma history is consistently associated with increased risk for substance abuse among young women, the trauma–substance-use disorders link is less clear for men. For instance, both childhood trauma and cumulative lifetime trauma predicted alcohol and drug relapse in alcohol-dependent women, but not men (Heffner, Blom, & Anthenelli, 2011). In contrast, Danielson et al. (2009) found that exposure to traumatic events increased risk for substance abuse among both men and women. Because some forms of interpersonal trauma, such as sexual assault, are more common among women (Kelley et al., 2014; Xu et al., 2013), they may have greater associations with women's subsequent mental health and substance-use disorders. Furthermore, in a study of women recruited from community violence agencies, women with recent incidents of intimate partner violence were more likely to report using alcohol as a coping behavior (Kaysen et al., 2007). Moreover, sexual assault during adulthood appears associated with substance use. For instance, in a sample of women applying for a protection order from a domestic violence unit, those who experienced more than one sexual assault from a partner, as compared to one, were 3.5 times as likely to start or escalate substance use (McFarlane et al., 2005).

It also appears that male significant others who use drugs strongly influence women's patterns of substance use. In comparison with women, men are more likely to introduce their partners to the use, initiate their female partners into injecting drugs, and supply drugs to their partners (e.g., Simmons, Rajan, & McMahon, 2012). Moreover, women frequently report being pressured to use by their partners (Walitzer & Dearing, 2006; Zywiak et al., 2006). Marital stress is more strongly associated with substance use for women (Walitzer & Daring, 2006; Zywiak et al., 2006) and partner alcohol or drug use are more strongly related to relapse for women than for men (Grella, Scott, Foss, Joshi, & Hser, 2003). Taken together, these findings suggest that women may be more likely to rely on substance use as a form of self-medication for mood disturbances and interpersonal problems, which has been termed "effortful avoidance" (Feuer, Nishith, & Riseck, 2005).

The Stigma of Addiction for Women Sociocultural factors also play important roles in the substance use patterns of men and women. In nearly all societies in which alcohol or other drugs are consumed, the cultural norms, attitudes, stereotypes, and legal sanctions often differ for men and women. Most cultures expect that women will drink less alcohol than men. These expectations may serve as a protective factor, reducing the incidence of alcohol-use disorders among women (e.g., Kubicka, Csemy, & Kozeny, 1995).

The intense stigma associated with drinking and drug use by women can also create serious social problems. Society tolerates some behaviors exhibited by intoxicated men, but views the same behavior as scandalous and morally objectionable if exhibited by intoxicated women. As Blume (1991) argues, women who are intoxicated are often subjected to unwanted sexual overtures, and men believe that intoxicated women who say "No" to their advances really mean "Yes." In a study of beliefs about rape, participants viewed rapists who were intoxicated as less responsible for their crimes than sober rapists and viewed rape victims who had been drinking as more to blame for the rape than victims who had not been drinking (Richardson & Campbell, 1982). Thus, it is not surprising that women who are alcoholics are much more likely to be victims of violent crime, including rape, than their sober counterparts (e.g., McClelland & Teplin, 2001). Some contend that society at large views women who abuse alcohol or other drugs as partially or fully responsible for sexual advances made by men, thus making these women acceptable targets for physical and sexual aggression (e.g., Stormo, Lang, & Stritzke, 1997).

An important result of the stigma associated with women's alcohol and drug use is denial by the person, the family, and society. These various forms of denial, in turn, are barriers that may prevent women from seeking treatment. As noted by Schober and Annis (1996), women often are reluctant to acknowledge a substance use problem publicly by seeking treatment because of intense fear of

being stigmatized as a substance abuser. In comparison to their male counterparts, fears about the stigma associated with the label of *alcoholic* or *substance abuser* may be a more powerful disincentive for women to seek treatment than denial of having a problem or accessibility and cost of treatment (Marlatt, Tucker, Donovan, & Vuchinich, 1997).

Along with personal denial, family denial is also a significant barrier to women entering treatment for substance abuse. In fact, the response by family members to women entering treatment appears to be more negative than it is for men. For example, Beckman and Amaro (1986) found that nearly 25% of women in a treatment sample reported opposition from family or friends, compared to only 2% of men. In addition, childcare and child custody issues influence the decision to seek treatment.

Women often fear that entering treatment will lead to the perception that they are unfit as mothers and may be deprived of custody of their children. These fears are not unfounded. Women who use alcohol and other drugs during pregnancy have been prosecuted on charges of prenatal child abuse or delivery of a controlled substance to a minor (via the umbilical cord). Such practices have not prevented substance use during pregnancy, but have discouraged pregnant substance users from seeking prenatal or addiction treatment (Blume, 1997).

In addition to these personal and familial barriers, several factors related to the type and process of treatment have increased women's reluctance to seek help. Less than 14% of all women who need treatment for substance abuse receive it; less than 12% of pregnant women receive help (Center on Addiction and Substance Abuse, 1996). Dawson, Grant, Stinson and Chou (2006) found that individuals who seek treatment differ in many ways, with men being more likely to seek help than women. Unfortunately, these findings are not surprising; most treatments for addictive behaviors have been developed largely to meet the needs of men and thus may not be as effective for women (e.g., Ramlow, White, Watson, & Luekefeld, 1997). A major obstacle to more women engaging in treatment is that most programs fail to provide services (e.g., child care, obstetrician services) that make it easier for women to enter treatment (Nelson-Zlupko, Dore, Kauffman, & Kaltenbach, 1996; Stewart, Gossop, & Tranaka, 2007). Some studies do, in fact, suggest that treatment programs designed specifically to meet the treatment needs of substance-abusing women have higher retention rates and better outcomes than standard outpatient or residential treatment programs (e.g., Dahlgren & Willander, 1989; Roberts & Nishimoto, 1996). Yet, gender-specific treatment programs are not widely available. In a review of the Alcohol and Drug Services Study conducted by the U.S. Department of Health and Human Services, Brady and Ashley (2005) found that women-only treatment availability ranged from about 2% of outpatient nonmethadone facilities to 21% of nonhospital residential facilities.

Sexual Orientation and Alcohol and Drug Use Disorders

Lesbian and Bisexual Women The most recent Institute of Medicine (2011) report on the health of sexual minorities reported that compared with heterosexual individuals, those that have same-sex partners have higher rates of substance use and alcohol consumption. For instance, Wilsnack et al. (2008) found that in comparison to heterosexual women, lesbian, bisexual, and questioning women drink more often and more heavily. Similarly, based on estimated from the 2004–2005 National Epidemiologic Survey on Alcohol and Related Conditions (McCabe et al. (2009) found that rates of past-year heavy drinking (defined as four drinks for women in a 2-hour period in the past year) were 20% and 25%, and alcohol dependence was 13% and 16% among lesbian and bisexual women, respectively. In contrast, 8.4% of heterosexual women reported past-year heavy drinking and 2.5% met the criteria for alcohol dependence. Past-year drug use and dependence were also notably higher among women who self-identified as lesbian or bisexual (McCabe et al., 2009). For instance, past-year drug use (defined as cocaine or crack, heroin, hallucinogens, ecstasy, inhalants, sedatives, stimulants, etc.) was 12.6% and 14.1% and past-year other drug dependence was 5.7% and 3.0% for lesbian and bisexual women. In contrast, 3.1% and 0.4% of women who self-identified as heterosexual reported past-year drug use or dependence, respectively. Lesbian women are also more likely to have co-occuring mental health problems and substance-use disorders (Lipsky et al., 2012) which may escalate risk for substance-use disorders.

Gay and Bisexual Men Estimations from the 2004–2005 National Epidemiologic Survey on Alcohol and Related Conditions (McCabe et al., 2009) found that 18.1% and 16.4% of gay and bisexual men, respectively, meet the criteria for alcohol dependency (defined as five or more drinks in a 2-hour period in the past year). Among heterosexual men, 13.7% and 6.1% met heavy drinking and alcohol dependence criteria. Among gay or bisexual men, past-year drug use and drug dependence was 16.8% and 17.7% and 3.2% and 5.1%, respectively, whereas, men who self-identified as heterosexual reported 4.5% and 0.5%, respectively.

In addition to the consistently higher rates of substance use among lesbian, gay, and bisexual individuals, an equally alarming concern is the growing body of literature indicating that sexual minority youth, and particularly lesbian and bisexual adolescent girls, have higher rates of initial substance use and experience steeper substance use trajectories than do heterosexual youth (Marshal, Friedman, Stall, & Thompson, 2009). In a meta-analysis, Marshal and colleagues (2008) found that the prevalence of substance use for young, non-heterosexual women were, on the average, 400 times higher than for heterosexual youth. In a study of 1,514 high school students.

Orenstein (2001) found that gay or lesbian youth were approximately twice as likely as heterosexual students to have ever used marijuana; however, for "hard" drugs differences were more substantial. For instance, 2% of heterosexual students have ever used cocaine or crack whereas 27% of gay and lesbian students reported cocaine or crack use. Similarly, 1% of heterosexual adolescents surveyed indicated that they had used an intravenous drug, whereas 9% of gay or lesbians indicated they had injected a drug. Findings from previous research led Marshal and colleagues (2009) to conclude that a significant proportion of lesbian, gay, and bisexual youth are on high-risk substance-use trajectories that begin in young adulthood.

The growing awareness of substance use disparities between lesbian, gay, and bisexual individuals as compared with heterosexual peers has led to research aimed at understanding the causal mechanisms associated with these differences. Importantly, the Centers for Disease Control (Milstein, 2006) has suggested that these disparities may be due to "syndemics"; that is, multiple factors may interact to contribute to greater burden of disease in a population. For instance, pressure to conform during childhood may lead to stress. Moreover, children who are viewed as different may experience greater bullying or peer rejection. As young adults, sexual minorities may begin to socialize in bars and clubs in an attempt to seek social support and community identification, thereby increasing their exposure to environments where alcohol and drugs are widespread (Marshal et al., 2009). In addition, sexual minority stress, a multifaceted construct that includes stress related to identity concealment and confusion, experienced and anticipated rejections and discrimination, and internalized homophobia may also contribute to substance use and misuse (Lewis, Kholodkov, & Derlega, 2012; Meyer, 2013).

Etiology of Alcohol and Other Drug Use Disorders

Research on the etiology of substance-use disorders is a multidimensional, multidisciplinary effort, and findings that have appeared in the literature are truly voluminous; this is a consequence of the multiple theoretical conceptualizations of the development and maintenance of these disorders. For instance, investigators who view alcoholism and drug abuse as diseases are often most interested in examining genetic and biological contributions. Behavior-oriented researchers are more apt to explore antecedent and consequent events that may serve to initiate and reinforce substance use. Sociologists examine macro-level variables, such as peer and societal influences that contribute to the onset and maintenance of addictive behavior. To be clear, development of an addiction to alcohol or other drugs is a complex process involving many factors; however, the exact role of each of these ingredients has not been fully determined and operates differently for each individual. Genetics, biology, environment, sociocultural factors, and the biochemical properties of the psychoactive substances themselves appear to contribute substantially to the process (e.g., Sandbak, Murison, Sarviharju, & Hyytiae, 1998).

Neurobiology of Addiction Neurobiological research on addiction tends to focus on identifying neuroadaptive mechanisms within specific brain circuits that mediate the transition from infrequent and controlled substance use to chronic addiction (Cruz, Bajo, Schweitzer, & Roberto, 2008). Central to the neurochemical process underlying addiction is the means by which alcohol or other drugs become reinforcing. Most neurobiological studies of addiction have focused on the dopamine system, which is considered to be the neurotransmitter system through which most drugs of abuse exert their reinforcing effects (Kreek et al., 2012). However, other pathways may be involved in addiction as well, including the opioid peptide, gamma-aminobutyric acid, and serotonin pathways (Koob & Simon, 2009). A reinforcer is operationally defined as an event that increases the likelihood of a subsequent response; drugs of abuse, which appear to have substantial effects on the dopamine system, are much stronger reinforcers than natural reinforcers, such as food and sex (Wightman & Robinson, 2002).

A network of four circuits may be largely responsible for drug abuse and addiction: (a) reward, located in the nucleus accumbens and the ventral pallidum; (b) motivation/drive, located in the orbitofrontal cortex and the subcallosal cortex; (c) memory and learning, located in the amygdala and the hippocampus; and (d) control, located in the prefrontal cortex and the anterior cingulate gyrus (Feltenstein & See, 2008). These four circuits receive innervations (i.e., are stimulated into action) from dopaminergic neurons, but are also connected to one another through direct and indirect pathways with the ventral striatum being key structures (Taber, Black, Porrino, & Hurley, 2012). The circuits work together and can be changed with experience. Thus, during the development and maintenance of addiction to a psychoactive substance, the enhanced value of the substance in the reward, motivation, and memory circuits eventually overcomes any inhibitory control exerted by other parts of the brain (e.g., the prefrontal cortex). This, in turn, creates a positive feedback loop that is initiated by the ingestion of the psychoactive substance and then perpetuated by the activation of the motivation/drive and memory circuits. For individuals addicted to drugs, the value of the drug of abuse is enhanced in the reward and motivation/drive circuits; increases in dopamine induced by drugs are three to five times higher than those of natural reinforcers (Wise, 2002). Some studies have supported the hypothesis that genetic polymorphisms of dopamine receptors and transporters actually increase the risk of addictive disorders (Gorwood et al., 2012).

Genetic Propensity Many studies have examined the genetic predisposition to substance abuse and addiction,

and a substantial literature suggests addiction problems and disorders have a genetic component (Dick & Agrawal, 2008). The familial nature of alcoholism has long been recognized and is well documented (e.g., Merikangas et al., 1998; Nurnberger et al., 2004). While alcoholism appears to run in families, it is difficult to disentangle genetic factors from environmental influences because members of nuclear families typically share both genetic and environmental factors. As an example, a number of genes are believed to contribute to an individual's susceptibility to substance dependence; thus, there may be many idiographic combinations of these genes resulting in different combinations for different people. Environmental influences, in particular the environment–gene interaction, may also impact alcohol and drug use (Heath et al., 2002). With this caveat in mind, studies of twins also support the role of genetic factors in the devel-opment of alcoholism. The level of twin pair concordance for a disorder or trait can be compared between identical (monozygotic) and fraternal (dizygotic) twins. A higher concordance for monozygotic than dizygotic twins is an indication of genetic heritability; 100% heritability indicates a disorder is entirely genetic, 50% heritability means there is a substantial contribution from genetic and environmental factors, and 0% indicating genetic factors do not play a role. In general, adult twin studies of alcohol dependence show a heritability of 50–60% (Hasin, Hatzenbuehler, & Waxman, 2006).

Only a few studies have sought to determine a familial influence in the development of substance-use disorders other than alcohol. Compton, Thomas, Conway, and Colliver (2005) estimated that 40–60% of variability in the risk of addiction is accounted for by genetic factors (including genetic–environment interactions). Similarly, a population-based twin study of abuse/dependence found that the median heritability estimate was 53% for males and 55% for females, respectively (Kalaydjian & Merikangas, 2008). In addition, a preference for specific types of drugs may occur among family members of drug abusers; monozygotic twins appear to display a greater similarity than dizygotic twin pairs in their preference for, and response to, certain types of drugs (Schuckit, 1987).

Examining the genetic link for the abuse of drugs other than alcohol is often far more difficult than for alcohol because individuals must be exposed to the psychoactive substance for the behavioral manifestation of the genetic propensity toward a certain behavior to appear. Most individuals in the world have exposure to alcohol; this is not the case for many other drugs of abuse (e.g., heroin, cocaine).

Family Environment Influences Not only do individuals who abuse alcohol and other drugs often develop physiological dependence, they can also develop a strong psychological dependence. Essentially, they become dependent on the drug to help them cope with negative emotional states and stressful social situations. Because substance abuse often leads to significant problems for people across multiple domains of functioning, we must ask, "How is psychological dependence on alcohol or other drugs learned?"

As has been argued by many investigators, the family is an extremely important molding influence for children across a variety of domains. Although the misuse of alcohol and other psychoactive substances by adults often has serious physical, emotional, behavioral, and economic consequences, the ancillary short- and long-term negative effects on those who live with these adults are often no less destructive. In particular, children who live with parents who abuse alcohol and other drugs may be victimized by the deleterious environments these caregivers frequently create. Generally, there is an inverse relationship between parental monitoring and youth behavior problems (Koning et al., 2012). However, in comparison to matched controls, alcohol- and drug-abusing parents are less able to monitor their children's behavior (Fals-Stewart, Kelley, Fincham, Golden, & Logsdon, 2004). Robertson, Baird-Thomas, and Stein (2008) found that alcohol-use disorders were associated with lower levels of parental monitoring which seemed to significantly impact youth alcohol and marijuana use, hard drug use (e.g., cocaine), sexual risk behavior, and delinquency. Stress and negative affect, which are comparatively high in families with an alcoholic family member, are associated with alcohol use in adolescents (Fagan, Van Horn, Hawkins, & Jaki, 2013). Also, there is a correlation found between high parental monitoring and significantly less use of alcohol and drugs in adolescents (Clark, Shamblen, & Ringwalt, 2012).

Personality and Psychiatric Factors A theory that is often used to explain substance abuse (most often in the popular press) is the idea of an alcoholic personality—a type of character organization that predisposes a person to use alcohol rather than some other strategy for coping with emotional and social stress. Some investigators have found that persons at high risk for developing alcoholism are significantly more impulsive and aggressive than those at low risk for abusing alcohol (e.g., Morey, Skinner, & Blashfield, 1984). However, results of studies regarding a predisposition to drinking or other drug use have been mixed, and most alcoholism and drug abuse investigators have largely dismissed the traditional notion of the existence of an alcoholic personality. Yet, there is strong evidence that certain disorders and substance abuse are linked. For example, roughly half of individuals diagnosed with schizophrenia are also either alcohol or drug dependent (Kosten, 1997). The relationship between antisocial personality and substance use is strong (Harford & Parker, 1994; Kwapil, 1996), although the direction of the causal link (if there is one at all) is unclear (Carroll, Ball, & Rounsaville, 1993). Some investigators have suggested that there is a strong relationship

between depressive disorders and alcoholism (Kranzler, Del Boca, & Rounsaville, 1997) and that this relationship may be stronger among women than among men (Moscato et al., 1997).

Although the nature of the relationship between substance abuse and other mental disorders is unclear, comorbidity is important to consider in treatment planning. Providing the best possible treatment for substance-abusing clients who have co-occurring psychological disorders requires (a) more cross-disciplinary collaboration; (b) greater integration of substance abuse and mental health treatments; and (c) more comprehensive training to treatment providers in the assessment of and intervention with common co-occurring conditions.

Behavior-Oriented Explanations The first learning theories for substance abuse were based on two fundamental assumptions: (a) substance use is a learned behavior; and (b) substance use is reinforced because it reduces anxiety and tension. To test these assumptions, Masserman, Yum, Nicholson, and Lee (1944) induced an experimental neurosis in cats. After the cats were trained to eat food at a food box, they were given an aversive stimulus (e.g., an air blast to the face or an electric shock) whenever they approached the food. In turn, the cats stopped eating and displayed various symptoms, including anxiety, psychophysiological disturbances, and peculiar behaviors. When the cats were given alcohol, however, their symptoms were alleviated and they were able to eat again. Based on these findings, it was concluded the anxiety-reducing properties of the alcohol are reinforcing and are thus responsible for maintaining drinking behavior.

In general, however, the tension reduction model of substance use is difficult to test, and research with alcoholic participants has produced conflicting findings. Paradoxically, in some individuals, prolonged drinking is associated with increased anxiety and depression (e.g., McNamee, Mello, & Mendelson, 1968). Along these lines, if tension reduction alone explained the development of substance-use disorders, anyone who finds drugs or alcohol tension-reducing would be in danger of becoming a substance abuser. We would expect alcoholism and drug abuse to be far more common than it is because psychoactive substances tend to reduce tension for most people who use them.

Several investigators have concluded that expectancies play a central role in the initiation and maintenance of alcohol and drug use disorders (Mendelson & Mello, 1995; Marlatt et al., 1998; Read & Curtin, 2007). Expectancies of the positive effects of substance use develop from repeated pairings of alcohol or other drugs with their reinforcing effects. Expectancies can be conceptualized as conditioned cognitions, which can themselves be associated with positive experiences, or positive subjective responses, to alcohol or other drugs. Positive expectancies can facilitate more frequent use and thus contribute to the development of dependence (Rotgers, 1996). Expectancy theory has been supported by research. For example, expectancies of social benefit can influence adolescents' decisions to start drinking and predict their consumption of alcohol (Christiansen, Smith, Roehling, & Goldman, 1989) and alcohol-related problems (Moulton, M., Moulton, P., Whittington, & Cosio, 2000).

Sociocultural Factors in the Etiology of Substance Abuse What is ultimately labeled alcoholism or drug addiction varies based on the temporal, geographic, and religious context. In the mid 1800s, the average American consumed roughly three times more alcohol than he or she consumes today. Thus, what would be considered alcoholic drinking then would differ substantially from our present-day definition. During the 1600s and much of the 1700s, the alcohol and brewing industry was at its peak in Massachusetts Bay Colony and alcohol was not even seen as an addictive substance and habitual drunkenness was not, by and large, seen as problematic (McWilliams, 1998). The heavy use of alcohol was also seen in New Zealand in the 18th century (McEwan, Campbell, & Swain, 2010).

Additionally, the effect of cultural attitudes toward drinking is well illustrated by Mormons and Muslims, whose religious values prohibit the use of alcohol. As such, the prevalence of alcoholism, using standard diagnostic systems is extremely low in these groups. Any drinking is considered problematic use by members of these religious groups. This is not dissimilar from many cultures that consider any use of "hard drugs" (e.g., heroin, cocaine) to be problematic use.

Although alcohol plays a significant role in some Jewish family rituals (Lawson, A. & Lawson, G. 1998), excessive consumption is viewed as inexcusable behavior. Thus, within the Jewish culture, norms are for frequent drinking of alcohol, but in small amounts. In general, among cultures where drinking is integrated into religious rites and social customs and where self-control, sociability, and "knowing how to hold one's liquor" are important, alcoholism is rare (Drerup, 2005).

In comparison, the prevalence of alcoholism is high in Europe and those countries that have been highly influenced by European culture (i.e., Argentina, Canada, Chile, Japan, the United States, and New Zealand). Although these countries make up less than 20% of the world's population, the adults, ages 15 years and older, consume 80% of the alcohol (World Health Organization, 2011). In particular, the French have the highest rate of alcoholism in the world (i.e., roughly 15% of the population); France is also marked by the highest per capita alcohol consumption and the highest death rate from cirrhosis of the liver.[1]

It is also generally accepted that Irish Catholics have a comparatively high rate of alcoholism (Lawson & Lawson, 1998). Vaillant (1983) found that Irish subjects in his study on the longitudinal course of alcoholism were

more likely to develop alcohol problems than were those from other ethnic groups. Interestingly, Irish subjects were also more likely to abstain as a way of controlling drinking (O'Dwyer, 2001). As noted by Vaillant (1983), "It is consistent with Irish culture to view alcohol in terms of black or white, good or evil, drunkenness or complete abstinence" (p. 226). Viewing drinking behavior dichotomously, as either good or sinful, may serve to eliminate models of social drinking.

Studies have found consistent differences between cultures that generally engage in moderate drinking and those where a disproportionately large percentage of its members appear to have drinking problems (Maloff, Becker, Fonaroff, & Rodin, 1982; Peele & Brodsky, 1996). Cultural groups with comparatively low rates of alcoholism share four characteristics regarding alcohol use. First, drinking is accepted and is governed by social custom; thus, individuals in these cultures learn constructive norms for drinking. Second, differences between "good" and "bad" patterns of drinking are explicitly taught. Third, skills for drinking responsibly are taught. Fourth, drunkenness and misbehavior under the influence of alcohol are disapproved. In cultures where alcohol consumption is more problematic, agreed-on social standards for alcohol use have not been established, so drinkers must rely on an internal standard or their peer group's standards. Finally, people in these cultures expect that alcohol will overpower the individual's capacity for self-management.

Social customs and cultural norms have an enormous influence on substance use. In addition, the behavior that is displayed while under the influence of psychoactive substances also appears to be affected by cultural factors. For example, Lindman and Lang (1994) studied alcohol-related behavior in eight countries and found that although participants generally believed aggression would follow many alcoholic drinks, there were significant national differences in the expectancy that excessive alcohol consumption leads to aggression. These differences were unrelated to self-reported alcoholic beverage preference, frequency of drinking to intoxication, or rates of personal involvement in episodes of alcohol-related aggression. Thus, the expectation that drinking leads to aggression is determined to a significant extent by contextual factors and cultural traditions related to alcohol use. Previous studies using real and mock alcoholic beverages have shown that individuals who believe they have consumed alcohol begin to act more aggressively regardless of the beverage they actually consumed (Bushman, 1997).

Thus, how we learn to drink alcohol and consume other drugs is determined largely by the drinking and drug use we observe and by the people with whom we engage in these behaviors. There is a strong interdependence between the drinking and drug use patterns of those who associate with each other regularly in the same social circle. Indeed, in the broadest sense, each individual is linked, to a greater or lesser extent, to all other members of his or her culture. It is within this social fabric that patterns of belief and behaviors about alcohol and other drug use are modeled through a combination of examples, reinforcements, punishments, encouragement, and the many other means that societies use to communicate norms, attitudes, and values (Heath, 1982).

It is important to note that alcohol and drug abuse are not mutually exclusive (Hasin, Stinson, Ogburn, & Grant, 2007). As noted by the National Epidemiological Survey of Alcohol and Related Conditions, 5.6% of adults in the U.S. population reported both alcohol and drug use in the past year. Moreover, findings also revealed that co-use of alcohol and drugs was highest among the 18–24 age group.

Treatment

Given that conceptualizations of addiction have varied throughout history, different treatment approaches have predominated based largely on the theoretical model holding sway. At present, the range of treatment possibilities is fairly extensive, varying greatly in terms of philosophy and general treatment goals. Consequently, those seeking help now have a broad variety of choices in their attempts to resolve substance abuse problems.

Pharmacotherapy Intuitively, a reasonable solution to drug or alcohol addiction is to identify molecules that oppose the actions of these substances. Much scientific effort has been put forth based on this idea and the results are manifested in the large variety of pharmacotherapies now available to treat addictive behavior. These include medications to reduce cravings to use drugs, to reduce the reinforcing effects of the psychoactive substances, to make taking drugs aversive, and to treat co-occurring mental disorders that may potentially underlie the drinking or drug use (Romach & Sellers, 1998).

Several pharmacotherapies have been developed for the treatment of alcoholism. Disulfiram (Antabuse®) produces a sensitivity to alcohol which results in a highly unpleasant reaction when the patient ingests even small amounts of alcohol. Disulfiram blocks the oxidation of alcohol at the acetaldehyde stage. Accumulation of acetaldehyde in the blood produces a complex of highly unpleasant reactions such as flushing, throbbing in head and neck, throbbing headache, respiratory difficulty, nausea, copious vomiting, sweating, thirst, chest pain, and so forth.

The effectiveness of disulfiram has been mixed, however, particularly if used as the sole intervention. Because it is generally self-administered, the alcoholic client can simply cease taking the disulfiram, wait for the drug to leave his or her system (roughly 2 weeks), and resume drinking. In fact, controlled clinical trials indicate that, unless its administration is supervised (e.g., by a family member or treatment provider), disulfiram does not significantly improve abstinence rates compared to placebo

(e.g., Hughes & Cook, 1997). In general, patients that seem to respond well to disulfiram treatment are characterized as being older, possessing longer drinking histories, having social stability, and motivated for recovery (Fuller & Gardis, 2004; Suh, Pettinati, Kampman, & O'Brien, 2006). Given the potential serious adverse response resulting when the medication is combined with alcohol, some have suggested that disulfiram be used only in abstinence-based programs (Arias & Kranzler, 2008).

Another medication used to treat alcoholism is naltrexone, which helps to reduce craving for alcohol by blocking the pleasure-producing effects of ethanol. O'Malley and colleagues (1996) have shown that use of naltrexone reduced alcohol intake and lowered the incentive to drink for alcoholics compared with those given a placebo. Naltrexone has been most consistently found to be effective in reducing heavy drinking, with less support for reductions in percentage of drinking days or in increasing the likelihood of abstinence (Bouza, Angeles, Munoz, & Amate, 2004; Srisurapanont & Jarusuraisin, 2005). Although some studies have found naltrexone to be ineffective (Krystal, Cramer, Krol, Kirk, & Rosenheck, 2001), the majority of trials to date support its efficacy and safety (see Bouza et al., 2004).

Acamprosate has also demonstrated effectiveness in treating alcohol addiction. In a meta-analytic review of 17 studies, 6-month continuous abstinence rates were significantly higher for those taking acamprosate compared to a placebo (Mann, Chabac, Lehert, Potgieter, & Henning, 2004). Acamprosate appears to reduce craving for alcohol, the mechanism underlying acamprosate's action remains unknown, although some evidence suggests that it blocks the glutamate receptor (Harris et al., 2002).

Finally, antidepressant medications have been used to treat alcohol-dependent individuals with co-occurring depression. Mason, Kocsis, Ritvo, and Cutler (1996) reported that tricyclic antidepressants reduced depressive symptoms, and, to a certain extent, drinking behavior. Others have found that selective serotonin reuptake inhibitors, such as fluoxetine (Prozac®), reduce the frequency of drinking among alcohol-dependent patients with major depression (e.g., Cornelius et al., 1997).

Significant progress also has been made in the pharmacotherapy of opiate addiction. The most effective and commonly used pharmacotherapies for opiate addiction involve the use of agonists (i.e., drugs that occupy and activate the same receptors as opiates). The approach involves administration of drugs with similar action to those of the abuse drug, but with different pharmacotherapeutic effects (e.g., longer acting, less reinforcing, decreased euphoria).

Two widely used agonists, methadone and L-alpha-acetylmethadol (LAAM), have been approved in the United States to treat opiate dependence and are only available through licensed programs that closely monitor the patient's substance use and may also provide psychiatric help as needed (Arias & Kranzler, 2008). Methadone and LAAM reduce the subjective effects of heroin and other opiates through cross-tolerance. The usefulness of these two agonists lies in the fact that they satisfy the craving for heroin or other opiate-based illicit drugs; however, it is important to note that they are equally physiologically addictive. Thus, the advantage of methadone and LAAM over heroin is that methadone and LAAM are administered in a controlled environment as part of treatment. Methadone and LAAM are often combined with other psychosocial treatments to be fully effective (e.g., McLellan et al., 1993; Fals-Stewart, O'Farrell, & Birchler, 2001). LAAM is similar to methadone, but is administered every 3 days rather than daily. The main difficulty with LAAM, however, is that it takes time to achieve initial stabilization, thus increasing relapse risk (Jaffe, 1995).

Both LAAM and methadone also reduce such concomitant negative behaviors as crime and needle sharing and seem to enable those dependent on opiates to function well enough to maintain employment and other social obligations. Many who are involved in LAAM or methadone maintenance programs are also able to function in their communities and within their families. The quality and dosing of LAAM and methadone is strictly controlled via well-monitored government standards compared with that of street heroin and other illegal opiate-based substances. Yet, the practice of weaning drug abusers from heroin only to addict them to a government-controlled substance is considered by many to be morally and ethically questionable. Moreover, LAAM and methadone maintenance treatments are often difficult for participants to manage because they may require secrecy from employers, friends, and family members, owing to concerns about the stigma associated with being maintained on these drugs. It can also be difficult for participants to develop a drug-free social network—a task that many see as a crucial part of successful recovery.

Buprenorphine, an opioid agonist approved in 2002 by the Food and Drug Administration (FDA) for the treatment of opiate addiction, blocks the subjective effects of heroin and other opiates and has been shown to reduce heroin use and increase compliance with psychosocial treatment (Strain, Stitzer, Liebson, & Bigelow, 1994). Compared with methadone, an advantage of buprenorphine is that it has less potential for abuse and a lower risk of overdose. In addition, it can be obtained by prescription from a psychiatrist or primary-care physician who has completed approved training, rather than through attendance at a specialized methadone clinic (O'Malley & Kosten, 2006).

Naltrexone is another approved treatment for opiate addiction. Naltrexone is a competitive antagonist (i.e., it binds to receptors), but instead of activating these receptors as agonists do, it blocks them. Thus, naltrexone works by preventing receptors from being activated by heroin and other opiates. Yet, unlike its use in alcohol treatment, naltrexone has not been very successful in treating opiate dependence, largely because of poor patient compliance (O'Brien & McLellan, 1996). Naltrexone appears to be

effective with individuals highly motivated to quit, particularly those in high-level professional positions (attorneys, physicians, etc; Meandzija & Kosten, 1994), and as part of other psychosocial treatments that include patient monitoring (e.g., Fals-Stewart & O'Farrell, 2003).

Although over 50 different medications have been tried as treatments for cocaine and amphetamine addiction, none is presently FDA approved and none has been demonstrated to be effective (e.g., Mendelson & Mello, 1996). No medications are yet available to treat clients suffering from abuse of other drugs, such as cannabis, phencyclidine, and inhalants (Wilkins & Gorelick, 1994).

Psychosocial Interventions Although medications are an important part of the treatment armamentarium of clinicians working with clients suffering from psychoactive substance-use disorders, most experts agree that the mainstay of treatment for addiction is some form of peer support or psychosocial therapy. AA and other 12-step peer support groups (e.g., Narcotics Anonymous, Cocaine Anonymous) are the largest and most widely known self-help support groups for treating persons with alcohol and other drug problems. These programs operate primarily as self-help counseling programs in which both person-to-person and group relationships are emphasized. Meetings consist mainly of discussions of participants' problems with alcohol and other drugs, with testimonials from those who have recovered. Participants are encouraged to "work" the 12 steps of AA, which include admitting powerlessness over alcohol, believing a higher power can restore sanity, and developing a searching and fearless moral inventory of oneself (Alcoholics Anonymous, 1976). AA promotes total abstinence from alcohol and drugs. Although widely used, evidence for the effectiveness of AA and the related peer support groups is mostly anecdotal rather than based on well-controlled studies, largely because AA does not endorse participation in research. However, Fiorentine (1999) reported that affiliation with AA after outpatient treatment was associated with better outcomes than non-AA involvement.

A commonly used formal treatment approach derived from AA and the disease model is 12-Step Facilitation (TSF). In this approach, substance dependence is viewed not as symptomatic of another illness, but as a primary problem with biological, emotional, and spiritual underpinnings and presenting features. Alcoholism and drug abuse are seen as progressive illnesses, marked largely by denial. The primary goals of treatment are to encourage clients to work through their denial and work the 12 steps of AA. This is typically done in the context of individual and group therapy and involves strong encouragement to attend twelve-step self-help groups on a regular basis. Along with individual and group counseling, medical and religious services are also considered important parts of treatment because the disease of alcoholism and other drug use is viewed as affecting the biological and spiritual realms, as well as psychosocial functioning. Despite being the most common form of treatment in the United States, relatively little research has been conducted on the efficacy of TSF as compared with other forms of treatment. In a review of TSF as compared to other psychosocial interventions, Ferri Amato, and Davoli (2006) found limited evidence that AA participation is more effective than alternative treatments.

One of the strongest predictors of success of treatment for alcoholism has been motivation to change. A treatment intervention that has grown out of this observation is motivational enhancement therapy (MET), which attempts to engage clients who are resistant to behavior change and is considered the most acceptable approach for new patients (Arias & Kranzler, 2008). A central technique of MET is motivational interviewing (Gray & Zide, 2006), which is defined as a directive, client-centered therapy style designed to elicit change by assisting clients with exploring and resolving ambivalence. Several studies have now demonstrated that MET is an effective treatment for alcoholism and other drug abuse (e.g., Vasilaki, Hosier, & Cox, 2006).

Cognitive and behavioral treatments have been among the most widely used and investigated psychosocial treatments for substance dependence. Cognitive-behavioral therapy (CBT) teaches clients coping skills to reduce or eliminate drinking or substance use. Techniques that characterize CBT include identifying high-risk situations for relapse, instruction and rehearsal strategies for coping with those situations, self-monitoring and behavioral analysis of substance use, strategies for recognizing and coping with cravings, coping with lapses, and instruction on problem solving (Carroll & Onken, 2005).

It has long been maintained by providers and researchers alike that treatment for substance-use disorders would be more effective if important patient characteristics were taken into account in selecting treatments (e.g., Mattson et al., 1994). This hypothesis was tested in the most comprehensive study of patient–treatment matching for alcoholism, Project MATCH. In this investigation, the efficacy of TSF, MET, and CBT were compared. Overall, the study involved 1,726 participants who were treated in 26 alcohol treatment programs in the United States by 80 different therapists. The investigators evaluated patients on 10 characteristics shown to predict treatment outcome (Project MATCH Group, 1997): diagnosis, cognitive impairment, conceptual ability level, gender, desire to seek meaning in life, motivation, psychiatric severity, severity of alcohol involvement, social support for drinking versus abstinence, and the presence of antisocial personality disorder. The results of the study were somewhat surprising to many in the treatment and research communities—matching the patients to particular treatments did not appear to influence treatment effectiveness. TSF, MET, and CBT were shown to be equally effective across multiple domains of functioning. One conclusion drawn from these findings was that clients receiving interventions in competently run

programs will do equally well with any of the three treatments (Cooney, Babor, DiClemente, & Del Boca, 2003).

Relapse Prevention Without question, one of the most vexing problems facing substance abuse treatment providers is relapse. Polich, Armor, and Braiker (1981) found that, over a 4-year post-treatment period, only 7% of their total sample (i.e., 922 males) abstained from alcohol during the entire follow-up interval. Given that the goal of treatment was abstinence, 7% is a disturbingly low rate of success. Equally disturbing, 54% continued to show alcohol-related problems. Thus, increasing the long-term benefits made during any primary intervention has become a major part of overall treatment planning. An effective cognitive-behavioral approach to preventing relapse, introducing "mindfulness meditation," is described in the work of Witkiewitz, Marlatt, and Walker (2005), in which relapse is viewed as a key behavior in substance abuse treatment. The behaviors underlying relapse are seen as indulgent behaviors based on an individual's learning history. When a person is abstinent, he or she gains a greater sense of personal control over the indulgent behaviors. The longer the person stays abstinent, the greater his or her sense of self-efficacy. In this model, relapse is a process that begins with a series of small, seemingly irrelevant decisions, even while maintaining abstinence. These decisions make relapse inevitable. For example, an alcoholic who buys beer to keep in his house to be prepared for visits by friends who drink has made a decision that may ultimately lead to relapse. Hodge (2011) introduced an approach that incorporates spiritual beliefs and practices into treatment with cognitive-behavioral therapy, enhancing its effectiveness.

Another relapse behavior that is also often observed involves the abstinence violation effect, in which any use of drugs or alcohol is viewed by the substance abuser as complete failure. In many respects, this notion is advocated by a large number of treatment programs and grows from an axiom often heard at AA meetings: "One drink, one drunk." Thus, when a substance user who has been abstinent for an extended period drinks or uses drugs, because the goal of complete abstinence has been violated, the individual's self-efficacy for abstinence plummets, and he or she may assume that a return to regular use of the drug is inevitable and then behave in a way that fulfills this prophecy.

As part of the relapse prevention program, clients are taught to recognize the apparently irrelevant decisions that serve as warning signals of the possibility of relapse. High-risk situations are identified and targeted, and the individuals learn to assess their own vulnerability to relapse. To counter the abstinence violation effect, clients are also taught not to become excessively discouraged if they do relapse. In this respect, clients are taught that relapse is part of the recovery process and are encouraged to develop plans to address relapse when it happen.

Controlled Drinking: A Controversial Outcome Some interventions for drug and alcohol abuse are based on the hypothesis that some individuals need not give up drinking or drug use, but can learn to use moderately (Lang & Kidorf, 1990; Sobell & Sobell, 1995). Several approaches have been used to teach controlled drinking, and some research has suggested that certain alcohol-dependent individuals can control their drinking. For example, self-control training techniques (e.g., behavioral self-control training), in which the goal of treatment is to have alcohol-dependent clients reduce their drinking without necessarily abstaining from alcohol, has much appeal for some clients (Walters, 2000). A computer program that provides instruction on self-control training and has been shown in a well-controlled study to reduce problem drinking (Hester & Delaney, 1997). Improved working memory through training of the working memory restores control over drinking behavior and has been found to reduce alcohol consumption, particularly in those with strong automatic impulses for drinking (Houben, Wiers, & Jansen, 2011). In general, the best candidates for this type of intervention are young, motivated clients with no biomedical impairment from alcohol abuse (Thombs, 2006).

In the United States, the vast majority of treatment professionals have rejected the idea that substance abusers can control their drinking or drug use and thus insist on a total abstinence approach. However, controlled drinking interventions are less controversial in other parts of the world (e.g., the Netherlands, Australia), and have been used effectively in other countries (e.g., Dawe & Richmond, 1997). The debate regarding controlled drinking as an acceptable treatment goal has raged for over 25 years. Some investigators have noted that controlled drinking can be efficacious (Heather, 1995; Kahler, 1995), whereas others have argued that alcohol-dependent individuals cannot maintain control over drinking and, as such, controlled drinking is not an acceptable treatment objective (Glatt, 1995).

Conclusion

Substance-use disorders are among the most pressing and intransigent mental health problems facing society today. Concerns about abuse of alcohol, tobacco, and other psychoactive substances date as far back as Biblical times: "He shall separate himself from wine and strong drink, and shall drink no vinegar of wine, or vinegar of strong drink, neither shall he drink any liquor of grapes, nor eat moist grapes, or dried" (Numbers 6:3). Despite such a long history, scientific scrutiny of these problems is a fairly recent phenomenon. However, a large and evolving body of literature about the epidemiology, etiology, neurobiology, and treatment of addictive behavior has accumulated. These and other aspects of addictive behavior continue to be the focus of extensive clinical and experimental research.

The study of substance use and misuse has been marked by extensive controversy and heated debate. These

conflicts have been fueled by a fundamental disagreement among scientists, clinicians, social policy makers, and the public about whether to view addiction as a disease in need of medical treatment, as sin in need of punishment and containment, or as learned behaviors that can be modified by contingencies. The debate has important implications for research, treatment, and social policy. For example, interventions shown to be effective in research settings are often ignored in the treatment community because they are viewed as philosophically opposed to a conventional wisdom about what works best. Unfortunately, the beliefs about what constitutes effective treatment are often not supported by the empirical research. Those who view alcoholism and other drug use as diseases are ethically and, in some instances, morally opposed to the use of controlled use treatments, which they view as irresponsible. Drug users fill our criminal justice system, while treatment providers lament the criminalization of these "diseases." Yet, proponents of legalization of drugs, who are in the minority, note that legalization will help contain many of the social ills associated with substance use. Others see such a stance as irresponsible and immoral because it would increase the exposure of those with a genetic propensity for addiction to the substances that would activate the addictive process. In turn, they believe that such policies would contribute to greater social decay. Because the emotional, legal, and economic stakes in the debate are so high, no end to the debate is in sight.

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Note

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15

Somatic Symptom and Related Disorders

Michael J. Zvolensky, Georg H. Eifert, and Lorra Garey

Individuals exhibiting multiple somatic symptoms often present to medical practitioners believing that they are physically ill, yet upon evaluation, they are informed that there is no known physiological source underlying their reports of distress. Although many of these patients will be satisfied with negative medical examination results, a significant subgroup will anxiously continue to worry about these physical symptoms—a phenomenon traditionally known as somatization. Somatization denotes the presence of physical symptoms (e.g., chest pain) for which a demonstrable disease process or bodily oriented pathology is not identified, but which cause distress for and impairment to the individual. Individuals who do not receive a medical diagnosis for their symptoms are likely to continue to seek help for their physical symptoms, demand more physical examinations and specialist referrals, undergo costly laboratory tests, and in rare cases, even end up on an operating table (Harth & Hermes, 2007; Warwick & Salkovskis, 1990). At the extreme, such somatization behavior can interfere with life activities and goals, resulting in clinically significant impairment—a phenomenon typically classified by the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) as *somatic symptom disorder*. Yet, somatization processes frequently occur in other "somatic disorders," including conversion disorder, illness anxiety disorder, and factitious disorder, as well as many other psychiatric conditions (e.g., panic disorder, major depressive disorder; DSM-5, 2013).

Somatization is a relatively common occurrence in the general medical system. Moreover, several studies suggest that approximately 30% of somatic symptoms in primary care and related clinic settings are medically unexplained (Khan, Khan, & Kroenke, 2000; Kroenke & Price, 1993; Marple, Kroenke, Lucey, Wilder, & Lucas, 1997; Narrow, Rae, Robins, & Regier, 2002; Smith et al., 2003). Further, somatization appears to maintain cross-cultural and cross-national applicability (Zvolensky, Feldner, Eifert, Vujanovic, & Solomon, 2008).

Somatization is associated with a high degree of personal suffering measured in both human and financial terms. For example, Katon, Sullivan, and Walker (2001) analyzed several studies with community, primary care, and medical specialty samples and determined that individuals with unexplained somatic symptoms experienced severe personal distress and corresponding life impairment. Somatizing individuals have higher rates of inpatient and outpatient healthcare utilization, and incur correspondingly higher inpatient and outpatient medical costs (Barsky, Orav, & Bates, 2005; Hansen, Fink, Frydenberg, & Oxhoj, 2002). In the United States, some estimates suggest the cost associated with treating somatization at approximately $256 billion a year (Barsky et al., 2005).

Importantly, somatization does not rule out the possibility of a true physical illness. In fact, many physical healthcare problems, typically quite mild, can be found among individuals with somatization problems (e.g., hypertension). Thus, somatization denotes an excessive degree of worry about physical health and overuse of medical services, relative to the severity of *identifiable* illness. Perhaps not surprisingly, those individuals without a medical explanation for their suffering seem to experience more anxiety and depression than others with similar illness experiences that do have diagnoses (Katon et al.,

2001). This difference even held in one study that examined individuals who sacrificed extensive amounts of energy and time to their care (individuals in the top 10% of frequent primary care visitors); those without an identifiable illness reported a higher degree of physical, social, and mental impairments compared with their "diagnosable" counterparts (Vedsted, Fink, Sørensen, & Olesen, 2004). Typically, these impairments are long-standing (occur across most phases of life) and tend to be exacerbated by concurrent stressors in everyday life. Although somatization is indeed a vexing healthcare problem, this domain represents an exciting opportunity for researchers from diverse disciplines to work together in a collaborative manner to better understand the relations between "body and mind" and health and disease.

The main goal of the present chapter is to provide a contemporary overview of the process of somatization as it applies to somatic symptom disorders. To some extent, our discussion is necessarily organized around the categories and classification of somatic symptom and related disorders in DSM-5 (2013). It should be noted, however, that while the presented classifications of somatic symptom disorders are based on DSM-5, we incorporate elements of DSM-IV (American Psychiatric Association, 1994). The inclusion of the DSM-IV was necessary, given the very limited research on DSM-5 to date. Furthermore, we attempt to move beyond DSM categories toward a more function-based dimensional perspective of somatization problems. We believe that such an approach is potentially useful because it targets analyses towards fundamental biobehavioral processes and thereby provides information that is likely to be directly useful for the design of clinical interventions.

The first section of the chapter briefly reviews somatic symptom and related disorders from the DSM-5 perspective, highlighting the prevalence, nature, and the diagnostic validity of such diagnoses. We then outline how a dimensional perspective that focuses on key biobehavioral processes may be a more useful approach for understanding somatic disorders than the DSM's categorical approach. Next, we address some key vulnerability processes for somatic symptom disorders. Finally, we address how a *dimensional perspective* and a focus on dysfunctional processes related to illness behavior can be translated into treatments for somatic symptom disorders.

Classification, Prevalence, and Course of Somatic Symptom and Related Disorders

Considering the extensive DSM revisions regarding somatic symptom and related disorders, we believe it is necessary to highlight the most consequential changes early in this chapter. The somatic symptom and related disorders (DSM-5) category was previously titled *somatoform disorders* (DSM-IV). According to the DSM-5 (APA, 2013), *somatic symptom disorders* may or may not accompany diagnosed medical disorders; a diagnosis of a somatic symptom or related disorder is based on the endorsement of symptoms rather than the absence of a medical explanation. Further, DSM-5 criteria emphasizes that the diagnosis should be made in consideration of the way persons present and interpret the somatic symptoms. DSM-5 also presents updated, more distinct categories for this and related disorders, and no longer includes somatization disorder, hypochondriasis, pain disorder, or body dismorphic disorder as categories of somatic symptom disorder.

DSM-5 defines the common feature of somatic symptom disorders as the conspicuousness of somatic symptoms associated with significant distress and impairment. Abnormal and/or excessive thoughts, feelings, or behaviors regarding the somatic symptoms must result in significant distress or significantly disrupt daily life.

Although the exact prevalence of specific somatic symptom disorders is not known, it is estimated that around 5–7% of the general population has somatic symptom disorder (American Psychiatric Association, 2013). DSM-5 distinguishes between five somatic symptom and related disorders: somatic symptom disorder, illness anxiety disorder, conversion disorder, psychological factors affecting other medical conditions, and factitious disorder. We begin our discussion with a brief overview of these categories.

Somatic Symptom Disorder: Somatic symptom disorder is characterized by many physical complaints, with or without clear or known physical causes, accompanied by excessive thoughts, feelings, and behaviors regarding the physical complaints. The condition may last for many years and, in some cases, extend over the entire adult life span. To meet the DSM-5 diagnostic criteria, an individual needs to present distressing somatic symptoms or somatic symptoms that significantly interfere with the individual's ability to function in daily life, and maladaptive thoughts, feelings, or behaviors about the somatic symptoms. Additionally, symptoms must be persistent for typically more than 6 months. These symptoms lead to frequent and multiple medical consultations, complex medical history, and to alterations of the person's life-style. Physical and laboratory findings may or may not detect a plausible medical condition as the cause of the somatic symptoms. Its onset is in early adulthood, course is often chronic, and prognosis is generally regarded as poor.

Illness Anxiety Disorder: A new disorder, although somewhat similar to hypochondriasis, illness anxiety disorder is characterized by a preoccupation with having or acquiring a serious illness accompanied by substantial anxiety about health and disease. Somatic symptoms, if present, are only mild in intensity, but somatic symptoms may not be present. Patients frequently seek reassurance, check their bodies, and avoid illness-related situations. Informing patients of the absence of a disease process, or explaining the benign nature of the symptoms, does not reassure the patient, and may even heighten the individual's concerns (American Psychiatric Association, 2013).

Moreover, the renewed worry over symptoms may contribute to the individual continuing to overuse medical services. The onset of illness anxiety disorder is believed to be early to middle adulthood. The course of illness anxiety is presently unclear, but thought to be a chronic and relapsing condition.

Conversion Disorder (Functional Neurological Symptom Disorder): Conversion disorder is characterized by symptoms suggesting a neurological disorder with medical investigations and neurological examinations failing to identify a neurological or general medical disorder. At times, the particular symptoms may even be inconsistent with general neurological knowledge. Patients may present with any one or a combination of motor symptoms (e.g., paralysis), seizures or convulsions, and sensory deficits (e.g., blindness, anesthesia, and aphonia). An important requirement for the diagnosis is the temporal relation between conversion symptoms and a psychological stressor such as acute grief or victimization. Patients are typically unaware of any psychological basis for their symptoms and report being unable to control them. The diagnosis of conversion disorder is rare and difficult to establish with estimates ranging between 0.001% and 0.3% (1–300 per 100,000; American Psychiatric Association, 1994). Although conversion disorder may occur at any age, onset is typically in late childhood or early adulthood. Onset is often sudden and in response to conflicts or stressful situations such as unresolved grief and sexual trauma (Sar, Islam, & Öztürk, 2009).

Psychological Factors Affecting Other Medical Conditions: Psychological factors affecting other medical conditions are characterized by the adverse effect of one or more clinically significant psychological or behavioral factors on a medical condition. Specifically, psychological or behavioral factors such as psychological distress, patterns of interpersonal interaction, coping styles, and maladaptive health behaviors increase the risk for suffering, death, or disability (American Psychiatric Association, 2013). Psychological factors affecting other medical conditions can occur across the lifespan. The prevalence of the condition is unclear, but it is believed to be a more common diagnosis than somatic symptom disorder.

Factitious Disorder: Factitious disorder is the falsification of medical or psychological symptoms, associated with identified deception. Exaggeration, fabrication, simulation, and induction are ways in which individuals can falsify illness. Great psychological distress or functional impairment may develop in persons with factitious disorder, or those with factitious disorder may impose distress or impairment on others. Factitious disorder usually occurs in intermittent episodes. Onset is usually in early adulthood. The prevalence is estimated to be about 1% (American Psychiatric Association, 2013).

Diagnostic Validity of DSM-defined Somatic Symptom and Related Disorders As the diagnostic validity of DSM-5 has been relatively unexplored, we examine the diagnostic validity of DSM-IV. DSM-IV diagnostic validity of somatoform disorders (currently titled "Somatic Symptom and Related Disorders" in the DSM-5) in relation to each other as well as to other clinical syndromes was problematic and continues to be the focus of intense debates (Mayou, Kirmayer, Simon, & Sharpe, 2005; Löwe et al., 2008). Scholars frequently observed that conditions defined under somatoform disorders did not necessarily represent distinct conditions, and thus lead to difficulties distinguishing between symptoms of somatic disorders and physical health problems abound (Mayou et al., 2005). Developers of DSM-5 recognized and attempted to address this overlap, although it is unknown if the new classifications will provide clarity.

Furthermore, the distinctiveness of somatic disorders has been questioned repeatedly. As a category, somatic disorders continue to lack conceptual coherence and clearly defined diagnostic criteria. According to DSM-5, comorbidity is frequent and somatic symptom disorders diagnoses may accompany anxiety or depression disorders (American Psychiatric Association, 2013). Van der Feltz-Cornelis and van Balkom (2010) suggested that the DSM-5 committee completely abolish this category and simply re-categorize each subcategory so that it is subsumed under cosyndromal disorders (i.e., depressive disorders and anxiety disorders). Further, while categories are used to group items that share characteristics, scholars suggests that this category lacks a unifying principle (Sykes, 2012). The lack of a unifying principle adds further support for the abolishment of this category.

The general diagnostic criteria overlap between psychiatric disorders associated with somatic symptom disorders and somatic symptom disorders, leads to high rates of multiple diagnoses. Depression and somatization, for instance, are four times more likely to co-occur than not (Leiknes, Finset, Moum, & Sandanger, 2008). Löwe and colleagues (2008) reviewed several studies and concluded that overlap with depressive and/or anxiety disorders occurred in as many as 26–59% of cases. Mayou and colleagues (2005) point out that the subcategories are unreliable and arbitrary. Many of the subcategories fail to achieve acceptable standards of reliability, which can negatively affect patient's rights to medical-legal and insurance entitlements.

Moreover, Bass and Murphy (1995) questioned the distinction between somatic symptom disorders and personality disorders, given the high rates of comorbidity between the two. More recent research by Garcia-Campayo, Alda, Sobradiel, Olivan, and Pascual (2007) showed that individuals with somatic disorders were 2.2 times more likely to have a personality disorder than other individuals with psychiatric diagnoses. Chaturvedi, Desai and Shaligram (2006) note that somatization and *abnormal illness behavior* are intricately related. Perhaps new diagnostic standards should pay greater attention to an individual's actions, but then again, abnormal illness

behavior can develop anywhere, irrespective of a person's psychiatric diagnosis.

First (2011) argued that proposed revisions to DSM-5, which were eventually slightly revised, accepted, and integrated, present ambiguous and overlapping criteria for assessing somatic symptom disorder. Specifically, criteria B lists three items that are conceptually similar, leading to endorsement of multiple, related items. Given that DSM-5 retains the arbitrary threshold for number of symptoms to determine severity, clinicians may inappropriately specify the severity of the disorder due to the conceptual overlap in criteria.

Cultural influences on somatization processes are well documented (Ryder et al., 2008; Zvolensky et al., 2008). Based on a review of data from cross-cultural studies, Escobar and colleagues (2001) initially concluded that there is "considerable cultural variation in the expression of somatizing syndromes" (p. 226). Kirmayer and Sartorious (2009) have explained this variation as a function of different "symptom schemas" that reflect cultural conceptions of suffering and distress, and which are rooted in cultural causal explanations (e.g. cellular biology in Western medicine; the balance of the body's basic constituents—fire, earth, metal, water, wood—in traditional Korean culture; preservation of vital energy in Indian culture, etc.; p. 23). Thus, the specific symptoms of certain disorders often appear to be more a function of the individual's culture than of some underlying (distinct) biologically based disease process. For example, it would be helpful for clinicians to know that for some individuals of Indian descent, the experience of semen in the urine ("*dhat*") is frequently not a delusion but a somatization related to fatigue and depression, a representation of feeling sapped of "vital energy"; likewise, it would also be helpful to know that epigastric burning among individuals of Korean descent may not be heartburn but a somatic experience of intense, culturally inappropriate emotion ("*hwa-byong*" or "fire illness") (Kirmayer & Sartorious, 2009). Symptom lists for Europe and North America would, by contrast, focus on the most frequent areas of concern in these locations, heart disease and cancer (see López and Guarnaccia, Chapter 4 in this volume).

Mayou and colleagues (2005) note that the terms "somatoform" or "somatization" are often unacceptable to patients because they imply that the patients' symptoms are in their mind and "not real." Patients doubt that clinicians appreciate the reality and authenticity of their symptoms. At a more fundamental level, Mayou and associates (2005) criticized the distinction between somatic and psychological problems as inherently dualistic, which does not translate well into other cultures that have a less dualistic view of mind and body (e.g., Asia and China) than Western culture. The notion that problems can be neatly divided into those that can and cannot be explained by disease is indeed unlikely at best. Several authors also point to potential pathophysiological mechanisms that may underlie unexplained physical symptoms such as chest pain (Sharpe & Bass, 1992; Pilgrim & Wyss, 2008; Yilmaz et al., 2008). We often just do not understand them very well—and that is what we should tell patients, rather than using terms such as non-organic when describing their symptoms. We also need to recognize that there is frequently a reciprocal relation or "looping effect" between health anxiety and somatic symptoms, in which attending to sensations increases their salience and intensity (Kirmayer & Sartorious, 2009). This could occur either indirectly, through psychological processes, or directly, involving the behaviors designed to reduce anxiety over physical symptoms paradoxically increasing physical symptoms (e.g. constantly checking/scratching a lump increasing its size and irritation).

Although the DSM-5 attempts to address issues such as overlap and lack of clarity regarding boundaries for diagnoses, it still has many issues to overcome. DSM-5 recognizes the possible comorbidity between each category of somatic symptom disorders and other disorders, and proposes that concurrent diagnoses should be explored. Further, the differential diagnosis section tries to disentangle the overlapping features; however, the lack of clear, solid boundaries may result in patients being inappropriately diagnosed with a mental disorder and thus receiving inappropriate treatment (Sykes, 2012).

Toward a Dimensional Framework for Understanding Pathogenic Processes In light of the long-standing diagnostic problems with the somatic disorder category, Mayou and colleagues (2005) suggested abolishing this category in DSM-5 because the concept and category has consistently failed clinicians and patients alike. They suggested redistributing the somatic disorders to other diagnostic categories. More recently, Van der Feltz-Cornelis and Van Balkom (2010) suggested a similar notion. Löwe and colleagues (2008), on the other hand, have argued against a complete abolition of the somatic disorder category on the basis that, faulty or not, it has come to have huge personal, clinical, and societal importance, and abolishing the category would effectively abandon a large group of patients and exclude them from treatment. They argued for revisions that would integrate somatic disorders into the greater medical field, so that mental and physical health workers have identical, interdisciplinary diagnostic and treatment standards.

We previously argued (Eifert, Lejuez & Bouman, 1998; Eifert, Zvolensky & Lejuez, 2000) that overlap between related categories is not a problem of "comorbidity" or inaccurate definitions, but rather, a result of similar psychological processes involved in these conditions. Accordingly, we suggest adopting a dimensional approach to understanding illness-related concerns that can identify key biobehavioral processes. To illustrate this approach, we discuss focal dimensions of health anxiety, a psychological process that characterizes, in part, many somatic symptom disorders as well as related conditions

(e.g., panic disorder). We view health anxiety as a psychological process where persons present with problems that fall on a continuum along four dimensions:

1. Preoccupation with the body and its functioning. Such bodily preoccupation, especially when coupled with somatic complaints, may produce a state of somatic uncertainty and form the basis for the other three dimensions of the disorder.
2. Disease suspicion or conviction. The person has the suspicion or is convinced of having a serious physical disease; suspicion and conviction are on a continuum of strength, and in rare cases the conviction may reach delusional intensity.
3. Disease fear. The person fears having a serious physical disease.
4. Safety-seeking behavior such as repeated requests for medical examinations and tests, bodily checking, verbal complaints, and seeking reassurance. The function of such behavior is to reduce worry and anxiety over physical illness (Eifert et al. 2000; Salkovskis & Warwick, 2001).

A person could obtain a high score on any one or all four dimensions of health anxiety. For example, disease suspicion/conviction may or may not be accompanied by a strong fear of the suspected disease. Clinically, this feature is most apparent in patient's resistance to medical reassurance that nothing is wrong. This is particularly evident in a study by Rief, Heitmüller, Reisberg, and Rüddel (2006), which found that when patients with medically unexplained symptoms were asked to recall meetings with their doctor, the patients remembered a higher likelihood of medical explanations than their doctors actually gave. Accordingly, a dimensional classification system could help overcome some challenges inherent to a traditional diagnostic perspective of somatic disorders. Moreover, identifying dimensions that allow a classification of illness behavior based on the function that such behavior serves, rather than just its topography, might lead to a better understanding and improved treatments of persons with somatic problems (Eifert & Lau, 2001).

General Vulnerability Processes for Abnormal Illness Behavior

Given our previous discussion as to prototypical characteristics of health anxiety, the next logical question pertains to the types of processes that increase or decrease the risk for developing abnormal illness behavior. As discussed at the onset of this chapter, just about all people experience distressing physical sensations at some point in their lives. Moreover, a substantial percentage will even experience robust internal physical (interoceptive) reactions in the form of panic attacks, limited symptom panic attacks, gastrointestinal distress, respiratory infections, strained muscles, and so on. In fact, such bodily distress is so common to the human experience that it seems almost inconceivable to imagine a person going through life without experiencing at least some significant somatic disturbance. These normal experiences of physical symptoms become problematic when they begin to interfere with a person's life due to obsessive preoccupation with them and excessive rigid behaviors designed to control, reduce, or escape from them. Such pervasive experiential avoidance behavior seems to be at the core of many anxiety-related clinical problems (Forsyth, Eifert, & Barrios, 2006; Walker & Furer, 2008)—in fact, maladaptive avoidance behavior is a core feature of all anxiety disorders (Eifert & Forsyth, 2005; see also Williams, Chapter 9 in this volume).

Although systematic knowledge about causes is lacking, factors such as parental modeling, stressful life events, biological or genetic components, and greater cultural acceptance of physical versus mental illness appear to be related to the development of somatic disorders (Heinrich, 2004). One finding, that there is a decreased likelihood of somatic symptom diagnoses when primary care physicians have a more personal and long-term relationship with patients, also suggests that open, honest doctor–patient relationships protect against somatization diagnoses (Gureje, 2004). Difficulty in tolerating emotions (experiential avoidance) has also been shown to be associated with the development of somatization symptoms (Chawla & Ostafin, 2007). Difficulty expressing emotions (alexithymia) and negative affect/neuroticism are associated with symptom increase and persistence (De Gucht, 2002).

As these studies indicate, there are a number of biopsychosocial processes that increase vulnerability for the development of somatic symptom pathology or abnormal illness behavior generally. The processes that we focus on in this section include: (1) an inherited risk for emotional responsivity to physical sensations; (2) deficits in emotion regulatory skills; and (3) language-based and observational learning.

Before this discussion, however, we need to briefly define what we mean by the term *abnormal illness behavior*. Pilowsky (1993, p. 62) defined abnormal illness behavior as the "persistence of a maladaptive mode of experiencing, perceiving, evaluating, and responding to one's own health status" despite the fact that a doctor has conducted a comprehensive assessment of relevant biological, psychological, social and cultural factors and provided the patient with feedback about the results of these assessments and opportunities for discussion and clarification of the results. Thus, abnormal illness behavior essentially refers to the disagreement between the doctor and patient about the sick role to which the patient feels entitled. The concept of abnormal illness behavior is valuable for understanding not only patients with functional somatic symptoms but also the behavioral aspects of all illness (Chaturvedi et al., 2006).

Inherited Risk for Emotional Responsivity to Physical Sensations There is consistent empirical evidence for a strong inherited component or substrate for negative emotionality. Researchers have referred to this inherited disposition by a number of different terms, including *negative affectivity*, *trait anxiety*, and *neuroticism* (Barlow, 2001; Lau, Ely, & Stevenson, 2006). These synonymous labels are intended to capture a general predisposition to experience negative affect (e.g., anxiety) and perhaps abrupt emotional reactivity (e.g., panic) to challenging or stressful life events.

Although the characteristics of negative affectivity vary across specific negative emotional experiences (e.g., panic versus anger), they all posit that high degrees of negative emotionality are associated with a lower threshold of initial affective response, slower recovery to baseline, and greater reactivation of arousal with repeated exposure to stressful events. High reactivity is generally associated with inhibited temperament, whereas low reactivity is associated with uninhibited temperament (Kagan, 1989; Letcher, Smart, Sanson, & Toumbourou, 2009). Thus, individual differences in temperament are often related to the experience and regulation of affect because they constrain or facilitate certain types of responding. For example, the temperamental characteristic of neuroticism is related to tendencies to experience low levels of self-esteem in situations of interpersonal conflict, and global and day-to-day perceptions of relationships are more dependent on self-esteem in highly neurotic individuals than in others (Denissen & Penke, 2008).

As behavioral genetic research continues to make important strides in our understanding of emotional functioning, we may eventually have specific models articulating the extent to which a specific gene or combination of genes contributes to a specific anxiety disorder or health disorder. At this stage, however, it appears that a general disposition for negative affectivity is inherited. The contribution of this inherited component is estimated to be at approximately 30% in the development of somatic-related disorders (Kendler, Walters, Truett, & Heath, 1995). No research has documented the exact proportion of explained variance for the development of a specific disorder, although there is some evidence that there are both shared and specific genetic vulnerabilities across anxiety-related disorders (Smoller, Block, & Young, 2009). It is likely that a genetic predisposition creates the biological conditions that make people prone to respond with anxiety and panic reactions to certain bodily changes and processes (Walker & Furer, 2008, p. 369) One research challenge in this domain will be to specify how genetic vulnerabilities (once they are identified) might influence the pathogenesis of a specific disorder.

Development of Emotion Self-Regulatory Skills Research has shown that early in life humans develop a repertoire of regulatory oriented skills to deal with elevated levels of bodily arousal and concomitant emotional distress. For example, parents often distract distressed infants by orienting them to other stimuli, and children eventually learn to reorient and soothe themselves (Rueda & Rothbart, 2009, p. 26). Thus, the effective management of bodily states, particularly negative emotional experiences, is a critical early step in psychological development. As children mature, they learn to approach and avoid salient environmental stimuli, and the members of society increasingly expect them to gain better emotion regulatory skills (e.g., suppression of crying in school). By adulthood, the individual is expected to have sophisticated emotional control skills and the ability to learn new regulatory skills for a variety of changing sociocultural contexts. For example, adults are not supposed to demonstrate signs of emotional distress in social, performance, or work settings. Adults without emotion regulation skills often suffer in modern society. An interesting series of studies by Kantor-Martynuska (2009) provides some insights why this may be the case. She found that highly emotionally reactive individuals were more likely to prefer music at low volumes and to recall music as having been played loudly. This suggests that reactive individuals may have an increased sensitivity to arousing stimuli (music) and a tendency to experience it as aversive.

The preceding discussion highlights the notion that emotion regulation skills are expected to increase consistently across the lifespan and are an integral component of mental health (Stewart, Zvolensky, & Eifert, 2002). When individuals lack the ability to effectively alter their emotional experiences, they are more susceptible to physical discomfort, negative affect, and anxiety (Rothbart, Ziaie, & O'Boyle, 1992). An important way in which children learn emotional regulation skills is through exercising their capacity to explore their world, both literally and metaphorically. Developmental researchers originally referred to these experiences as *mastery learning opportunities* (Rothbart, 1989). Impoverished environments where parents or caregivers (e.g., teachers) respond to children in a non-contingent manner produce more emotional distress than do environments characterized by contingent outcomes. For example, van der Bruggen, Stams, Bögels, and Paulussen-Hoogeboom (2010) found that maternal rejection mediated a relationship between parent perceptions of children's negative emotionality and the children's anxiety/depression. The researchers hypothesize that maternal rejection instills a sense of helplessness in children, making them less likely to experience mastery of their negative feelings, and thereby increasing the likelihood that negative emotions eventually lead to symptoms of anxiety and depression. Similar findings have been observed in studies of non-human animals raised in environments with little control over important outcomes (e.g., availability of food; Mineka, Gunnar, & Champoux, 1986; Roma, Champoux, & Suomi, 2006). Such findings are important to understanding somatic disorders because greater degrees of perceived uncontrollability

are predictive of the tendency to view ambiguous internal stimuli as threatening (NASA Scientific and Technical Information Program Office, 2004; Zvolensky, Eifert, Lejuez, & McNeil, 1999).

Coping with emotional distress is, of course, a multidimensional process. Research indicates that coping responses are best viewed from a hierarchical model that includes first-order and higher-order dimensions (Compas et al., 2001). Indeed, researchers increasingly suggest that coping with emotional distress involves strategic (voluntary) and automatic (involuntary) responses. Additionally, Compas, Conner-Smith, Saltzman, Thomsen, and Wadsworth (2001) have categorized these coping responses along the dimensions of engagement and disengagement. Engagement responding includes active, primary control-oriented responding aimed at altering the immediate situation in some sort of direct manner (e.g., leaving a situation that one finds uncomfortable because of cardiac-related distress and tension). *Disengagement* responding includes secondary control-oriented responding aimed at adapting to an uncontrollable situation by purposively altering one's cognitive-affective response to that situation (indirect responding). For instance, an individual might adapt to pain or other aversive bodily sensations by altering their cognitive response to such events (e.g., acceptance, distraction, reframing). Overall, it is likely that individuals will develop a variety of emotion regulatory skills across the lifespan, and these skills are likely to be a product of early learning experiences.

Language-Based Learning Aside from direct forms of learning, individuals also will experience affective responses to body-related events and sensations through the utilization of language. Language serves important symbolic functions by providing humans with emotional experiences without exposure to the actual physical stimuli or events that ordinarily elicit those responses (Luoma, Hayes, & Walser, 2007; Staats & Eifert, 1990). For instance, both "knowing what to do" and "knowing what to feel" involve verbal understanding of the relation between them. Thus, the meaning of health-related anxiety in a psychological sense represents a complex act of relating largely arbitrary verbal symbolic events with other events and psychological functions within a particular context. For instance, words such as anxiety and fear either implicitly or explicitly establish relations with other events such as "I am anxious or afraid of . . . something, some event, or someone."

The relational quality of terms denoting emotions, in turn, must be tied to descriptions of behavior and events with a variety of stimulus functions (e.g., eliciting, evoking, reinforcing, and punishing) and meanings (e.g., good, bad, pleasant, unpleasant, painful). In turn, people often describe their emotional experiences metaphorically in ways that others can understand (e.g., "When I feel anxious it's like a knife going through my chest"). These metaphorical extensions have no real counterpart inside the person. Instead, they function to communicate the meaning of emotional experience (feeling threatened to the point of fearing death) by identifying and relating events with known stimulus functions (a sharp knife can cut into a chest and cause death).

Society determines what kind of stimuli and events are placed in relation to each other and the nature of that relation (Hayes & Wilson, 1994). These arbitrary relations are learned and function in a variety of ways (Sidman, 1994; Staats & Eifert, 1990). Individuals become anxious about particular health-related experiences or "symptoms" because they read and hear about them in the specific cultural context in which they live. This may be one of the main reasons why cultural variations in somatic disorders are widespread and can be observed with such great regularity (Escobar et al., 2001). For instance, people in Western societies become anxious when they notice a fast or irregular heart beat because they have seen or heard many times that this event may be a sign of a heart attack. In Southeast Asian and African cultures, it is quite common to observe a phenomenon that has been labeled "*koro*," which describes an individual overcome with the belief that his penis—or, in females, her nipples—are retracting or shrinking, with fear that the organ will disappear (Barlow, 2002). In contrast, this phenomenon is quite rare in Western culture. Thus, the overwhelming finding from cross-cultural studies is that the somatic manifestation and expression of emotional distress is universal but the focus of somatic concern may vary in different cultures (see López and Guarnaccia, Chapter 4 in this volume).

The most important point is that persons with somatic disorders have likely developed complex repertoires of verbal and other symbolic responses that elicit negative affect and serve as discriminative stimuli for escape or avoidance behavior (Staats & Eifert, 1990). Thus, for otherwise healthy people, the sensations of a beating heart or chest pain may lead to a sequence of verbal and autonomic events that result in the belief that they are having a heart attack (Eifert, 1992). In this instance, a fast or irregular heartbeat is not just a felt beating heart. Instead, it is an acquired and verbally mediated formulation of what it *means* to have a fast or irregular heartbeat or chest pain (e.g., "I have heart disease" or "I am suffering from a heart attack"). Not only may the person respond to such sensations by rushing to an emergency room, but also any other public or private stimulus events associated with this response may now acquire similar negative functions (e.g., physical exercise, smoking, working hard). In this way, a variety of behaviors and events can come to elicit the physiological event that the person then misconstrues as dangerous.

Observational Learning It is also likely that persons who develop somatic symptom disorders have been exposed to negative health-related events to a greater degree than

persons who do not develop these disorders. For example, some studies indicate that a significant number of persons with cardiophobia, defined as an irrational fear of heart disease, have observed heart disease and its potentially lethal effects (e.g., death) in relatives and close friends (Eifert et al., 2000). These persons had been exposed directly to the physical and emotionally painful consequences of heart disease. As a result, they may also have had more exposure to heart-focused perceptions and interpretations of physical symptoms and physiological processes.

Observational learning is strongly involved in learning pain tolerance, pain ratings, and non-verbal expressions of pain (Flor, Birbaumer, & Turk, 1990). Such observational learning may increase the likelihood of expressing and interpreting arousal and pain in later life as a heart problem because socially acquired perceptions and interpretations of symptoms largely determine how people deal with illness. For instance, if one or both parents have heart disease, children might observe their parent's response to a heart problem. If the behavior that is modeled is maladaptive (e.g., excessive illness behavior), these children will not only be more likely to respond to stress with increased cardiovascular activity, but they will have also learned maladaptive labeling and interpretation of such symptoms and have fewer adaptive coping skills.

Individuals afflicted with somatic symptom disorders also report poorer overall health (Eifert et al., 2000). Learned maladaptive coping skills may contribute to this observation. For example, men with more somatic symptoms tend to drink more alcohol (Vijayasiri, Richman, & Rospenda, 2012). This maladaptive coping strategy was possibly observed as a child and then carried forward in life as a means to cope, especially given that parental alcohol use predicts adolescent alcohol use (Kerr, Capaldi, Pears, & Owen, 2012) and adolescent alcohol use in turn predicts adult alcohol use (Patrick, Wray-Lake, Finlay, & Maggs, 2010). The true underlying process between maladaptive coping skills, health behaviors, and somatic symptoms, however, is complex and more research is needed to disentangle these relations.

Taken together, research suggests a variety of factors may promote the development of the type of abnormal illness behavior found in somatic symptom disorders. These processes are likely nonspecific in the sense that they increase the chance of negative emotional responding and poor affect regulatory strategies. Exposure to specific illnesses or to persons who model the potential dangers of certain physical disorders may increase an individual's general vulnerability. Continued research in each of these general domains will improve our ability to predict who will develop a specific type of somatic disorder.

General Treatment Strategies for Somatic Symptom Disorders

Cognitive-behavioral theories and research have been helpful in providing a fledgling basis for a better understanding and treatment of persons with somatization problems. Important progress has been made in particular for persons with health anxiety (Eifert & Lau, 2001; Salkovskis & Warwick, 2001) and chronic pain (e.g., Flor et al. 1990; Kerns, Thorn, & Dixon, 2006; Schermelleh, Eifert, Moosbrugger, & Frank, 1997). We now overview these treatment strategies at a general level and how they can be applied to specific types of somatic disorders.

Psychologically distressed patients who present with unexplained somatic symptoms are high users of medical care and their doctors regard them as frustrating and difficult to treat (Mayou, 2009). There is often a mismatch between the expectations of these patients and their doctors' abilities and communication skills. For instance, terms such as *functional heart problem*, *nervous heart*, *atypical chest pain*, and *pseudoangina*, when used to "diagnose" unexplained chest pain, can easily be misinterpreted by a patient who is determined to believe that some significant cardiac disease is being described (Eifert et al., 2000). Healthcare providers often feel frustrated and emotionally drained because these patients obviously need psychological support but resent being referred to a psychologist or psychiatrist.

Patients often perceive the use of diagnostic labels such as hypochondriasis as an insult because these labels seem to imply that the patients' problems are not real and "just in their head." The controversy surrounding hypochondriasis supported the removal of it and, to a degree, the placement of it with illness anxiety disorder (Mayou et al., 2005). This new "recategorization," however, may not yield a large degree of improvement.

Wainwright, Calnan, O'Neil, Winterbottom, and Watkins (2006) attribute the patient perceived unacceptability of a hypochondriasis diagnosis to modern society's moral "hierarchy of illness" in which observable, stoically born physical pathology is elevated and legitimized, and somatization is denigrated and thought of as "faking it" or malingering (p. 79). They therefore argue that accepting and understanding, rather than refuting or arguing with the patient's symptoms, is the more important strategy for engaging the patient in a therapeutic working relationship. Lipsitt and Starcevic (2006) similarly state that it is extremely important for healthcare providers to treat the somatizing patient in a warm, respectful manner, without prejudice based on previous assessments from other clinicians. Healthcare professionals can help make the patient feel listened to and let them know that they regard their symptoms as "real" and do not view them as "crazy." They can also ease the patient's anxieties and enhance the relationship with the patient by telling the patient that worrying about their symptoms is reasonable and legitimate. A solid and trusting working relationship is essential for optimal help. In the engagement stage of treatment, patients can also be told that there may be alternative explanations for the difficulties they are experiencing (e.g. pathophysiological pathways; Pilgrim & Wyss, 2008; Yilmaz et al., 2008). The general treatment strategy

is to test such alternative or benign medical explanations for symptoms and to conduct therapy in the context of an experiment that provides an opportunity for testing alternative hypotheses (Eifert & Lau, 2001; Salkovskis, 1996).

Rather than merely telling patients there is no "organic" reason for their chest pain, an explanation of symptoms that overcomes the non-organic–organic dualism provides the patient with a more acceptable rationale and reassurance (Eifert, Hodson, Tracey, Seville, & Gunawardane, 1996). For instance, to provide a patient with a credible explanation of how anxiety and chest-wall muscle tension can result in chest pain, patients may be given a chest-focused relaxation with electromyographic feedback that literally shows them how they can change their chest tension levels. Salkovskis and Warwick (2001) found that reassurance which only informs the patients that there is nothing organically wrong with their bodies will actually increase future reassurance-seeking rather than decrease it. Appropriate reassurance and feedback that provides the patient with new and alternative explanations is the key to successful treatment and may help prevent the development of chronic somatization problems. In a controlled case study, Eifert and Lau (2001) found that behavioral experiments were not only useful in developing alternative symptom explanations but also in teaching the patient to reassure herself rather than seeking therapist reassurance. The treatment strategies and techniques targeting the dimensions of abnormal illness behavior related to heart-focused anxiety are summarized in Table 15.1.

Medical professionals need to be trained to provide alternative explanations rather than vague pseudo-medical labels and to acknowledge rather than dismiss patients' concerns. At the same time, withholding unnecessary medication and medical examinations (response prevention for safety-seeking behavior) is a crucial part of treatment. For instance, in their interviews with British physicians, Wainwright and colleagues (2006) describe how some doctors have adopted patient-centered philosophies and view diagnosis as a negotiated process that can approach psychosocial causality covertly over several sessions, thereby balancing a doctor's ethical commitment to scientifically rigorous standards of practice while accommodating a patient's resistance to psychological explanations. The researchers also found, however, that many physicians still struggle when dealing with these issues, so the focus should remain on developing programs for health care providers that help them communicate more effectively with their patients. Early work focused on arming physicians with basic psychological intervention skills, such as empathic listening to complaints and symptoms (Goldberg, Gask, & O'Dowd, 1989; Rost, Kashner, & Smith, 1995; Smith, Rost, & Kashner, 1995). Physicians were also taught not to reinforce patients' abnormal illness behavior by providing expensive diagnostic procedures, surgeries, and hospitalizations (Rost et al., 1995; Smith et al., 1995). Withholding such procedures also prevents iatrogenic diseases and counteracts patients' disease convictions. Mayou (2009) recommends the use of a stepped-care approach, which involves mixing therapeutic intervention with medical reassessment at various set intervals. This should also help to counteract patients' disease convictions while still moving them forward in their treatment. The results of these physician-focused interventions suggest that this balanced approach to patient care can reduce healthcare costs and at the same time increase the psychological adjustment of patients.

Finally, it is essential that we move patients from an almost exclusive focus on symptoms, which may or may not be changeable and/or controllable, to goals and behaviors that are controllable and changeable. After an initial empathic discussion of symptoms, treatment needs to shift toward more changeable behavioral targets, such as occupational problem solving, social skills training,

TABLE 15.1

Key Treatment Components Targeting Various Dimensions and Symptoms of Heart-Focused Anxiety (adapted from Eifert and Lau, 2001)

Dimensions	Treatment Strategy	Techniques
Preoccupation with heart	Demonstrate that chest pain/heart sensations are not dangerous	Reduce avoidance/expose to cardiac-related stimuli
Disease fear	Extinction of fear and exposure to avoided activities	Exposure to interoceptive (particularly cardioceptive) cues
		Reinforce "dangerous" behavior (e.g., strenuous exercise)
Disease conviction	Testing alternative symptom explanations	Explain impact of anxiety and tension on body (chest pain)
		Conduct behavioral experiments to test hypotheses
		Review evidence for/against heart disease
		Review evidence for cardiac vs. tension chest pain
Safety/reassurance-seeking	Extinction of help and reassurance-seeking	Review results of previous tests
		Withhold reassurance
		Refuse further tests
		Do physical exercise while preventing pulse checking
Physical (panic) symptoms	Reduce chronic tension and overbreathing	Chest muscle relaxation
		Teach slow diaphragmatic vs. thoracic breathing

Note: Although this example deals with heart-focused anxiety, the process dimensions are adaptable and applicable to other somatization problems.

and quality of life enhancement (Rief, Hiller, Geissner, & Fichter, 1995; Mayou, 2009). Behavioral activation treatment programs (Lejuez, Hopko, & Hopko, 2001) appear to be valuable therapists' tools to help move patients in their "valued direction" (Hayes, Strosahl, & Wilson, 2000) and at the same time reduce negative affect.

Acceptance and commitment therapy (ACT), a new acceptance-based behavior therapy, has shown great promise in the treatment of chronic pain (Vowles, Wetherell, & Sorrell, 2009) and anxiety disorders (Eifert & Forsyth, 2005). In future, this approach could easily be extended to people suffering from all types of health-related anxiety and concerns because it directly targets the experiential avoidance behavior that tends to interfere with people's daily functioning. ACT helps people to refocus their efforts on areas in their lives where they can affect change, and where they are in control, rather than wasting their time with fruitless attempts to control or change bodily sensations and their evaluations of them, which are very difficult, if not impossible, to change. ACT provides very useful therapeutic tools for helping people reorient their lives away from managing symptoms, pain and disability to moving forward and living a life guided by their values—what truly matters to them (McKay, Forsyth, & Eifert, 2010).

Specific Treatment Recommendations

Somatic Symptom Disorder It may be most useful to think of somatic problems as requiring management rather than treatment (e.g., Bass & Benjamin, 1993). Early diagnosis and the prevention of unnecessary medical and surgical investigation are of primary importance. For instance, a study by Hoyer and colleagues (2008) has shown that the specific assessment of heart-focused anxiety may help identify individuals with elevated levels of heart-focused anxiety who might benefit from interventions to help them adjust to the effects of surgery and lingering cardiac problems. Most somatic symptom patients have specific expectations regarding treatment goals and procedures and try to persuade their doctors to follow their wishes for further medical investigations and treatments. Bass and Murphy (1990) state that treatment often involves long-term supportive psychotherapy and must be directed toward controlling the demands on medical care as well as the treatment of symptoms and social disability. They recommend the following four steps:

a. encourage a long-term supportive relationship with only one understanding primary care physician to prevent doctor-shopping and to coordinate all actions;
b. see patients on regular appointments rather than on demand to prevent reinforcement of illness behavior;
c. view patient's physical complaints as a form of communication rather than as evidence of disease; and
d. minimize the use of psychotropic drugs and/or analgesic medication.

In general, adaptive behavior is encouraged and promoted, whereas sick role behavior is ignored as much as possible. Kashner, Rost, Cohen, Anderson, and Smith (1995) focused on coping with the nature and consequences of the physical symptoms, general problem solving, and helping patients take more control of their lives. These authors found that eight sessions of brief group therapy improved physical and mental health at 1-year follow-up.

A randomized controlled trial examining a comprehensive cognitive-behavioral approach for dealing with medically unexplained physical symptoms was conducted by Speckens and colleagues (1995). A cognitive-behavioral intervention group of 39 general medical outpatients was compared with a control group of 40 patients receiving optimized medical care. Treatment included imaginary exposure and distraction techniques to break the vicious circles of cognitive avoidance and preoccupation; activity scheduling, exposure *in vivo*, and response prevention to decrease avoidance behavior; relaxation training, breathing exercises, and physical exercises; and problem-solving or social skills training to overcome any problems in interpersonal relationships. At both 6- and 12-month follow-up, the intervention group reported lower intensity and frequency of symptoms, reduced illness behavior, less sleep impairment, and fewer limitations in social and leisure activities than did the control group.

More recently, Allen, Woolfolk, Escobar, Gara, and Hammer (2006) and Escobar and colleagues (2007) conducted randomized controlled trials investigating the efficacy of cognitive behavior therapy interventions for the treatment of somatic symptom disorder. Both treatments were administered in a primary care setting, but in contrast to earlier studies, patients had to exhibit more severe somatic symptoms. Allen and associates (2006) found that trained, independent raters reported less severe somatic symptoms at follow-ups among patients treated with cognitive behavioral therapy. Escobar and associates (2007) observed similar finding in that patients treated with cognitive behavioral therapy had significant improvements in clinician-rated physical symptoms and depression.

Conversion Disorder An important first step in the treatment of conversion symptoms is their early recognition in which a physical examination plays a crucial role. In many cases, a positive diagnosis can be made on the basis of the rather untypical or bizarre symptoms. Since conversion symptoms vary widely across patients, treatment needs to be individualized. Identifying precipitating stressors is crucial so that patients can be taught more adaptive ways of coping with these stressors. Occasionally, manipulation of the patient's social environmental is necessary to reduce the influence of secondary gain, such as attention from family and friends. Partners and significant others may have to learn how to reinforce the patients non-symptomatic behavior.

Behavior therapy has shown some efficacy in the treatment of conversion disorder, particularly for patients that

primarily exhibit motor symptoms and gait disturbances (Lipsitt & Starcevic, 2006; Speed, 1996). The program consisted of providing or withholding reinforcement depending on the patient's attempt at normalizing gait and increasing motor movement through specific exercises.

Researchers have successfully utilized a variety of other treatments, including psychotherapy and hypnotherapy, to treat conversion disorder (see Rosebush & Mazurek, 2011). Moene, Spinhaven, Hoogduin, and VanDyck (2003) administered hypnosis to patients diagnosed with conversion disorder, motor type, and observed a significant improvement in behavioral symptoms and motor disability compared with a control group. It is worth noting, however, that the efficacy of conversion disorder treatments is inconclusive and inconsistent (Kroenke, 2007; Rosebush & Mazurek, 2011).

Conclusions

Somatic symptoms, complaints, and concerns are very common in the general population. These problems are costly to the individuals concerned in terms of distress and financial expense, as well as to society in terms of lost productivity and health care costs. Compared with other common psychological dysfunctions (e.g., anxiety and depression), our present conceptual understanding of somatic symptom and related disorders is poor, and comprehensive integrative models are still lacking.

One factor that has impeded a better understanding of the somatic symptom disorders is the unsatisfactory and somewhat arbitrary nature of the DSM-IV classification. In view of the conceptual and diagnostic confusion, vagueness, and imprecision detailed in the DSM-IV, we greatly anticipate the research to be done exploring the new classifications and appropriateness of language in the new DSM-5. "Comorbidity" of somatic symptom disorders with anxiety disorders and depression is not a diagnostic problem but an indication that there are similarities in the underlying psychopathological processes (Aikens, Zvolensky, & Eifert, 2001). We have outlined some commonalities in emotional dysregulation processes particularly in relation to anxiety. A practical consequence for researchers and clinicians is to give up their focus on the individual disorders and increase their efforts at identifying the common functions of symptoms in persons with different somatic problems (Eifert, 1996; Kirmayer & Sartorious, 2009).

The complex relationships between the physical and psychological aspects of somatic symptom disorders has led to much confusion. We caution against an overreliance upon medical diagnostic procedures and medical theory. At the same time, research and service delivery would benefit from a more balanced approach. This approach should focus not just on finding or excluding somatic abnormalities but also on combining current medical knowledge and diagnostic techniques with the psychological assessments of a patient's behavior, cognitive processes, and social relationships (cf. Fink, 1996; Löwe et al., 2008). In our work with cardiac patients we have observed how a simple reliance on one source of information (medical or psychological) was inadequate for many patients. Instead, it was the combination of sophisticated medical tests and psychological information that yielded the type of knowledge that was most useful for recommending and designing the most appropriate treatment for the individual patient.

Hence, perhaps one of the most compelling conclusions arising from this chapter is that somatic symptom disorders cannot be adequately understood, assessed, and treated from a single perspective. Both the classification and research could be improved by adopting a multidisciplinary approach and an integrated biopsychosocial perspective. For example, Mayou, Bass and Sharpe (1995) proposed a multidimensional classification of patients with functional somatic symptoms along five dimensions: (1) Number and type of somatic symptoms; (2) mental state (mood and psychiatric disorder); (3) cognitions (e.g., symptom misinterpretations, disease conviction); (4) behavioral and functional impairment (illness behavior, avoidance, use of health services); and (5) pathophysiological disturbance (organic diseases, physiological mechanisms such as hyperventilation). As described in the section on health anxiety, Löwe and colleagues (2008) present an alternate diagnostic system that focuses on the presence of behaviors, some of which are abnormal illness behaviors. Individuals should be assessed for: (1) Dysfunctional cognitions; (2) excessive healthcare use; (3) selective attention to bodily signals; (4) persistent attribution of symptoms to undiagnosed conditions; (5) avoidance and decreased activity; and (6) functional impairments due to somatization. These criteria are behavioral and would be of more immediate relevance for treatment than diagnosing.

Although DSM-5 integrated some elements similar to the recommendations described above, the utility and appropriateness of the wording and classification is still unknown. The issues outlined throughout this chapter regarding DSM-IV and DSM-5 support assessing somatic symptom disorder using tools other than the DSM. Thus, rather than attempting to find the "correct diagnosis," we recommend assessment along the crucial dimensions involved in the regulation of maladaptive illness behavior and devising treatment programs based on such assessments. This may be more valuable than diagnosing according to the number and type of physical complaints.

A multicausal perspective suggests several potentially fruitful lines for future psychological research into somatic disorders, such as an increased focus on information processing behavior (attribution, attention, and memory), and environmental contingencies for illness behavior (e.g., social, occupational, medical). Psychoneuroimmunological studies may help to clarify particular aspects of the nature of the interface between pathophysiological changes and individual responses

to such changes as exemplified in some chronic pain research (cf. Flor et al., 1990). Although the past emphasis on the problems of patients with no demonstrable physical pathology was worthwhile and deserves continued attention, the gray area of persons with some organic pathology, bodily symptoms, and psychological distress deserves greater recognition and needs to be investigated more carefully.

Treatment programs and outcomes are likely to be enhanced by an improved conceptual understanding of these problems. The need for better theories and treatments is even more pressing for those somatic problems that have been particularly neglected in the past such as conversion problems. The relative success of recent cognitive-behavioral treatment programs for persons with unexplained physical symptoms, health anxiety, or chronic pain is promising. These treatment successes may help change the common perception of healthcare providers that people with such problems are just a "pain in the neck" and invariably difficult, or even impossible, to treat.

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Dissociative Disorders

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From Jekyll and Hyde like shifts of feelings and behaviors to feelings of unreality, like "all the world's a stage" for actors seemingly playing a part, to amnesia for events that one should, by all rights remember, the symptoms of dissociative disorders have proven to be as fascinating as they are perplexing and controversial. Janet (1973) was perhaps the first to claim, in 1889, that dissociation (or "desagregation" as he termed it) originated in a defensive response to traumatic events and to appreciate the importance of studying dissociation in order to comprehend the full range of everyday and anomalous experiences (Cardeña, Lynn, & Krippner, 2014). Janet's contention that dissociation represents a coping strategy in response to highly aversive events continues to provoke vigorous debate, just as the notion of multiple personality disorder (now termed dissociative identity disorder) sparked Freud's skepticism in Janet's time. In this chapter, we examine the three major dissociative disorders—dissociation/derealization disorder, dissociative amnesia, and dissociative identity disorder, in turn. More specifically, we describe their symptoms, prevalence, and assessment, as well as current controversies regarding the genesis of dissociation and competing theories of their nature and origin and efforts to treat their vexing symptoms.

Dissociation and Dissociative Disorders

As described in the DSM-5 (American Psychiatric Association, 2013), dissociation can be defined as a "disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior" (p. 291). Dissociative experiences range from the mundane, such as occasional lapses or divisions in attention and memory, to fantasizing, daydreaming, becoming absorbed in movies and what is commonly referred to as "highway hypnosis" ("losing" lengthy periods of time while driving), to the profound and sometimes unpredictable shifts in consciousness that mark dissociative disorders. In some cases, it is difficult to distinguish pathological from nonpathological dissociation (Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008; Modestin & Erni, 2004; Waller, Putnam, & Carlson, 1996; Waller & Ross, 1997), and it is not altogether clear whether milder manifestations of dissociation share biological and etiological roots with more dysfunctional manifestations of dissociation (see Cardena, 1994; Lynn et al., 2014).

Although controversy persists regarding the origins of both mild and more pathological dissociation, there is little dispute that some individuals present with symptoms that fall under the rubric of "dissociation," as codified in DSM-5. In brief, the three major disorders in DSM-5 are:

1. *Depersonalization/derealization disorder* (with depersonalization being experiences of unreality, detachment, outside observer of one's thoughts, feelings, sensations or actions, and derealization being experiences of unreality or detachment with respect to surroundings);
2. *Dissociative amnesia*, the inability to recall important autobiographical information, usually of a traumatic or stressful nature, inconsistent with ordinary forgetting; and
3. *Dissociative identity disorder* (DID; formerly called multiple personality disorder), a striking disruption of identity characterized by two or more distinct personality states and recurrent gaps in the recall of everyday events.

To merit a diagnosis of a dissociative disorder, dissociative experiences or symptoms must (a) interfere with important areas of functioning, such as work and social relationships, (b) engender subjective distress, or both. Spiegel and colleagues (2011) classified pathological dissociative symptoms as "positive" or "negative," with positive symptoms comprising flashbacks and intrusions of an "aspect of identity" into awareness, and negative symptoms comprising "deficits in memory, sense of self, and/or the ability to sense or control different parts of the body" (p. 826).

Some researchers have suggested that a dissociative subtype of schizophrenia can be identified with prominent dissociative features, including amnesia and depersonalization/derealization (see Ross, 2008; Vogel, Braungardt, Grabe, Schneider, & Klauer, 2013). However, it is not clear to what extent dissociative symptoms merely reflect nonspecific symptoms associated with substantial comorbidity due to a psychotic disorder (Laferrière-Simard, Lecomte, & Ahoundova, 2014), or whether a dissociative subtype of schizophrenia will emerge as a valid entity pending future research.

Prevalence of Pathological Dissociation: Nonclinical and Clinical Samples Studies of the prevalence of people in nonclinical populations who report clinically significant symptoms of dissociation using well-validated measures of dissociation or structured interviews provide variable estimates of often non-trivial rates of dissociation. More specifically, the rates of pathological dissociation generally range from 0.3% (Spitzer, Barnow, Freyberger, & Grabe, 2006) to 2–3% in the general population (Seedat et al., 2003; Vanderlinden, Van Dyck, Vandereycken, & Vertommen, 1991; Waller & Ross, 1997), although Sar, Akyüz, and Dogan (2007) reported an "outlier" estimate of 18.3% lifetime prevalence of dissociative disorders among Turkish women sampled in the community, a rate perhaps attributable to unknown cultural factors.

Waller and Ross (1997) conducted an analysis of pathological dissociation in the general population in two North American samples and used biometric and taxonomic statistical procedures to determine that approximately 3.3% of the population belongs to a pathological dissociative taxon, as measured by an 8-item scale (DES-T) derived from the Dissociative Experience Scale (DES; Bernstein-Carlson & Putnam, 1986). In psychopathology, a taxon is a natural class; that is, a group that differs in kind rather than in degree from normality. Nevertheless, Watson (2003) found the taxon scores to be only modestly stable (test re-test correlation $r = 0.34$ after approximately 2 months), and most individuals identified as taxon members on one occasion failed to be so classified at re-test. Generally speaking, little is known about the test–re-test reliability of diagnoses of dissociative disorders and the impact of potential low reliability on prevalence estimates.

Few studies have examined the rates of dissociative disorders in college students. Sandberg and Lynn (1992) identified 33 female college students who scored in the upper 15% on the DES, a widely used self-report measure of dissociation (Bernstein-Carlson & Putnam, 1986). Two of these students met criteria for a dissociative disorder (i.e., psychogenic amnesia, or what would today be called dissociative amnesia; multiple personality disorder, or what would today be called dissociative identity disorder), although none of the 33 students who scored below the mean on the DES met criteria for a dissociative disorder. Eight participants who scored in the upper 2% on the DES did not meet criteria for a dissociative disorder.

The prevalence of dissociation in clinical populations tends to be much higher than in nonclinical populations, although significant variability is also evident in the former samples, ranging from lows of 12–17% (Lipsanen et al., 2004; Sar, Tutkun, Alyanak, Bakim, & Barai, 2000; Saxe et al., 1993) to higher estimates of lifetime prevalence of 28–40.9% in an inpatient setting (Ross, Duffy, & Ellason, 2002) to the highest estimate 34.9% in a psychiatric emergency facility (Sar, Akyüz, & Dogan, 2007). Prevalence rates of dissociative disorders also run high in special populations, with rates reported of all dissociative disorders of 80% (35% diagnosed with DID) in exotic dancers and 55% in prostitutes (Ross, Anderson, Heber, & Norton, 1990), and 17–39% in individuals with substance use disorders (for a review, see Sar, 2011). Overall, the findings regarding gender differences in dissociative diagnoses are mixed, with some studies finding a higher prevalence among women, and other studies finding no differences (Sar, 2011).

Suicide and self-mutilatory behaviors tend to be higher (completed suicide 1–2%) among patients with dissociative disorder, although the interpretation of such elevated rates is often complicated by substantial "comorbidity" (co-occurrence) of dissociative disorders with borderline personality disorder and other serious manifestations of psychopathology, including other personality disorders, anxiety disorders, and depressive disorders (Johnson, Cohen, Kasen, & Brook, 2006; Lynn et al., 2014). Sar Akyüz, Öztürk, and Alioğlu (2013) sampled 628 women in a Turkish community and identified "dissociative depression" among 40% of depressed women, a condition marked by symptoms of depression, increased suicidality and self-mutilation, and reports of childhood trauma. Most of the women did not meet diagnostic criteria for borderline personality or posttraumatic stress disorder (PTSD).

Reasons for the disparities of prevalence rates among studies are not clear, although such rates almost certainly vary as a function of the assessment instrument used, diagnostic base rates at different facilities, the presence and nature of comorbid symptoms, and examiner beliefs and biases regarding dissociation. Generally speaking, experimenter blindness regarding patient characteristics lowers the rate of diagnoses of dissociative disorders (Friedl, Draijer, & de Jonge, 2000).

Genetics and Dissociation Research supports a genetic substrate for the propensity toward dissociation, although

the evidence is not consistent. Jang, Paris, Zweig-Frank, and Livesley (1998) reported that 48% of the variability in DES-T scores is attributable to genetic influences,, and that the remainder of the variance (52%) can be attributed to nonshared environments; that is, environmental experiences that make members of the same family different from one another. When the researchers excluded taxon items and considered nonpathological dissociation scores, genetic influences accounted for 55% of the variance, whereas nonshared environmental influences accounted for 45% of the variance. In a study of children and adolescents, Becker-Blease and colleagues (2004) found a substantial genetic (59% genes, 41% nonshared environment) contribution to dissociation scores, although Waller and Ross (1997) found no evidence for genetic influences based on their research with 280 identical twins and 148 fraternal twins. Unfortunately, no genetic studies of diagnosed patients with dissociative disorders have been conducted. Twin and adoption studies would help to clarify the extent to which the familial clustering of dissociative disorders is due to genes, shared environment (environmental factors that boost family resemblance), or both.

Physiological and Neuroimaging Findings Research has not produced definitive conclusions regarding (a) the relation between dissociation and cortisol response, a commonly used measure of stress reactivity, and (b) the relations among dissociation, cardiovascular (heart rate), and autonomic (e.g., skin conductance response) reactions in response to trauma reminders (Dalenberg et al., 2012). Similar inconsistencies are present in neuroimaging studies in which "either increased or decreased response in medial prefrontal cortex and limbic regions accompanying dissociative symptoms" has been observed (p. 579). Moreover, individuals who score highly on measures of dissociation usually fall within the normative range on standard neuropsychological tests and tests of intellectual ability (Giesbrecht et al., 2008; Schurle, Ray, Bruce, Arnett, & Carlson, 2007). Although trait dissociation, in contrast to PTSD, is associated with increases in gray matter volume in prefrontal, paralimbic, and parietal cortices, as determined by magnetic resonance imaging (MRI), the meaning of these findings is unclear (Nardo et al., 2013). In many studies in which differences in physiological responses vary as a function of dissociation, researchers have neglected to control for general distress and openness to experience, which are both moderately associated with dissociation, making it difficult to interpret the findings (Lynn et al., 2014). Nevertheless, as our discussion proceeds, we see that different dissociative disorders are related to different cognitive deficits and physiological responses.

Depersonalization/Derealization Disorder

We begin our discussion of the three major dissociative disorders with depersonalization/derealization disorder (hereafter referred to as DPD), the most common and least controversial of the dissociative disorders. Unlike earlier versions of the DSM, in which depersonalization and derealization were catalogued as separate disorders, DSM-5 groups depersonalization and derealization experiences together as DPD. The conjoining of depersonalization and derealization appears to be warranted, as they share similar features with respect to demographics, comorbidity with other disorders, and course (Simeon, 2009).

Description Ludovic Dugas is often credited with first using the term "depersonalization" in the late 1890s (Simeon & Aubugel, 2006). The hallmark of depersonalization is an altered sense of selfhood. Depersonalization symptoms are varied and span feeling as though one is outside of or observing one's body, feelings, sensations, and actions; emotional or physical numbing; time distortion; and an absent or unreal sense of self. In contrast, derealization pertains to feelings of unreality with regard to one's surroundings, in which objects or people may appear to be visually distorted or experienced as foggy, dreamlike, robotic, or lifeless. Understandably, such strange, unusual, and often unexpected experiences of depersonalization/derealization can evoke considerable anxiety, even to the extent of fearing losing control or becoming psychotic. Simeon and colleagues (2008) conducted a factor analysis which revealed that depersonalization could be described in terms of five symptom dimensions: Numbing, unreality of self, perceptual alterations, temporal disintegration, and unreality of surroundings, providing further support for combining depersonalization and derealization symptoms. Individuals with DPD may experience not only alexithymia (i.e., difficulty identifying feelings; Simeon, Giesbrecht, Knutelska, Smith, R. J., & Smith, L. M., 2009), but also feelings of unreality produced by difficulties in discriminating emotional from neutral stimuli (Quadflieg et al. 2013) and a dampening, numbing, or shutting out of emotional responses (Medford, 2012).

Diagnosis The onset of DPD varies from gradual to sudden and from chronic to episodic, and may last from hours to weeks or months and extend to years or even decades in rare cases (Simeon, 2009). Freud described experiencing transient depersonalization/derealization during a visit to the Athens Acropolis in 1904, which he found difficult to experience as real (Simeon & Aubugel, p. 11). Freud's experience is not atypical: Episodes of depersonalization/derealization are commonly brief and may occur only a few times in a person's lifetime. Estimates suggest that the annual prevalence of transient depersonalization/derealization is 23% (Aderibigbe, Bloch, & Waler, 2001), with a lifetime prevalence between 26% and 74% (Hunter, Sierra, & David, 2004).

DPD often occurs in the context of highly stressful events, unfamiliar environments, threatening social interactions, or the use of drugs such as marijuana, ketamine,

and other hallucinogens. DPD has been associated with childhood parental rejection and punishment, controlling for age and severity of depression and anxiety (Michal et al., 2009), and with emotional abuse in childhood in clinical and nonclinical samples (Michal et al., 2009; Simeon, Guralnik, Schmeidler, Sirof, & Knutelska, 2001).

However, when DPD symptoms are persistent, recurrent (at least once a month), disturbing, and/or interfere with daily functioning, and do not occur exclusively in the context of another disorder, such as an anxiety disorder or a medical condition (e.g., seizures), they qualify for a diagnosis of DPD. The first episode of depersonalization/derealization is likely to occur between adolescence (age 16; Simeon et al., 1997) and young adulthood (age 22; Baker, Hunter, & Lawrence, 2003), although treatment may not be sought until late adulthood. In approximately two-thirds of cases, the course of DPD is chronic, with symptoms present most or all of the time (Simeon, 2009). To rule out a diagnosis of schizophrenia or another psychotic condition, for a diagnosis of DPD, reality testing must remain intact during episodes of depersonalization/derealization. Moreover, DPD should not be diagnosed when symptoms are restricted to meditative or trance practices.

Prevalence and Comorbidity Estimates of the prevalence of diagnosed DPD in the general population converge in the range of 1–3% (Lynn et al., 2014). In inpatient samples, the rates of depersonalization and derealization diagnoses run as high as 16% (Hunter et al., 2004).

DPD often occurs in the presence of other disorders. For example, DPD is especially common in panic disorder, with prevalence as high as 82%. The relation between DPD and anxiety has compelled some European authors to classify DPD as an anxiety disorder (Lynn, Merckelbach et al., 2015). Moreover, depersonalization/derealization is included among the diagnostic criteria for panic disorder in DSM-5. Nevertheless, the lumping of anxiety and depersonalization/derealization has been questioned. Although one study found that the only childhood risk factor identified for adult DPD is a history of anxiety (Lee, Kwok, Hunter, Richards, & David, 2012), another found that the correlation between measures of anxiety and depersonalization in adulthood is statistically significant but low (Sierra, Medford, Wyatt, & David, 2012). Moreover, mood-stabilizing medications typically used to treat anxiety conditions have little therapeutic effect on DPD (Simeon, 2009). Additionally, DPD occurs commonly in many conditions, not identified solely with anxiety, including major depression, somatoform disorders, substance use disorders, and various personality disorders (e.g., borderline, avoidant; Belli, Ural, Vardar, Yesilyurt, & Oncu, 2012; Lynn et al., 2014). For example, the rate of depersonalization/derealization symptoms in major depression has been reported to be as high as 60% (Noyes, Hoenk, Kuperman, & Slymen, 1977). Finally, although hyperarousal may trigger episodes of depersonalization/derealization (Sterlini & Bryant, 2002), depersonalization/derealization itself may engender anxiety, as mentioned earlier, and hyperventilation may bring about symptoms of both anxiety and depersonalization/derealization (Lickel, Nelson, Hayes, Lickel, & Deacon, 2008). In sum, anxiety and depersonalization appear to be related to some extent, but are largely distinguishable (Sierra et al., 2012).

In acute stress disorder, which often precedes PTSD, depersonalization/derealization is one of 14 symptoms listed in DSM-5, and is present in as many as 30% of cases of PTSD (Michal et al., 2009). Moreover, in DSM-5, it is possible to specify whether depersonalization and derealization accompany PTSD (American Psychiatric Association, p. 274). The dissociative subtype of PTSD can be identified in civilian and noncivilian populations (Lanius et al., 2012), with the prevalence of the subtype ranging from 14% (Stein et al., 2013) to 25% (Steuwe, Lanius, & Frewen, 2012), to 30% (females; Wolf et al., 2012). The fact that dissociative symptoms follow in the wake of trauma in only a minority of cases implies that exposure to highly aversive events is but one of a number of potential variables associated with dissociation, a contention that figures in our discussion below of theories of dissociation.

Physiological and Neuroimaging Findings Although trauma exposure may be a distal cause of depersonalization, researchers have implicated a mismatch in perceptual/sensory signals and alterations in body schemas as culprits in engendering DPD. For example, investigators (Simeon et al., 2009) have used MRI and positron emission tomography of the brain to reveal that DPD patients, compared with healthy controls, exhibit abnormalities in areas of the sensory cortex related to somatosensory, visual, and auditory experiences and areas of the cortex that subserve the integration of body schemas. Simeon et al. (2009) suggested that these findings imply that DPD involves a dissociation of perceptions that give rise to the symptoms of DPD. Additionally, vestibular stimulation produced by caloric irrigation of the ear labyrinths produces feelings of depersonalization in healthy participants, including strange bodily feelings, feeling "spaced out," and not in control of the self (Jáuregui-Renaud, Green, Bronstein, & Gresty, 2006). Moreover, patients experiencing peripheral vestibular disease (Jáuregui-Renaud, Sang, Gresty, Green, & Bronstein, 2008) and patients with retinal disease (Jáuregi-Renaud, Ramos-Toledo, Bolaños, Montaño-Velazquez, & Pliego-Maldonado, 2008) are more likely to experience depersonalization/derealization compared with healthy controls. Accordingly, DPD symptoms may ensue when there is a lack of integration or mismatch between multisensory inputs (e.g., vestibular, visual proprioceptive) that impairs neural representations that generate an altered sense of reality and the self

(Aspell & Blanke, 2009; Lynn et al., 2014). Alternatively, among predisposed individuals, DPD symptoms may tend to arise whenever there are markedly unexpected bodily or perceptual experiences.

Additional evidence of a link between DPD symptoms and physiology comes from studies of out-of-body experiences—a sense of physical separation from the self—sometimes reported by people with DPD. Researchers have shown that out-of-body experiences stem from a mixing or scrambling of the senses (e.g., vision and touch) when the sense of the physical body is disrupted. When physical sensations and visual impressions combine in atypical ways and there is a disruption in somatosensory signals, it can create the experience of feeling outside of one's body (Cheyne & Girard, 2009; Terhune, 2009). Ehrrson (2007) generated an out-of-body experience in the laboratory by creating the illusion that participants' bodies were standing in front of them. This was accomplished by participants donning goggles that displayed a video image of themselves provided by a camera behind them. Participants reported that they could experience themselves being touched in a location outside their physical bodies when they were touched with a rod on the chest at the same time the experimenters used the camera set-up to make it appear that their visual image was touched (see also Aspell, Lenggenhager, & Blanke, 2009; Lenggenhager et al., 2007). Additionally, investigators have produced out-of-body-like experiences by stimulating where the brain's parietal and right temporal lobes join, the vestibular cortex, and the superior temporal gyrus (see Lynn et al., 2014). Perhaps not coincidentally, Sierra, and colleagues (2014) compared DPD patients with controls and reported gray matter changes in the frontal, temporal, and parietal lobes associated with DPD based on MRI findings. The authors note that additional research is necessary to determine whether these changes are vulnerability or disease markers.

Transcranial magnetic stimulation (TMS)—a method of inducing an electrical field in select portions of the cortex by passing a magnetic field through the skull (Barker, Jalinous, & Freeston, 1985; O'Shea & Walsh, 2007)—has shown promise in elucidating pathological variations in cortical excitability associated with depersonalization (e.g., Sierra & Berrios, 1998). Jay, Sierra, Van den Eynde, Rothwell, and David (2014) used repetitive TMS (rTMS) to evaluate a neurobiological model of depersonalization proposed by Sierra and Berrios (1998) in which the ventrolateral prefrontal cortex (vlPFC) inhibition of the insula (a brain area involved in the processing of bodily sensations) contributes to the emotional numbing and altered sense of self associated with depersonalization. Jay and colleagues hypothesized that inhibition of the vlPFC engendered by rTMS would disinhibit insula activity and would allow for increased arousal and reduced depersonalization symptoms. Among patients with medication-resistant depersonalization disorder, a single session of rTMS produced reductions in depersonalization symptoms. However, although rTMS inhibited vlPFC activity and increased insula activity as hypothesized, symptom reductions also occurred after patients received rTMS targeting the temporal parietal junction, a known neural substrate of out-of-body experiences. The authors concluded that their findings support the neurobiological model proposed by Sierra and Berrios (2000). Nevertheless, they observed that, in their uncontrolled trial, the therapeutic effects of rTMS were independent of increased arousal (i.e., insula activity) and thus nonspecific and potentially attributable to placebo effects.

Other investigations of the effects of TMS and rTMS on depersonalization symptoms include two case studies and one clinical trial. Keenan, Freund, and Pascual-Leone (1999) treated a female patient with comorbid major depression and depersonalization disorder with rTMS and reported decreases in depersonalization symptoms in tandem with increases in self-awareness. In this case, rTMS targeted the patient's right frontal lobe, which showed hyperactivity in a single PET scan. A second case study (Jiménez-Genchi, 2004) of a male patient with comorbid medication-resistant DPD and major depressive disorder showed a 28% reduction in depersonalization symptoms after six sessions of rTMS delivered to the dorsolateral prefrontal cortex. Finally, Mantovani and colleagues (2011) conducted the first uncontrolled open clinical trial examining effects of inhibitory rTMS delivered to the temporal parietal junction in patients with depersonalization disorder. The authors observed that 6 of 12 patients demonstrated symptom improvement after 3 weeks, and 5 of the 6 responders showed 68% improvement in DPD symptoms after an additional 3 weeks of treatment (see also Christopeit, Simeon, & Mantovani, 2013). Randomized and placebo controlled trials are essential to further evaluate the specific and nonspecific effects of rTMS.

Dissociative Amnesia

As mentioned earlier, in DSM-5, dissociative amnesia (formerly called psychogenic amnesia) is diagnosed when there is substantial memory loss for important autobiographical information that is not the product of a neurological or other medical condition (e.g., seizures, memory loss associated with age, or brain injury) or substance abuse. Forgetting can pertain to everyday circumstances and is not limited to amnesia for traumatic or highly stressful events. Moreover, the symptoms of dissociative amnesia cannot be attributable to another disorder, such as acute stress disorder or PTSD, somatic symptom disorder, a neurocognitive disorder, or DID. It is believed to frequently follow a traumatic event and may be classified in terms of localized amnesia related to a specific time period (e.g., a vacation), selective amnesia for some but not all events from a specific time period, generalized amnesia for all life events, continuous amnesia for new events, and systematized amnesia for specific categories

of information, such as childhood sexual abuse. Based on an analysis of 42 memory and amnesia items from the Multidimensional Inventory of Dissociation, Dell (2013) identified three amnesia factors: discovering dissociated actions, lapses of recent memory and skills, and gaps in remote memory.

Despite failure to recall important events, people with dissociative amnesia typically retain implicit memories and habits related to these events, and some people with dissociative amnesia continue to experience a sense of familiarity with respect to previously known people, objects, and places. Moreover, in cases of dissociative amnesia, perception and immediate memory are preserved, although disorientation in time and impairment in new learning may occur (Staniloiu & Markowitsch, 2012).

Dissociative amnesia varies in terms of whether (a) it is experienced in childhood or adulthood (even young children may experience significant memory losses); (b) amnestic episodes last only minutes or persist for years and range from an isolated episode to recurrent episodes; and (c) significant or minimal functional impairment occurs before, during, or after the amnestic episode. Most cases of dissociative amnesia occur in the 30s and 40s, last between 1 and 5 days, and are equally common among females and males (American Psychiatric Association, 2013; Lynn et al., 2014). In two classic studies, in 63 cases, 94% (59/63) resolved within a week, and only 6% (4/59) of people took 3 weeks or more to recover (Ables & Childer, 1935; Herman, 1938). Because of differences in symptom presentation, age of onset, and functional impairment, it is likely that dissociative amnesia comprises a heterogeneous set of conditions with different etiologies.

Prevalence and Comorbidity The prevalence rates for dissociative amnesia range from 0.2% (China), to 3.0% (Canada), to 7.3% (Turkey), with unspecified cultural factors and differences in criteria for evaluating amnesia across studies probably playing a role in disparate diagnostic base-rates across samples (Dell, 2009; Lynn et al., 2014). Ross (2009) reported that among 3000 trauma patients he treated, only one exhibited dissociative amnesia. Still, individuals in the general population often report significant forgetting of events. In one study, 20.6% of college students reported some degree of impairment associated with one or more symptoms of dissociative amnesia based on their responses to a questionnaire (Sar, Alioğlu, Akyüz, & Karabulut, 2014). Researchers have reported that dissociative amnesia often occurs in response to multiple stressors and in the presence of mood, somatoform, eating, and personality disorders (Maldonado & Spiegel, 2008).

Controversy Dissociative amnesia has attracted considerable controversy, spurred in recent decades by Loftus's (1993) critique that questioned whether memories of traumatic events are repressed or dissociated from consciousness. Spiegel (1997) contended that dissociative amnesia is "more, rather than less common after repeated episodes; involves strong affect; and is resistant to retrieval through salient cues" (p. 6). Yet intense affect and repetition ordinarily improve memory, increasing access of memory through salient cues (McNally, 2003). Traumatic or stressful memories are often better remembered than less stressful memories (Berntsen & Rubin, 2014; Lynn et al., 2014; McNally, 2004), as is the case in PTSD, when patients experience intrusive and distressing memories (Lynn et al., 2014; Porter & Peace, 2007). Studies of concentration camp survivors, witnesses of homicide, and children who have been kidnapped indicate that memories of such highly aversive events are typically remembered vividly with little or no hint of amnesia (Merckelbach, Dekkers, Wessel, & Roefs, 2003). Memories of traumatic events may be better remembered for their salience, enhanced by attendant physiological arousal (e.g., Jelicic, Geraerts, Merckelbach, & Guerrieri, 2004).

Findings of facilitated recall for emotionally charged memories render implausible many claims of crime-related amnesia—reported in 25–40% of homicide cases and severe sex offenses—in which perpetrators claim they have little or no recall of crimes they committed (Moskowitz, 2004). Van Oorsouw and Merckelbach (2010) contend that malingering probably accounts for most claims of crime-related amnesia.

Although Brown, Scheflin, and Hammond (1998) argued that the empirical case for dissociative amnesia is compelling, Kihlstrom (2005) asserted that supportive studies lack methodological rigor and that longitudinal studies are both scant and do not rule out alternate explanations for amnesia, such as seizures and traumatic brain injury (see also Piper, Pope, & Borowiecki, 2000). Additionally, researchers have suggested that it is problematic to assume that the failure to report memories associated with highly aversive events, such as childhood abuse, is produced by dissociation and have asserted that studies of corroborated traumatic events have failed to uncover evidence that people "encode trauma, yet become incapable of recalling it through the mechanism of dissociative amnesia" (Lynn et al., 2014, p. 906; see also McNally, 2004; Pope, Oliva, & Hudson, 1999).

Specifically, the failure to report memories of events such as childhood sexual abuse may occur for reasons entirely unrelated to a special dissociative mechanism. For example, individuals may not have labeled the physical contact as "abuse" at the time it occurred, and not have thought about the event for some time, or they may presently be reluctant to disclose the abuse to a particular interviewer (Loftus, Polonsky, & Fulliove, 1994; McNally, 2004). Ordinary memory mechanisms, such as motivated forgetting or memory suppression, could account for failure to recall or report important autobiographical information, particularly when the information to be recalled or reported produces anxiety, shame,

or embarrassment. Indeed, feeling greater responsibility for the abuse, or fearing negative consequences of disclosure, rather than dissociative amnesia, provide a plausible account for the nondisclosure of some cases of childhood sexual abuse. Goodman and colleagues (2003) repeatedly interviewed 175 individuals with documented child sexual abuse approximately 13 years after the event. On initial interview, 18.9% of the participants did not report the event. Yet, by the third interview, which occurred in person, only 8% of the respondents failed to disclose the abuse, implicating reluctance to report, rather than a dissociative mechanism, for failure to report.

A fine line may divide active and passive or unconscious memory suppression, as exemplified by the case of FF, who, after a series of traumatic events (i.e., serious motorcycle accident and being the victim of a robbery and shooting) and earlier problems with his business and impending divorce "decided to get away from all of these problems by coming (from Germany) to the United States and starting anew" (p.1145), after which he experienced fugue symptoms. He allegedly lost access to his native German language and confided to researchers,

> I was aware of knowing German in written and spoken form . . . It was a part of my life I just wanted to lock away in a dark chamber. I can't even say if it was an active will or passive defense . . . The point where this disorientation was replaced by neglecting the truth is not easy to find and somehow undefined.
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> (Glisky et al., 2004, p. 1146)

Pope, Poliakoff, Parker, and Boynes (2007) argued that dissociative amnesia is a culture-bound phenomenon and does not appear in literature until the late 18th century. Only one individual from among over 100 entrants in a contest was able to find an example of dissociative amnesia appearing in a fictional or nonfictional work before 1800, and thereby claim a $1,000 prize that Pope and colleagues offered for such evidence. The recency of reports of dissociative amnesia raises troubling questions about its existence as a natural category or entity, and suggests that social, cultural, and historical factors are implicated in reports of dissociative amnesia (Lynn et al., 2014).

Dissociative Fugue In DSM-IV-TR (American Psychiatric Association, 2000), dissociative fugue was classified as a separate disorder, but in DSM-5 it is coded as a condition that can accompany dissociative amnesia. Dissociative fugue (formerly called psychogenic fugue, and based on the Latin word for flight, which has the same root as the English word "fugitive") is a puzzling, rare (0.2% of the general population; Sadock & Sadock, 2005), and reversible amnesia for personal identity that involves wandering or unplanned travel. Some individuals with fugue assume a new identity in a new location, and in so doing, may travel thousands of miles and cross national boundaries (Santos & Gago, 2010). Still, in most cases, fugue is limited in duration to days or weeks and to local travel. Cases of fugue are often associated with highly aversive events, including natural disasters and war, and with avoidance of other stressful life events, including legal, financial, and marital problems.

The association of fugue with stressful events, physical escape from anxiety-eliciting surroundings, the assumption of a "new identity," and the fact that serious psychopathology is rarely manifested in the new identity, point to the purposeful, goal-directed nature of fugue-related behaviors and the possibility of malingering and factitious disorder. Clearly, these possibilities are essential to rule out in arriving at the conclusion that fugue is present. Moreover, it is important for clinicians to evaluate the hypothesis that fugue-like behaviors arise from brain injury and other medical complications, as fugue-like behaviors have been related to prednisone use (Gifford, Murawski, Kline, & Sachar, 1976), limbic system dysfunction (Mohan, Salo & Nagaswami, 1975), cervical plexus block (Goldberg, 1995), and changes in frontal, temporal, and parietal brain activity (Hennig-Fast et al., 2008).

Fugue, like dissociative amnesia, has stirred considerable controversy and skepticism. Hacking (1998), for example, described fugue as a phenomenon that varies with historical and cultural circumstances, given that it first appeared in the 19th century. Across cultures, fugue-like symptoms, including running, fleeing, energized activity, and amnesia for the duration of the episode are manifested in such culture-bound syndromes as amok in Western Pacific cultures (the source of the popular term "running amok"); *pibloktok* in native cultures in the Arctic, and Navajo "frenzy" witchcraft (Lynn et al., 2014).

Dissociative Identity Disorder

DID is arguably the most fascinating and controversial of the dissociative disorders. The diagnostic criteria for DID have changed considerably over the years, reflecting changes in the conceptualization of dissociative symptoms. The term "multiple personality" first surfaced in DSM-II (American Psychiatric Association, 1968), listed under the diagnosis hysterical neurosis, dissociative type. In DSM-III (American Psychiatric Association, 1980), the diagnosis fell under the rubric of "dissociative disorders" and retained the term multiple personality. In DSM-IV (American Psychiatric Association, 1994), the diagnosis was renamed to "dissociative identity disorder" to emphasize changes in identity and to deemphasize the notion that individuals with DID house separate personalities. In DSM-5, dissociative amnesia was broadened to encompass gaps in memories for everyday events, no longer being largely restricted to forgetting of traumatic or stressful events. In DSM-5, the criterion that "two or more distinct identities or personality states" (p. 529) must be present was replaced with "two or more distinct personality states" (p. 292), creating clear distance from the

DSM-IV view of DID as involving the invasion or intrusion of identities or personalities (sometimes called alters) that "recurrently take control of the individual's behavior" (p. 519). The latest diagnostic scheme also specifies that the "signs and symptoms may be observed by or reported by the individual" (p. 292), and that a person is eligible for a diagnosis when there are "sudden alterations or discontinuities in sense of self or agency and recurrent dissociative amnesias" (p. 293). Given the widespread use of the terms "personalities" and "identities," and the fact that these terms encompass "personality states," we continue to use all three terms in this chapter (see also Lynn et al., 2014).

DSM-5 notes that, in some cultures, personality states may be described as an experience of possession by a spirit, supernatural entity, or outside person taking control, such that the individual begins speaking or acting in a distinctly different manner (American Psychiatric Association, 2013, p. 293). In cases of possession, the personality states must be unwanted, involuntary, recurrent, distressing or impairing, and not be a part of accepted cultural/religious practices. According to DSM-5, discontinuities supposedly associated with different personality states may involve rapid and unusual shifts in attitudes, food preferences, perceptions of the body as a small child or member of the opposite gender, perceptions of internal voices, crying, and a sense of loss of self. Still, in only a "small proportion of non-possession-form cases, manifestations of alternate identities are highly overt" (p. 292) and individuals may "often conceal, or are not fully aware of . . . amnesia or other dissociative symptoms" (p. 294).

Although DID symptoms are typically more florid (e.g., flashbacks, amnesia, fugue, neurological symptoms) in women compared with men, men exhibit more criminal or violent behavior. DSM-5 specifies that the symptoms that qualify for a diagnosis of DID "are not attributable to the physiological effects of a substance (e.g., blackouts or chaotic behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures)" (p. 292) and that "the disturbance is not a normal part of a broadly accepted cultural or religious practice" (American Psychiatric Association, 2013; p. 292).

The shifts in diagnostic criteria over the years pose significant problems for diagnosticians and will probably increase the prevalence rates of DID for the following five reasons:

1. The criteria leave open to interpretation: (a) What is a "personality state," especially when it typically is not "overt," and (b) alterations or discontinuities in the sense of self or agency may be difficult to specify because behaviors and emotions are often highly variable and experienced with little sense of personal agency in everyday life (Kirsch & Lynn, 1998).
2. Relatedly, in cases of possession, the judgment of whether personality states are "involuntary" is highly subjective.
3. In some cases, it may be difficult to differentiate "ordinary forgetting" and "memory gaps" from clinically significant amnesia.
4. Including both individuals' and outside observers' evaluation of shifts in personality states as diagnostic indicators further liberalizes the criteria for diagnosing DID.
5. Because some individuals purportedly conceal amnesia or other dissociative symptoms, the diagnosis may be highly dependent on the evaluator's impressions, judgments, and beliefs about dissociation.

Prevalence and Comorbidity The prevalence rates of DID vary widely in terms of general versus clinical populations. General population studies of participants in Turkey (Akyüz Do an, Sar, Yargic, & Tukun, 1999), Canada (Ross, 1991), and North America (Loewenstein, 1994) converge on a prevalence rate of approximately 1% for DID, with approximately equal rates among males and females (American Psychiatric Association, 2013). In contrast, in inpatient settings, with the exception of a study by Rifkin, Ghisalbert, Dimatou, Jin, and Sethi (1998), the reported prevalence rates equal (Bliss & Jeppsen, 1985) or exceed (Latz, Kramer, & Hughes, 1995; Ross et al., 2002) 10%, with Ross and colleagues (1992) reporting a 14% lifetime prevalence of DID in 100 chemically dependent patients. In adult clinical settings, females predominate, although gender equality in prevalence rate is common in child clinical settings (DSM-5). Selection and referral biases may account for the imbalanced sex ratio among adults in inpatient settings: A large proportion of males with DID are treated in forensic settings or incarcerated (Lilienfeld & Lynn, 2015; Putnam & Loewenstein, 2000).

The prevalence of DID has generated much controversy, sparked by literature reviews contending that the rates of adult and childhood DID are inflated and limited to a small number of practitioners in only a few countries (Boysen, 2011; Boysen & VanBergen, 2013; see also Piper & Merskey, 2004). For example, Boysen (2011) reported that four research groups in the United States accounted for two-thirds of all 255 child cases reported since 1980, which prompted his conclusion that childhood DID is extremely rare.

The number of reported cases of DID has increased dramatically over the years. From 1970, in which approximately 80 cases were reported, the number had skyrocketed to approximately 6,000 by 1986. Over roughly the same time period, the number of alters per patient also dramatically increased from 2 to 3 to approximately 16 (Lynn et al., 2014). Paris (2012) claimed that the diagnosis of DID is little more than a fad. Nevertheless, other researchers have vigorously challenged the rarity of DID, counterpunching that DID is massively underdiagnosed (Brand, Loewenstein, & Spiegel, 2013; Ross, 2013). Studies that implement standardized assessment of DID using structured interviews with evaluators trained to a high degree of reliability and blind to symptom status and patient history are imperative to securing more accurate

prevalence estimates. Nevertheless, estimating the prevalence of DID will remain a challenge in the absence of clear-cut validating variables for the presence or absence of DID (Lilienfeld & Lynn, 2015; Robins & Guze, 1970).

DID is comorbid with many disorders, raising the question of whether the disorder is a marker for severe psychopathology or negative emotionality (Lynn et al., 2014; North, Ryall, Ricci, & Wetzel, 1993). Indeed, many disorders typically occur in conjunction with a DID diagnosis, with one study (Ellason, Ross, & Fuchs, 1996) finding that DID patients qualified for an average of 4.5 personality disorders and eight other mental disorders. Up to three-quarters of patients with DID meet diagnostic criteria for borderline personality disorder and substance abuse, and as many as 90% of patients with DID meet criteria for major depression (see Lynn et al., 2014). DID is also comorbid with PTSD, schizoaffective disorder; schizophrenia; sexual, sleep, and eating disorders; and avoidant and obsessive-compulsive personality disorders (Lynn et al., 2014). Moreover, DID is associated with markedly increased risk of attempted suicide (more than 70% of patients so diagnosed have attempted suicide), self-mutilation, and aggressive behaviors (American Psychiatric Association, 2013).

Assessment of Dissociation

Researchers have at their disposal a number of assessment instruments to evaluate trait and state dissociation based on self-report and interview modalities. In this section, we review the most widely used and best validated measures of dissociation, starting with self-report trait measures.

First developed in 1986 (Bernstein-Carlson & Putnam, 1986) and revised in 1993 (Bernstein-Carlson & Putnam, 1993), the 28-item Dissociative Experiences Scale (DES) is today the most widely used measure of trait dissociation in both applied and research settings. Participants rate the percent of the time they experience a given symptom of dissociation from 0–100% of the time at 10% intervals. Test–re-test reliabilities (Bernstein-Carlson & Putnam, 1993) range from $r = .79$ to $r = .95$, split-half reliabilities of $r = .83$ to $r = .93$, and Cronbach's alpha = .96 (Condon & Lynn, 2014; van Ijzendoorn & Schuengel, 1996). The DES has demonstrated convergent correlations with related constructs, diagnostic interview scores, and other dissociation measures (van Ijzendoorn & Schuengel, 1996). In a multicenter study (Carlson et al., 1993), a cutoff of 30 on the DES was thought to index more pathological dissociative psychopathology. With this cutoff, the DES correctly identified 80% of participants without DID and correctly identified 74% of patients with depersonalization disorder. Although research has generated mixed findings regarding the factor structure of the DES, researchers typically use three factor analytically derived scales that measure absorption, amnesia, and depersonalization (Carlson et al., 1991; Ross, Ellason, & Anderson, 1995; Sanders & Green, 1994). In nonclinical populations, the DES distribution tends to be highly skewed and vulnerable to floor effects (Wright & Loftus, 1999). Although the DES-T derivative measure introduced earlier yields a high proportion of false positive diagnoses (Giesbrecht, Merckelbach, & Geraerts, 2007), the scale is capable of distinguishing patients with DPD from control participants and has been employed in studies of dissociation, memory, and cognition (Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008).

Researchers have developed a 30-item Adolescent Dissociative Experiences Scale (A-DES; Armstrong, Putnam, Carlson, Libero, & Smith, 1998) designed to screen for dissociative disorders and assess changes in dissociation over time. Participants rate items grouped into four subscales (i.e., absorption and imaginative involvement, passive influence, depersonalization/derealization, and dissociative amnesia) on an 11-point Likert-type scale. Norms exist for both patients and healthy adolescents, and the total score and scales are consistent internally ($\alpha = .72–.93$, total score; see Giesbrecht et al., 2008). Still, the A-DES appears to be related to anxiety as well as dissociation, so its discriminant validity is questionable (Muris, Merckelbach, & Peeters, 2003).

Researchers have developed additional self-report scales of dissociative experiences and symptoms that have been used as screening instruments and research tools. These measures possess adequate-to-excellent internal consistency, test–re-test reliability, and discriminant and convergent validity, and include the Dissociation Questionnaire (63 items, $\alpha = .96$; Vanderlinden et al., 1991), which has seen the widest use, the Questionnaire of Experiences of Dissociation (Riley, 1988; 26 items, $\alpha = .80$; Watson, 2003), the Dissociative Processes Scale (Harrison & Watson, 1992; Watson, 2003; 33 items; $\alpha = .93$), and the Perceptual Alterations Scale (Sanders, 1986; 60 items; $\alpha = .95$). Unlike the aforementioned measures, the Multidimensional Inventory of Dissociation (MID; Dell, 2006; median $\alpha = .91$) assesses only pathological dissociation. The 218-item MID provides scores on 23 diagnostic scales and five validity scales that evaluate participants on criteria for DID proposed by Dell (2002). The MID possesses good-to-excellent 4–8-week test–re-test reliability; $r = .82–.97$), convergent validity (correlations with the DES = .90), and distinguishes among patients with DID, dissociative disorder not otherwise specified, mixed psychiatric patients, and nonclinical adults (Lynn et al., 2014).

The 29-item Cambridge Depersonalization Scale (Sierra & Berrios, 2000; $\alpha = .89$) assesses the frequency and duration of depersonalization symptoms. Discriminative validity is excellent, with the scale distinguishing patients with DPD from healthy patients and patients with other disorders, including anxiety disorders and epilepsy (Sierra & Berrios, 2000).

The 20-item Somatoform Dissociation Questionnaire (SDQ-20; Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1996; $\alpha = .95$) evaluates somatoform responses (e.g., unexplained neurological symptoms, sensory loss) associated with dissociative states with no

identifiable medical basis and has demonstrated success in distinguishing outpatients with dissociative disorders from nondissociative psychiatric outpatients. A five-item scale derived from the SDQ-20 is available to screen for dissociative disorders (SDQ-5, 1997) with a high degree of sensitivity (94%) and specificity (98%) among psychiatric patients (Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1998).

Researchers have devised so-called "state" measures to evaluate relatively transient dissociative experiences. The Clinician Administered Dissociative States Scale (CADSS; Bremner et al., 1998; α = .94; Condon & Lynn, 2014; α = .80) is composed of 19 self-report items and eight observer-rated items. The self-report items are grouped into three subscales: amnesia, depersonalization, and derealization. The CADSS is sensitive to experimental manipulations intended to increase levels of dissociation (e.g., staring at a dot). Moreover, research has garnered evidence of construct validity, including high correlations with the DES (r = .56, Condon & Lynn, 2014) and discriminative validity, including the ability to distinguish among patients with PTSD, healthy adults, and patients with schizophrenia and mood disorders (Bremner et al., 1998).

The 56-item State Scale of Dissociation (SSD; Krüger & Mace, 2002; α = .97) comprises seven subscales: derealization, depersonalization, identity alteration, identity confusion, conversion, amnesia, and hypermnesia. The SSC and the DES correlate at r = .81 among people with a dissociative disorder and r = .57 among healthy adults.

Structured interviews provide a more fine-grained analysis of dissociative symptoms in terms of psychological diagnoses, with the 250-item Structured Clinical Interview for DSM-IV Dissociative Disorders-Revised (SCID-DR; Steinberg, 1994) and the 131-item Dissociative Disorders Interview Schedule (DDIS; Ross, Heber, Norton, & Anderson, G., 1989) the most widely used to discriminate dissociative disorders from other disorders. Interrater reliability for both measures is adequate to excellent, with interrater reliability ranging from r = .68 (Ross, Heber, Norton, Anderson, D. et al., 1989) to .72–.86 (weighted κ, SCID-D; Steinberg, 1994), with the exception of lower interrater reliability (r = .56) for depersonalization disorder on the DDIS, which cannot be reliably diagnosed in this context (Ross, Anderson, Fraser, Bjornson, Miller, & Reagor, 1992). Both structured interviews have demonstrated success in distinguishing among patients with dissociative disorders and nondissociative disorders, and healthy participants. Nevertheless, the SCID poses some difficulties in discriminating among dissociative disorders, bipolar disorder, borderline personality disorder, and schizophrenia (Kihlstrom, 2005).

Models of Dissociative Disorders

No discussion of dissociation and dissociative disorders would be complete without consideration of the controversies that have riven the field of dissociative disorders. In this section, we describe the two major accounts of dissociation that vie for empirical attention and support: the posttraumatic model (PTM) and the sociocognitive model (SCM), the latter called by some the fantasy model (Dalenberg et al., 2012). We suggest that a multifactorial perspective, which provides a modicum of integration of these models, provides the most comprehensive, balanced, and accurate account of dissociative experiences and disorders.

The PTM (Dalenberg et al., 2012, 2014; Gleaves, 1996; Ross, 1997) holds that dissociation is a defensive response to severe physical, sexual, or emotional abuse and/or other highly aversive events that often date to childhood. More specifically, to ward off negative emotions that would engender or intensify anxiety and suffering, or to escape the implications of abuse (e.g., "I am unlovable, bad, and so forth"), the individual either distances him or herself from reality or a sense of selfhood, as in DPD, or develops what are variously called separate selves, identities, personality states, ego states, personalities, or alters to contain, manage, and delimit threatening memories and affect, as in DID. In the latter case, the sense of self essentially fragments, with different aspects of experience and identity somehow splitting off from consciousness, disrupting the ordinary sense of continuity of experience, behaviors, and identity, thereby engendering dissociative experiences and symptoms.

The most persuasive evidence for the PTM comes from many studies that provide evidence for positive correlations between a history of highly aversive experiences and reports of dissociative symptoms and experiences. For example, in their meta-analysis, Dalenberg and colleagues (2012) reported that after excluding studies using college samples, and studies using subscales of the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986), they obtained an overall weighted r effect-size estimate of .32 (a medium-sized effect; Cohen, 1992). Moreover, Dalenberg et al. (2012) claimed that prospective studies of trauma, using objective measures, have yielded positive correlations between highly aversive events and later dissociation.

The SCM has challenged the central assumption of the PTM of a close link between highly aversive events and dissociation. According to this account, sociocognitive and other variables must be considered in a comprehensive account of dissociation. SCM theorists (Spanos, 1994, 1996; Lilienfeld et al., 1999; Lynn et al., 2014) have claimed that the following nine points cast doubt on the existence of a reliable link between highly aversive events and dissociation:

1. Many studies of the link between trauma and dissociation lack strong corroboration of child abuse or the index event and do not permit causal inferences, as they are based on cross-sectional designs that are subject to memory biases. In contrast, prospective studies with well-corroborated cases of abuse often, but not

always, find little or no statistical association between childhood abuse and dissociation in adolescence and adulthood (see Lynn et al., 2014, for a review).

2. As noted earlier, dissociative disorders often co-occur with manifestations of mild to severe psychopathology and negative emotionality. Nevertheless, the role of comorbid conditions is rarely examined in studies of dissociation and trauma, despite the fact that such conditions may contribute substantially to dissociation (Kwapil, Wrobel, & Pope, 2002; Muris et al., 2003) and render it difficult to isolate abuse as the central causal agent of dissociation (Lynn et al., 2014).
3. Selection and referral biases common in psychiatric samples may account for high levels of child abuse among DID patients. For example, patients who are abused are more likely than other patients to seek treatment (Pope & Hudson, 1995).
4. Reports of abuse often arise in the context of a stressful or pathogenic family environment. When perceptions of family pathology are controlled statistically, the correlations between abuse and psychopathology decrease appreciably or disappear entirely (Nash, Hulsey, Sexton, Haralson, & Lambert, 1993).
5. Correlations between highly aversive events and dissociative experiences and symptoms are highly variable and range between $r = .06$ (NS) to $r = .44$ ($P < 0.001$) in nonclinical samples, and from $r = .14$ (NS) to $r = .63$ ($P < 0.001$) in clinical samples (see Dalenberg et al., 2012). Moreover, 40% of the correlations Dalenberg et al. (2012) reported were below .30, and only 6% of the correlations equaled or exceeded .50, signifying a large effect (Lynn et al., 2014). The reasons for the differences observed among correlations are unknown.
6. Highly aversive events do not necessarily precede the onset of dissociative disorders. For example, in two studies, 39.1% (Sar et al., 2007) and 24.4% (Duffy, 2000) of DID patients reported no trauma or neglect of any kind (Lynn et al., 2014).
7. Some studies that purport to find a link between a history of trauma and dissociation (see Dalenberg et al., 2012) are compromised by a lack of blindness. More specifically, diagnoses of DID were not made blindly of trauma reports, made only after records were thoroughly reviewed, and made when standardized diagnostic interviews were not completed (Lynn et al., 2014).
8. Drugs such as ketamine and other hallucinogens produce dissociative reactions (e.g., depersonalization/derealization), implying that pathways to dissociation exist independent of exposure to highly aversive events.
9. Highly dissociative individuals tend to score highly on measures of symptom exaggeration, raising suspicions about the authenticity of some of these individuals' memories and symptoms (Lynn et al., 2014).

In sharp contrast to the PTM, the SCM requires no special "dissociative mechanism" to explain dissociative symptoms (Lynn, Knox, Fassler, Lilienfeld, & Loftus, 2004). Instead, the SCM posits that DID is largely a socially constructed or reinforced condition that occurs when people are exposed to media influences (e.g., books, film, television), broader sociocultural expectations (e.g., people cope with abuse by developing "multiple personalities"), and suggestive procedures in psychotherapy (e.g., leading questions, hypnosis, repeated questioning about abuse; Lynn, Krackow et al., 2015) that cue the presentation of DID. For example, mainstream techniques for treating DID often shape or reinforce patients' displays of "separate selves" by (a) posing questions such as, "To which part am I speaking now?" or "Is there another part of you that holds your anger?", (b) conversing with different alters, and (c) employing suggestive devices, such as charts and bulletin boards, to "map the personality system" (Putnam, 1989). Moreover, the SCM contends that certain vulnerabilities increase the likelihood of a DID diagnosis, including serious coexisting psychopathology (e.g., major depression, borderline personality disorder), ambiguous or puzzling psychological symptoms, as well as high suggestibility, fantasy proneness, cognitive failures (e.g., absent-mindedness), a disrupted sleep cycle, and negative emotionality (Giesbrecht et al., 2008, 2010; Lynn et al., 2014). The SCM draws on the following findings to support the hypothesis that sociocultural and cognitive variables provide an account of dissociation and to challenge key tenets of the PTM:

1. Over the past several decades, the media in the United States accorded prominent attention to DID (e.g., movies: *The Three Faces of Eve*, television programs: *Sybil*, *The United States of Tara*), promoting awareness of DID among clinicians and patients regarding the features of DID and its purported link with abuse. These developments coincided with greatly increased numbers of patients diagnosed with DID (Elzinga, van Dyck, & Spinhoven, 1998), as well as sharp increases in the number of alters per patient (Ross, Norton, & Wozney, 1989). In most cases of DID prior to the 1970s, only one or two personalities was the norm; yet not many years later, Ross, Heber, Norton, and Anderson (1989) observed that the mean number increased to 16 personalities.

   Curiously, that number was the same number reported by the woman who went by the pseudoname of Sybil (Acocella, 1999), whose treatment was memorialized in the bestselling book (Schreiber, 1973) by the same name and the Emmy-award winning television film starring Sally Fields. Yet the case of Sybil, which monumentally shaped the cultural narrative regarding the purported tie between dissociation and abuse, has come under critical fire, with serious and credible doubts expressed regarding whether claims of abuse in her case are genuine

and the diagnosis of DID is accurate (Nathan, 2002; Rieber, 2006).

2. The possibility that DID is co-created by patient-therapist interactions is supported by findings that (a) most DID patients show few or no clear-cut signs of this condition (alters) prior to psychotherapy (Kluft, 1984), and (b) the number of alters at the time of initial diagnosis appears to have remained stable while the number of alters has increased following psychotherapy (Lynn et al., 2014; Ross, Heber et al., 1989). Moreover, a small number of therapists distributed over only a few countries, many of whom specialize in treating DID, account for the majority of cases in the published literature and probably patients seen in clinical practice (Boysen & VanBergen, 2013; Mai, 1995). Finally, the diagnosis of DID has proliferated in other countries, such as the Netherlands, where it has been the focus of extensive media and professional attention and publicity.
3. The entrenched cultural narrative enfolding DID is captured in laboratory research demonstrating that nonclinical participants who are provided with appropriate cues and prompts can reproduce many of the overt symptoms of DID (e.g., alter personalities that respond differently to psychological tests; Spanos, Weekes, & Bertrand, 1995). Moreover, persons in laboratory studies instructed to role-play the symptoms of DID report serious and implausible abuse (e.g., satanic ritual abuse) when interviewed about their childhood, consistent with clinical and media reports regarding DID (Stafford & Lynn, 2002). Across most comparisons between people instructed to simulate or role-play DID and patients with DID, few or no significant differences have emerged on measures of self-reported dissociative experiences, memory, and event-related potentials (Boysen & van Bergen, 2014).
4. Some researchers have claimed to find striking differences among alters or personality states, including differences in pain tolerance, eyeglasses prescriptions, handedness, handwriting, allergies, and heart rates (Lilienfeld & Lynn, 2015). Yet such disparities may be attributable to mundane fluctuations in mood, differences related to conscious or unconscious enactment or role-playing of different identities, or both (Lilienfeld & Lynn, 2015). Indeed, similar intra-individual differences may arise when healthy participants (e.g., actors) are instructed to role-play alters (Boysen & Van Bergen, 2014; Merckelbach, Devilly, & Rassin, 2002) or may arise on the basis of chance (type I errors), given the many psychophysiological variables considered in many studies (Allen & Movius, 2000).
5. The SCM holds that at least some reports of childhood abuse may be exaggerated or based on inaccurate memories prompted by suggestive techniques in psychotherapy. Researchers have demonstrated that in a sizable minority or even majority of participants (25–75%; Garry, 2013), it is possible to implant false memories of events such as riding in a hot-air balloon, being the victim of bullying, and being subject to a vicious animal attack (Lynn et al., 2014), implying that false memory formation is possible in more intensive psychotherapy contexts that may extend for months, years, and even decades (Lynn et al., 2014). Moreover, psychotherapists who use hypnosis tend to consult with more DID patients compared with psychotherapists who do not use hypnosis (Powell & Gee, 1999), a finding of considerable interest as hypnosis is associated with higher rates of inaccurate memories and unwarranted recall confidence compared with nonhypnotically enhanced recall (Lynn, Boycheva, Deming, Lilienfeld, & Hallquist, 2009).
6. The propensity for pseudomemories and memory commission errors associated with dissociation may be mediated by heightened levels of fantasy proneness, suggestibility, and cognitive failures, although the findings pertaining to trait dissociation and memory errors are mixed and not consistently strong in magnitude (see Dalenberg et al., 2012).
7. Contrary to the PTM, researchers have not found consistent support for an amnesic barrier that separates identities or personality states (Dalenberg et al., 2012). When objective measures, such as event-related potentials or behavioral tasks are used, studies typically find clear evidence of transfer of information across identities or alters (see Giesbrecht et al., 2010; Lynn, Lilienfeld et al., 2015).
8. Highly dissociative individuals typically experience a breakdown in cognitive inhibition (Giesbrecht et al., 2009, 2010) and, compared with other individuals, exhibit better memory for to-be-forgotten sexual words in directed-forgetting tasks (Elzinga, De Beurs, Sergeant, Van Dyck, & Phaf, 2000). These findings constitute a strong challenge to the idea that amnesia and avoidance of threat-related information are core features of dissociation.

Objections to the PTM have not gone unchallenged by adherents of the model. Specifically, proponents of the PTM (e.g., Dalenberg et al., 2012, 2014; Gleaves, 1996) have (a) criticized the SCM as failing to provide evidence for a strong link between dissociation and fantasy and suggestibility/false memories; (b) contended that trauma accounts for variance in dissociation beyond that predicted by fantasy proneness, but not vice versa; (c) argued that some studies provide evidence of a link between trauma and dissociation, even when objective measures of trauma are used; and (d) suggested that findings from laboratory studies using role-players, for example, are not generalizable to clinical populations.

Possibilities for Rapprochement and Integration

Recently, proponents of the PTM (Dalenberg et al., 2012)

and SCM (Lynn et al., 2014) have made important concessions and converged, to some extent, in their thinking about dissociation. Consensus now exists that biological (e.g., genetic) vulnerabilities, family environment, social support, developmental factors, and psychiatric history, may play a role in dissociative experiences and symptoms. Some adherents of the PTM acknowledge that: (a) Individuals with DID come to mistakenly believe they are more than one person and that DID is in part a disorder of self-understanding (Dalenberg et al., 2012, p. 568), views compatible with the SCM; and (b) "fantasy proneness—among other factors—may lead to inaccurate trauma reports" (p. 551); (c) the effects of a pathological family environment may be difficult to isolate from the effects of trauma on dissociation; and (d) therapists should eschew suggestive methods in psychotherapy.

SCM theorists (Lynn et al., 2014), in turn, have conceded that trauma may play a nonspecific causal role in dissociation by increasing stress levels and negative emotionality, particularly with regard to depersonalization/derealization in the face of high-impact aversive events (e.g., natural disaster). Moreover, the SCM acknowledges that brief dissociative reactions may persist on a longer-term basis in individuals predisposed to negative emotionality (e.g., trait anxiety, depression), especially when accompanied by comorbid psychopathology (Lynn et al., 2014). Another possibility is that fantasy proneness, suggestibility, and cognitive failures may contribute to an overestimation of a genuine, albeit weak or modest, association between dissociation and trauma (Lynn, Lilienfeld, Merckelbach, Giesbrecht, & van der Kloet, 2012). Alternatively, early trauma may predispose individuals to develop high levels of fantasy proneness or absorption, which may increase vulnerability to the iatrogenic (therapist-induced) and cultural influences posited by the SCM, thereby increasing the likelihood that DID will be diagnosed following exposure to these influences. Finally, the SCM and PTM concur that individuals with DID typically do not consciously role-play or feign the condition, but actually come to believe they possess multiple selves. In short, the subjective experience of "multiplicity" is real (Lynn et al., 2014).

Sleep and Dissociation: An Integrative Model

Recent findings regarding the link between sleep and dissociation may pave the way of integrating elements of the PTM and the SCM in a more encompassing perspective. van der Kloet, Merckelbach, Giesbrecht, & Lynn (2012) reviewed 23 clinical and nonclinical studies using a variety of measures of dissociation that assess sleep and dissociation. With only one exception, the 23 studies yielded correlations between measures of sleep disturbance and dissociation in the range of r = .30–.55. van der Kloet and colleagues (2013) tested patients experiencing insomnia who stayed one night in a specialized sleep clinic and obtained measures of a range of sleep electroencephalograph parameters along with self-report measures of dissociation, unusual sleep experiences, sleep quality, and trauma history. Dissociative symptom levels were elevated in patients suffering from insomnia, and dissociative symptoms were correlated with unusual sleep experiences (e.g., narcoleptic symptoms, sleep paralysis, nightmares) and poor sleep quality. Moreover, longer rapid eye movement (REM) sleep periods predicted dissociation.

Additional studies suggest that dissociation and sleep-related disturbances are causally related. After one night of sleep deprivation, sleep loss engendered a substantial increase in dissociative symptoms in participants that could not be attributed to changes in mood or demand characteristics (Giesbrecht, Merckelbach, & Geraerts, 2007). van der Kloet, Merckelbach, and colleagues (2012) found that when volunteers were deprived of 36 hours of sleep, dissociative symptoms, sleepiness, and mood deterioration all followed the same oscillating pattern of the sleep–wake cycle, remaining stable during the day and increasing during the night. Moreover, feelings of sleepiness preceded an increase of dissociative symptoms and deterioration of mood. van der Kloet, Giesbrecht, and colleagues (2012) examined the relation between sleep experiences and dissociative symptoms in a mixed inpatient sample at a private clinic evaluated on arrival and at discharge 6–8 weeks later. Following participation in a sleep hygiene program, decreases in unusual sleep experiences (e.g., narcoleptic symptoms, hypnagogic imagery) accompanied a reduction in dissociative symptoms. van der Kloet, Giesbrecht, Merckelbach, and Soontiens (2015) replicated decreases in dissociation in a college sample following 4 days of implementing rules of sleep hygiene. Combined, these findings imply a causal relation between sleep experiences and dissociation.

Based on the available evidence, researchers (Giesbrecht et al., 2008; Koffel & Watson, 2009a; van der Kloet, Merckelbach et al., 2012) have proposed that individuals with a labile sleep–wake cycle and unusual sleep experiences (e.g., hypnagogic hallucinations) experience intrusions of sleep phenomena (e.g., dreamlike experiences) into waking consciousness that promote fantasy-proneness linked with dissociation (see Giesbrecht et al., 2008; Koffel & Watson, 2009) and symptoms of depersonalization/derealization (see also Soffer-Dudek & Shahar, 2011). Disruptions of the sleep–wake cycle also impair memory and attentional control, producing the attention deficits, cognitive failures, and memory fragmentation experienced by highly dissociative individuals and patients (Lynn et al., 2014). Sleep disruptions, particularly in REM sleep, which affects the processing of emotional information (Walker & van der Helm, 2009), may account for the breakdown in cognitive inhibition in an emotional context that is evident in dissociative patients (Dorahy, McCusker, Loewenstein, Colbert, & Mulholland, 2006). Deficient processing of stressful events due to sleep loss also may explain the chronically increased levels of

anxiety and physiological arousal, which Giesbrecht et al. (2010) observed in patients with DPD. Moreover, daily stress interacts with trait dissociation to predict dissociative phenomena (e.g., hypnagogic hallucinations, nightmares) associated with sleep (Soffer-Dudek & Shahar, 2011). According to Lynn and colleagues (2014), this integrative perspective may explain (a) how highly aversive events or daily stressors disrupt the sleep cycle and promote errors in memory, (b) the intrusion of dissociative experiences into consciousness (e.g., fantasy and daydreaming), and (c) cognitive failures—all of which the SCM posits increase sensitivity to sociocognitive influences (e.g., suggestive psychotherapeutic techniques, media influences) and the likelihood of a diagnosis of a dissociative disorder.

Dissociation as Hyperassociation: A Novel Hypothesis

In this section, we turn the idea of dissociation on its proverbial head by positing that the tendency to hyperassociate increases the likelihood of garnering a DID diagnosis and may be an important substrate or mechanism of dissociative symptoms. The first author's (SJL) observations of six people in clinical and forensic contexts with the diagnosis of DID is the source of this hypothesis; yet there is also some support for this notion in the literature. SJL observed that all six patients exhibited a strong tendence to hyperassociate. That is, they often responded in a rapid-fire manner with associations to their thoughts, feelings, and behaviors in response to internal and external stimuli. Such hyperassociations were often accompanied by strong affect, or occurred in response to cues that elicited strong affect. At times, such associational shifts were marked by avoidance of the topic at hand and were followed by a period of silence, whereas at other times, the flow of associations led to the discussion of emotionally charged material. Occasionally, the conversation turned so far afield from the original topic that the patient lost the thread of the discussion and occasionally reported feeling "unanchored in reality" (i.e., depersonalization/derealization) or experienced difficulty in recalling elements of the conversation, implying a lack of cognitive control or coherence of associative processes. In the DID literature, this hyperassociative phenomenon has been called "switching," which might easily imply that the person is experiencing distinct "personality states."

Spitzer and colleagues (2007) observed that rapid shifting of attention in response to negative emotions during psychotherapy is why high dissociators do not achieve gains comparable with low dissociators in treatment. Hyperreactivity with respect to schema-related triggers, such as interpersonal situations and traumas, is also manifested in borderline personality disorder, a condition that is highly comorbid with DID (Saue, Arens, Stopsack, Spitzer, & Barnow, 2014). In borderline personality disorder, identity disturbance is correlated with affective instability and mood reactivity (Koenigsberg et al., 2001), which we posit are associated with poor impulse control and hyperassociativity.

Other evidence is consistent with the hyperassociation hypothesis. van Heughten-van der Kloet, Merckelbach, and Lynn (2013) suggested that excessive REM sleep during the night and/or minor REM sleep episodes during the day "fuel the type of fluid and hyperassociative cognition that is typical for dissociative disorders" (p. 630). Chiu, Heh, Huang, Wu, and Chiu (2009) reported that when experiencing negative emotion, high dissociators are particularly adept at disengaging from one task to rapidly shift to another task. According to Soffer-Dudek's (2014) review, highly dissociative individuals, especially when confronted with negative emotion, display impairments in sustained attention, focused attention when exposed to distracting stimuli, and attentional control (e.g., decreased theta brain wave activity; Krüger, Bartel, & Fletcher, 2013), as well as deficits in inhibitory functions, implying difficulties in steering and modulating mental associations.

A particular dimension of fantasizing or fantasy proneness associated with psychopathology and the tendency to engage in vivid and compelling imagery overlaps with dissociation (Klinger, Henning, & Janssen, 2009) and can be conceptualized as a tendency to fluid thinking and hyperassociation to a degree that lacks clear boundaries and can be disconnected from reality (see also Lynn, Neufeld, Green, Sandberg, & Rhue, 1996). Starker (1979) observed that emotion-loaded imagery disrupts the normal flow of imagery that ordinarily remains intact. In extreme terms, as in schizophrenia, for example, Starker hypothesized that the emotional content of the imagery process is manifested in dissociated form as hallucinations. In short, people who hyperassociate will appear to be "dissociated," particularly in response to stressors, negative emotions, and sleep disruptions that tax limited cognitive resources and the ability to impose control over mentation.

More specifically, we suggest that aspects of fantasy proneness, cognitive failures and degraded executive control, heightened suggestibility, hyperarousal, and intrusions of sleep-related mentation into everyday consciousness, lower the threshold for hyperassociation (and thereby dissociation) and account for the link between dissociation and memory errors. The combination of (a) hyperassociative tendencies, (b) a therapist who interprets associative shifts as manifestations of dissociated selves, (c) the use of suggestive methods that reify the existence of distinct identities, (d) attempts to recover memories associated with "dissociated identities," and (e) puzzling coexisting psychopathology, may be a perfect recipe for the iatrogenic creation of DID in psychotherapy. Clearly, investigating hyperassociative mechanisms in dissociation and impulse control disorders (e.g., borderline personality, bipolar disorder) is an important avenue for future research.

Psychotherapy and Dissociation

Treatments for dissociative conditions have received scant empirical attention, almost certainly less than

interventions for most major psychological disorders. Dissociative disorders are notoriously difficult to treat with pharmacotherapies. Research on medication treatment for dissociative amnesia is nonexistent, and little is known about the response of DID patients to medications. Moreover, DPD has largely proven refractory to pharmacological treatments (Simeon, 2009).

No well-controlled psychotherapy studies of DPD exist, although case studies (e.g., family therapy, paradoxical intervention, flooding) are scattered throughout the literature (Lynn, Merckelbach et al., 2015). In a study of PTSD, women with high levels of depersonalization appeared to respond better to a multimodal cognitive processing therapy compared with cognitive therapy alone (Resick, Suvak, Johnides, Mitchell, & Iverson, 2012). TMS, a procedure discussed earlier, may be a promising treatment for DPD, but controlled studies are lacking.

Numerous case studies of the treatment of DID have been reported, representing a wide swath of therapies, and all reporting positive outcomes. Yet Brand, Classen, McNary, and Zaveri (2009) were able to identify only eight studies that evaluated treatment for DID and other dissociative disorders. None of these studies was a randomized controlled trial. Accordingly, it is not possible to evaluate the reasons for symptom reduction, stemming from many potential alternative explanations, including placebo effects, regression to the mean, and the passage of time (for additional reasons, see Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2014).

Since their earlier review, Brand and her associates (Brand, McNary et al., 2013) reported their findings of decreased levels of dissociation, PTSD, general distress, and depression over the course of a 30-month community treatment and follow-up study of patients with DID and dissociative disorder not otherwise specified. A subsample of patients aged 18–30 progressed at a faster rate than did their older counterparts (Myrick et al., 2012). In another reanalysis of the data, patients who experienced revictimization, stressors, or both revictimization and stressors over the course of treatment fared worse than did patients who did not experience such events (Myrick, Brand, & Putnam, 2013). Although the findings of this large noncontrolled study are promising, no clear-cut conclusions can be drawn from the broader outcome research because of the lack of randomized trials, dropout rates as high as 68%, the variability in treatments provided, and the failure to document clinically meaningful changes following treatment (Lynn et al., 2014).

Proponents of the SCM have roundly criticized some PTM-based interventions for being highly suggestive in recovering supposedly repressed memories, identifying and speaking with alters, and encouraging "personalities" to interact (Lynn, Condon, & Colletti, 2013). Support for such concerns comes from a study in which the majority of patients developed "florid posttraumatic stress disorder during treatment" (Dell & Eisenhower, 1990, p. 361). Moreover, after treatment commences with some PTM-based approaches, patients tend to report an increased frequency of suicide attempts, hallucinations, severe dysphoria, and chronic crises (Lynn et al., 2014). Nevertheless, Brand and Loewenstein (2014) contended that DID treatment, including interacting with "dissociated self states," improves clinical outcomes and that depriving DID patients of such treatment may cause "iatrogenic harm" (see also Brand, Loewenstein, & Spiegel, 2014). As noted elsewhere (Lynn et al., 2014), research that compares negative sequelae across classic DID and conventional therapies (e.g., cognitive-behavioral) would be worthwhile. Finally, the intriguing findings we reported regarding the links among sleep, dissociation, and hyperassociation imply that interventions that ameliorate insomnia, reduce the frequency of nightmares, address other unusual sleep-related problems (e.g., narcolepsy), and promote cognitive control and affect regulation should also be a high treatment and research priority.

Conclusion

Despite, or perhaps because of, increased empirical attention devoted to dissociative disorders over the past few decades, controversy persists. It is nevertheless encouraging that some proponents of the SCM and PTM have now called for wide-ranging investigation into dissociative phenomena and agree that a multi-pronged investigatory approach that considers multiple determinants of dissociative disorders is the best way forward. There is little doubt that tension between competing perspectives will continue to generate provocative questions, healthy debate, and ultimately a more comprehensive and nuanced understanding of dissociative disorders.

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Schizophrenia Spectrum and Other Psychotic Disorders

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Schizophrenia is among the most debilitating of mental illnesses. It is typically diagnosed between 20 and 25 years of age, a stage of life when most people gain independence from parents, develop intimate romantic relationships, plan educational pursuits, and begin work or career endeavors (De Lisi, 1992). Because the clinical onset usually occurs during this pivotal time, the illness can have a profound negative impact on the individual's opportunities for attaining social and occupational success, and the consequences can be devastating for the patient's life course, as well as for family members (Addington & Addington, 2005). Further, the illness knows no national boundaries. Across cultures, estimates of the lifetime prevalence of schizophrenia range around 1% (Keith, Regier, & Rae, 1991; Kulhara, & Chakrabarti, 2001; Torrey, 1987; Arajarvi et al., 2005), although there is some research indicating that the rate may be somewhat lower with more stringent measurement criteria (0.4%; Saha Chant, Welham, & McGrath, 2005). Studies also suggest that the prognosis may differ among countries, with better outcomes in developing nations (Kulhara & Chakrabarti, 2001). However, more recent evidence suggest that variation in prognosis may not be that straightforward as there appears to be differences within individual developing nations in course of illness and discrepancies in measurement; it may be that access to treatment is associated with better outcome across countries (Cohen, Patel, Thara, & Gureje, 2008).

The origins of schizophrenia have continued to elude researchers, despite many decades of scientific research. To date, no single factor has been found to characterize all patients with the illness. This holds for potential etiological factors, as well as clinical phenomena. Patients with schizophrenia vary in symptom profiles, developmental histories, family backgrounds, cognitive functions, and even brain morphology and neurochemistry. Although this has led some to express dismay at our chances of ever findings the cause of schizophrenia, there is reason to be optimistic. Research efforts have succeeded in revealing numerous pieces of what is now recognized as a complex puzzle of etiological processes.

Based on findings from various lines of research, the consensus in the field is that: 1) Schizophrenia is a brain disease; 2) its etiology involves the interplay between genetic and environmental factors; 3) multiple developmental pathways eventually lead to disease onset; and 4) brain maturational processes play a role in the etiological process. In this chapter, we provide an overview of the current state of our knowledge about schizophrenia. We begin with a discussion of history and phenomenology of the disorder, and then proceed to a description of some of the key findings which have shed light on the illness.

History and Phenomenology

Written descriptions of patients experiencing psychotic symptoms (i.e. symptoms indicative of an inability to discern what is reality), similar to those of what we now call schizophrenia, have been recorded since antiquity. However, because psychotic symptoms can be a manifestation of a variety of disorders, it is unclear whether schizophrenia, as we view it today, is an ancient or a relatively new phenomenon. In the mid-to-late 19th century, European psychiatrists were investigating the etiology, classification and prognoses of various types of psychosis. The term *psychosis* refers broadly to the presence of psychotic symptoms and may various diagnoses such as schizophrenia-related disorders, together with mood disorders with psychotic features. At that time, the most common cause of psychosis was *tertiary syphilis*, although

researchers were unaware that there was any link between psychosis and syphilis (Kohler & Johnson, 2005). The psychological symptoms of tertiary syphilis frequently overlap with symptoms of what we now call schizophrenia. The cause of syphilis was eventually traced to an infection with the spirochete, *Treponema pallidum*, and antibiotics were found to be effective for prevention and treatment of the disorder. This important discovery served to illustrate how a psychological syndrome can be produced by an infectious agent. It also sensitized researchers to the fact that similar syndromes might be the result of different causes.

Emil Kraepelin (1856–1926) was the medical director of the famous Heidelberg Clinic. He was the first to differentiate schizophrenia, which he referred to as *dementia praecox* (or dementia of the young) from manic-depressive psychosis (Kraepelin, 1913). He also lumped together "hebephrenia," "paranoia," and "catatonia" (previously thought to be distinct disorders), and classified all of them as variants of dementia praecox. He based this classification on their similarities in age of onset and the clinical feature of poor prognosis. Kraepelin did not believe that any one symptom was diagnostic of dementia praecox, but instead focused on the total clinical picture and changes in symptoms over time. If a psychotic patient deteriorated over an extended period of time (months/years), the condition was assumed to be dementia praecox.

Many contemporary mental health professionals continue to expect negative outcomes in those afflicted with schizophrenia, and this expectation infuses the mental health profession with an unfortunate sense of therapeutic nihilism. Yet, while it is true that the majority of patients manifest a chronic course that entails lifelong disability, this bleak scenario is not always inevitable (Carpenter & Buchanan, 1994). The story of Dr. John Nash, professor and mathematician at Princeton, as told in the movie *A Beautiful Mind*, illustrates this point quite well. Dr. Nash was able to function at a very high level in his academic field, despite his struggle with schizophrenia.

Historical and Modern Conceptions of Psychosis Classification The term *schizophrenia* was introduced at the beginning of the 20th century by Eugen Bleuler (1857–1939), a Swiss psychiatrist and the medical director of a mental hospital in Zurich (Howells, 1991, pp. xii, 95). The word is derived from two Greek words: *schizo*, which means to tear or to split, and *phren*, which has several meanings. In ancient times, the word *phren*, meant "the intellect" or "the mind." *Phren* also referred to the lungs and the diaphragm, which were believed to be the seat of emotions. Thus, the word schizophrenia literally means the splitting or tearing of the mind and emotional stability of the patient.

Bleuler classified the symptoms of schizophrenia into fundamental and accessory symptoms. The fundamental symptoms of schizophrenia are often reported in textbooks as the four As, although, in fact, there are six As and one D (Bleuler, 1950). The fundamental symptoms are listed in Box 17.1. According to Bleuler, these symptoms are present in all patients, at all stages of the illness, and are diagnostic of schizophrenia.

Box 17.1 Bleuler's Fundamental Symptoms of Schizophrenia

- Disturbances of **a**ssociation (loose, illogical thought processes).
- Disturbances of **a**ffect (indifference, apathy or inappropriateness).
- **A**mbivalence (conflicting thoughts, emotions or impulses which are present simultaneously or in rapid succession).
- **A**utism (detachment from social life with inner preoccupation).
- **A**bulia (lack of drive or motivation).
- **D**ementia (irreversible change in personality).

Bleuler's "accessory symptoms" of schizophrenia included delusions, hallucinations, movement disturbances, somatic symptoms, and manic and melancholic states. In contrast to fundamental symptoms, he believed that these accessory symptoms were not present in all patients with schizophrenia, and often occurred in other illnesses. For these reasons, the accessory symptoms were not assumed to be as diagnostic of schizophrenia.

Further refinements in the diagnostic criteria for schizophrenia were proposed by Kurt Schneider (1959) in the mid-1900s. Like Bleuler, Kurt Schneider thought that certain "key" symptoms were diagnostic of schizophrenia. In his classification, he referred to these diagnostic symptoms as "first-rank symptoms" (see Box 17.2). He believed that, after medical causes of psychosis were ruled out, one could make the diagnosis of schizophrenia if one or more first-rank symptoms were present. Schneider's descriptions of the symptoms were more detailed and specific than were Bleuler's fundamental symptoms. Subsequent diagnostic criteria for schizophrenia have been heavily influenced by Schneider's approach.

Box 17.2 The Schneiderian "First-Rank Symptoms"

- Thought echoing or audible thoughts (the patient hears his thoughts out loud).
- Thought broadcasting (patient believes that others can hear his thoughts out loud).
- Thought intrusion (patient feel that some of his thought are from outside; that is, not originating in his own mind).
- Thought withdrawal (patient believes that the cause of having lost track of a thought is that someone is taking his thoughts away).
- Somatic hallucinations (unusual, unexplained sensations in one's body).
- Passivity feelings (patient believes that his thoughts, feelings or actions are controlled by another or others).
- Delusional perception (a sudden, fixed, false belief about a particular everyday occurrence or perception.

Following longitudinal research in the 1960s, a German researcher by the name of Gerd Huber laid out a list of symptoms that were evident (through retrospective study) in the early course of illness as well as in the later stages of psychosis (Huber, Gross, Shuttler, & Linz, 1980). These symptoms are termed "basic symptoms" and generally refer to symptoms reported by the patient themselves and include impairment in cognition, perception, motor function, will, initiative, level of energy, and tolerance of stress (Olson & Rosenbaum, 2006).

In subsequent years, investigators began to make a distinction between "positive" and "negative" symptoms of schizophrenia (Harvey & Walker, 1987). The positive symptoms are those that involve an excess of ideas, sensory experiences or behavior. Hallucinations, delusions and bizarre behaviors fall in this category. Most of the first-rank symptoms described by Schneider are also considered to be positive symptoms. Negative symptoms, in contrast, involve a decrease in behavior, such as blunted or flat affect, anhedonia, and lack of motivation. These symptoms were highlighted by Bleuler.

During the middle of the 20th century, different diagnostic criteria for schizophrenia became popular in different parts of the world. The "Kraepelinian" tradition, with its longitudinal requirements for diagnosis, identified patients with poorer long-term prognosis. In contrast, the Bleulerian and Schneiderian diagnostic systems allowed for a wider range of psychotic patients to be diagnosed with schizophrenia. Thus, the patients diagnosed with these two systems tended to have a better prognosis than those diagnosed in the more stringent Kraepelinian tradition. Because of these discrepancies, the use of multiple diagnostic systems had a detrimental effect on research progress; research findings from countries using different diagnostic criteria were not comparable, thus limiting the generalizability of the results.

The next generation of diagnostic systems evolved with the intent of achieving uniformity in diagnostic criteria and improving diagnostic reliability. Among these were the "Feighner" or "St. Louis" diagnostic criteria (Feighner, Robins, & Guze, 1972), and the Research Diagnostic Criteria developed by Robert Spitzer and his colleagues (Spitzer, Endicott, & Robins, 1978). These two approaches to the diagnosis of schizophrenia strongly influenced modern-day diagnostic systems, most notably, the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013). The DSM is now the most widely used system for diagnosing schizophrenia and other mental disorders.

Although the recent introduction of DSM-5 in May 2013 did not reflect any major departures from the preceding DSM-IV-TR (American Psychiatric Association, 2000), there are several noteworthy minor revisions affecting the diagnosis of schizophrenia. First, schizophrenia is now included with a group of disorders under the category of "Schizophrenia Spectrum and Other Psychotic Disorders," reflecting a shift toward a dimensional understanding of psychosis (Johns & van Os, 2001). As with DSM-IV-TR, there are six criteria (Table 17.1). In contrast to DSM-IV-TR, there are two distinct changes in DSM-5 for criterion A, which highlights specific characteristic symptoms that must be present for a significant amount of time during a 1-month period (or less if treated successfully). First, DSM-5 criterion A states that there must be two characteristic symptoms present from: 1) hallucinations; 2) delusions; 3) disorganized speech (e.g., frequent derailment or incoherence); 4) grossly disorganized or catatonic behavior; and 5) negative symptoms (i.e., affective flattening, alogia, or avolition). This is different, owing to the elimination of allowing for only one of these symptoms to be sufficient if it is bizarre in nature or if it is a Schneiderian first-rank auditory hallucination (e.g., multiple voices conversing with each other) (Tandon et al., 2013). The reason for the change relates to a general consensus in the field that there was poor empirical evidence to support the prior stipulation. Some researchers have indicated that this increased threshold for symptoms may exclude approximately 2% of individuals who held a diagnosis of schizophrenia under DSM-IV-TR (Pagsberg, 2013). Secondly, DSM-5 now requires that at least one of the criterion A symptoms must be a core positive symptom such as hallucinations, delusions, or disorganized speech. The reason for this change is that these three symptom groupings are considered "core positive symptoms" that reflect psychotic pathology (Tandon et al., 2013).

TABLE 17.1
DSM-5 Criteria for Schizophrenia

Criterion	Category	Description
A	Characteristic symptoms	Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated). At least one of these should include 1, 2, or 3. 1. Delusions 2. Hallucinations 3. Disorganized speech 4. Grossly disorganized or catatonic behavior 5. Negative symptoms (i.e., diminished emotion expression or avolition)
B	Social/occupational dysfunction	For a significant portion of the time since the onset of the disturbance, level of functioning in one or more major areas, such as work, interpersonal relations, or self-care, are markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, failure to achieve expected level of interpersonal, academic, or occupational achievement).
C	Duration	Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by only negative symptoms or by two or more symptoms listed in Criterion A present in an attenuated form (e.g., odd beliefs, unusual perceptual experiences).
D	Schizoaffective and mood disorder exclusion	Schizoaffective disorder and depressive or bipolar disorder with psychotic features have been ruled out because either (1) no major depressive or manic episodes have occurred concurrently with the active-phase symptoms; or (2) if mood episodes have occurred during active-phase symptoms, they have been present for a minority of the total duration of the active and residual periods of the illness.
E	Substance/general medical condition exclusion	The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.
F	Relationship to global developmental delay or autism spectrum disorder	If there is a history of autism spectrum disorder or a communication disorder of childhood onset, the additional diagnosis of schizophrenia is made only if prominent delusions or hallucinations, in addition to the other required symptoms of schizophrenia, are also present for at least 1 month (or less if successfully treated).
	Course specifiers	1. First episode, currently in acute episode 2. First episode, currently in partial remission 3. First episode, currently in full remission 4. Multiple episodes, currently in acute episode 5. Multiple episodes, currently in partial remission 6. Multiple episodes, currently in full remission 7. Continuous 8. Unspecified

There are no changes to criterion B, C, D, or E. Criterion B emphasizes that, in addition to the presence of characteristic symptoms, there must be significant social/occupational dysfunction. Schizophrenia can be diagnosed with DSM-5 when these signs and symptoms of the disorder are present for 6 months (including prodromal and residual phases; Criterion C). Further, significant mood disorders, such as depression or mania, along with schizoaffective disorder must be ruled out to ensure that the symptoms present are not better accounted for by one of these diagnoses (Criterion D). Also, general medical conditions or substance use that might lead to psychotic symptoms must be ruled out (Criterion E). Finally, in DSM-IV-TR, Criterion F stipulated that if a pervasive developmental disorder (PDD) or autistic disorder was present, schizophrenia could only be diagnosed if prominent delusions or hallucinations were present for at least 1 month (or less if successfully treated; DSM-IV-TR, 2000). In DSM-5, this criterion is restated to include "*or other communication disorder of childhood onset*" along with autism to acknowledge that there are additional disorders that may warrant diagnostic differentiation (Dyck, Piek, & Patrick, 2011; Tandon et al., 2013). In sum, the changes to the criterion (A and F specifically) appear to be minor and justified by existing evidence, will not significantly affect those individuals already carrying a diagnosis of schizophrenia, and will likely allow for improved diagnostic clarification.

Notably, the four subtypes of schizophrenia described in DSM-IV-TR (paranoid, disorganized, catatonic and undifferentiated) are no longer included in DSM-5, owing to minimal diagnostic stability and utility, as well as poor reliability and validity (Tandon et al., 2013). In place of these subtypes, a dimensional rating of severity of core symptoms is included in Section III, a section of the manual that includes tools to enhance diagnosis. The schizophrenia workgroup had strongly recommended including this dimensional approach in the primary text section but

this decision was overturned at the last minute because the American Psychiatric Association (APA) was concerned that this would hamper communication between providers and insurance companies (Barch et al., 2013). In any case, the inclusion of this new dimensional approach in DSM-5 highlights the heterogeneous presentation of schizophrenia and in theory, negates the necessity of subtypes while providing the possibility for increased diagnostic description for more effective communication between providers (Pagsberg, 2013).

There are also eight course specifiers for schizophrenia in DSM-5 that define the acute and longitudinal nature of the illness. These specifiers indicate whether the episode being assessed is the first episode or one of multiple episodes, along with coding whether the episode is active (currently in acute episode) or in full or partial remission. Finally, there are two course specifiers that allow for some ambiguity through labeling the episodes as continuous (i.e. the episodes are too continuous to determine a specific course of symptoms) and unspecified (i.e. the information needed to clarify course of symptoms is lacking). In addition to several other considerations, the purpose of these changes was to reduce comorbidity and the incidence of the "not otherwise specified" diagnoses and to improve diagnostic communication between healthcare providers. It remains to be seen whether comorbidity and diagnostic ambiguity will lessen and communication improve through these changes; however, the specifiers do appear to add some specificity to course of illness description that was absent in previous diagnostic manuals.

Also new in Section III of DSM-5 is the inclusion of *attenuated psychosis syndrome* (APS). This category identifies individuals who do not meet full criteria for schizophrenia, but who exhibit attenuated (less intense or severe) characteristic symptoms and intact reality testing. These individuals are at heightened risk of developing a psychosis spectrum disorder—as many as 30% will go on to develop a psychosis in a 2-year period (Cannon et al., 2008). The decision to include the new diagnosis accompanied a fierce debate in the period leading up to publication. Although expert consensus for the diagnosis is still evolving, the impetus for including APS in DSM-5 arose from accumulating evidence that high-risk patients are currently ill and at elevated risk for more serious mental illness (Cannon et al., 2008), the criteria for a risk state can be assessed with reliability and validity (in a research setting), and no DSM-IV diagnosis accurately captured the current illness/future risk (Addington et al., 2007; Yung et al., 2007). The proponents argued that providing a DSM-5 diagnosis could minimize potential harm to patients and families and could improve general provider education (Woods et al., 2010). Those who argued against the new label suggested that inclusion was premature because of a lack of information from community-based trials and significant concern about stigma (Shrivastava et al., 2011). Opponents also questioned the clinical validity of the syndrome because APS is predictive of a number of disorders outside schizophrenia (e.g., affective psychoses) and therefore, the label is in a non-specific initial stage. These critics also noted that because a primary focus relates to risk for future illness, a high rate of false positives (i.e., those who do not transition to psychosis) is ethically problematic as it exposes a disproportionate number of youth to unnecessary medications and stigma (Corcoran, First & Cornblatt, 2010). The ultimate decision to place APS in the research section of DSM-5 speaks to the valid points on both sides of this debate, and to the complex and delicate issues accompanying the diagnosis.

Several other diagnoses under the 'schizophrenia spectrum and other psychotic disorders' group are worth noting. The first is *schizotypal personality disorder* (SPD), which now falls both under this grouping, as well as under personality disorders. The diagnostic criteria for SPD includes social anxiety or withdrawal, affective abnormalities, eccentric behavior, unusual ideas (e.g., persistent belief in extrasensory perception/phenomena, aliens), and unusual sensory experiences (e.g., repeated experiences with confusing noises with peoples' voices, or seeing objects move). Although the individual's unusual ideas and perceptions are not severe or persistent enough to meet criteria for delusions or hallucinations, they are recurring and atypical of the person's cultural context. An extensive body of research demonstrates genetic and developmental links between schizophrenia and SPD. The genetic link between SPD and schizophrenia has been documented in twin and family history studies (Kendler, McGuire, Gruenberg, & Walsh, 1995; Kendler, Neale, & Walsh, 1995; Raine & Mednick, 1995). The developmental transition from schizotypal signs to schizophrenia in young adulthood has been followed in several recent longitudinal studies, with researchers reporting that 20–40% of schizotypal youth eventually develop a schizophrenia spectrum disorder (Cannon et al., 2008; Miller et al., 2002; Yung et al., 1998). The inclusion of schizotypal personality disorder in this grouping again illustrates the shift towards conceptualizing psychosis on a dimensional continuum.

An additional category, *schizophreniform disorder*, is for individuals whose symptoms do not meet the 6-month criterion. This diagnosis is frequently made as a prelude to the diagnosis of schizophrenia, when the patient presents for treatment early in the course of the disorder. Some individuals who fall into this category, however, will recover completely and not suffer further episodes of psychosis.

It is important to emphasize that, despite advances in diagnosis, the diagnostic boundaries of schizophrenia are still quite unclear (Wolff, 1991). Moreover, the boundaries between schizophrenia and mood disorders are sometimes obscure. Many individuals who meet criteria for schizophrenia show marked signs of depression or manic tendencies. These symptoms are sometimes present before the onset of schizophrenia, and frequently occur in combination with marked psychotic symptoms. As a result, the DSM-5 includes a diagnostic category called *schizoaffective disorder*. This disorder can be conceived

of, conceptually, as a hybrid between the mood disorders (bipolar disorder or major depression with psychotic features) and schizophrenia. The two subtypes of schizoaffective disorder are the depressive subtype (i.e., if the mood disturbance includes only depressive episodes) and the bipolar subtype (i.e., where the symptoms of the disorder have included either a manic or a mixed episode). Interestingly, the prognosis for patients with schizoaffective disorder is, on average, somewhere between that of schizophrenia and the mood disorders. Of note, DSM-5 changes affecting schizoaffective disorder include diagnosis based on consideration of the life course rather than just the current episode, such that mood disorder must be present for the majority of the total disorder duration after criterion A has been met (American Psychiatric Association, 2013).

Cognitive and Social Processes Deficits in Schizophrenia

Among the most well-established aspects of schizophrenia are the cognitive impairments that accompany the illness. In fact, some experts argued that cognitive impairment should have been added as a characteristic symptom in the DSM-5 (Keefe & Fenton, 2007); however, the DSM-5 Task Force decided against making this recommendation because of its lack of diagnostic specificity. Patients with schizophrenia manifest performance deficits on a broad range of cognitive tasks, from simple to complex (Green, Kern, Braff, & Mintz, 2000; Bozikas, Kosmidis, Kiosseoglou, & Karavatos, 2006). One of the most basic is the deficit in the very earliest stages of visual information processing. Using a laboratory procedure called backward masking, researchers have shown that compared with both healthy individuals and psychiatric controls, patients with schizophrenia are slower in the initial processing of stimuli (Green, Nuechterlein, Breitmeyer, & Mintz, 1999, 2006). One interesting finding in patients with schizophrenia relates to perceptual functioning. Individuals with schizophrenia tend not to be susceptible to optical illusions such as perceiving two-dimensional objects in three-dimensional form, and these perceptual impairments can actually produce superior performance on certain tasks (Keane, Silverstein, Wang, & Papathomas, 2013). In general, research suggests that patients with schizophrenia show deficits in attention/vigilance, context processing, working memory, episodic memory, verbal learning, visual learning, and reasoning and problem solving (Nuechterlein et al., 2004; Barch & Ceasar, 2012). Of note, there is some evidence that nicotine is beneficial to such cognitive impairments; there is ongoing work evaluating its effectiveness in clinical trials (Hong et al., 2011; Lieberman et al., 2013).

There are also deficits in thinking about social phenomena. Studies of social-cognitive abilities in schizophrenia patients have consistently shown that patients are impaired in their ability to comprehend and solve social problems, processing of emotions, social perception, attribution of events, and theory of mind (the ability to perceive the judgments, beliefs, intentions, and emotions of others; Green & Horan, 2010; Hooley, 2010; Penn, Corrigan, Bentall, Racenstein, & Newman, 1997; Meherwan Mehta et al., 2013). Social-cognitive imaging studies consistently point to abnormalities in brain regions and circuitry associated with social processing, adding to the evidence showing that social-cognitive impairment is a hallmark feature of schizophrenia. Deficits in social cognition may be partially due to limitations in more basic cognitive processes, such as memory and reasoning (Kring & Elis, 2013). However, basic cognitive impairments do not account completely for the more pervasive and persistent social-cognitive dysfunction observed in schizophrenia, and there is increasing evidence to support the idea that social cognition is a separate construct (Meherwan Mehta et al., 2013). For example, imaging studies show that there is something unique in brain activation when an individual is involved in a social versus strictly cognitive task, along with other research that finds social-cognitive and cognitive tasks often load on different domains when evaluated together using a statistical technique called factor analysis (Billike and Abowitz, 2013).

One of the diagnostic criteria for schizophrenia is blunted or inappropriate affect. It is not surprising therefore, that patients show abnormalities in the expression of emotion in both their faces and verbal communications. These abnormalities include less positive and more negative emotion, as well as emotional expressions that seem inconsistent with the social context (termed inappropriate affect; Brozgold et al., 1998; Tremeau et al., 2005). Further, patients with schizophrenia are less accurate than unaffected comparison subjects in their ability to label facial expressions of emotion, with a particular difficulty in labeling fear and sadness (Penn et al., 2000; Walker, E., 1981; Martin, Baudouin, Tiberghien, & Franck, 2005; Bigelow et al., 2006; Amminger et al., 2012). Numerous studies have examined the link between social-cognitive impairments, such as emotional processing and social functioning, indicating a strong relationship between poor social-cognitive ability and social difficulties (Salva et al., 2013). Specifically, research indicates that social cognition may be more closely tied to functioning than other cognitive domains, suggesting that social-cognition may be especially informative for understanding etiology and developing treatment (Fett et al., 2011).

Research Domain Criteria Initiative In 2009, the National Institute of Mental Health (one of the primary funding sources for research on schizophrenia) created a working group to set in motion efforts to develop new ways of classifying psychopathology based on dimensions of observable behavior and neurobiological measures. The aim of this effort is to acknowledge the need for a new approach to research aimed at cutting across diagnostic labels to create improved classification of mental

disorders through understanding underlying dimensions of functioning. Further, a focus of this initiative is to integrate multiple levels of analysis including genes, behavior, and neurobiology with the hope of translating basic research into improved understanding of psychopathology and better targeted treatment. While DSM-5 is purely a diagnostic tool, the Research Domain Criteria Initiative provides an alternate framework for research and has the potential to redefine mental disorders in future versions of the DSM.

The Origins of Schizophrenia

Biological Theories Kraepelin, Bleuler, and other early writers on schizophrenia did not offer specific theories about the origins of schizophrenia. They did suggest, however, that there might be a biological basis for at least some cases of the illness. Likewise, contemporary ideas about the origins of schizophrenia focus on biological vulnerabilities that are assumed to be present in early development. Researchers have identified two sources of constitutional vulnerability: genetic factors and environmental factors (e.g., prenatal or obstetric complications, traumatic brain injury). Both appear to have implications for prenatal and postnatal brain development, which is a focus of our understanding of the development of schizophrenia.

The Genetics of Schizophrenia One of the most well-established findings in schizophrenia research is that a vulnerability to the illness can be inherited (Gottesman, 1991). Behavior genetic studies utilizing twin, adoption and family history methods have all yielded evidence that the risk for schizophrenia is elevated in individuals who have a biological relative with the disorder; the closer the level of genetic relatedness, the greater the likelihood the relative will also suffer from schizophrenia.

In a review of family, twin, and adoption studies conducted from 1916 to 1989, Irving Gottesman (1991) outlined the compelling evidence for the role of genetic factors in schizophrenia. Monozygotic twins, who essentially share 100% of their genes, have the highest concordance rate for schizophrenia. Among monozygotic co-twins of patients with schizophrenia, 25–50% will develop the illness. Dizygotic twins and other siblings share, on average, only about half of their genes. About 10–15% of the dizygotic co-twins of patients are also diagnosed with the illness. Further, as genetic relatedness of the relative to the patient becomes more distant, such as from first-degree (parents and siblings) to second-degree relatives (grandparents, half siblings, aunts, and uncles), the relative's lifetime risk for schizophrenia is reduced.

Adoption studies have provided evidence that the tendency for schizophrenia to run in families is primarily due to genetic factors, rather than the environmental stressors related to growing up in close proximity to a mentally ill family member. In a seminal adoption study, Heston (1966) examined the rates of schizophrenia in adoptees with and without a biological parent who was diagnosed with the illness. He found higher rates of schizophrenia, and other mental illnesses, in the biological offspring of parents with schizophrenia, when compared with adoptees with no mental illness in biological parents. Similarly, in a Danish sample, Kety (1988) examined the rates of mental illness in the relatives of adoptees with and without schizophrenia. He found that the biological relatives of adoptees who suffered from schizophrenia had a significantly higher rate of the disorder than the adoptive relatives who reared them. Also, the rate of schizophrenia in the biological relatives of adoptees with schizophrenia was higher than in the relatives (biological or adoptive) of healthy adoptees. These adoption studies provide ample evidence for a significant genetic component in the etiology of schizophrenia.

Findings from an adoption study in Finland indicate that genetic influences often act in concert with environmental factors. Tienari, Wynne, Moring, and Lahti (1994) found that the rate of psychosis and other severe disorders was significantly higher in adoptees who had biological mothers with schizophrenia than in the matched control adoptees who had no history of having a first-degree relative with psychosis. However, the difference between the groups was only detected in adoptive families that were rated as dysfunctional. The genetic vulnerability was mainly expressed in association with a disruptive adoptive environment, and was not detected in adoptees reared in a healthy, possibly protective, family environment. These findings, which highlight a genetic vulnerability interacting with environmental events, are consistent with the prevailing diathesis-stress models of etiology. Taken together, the findings from behavioral genetic studies of schizophrenia lead to the conclusion that the disorder involves multiple genes, rather than a single gene (Gottesman, 1991; Van Winkel et al., 2010).

Consistent with this assumption, attempts to identify a genetic locus that accounts for a significant proportion of cases of schizophrenia have not met with success. Instead, researchers using molecular genetic techniques have identified numerous genes that may account for a small proportion of cases. In the past decade, linkage studies using genome-wide association scans have evaluated over 1,000 genes for schizophrenia (Gejman, Sanders, & Kendler, 2011). Association studies compare variations in specific gene sequences between individuals with and without schizophrenia. Variants found with significantly different frequency among those with schizophrenia are considered to confer susceptibility to the disease. Results from association studies generally have very small effect sizes, owing to the large number of gene variants that can potentially be evaluated; thus, while replication is critical, efforts to do so have met with limited success (Gejman et al., 2011). However, through combining data from multiple studies, results have uncovered a number of notable common polymorphisms (variations in DNA sequence, such

as single nucleotide polymorphisms involving the alteration in a single nucleotide of the DNA sequence) and copy number variants (variations in DNA structure involving the number of copies of a section of DNA within an individual's genotype; Van Winkel et al., 2010; Insel, 2010; Gejman et al., 2011). Findings highlight the heterogeneity of schizophrenia in that thousands of polymorphisms and multiple rare copy number variants (larger alterations in the DNA structure occurring relatively rarely in the population) may underlie the disorder, suggesting that the risk accounted for by each individual variant is small (Purcell et al., 2009; van Winkel et al., 2010). Despite the large number of genetic variants involved, research postulates that approximately 32% of the underlying contribution to schizophrenia may be explained by such common polymorphisms (Purcell et al., 2009; Ripke et al., 2013).

Using quantitative genetic techniques with large twin samples, researchers have shown that there is significant overlap in the genes that contribute to schizophrenia, schizoaffective disorder, bipolar disorder, and other neurodevelopmental disorders such as autism (Cardno, Rijsdijk, Sham, Murray, & McGuffin, 2002; Fanous & Kendler, 2005; van Winkel et al., 2010). Based on these and other findings, many experts have concluded that genetic vulnerability does not conform to the diagnostic boundaries listed in DSM and other taxonomies (e.g., Boks, Leask, Vermunt, & Kahn, 2007; Pelletier & Mittal, 2012). Rather, it appears that there is a genetic vulnerability to psychosis in general, and that the expression of this vulnerability can take the form of schizophrenia or an affective psychosis, depending on other genetic and acquired risk factors. Clearly, more research is needed to understand the specificity for genetic liability for schizophrenia and mood disorders.

As mentioned above, we now know that the environment begins to have an impact before birth; prenatal events are linked with risk for schizophrenia, and some of these events are discussed below. Thus, in order to index environmental events that contribute to non-genetic constitutional vulnerability, we must include both the prenatal and postnatal periods. At this point, however, researchers are not in a position to estimate the relative magnitude of the inherited and environmental contributors to the etiology of schizophrenia. Moreover, we do not yet know whether genetic vulnerability is present in all cases of schizophrenia. Some cases of the illness may be solely attributable to environmental risk factors.

Neurotransmitters The idea that schizophrenia involves an abnormality in the brain first began with a focus on neurotransmission. Initial neurotransmitter theories focused on epinephrine and norepinephrine. Subsequent approaches have hypothesized that serotonin, glutamate and/or gamma-aminobutyric acid (GABA) abnormalities are involved in schizophrenia. But, compared with other neurotransmitters, dopamine has played a more enduring role in theorizing about the biochemical basis of schizophrenia. In this section, we review the major neurotransmitter theories of schizophrenia, with an emphasis on dopamine.

In the early 1950s, investigators began to suspect that dopamine might be playing a central role in schizophrenia. Dopamine is widely distributed in the brain and is one of the neurotransmitters that enables communication in the circuits that link subcortical with cortical brain regions (Jentsch, Roth, & Taylor, 2000). Since the 1950s, support for this idea has waxed and waned. In the past decade, however, there has been a resurgence of interest in dopamine, largely because research findings have offered a new perspective.

The initial support for the role of dopamine in schizophrenia was based on two indirect pieces of evidence (Carlsson, 1988): 1) Drugs that reduce dopamine activity also serve to diminish psychotic symptoms; and 2) drugs that heighten dopamine activity exacerbate or trigger psychotic episodes. It was eventually shown that standard antipsychotic drugs had their effect by blocking dopamine receptors, especially the "D2" subtype that is prevalent in subcortical regions of the brain. The newer antipsychotic drugs, or "atypical" antipsychotics, have the advantage of causing fewer motor side effects. Nonetheless, they also act on the dopamine system by blocking various subtypes of dopamine receptors.

The relationship between dopamine activity and psychotic symptoms can be demonstrated by studies examining compounds, such as levodopa, that are used to treat Parkinson's disease by increasing dopamine transmission. For example, motor abnormalities associated with Parkinson's disease (i.e., hypokinesias; slow jerking movements, rigidity) are related to low levels of dopamine characteristic of the disease. However, patients with Parkinson's disease, who are being treated with dopamine agonists (i.e., levodopa-induced elevated striatal dopamine activity) show drug-induced dyskinesias (i.e., involuntary bodily movements such as writhing or jerking; Hoff, Plas, Wagemans, & van Hilten, 2001), and in extreme cases, psychotic symptoms (Papapetropoulos & Mash, 2005). In a similar vein, other amphetamines such cocaine, increase dopamine activity and can cause both hyperkinesias and psychotic symptoms (Weiner, Rabinstein, Levin, Weiner, & Shulman, 2001). The interplay between dopamine activity and movement has also been seen in research examining genetics and drug responsivity in schizophrenia. For example, schizophrenia patients with the type *3 or *4 alleles of the CYP2D6 gene related to poor metabolization of neuroleptic drugs show a heightened rate of dyskinesias (Ellingrod, Schultz, & Arndt, 2002).

Early studies of dopamine in schizophrenia sought to determine whether there was evidence of excess neurotransmitter in patients with schizophrenia. But concentrations of dopamine and its metabolites were generally found not to be elevated in body fluids from patients with schizophrenia. When investigators examined dopamine receptors, however, there was some evidence of increased

densities. Both postmortem and functional magnetic resonance imaging studies of patients' brains yielded evidence that the number of dopamine D2 receptors tends to be greater in patients than normal controls (Kestler, Walker, & Vega, 2001). Controversy has surrounded this literature, because antipsychotic drugs can change dopamine receptor density. Nonetheless, even studies of never-medicated patients with schizophrenia have shown elevations in dopamine receptors (Kestler et al., 2001). Thus, the first version of the dopamine hypothesis was formed, which focused on hyperdomapinergic (increased levels of dopamine) activity in the brain based on noted increased transmission of dopamine and the blocking of receptors to treat psychosis; this hypothesis was further refined in the 1990s to highlight hyperdopaminergic activity in the subcortical regions of the brain and hypoactiviation in the prefrontal cortex (Howes & Kapur, 2009).

The role of dopamine in schizophrenia has been further clarified following research showing additional abnormalities in dopamine transmission. For example, dopamine synthesis and release may be more pronounced in the brains of people with schizophrenia than among unaffected individuals (Lindström et al., 1999). When patients with schizophrenia and normal controls are given amphetamine, a drug that enhances dopamine release, the patients show more augmented dopamine release (Abi-Dargham et al., 1998; Soeares & Innis, 1999). Further, there are a number of replicated studies showing elevated presynaptic dopamine availability in patients with psychosis (Howes & Kapur, 2009). In concordance with these results, more recent evidence suggests that the primary dopamine activity abnormalities for schizophrenia exists in the three areas involving presynapse, synapse and release of dopamine, rather than in the dopamine receptors themselves (Howes et al., 2012). At present, antipsychotic medications do not target these areas, and there is some suggestion to emphasize presynaptic synthesis and release in treatment efforts (Howes et al., 2012).

Glutamate, an excitatory neurotransmitter, also may play an important role in the neurochemistry of schizophrenia. Glutamatergic neurons are part of the pathways that connect the hippocampus, prefrontal cortex, and thalamus, all regions that have been implicated in schizophrenia. There is evidence of diminished activity at glutamatergic receptors among patients with schizophrenia in these brain regions (Carlsson, Hansson, Waters, & Carlsson, 1999; Coyle, 2006; Ghose, Gleason, Potts, Lewis-Amezcua, & Tamminga, 2009; Marsman et al., 2013). One of the chief receptors for glutamate in the brain is the N-methyl-D-aspartic acid (NMDA) subtype of receptor. Blocking NMDA receptors produces the symptomatic manifestations of schizophrenia in normal subjects, including negative symptoms and cognitive impairments. For example, administration of NMDA receptor antagonists, such as phencyclidine and ketamine, induces a broad range of schizophrenic-like symptomatology in humans, and these findings have contributed to a hypoglutamatergic (decreased levels of glutamate in the brain) hypothesis of schizophrenia (Coyle, 2006; Marsman et al., 2013). Conversely, drugs that indirectly enhance NMDA receptor function can reduce negative symptoms and improve cognitive functioning in schizophrenia patients. It is important to note that the idea of dysfunction of glutamatergic transmission is not inconsistent with the dopamine hypothesis of schizophrenia, because there are reciprocal connections between forebrain dopamine projections and systems that use glutamate (Grace, 2010). Thus, dysregulation of one system would be expected to alter neurotransmission in the other (Stone, Morrison, & Pilowsky, 2007). Furthermore, the progressive deterioration of brain tissue seen in schizophrenia may be tied to the dysfunction of the NMDA receptors and the glutamatergic system (Marsman et al., 2013).

There also is evidence of abnormalities in GABA neurotransmission in the dorsolateral prefrontal cortex (Lewis & Hashimoto, 2007; Lewis et al., 2012). Although the implications of GABA alterations remains unclear (Taylor, Demeter, Luan Phan, Tso, & Welsh, 2013), disruptions in GABA, an inhibitory neurotransmitter, may underlie the reduced capacity for working memory in schizophrenia. Current theories about the role of GABA in schizophrenia assume that it is important because cortical processes require an optimal balance between GABA inhibition and glutamatergic excitation (Costa et al., 2004). In addition to work highlighting the connection between GABA and cognition, recent research has identified potential clinical relevance of GABA. For example, the blockage of GABA receptor activity can create psychotic symptoms in individuals with schizophrenia who are not actively psychotic (Ahn, Gil, Seibyl, Sewell, & D'Souza, 2011). Furthermore, current evidence suggests that GABA receptors are linked with negative affect in schizophrenia (Taylor et al., 2013).

The true picture of the neurochemical abnormalities in schizophrenia may be more complex than we would like to assume. All neurotransmitter systems interact in intricate ways at multiple levels in the brain's circuitry (Carlsson et al., 2001). Consequently, an alteration in the synthesis, reuptake or receptor density, and/or affinity for any one of the neurotransmitter systems would be expected to have implications for one or more of the other neurotransmitter systems. Further, because neural circuits involve multiple segments that rely on different transmitters, it is easy to imagine how an abnormality in even one specific subgroup of receptors could result in the dysfunction of all the brain regions linked by a particular brain circuit.

Abnormalities in Brain Structure The first reports of abnormal brain structure in individuals with schizophrenia were based on computerized axial tomography (CAT), and showed that affected individuals had enlarged brain ventricles, especially increased volume of the lateral ventricles (Dennert & Andreasen, 1983). However, these signs

were viewed as non-specific because enlarged ventricles could reflect wide spread brain matter abnormalities (i.e., smaller volume is reflected in larger ventricles). As new techniques for brain scanning were developed, these findings were replicated, and additional abnormalities were detected (Henn & Braus, 1999). Magnetic resonance imaging, a technique capable of providing significantly greater detail, revealed decreased frontal, temporal and whole brain volume among people with schizophrenia (Lawrie & Abukmeil, 1998; Tanskanen et al., 2010). More fine-grained analyses demonstrated reductions in the size of the anterior cingulate, amygdala, thalamus, insula, and hippocampus (Shepard et al., 2012). Diffusion tensor imaging (DTI) has allowed investigators to examine neuronal connections and white matter tracts in the brain and has shown widespread pathology in multiple tracts. White matter integrity refers to the ability of water molecules to diffuse along the axon in one direction and thus indicates increased fiber integrity and level of myelination. Deficient white matter integrity may be particularly present in the fronto-temporal areas of the brain (Ellison-Wright and Bullmore, 2009). Furthermore, recent evidence suggests that white matter integrity may be related to negative symptoms of schizophrenia (Nakamura et al., 2012).

Current neurodevelopmental theory of schizophrenia postulates that there may be deficient myelination and interneuron activity in the brain along with excessive pruning of excitatory synapses (i.e. overstepping the normal maturational process of removing synapses no longer in use) and that the aggregate impact of these abnormalities may account for the progressive decrease in gray matter volume seen in schizophrenia (Insel, 2010). Furthermore, the deficient myelination may be related to abnormal connectivity within the brain, emphasizing the need to further study brain circuitry using methods such as DTI.

There is also a wealth of evidence to suggest that irregularities in neural development during the adolescent period (immediately before the mean age of onset) contribute to the abnormalities of structural and connective tissue observed in adults with schizophrenia. Although few longitudinal studies of high-risk individuals (prospective designs) have been conducted, results suggest a developmental pattern of declining gray matter structures in left inferior fontal, medial temporal, cerebral and cingulate regions (Job, Whalley, Johnstone, & Lawrie, 2005; Pantelis et al., 2007). Further, a recent meta-analysis of gray matter in high-risk patients who were not taking antipsychotics revealed decreased gray matter in the temporal and limbic prefrontal cortex along with reductions in temporal, anterior cingulate, cerebellar, and insular regions being associated with psychosis onset in first-episode patients (Fusar-Poli, Radua, McGuire, & Borgwardt, 2012). In regards to white matter, the results of high-risk research is very heterogeneous, noting numerous tracts and various lobes exhibiting white matter abnormalities. Researchers have observed DTI evidence that patients failed to show a normal pattern of increasing white matter integrity with age (Karlsgodt et al., 2009; Carletti et al., 2012) and findings indicate declining white matter integrity coinciding with progression to psychosis (von Hohenberg et al., 2013). Specifically, multiple studies have highlighted reduced white matter integrity in the superior longitudinal fasciculus, together with connections involving the frontal, fronto-temporal and fronto-limbic regions (Karlsgodt et al., 2009; Bloemen et al., 2010; Carletti, et al, 2012; Samartzis, Dima, Fusar-Poli, & Kyriakopoulos, 2013; von Hohenberg et al., 2013; Mittal et al., 2013; Dean et al., 2013; Bernard et al., 2014). Taken together, this accumulating evidence points to a prominent role of abnormal adolescent neurodevelopment and conductivity in the pathogenesis of schizophrenia and suggests that many of the noted structural and connective deficits may have been present prior to the formal onset of illness.

Despite the plethora of research findings indicating the presence of abnormalities in the brains of patients with schizophrenia, no specific abnormality has yet been shown to be definitely pathognomonic. In other words, there is no evidence that a specific morphological abnormality is unique to schizophrenia or characterizes all schizophrenia patients. The structural brain abnormalities observed in schizophrenia are, therefore, gross manifestations of the occurrence of a deviation in neurodevelopment that has implications for the functioning of neurocircuitry.

Psychosocial and Environmental Theories In the early part of the 20th century, psychosocial theories of schizophrenia dominated the literature. For example, Sigmund Freud, the father of psychoanalysis, believed that psychological processes resulted in the development of psychotic symptoms (Howells, 1991). In 1948, Frieda Fromm-Reichmann proposed a theory of schizophrenia which postulated that the disorder arose in response to rearing by a 'schizophrenogenic mother' (Fromm-Reichmann, 1948). Although this hypothesis has fallen into disfavor because of a lack of support from empirical research, it caused considerable suffering for families. Subsequently, family interaction models of the etiology of schizophrenia were offered by various theorists (Howells, 1991). Although these early psychosocial theories contributed relatively little to our understanding of the etiology of schizophrenia, they did highlight the importance of considering the role of the family in relapse prevention and recovery for the patient. There has also been considerable focus on other types of environmental stressors involving prenatal and perinatal factors.

Prenatal and Perinatal Factors There is extensive evidence that obstetric complications have an adverse impact on the developing fetal brain, and may contribute to vulnerability for schizophrenia. Birth cohort studies have

shown that patients with schizophrenia are more likely to have a history of exposure to obstetric complications (Brown & Derkits, 2010; Buka, Tsuang & Lipsitt, 1993; Dalman, Allebeck, Cullberg, Grunewald, & Koester, 1999; Forsyth et al., 2013; Takagai et al., 2006). Included among these are prenatal conditions, such as toxemia and preeclampsia, and labor and delivery complications. A meta-analysis of the literature on obstetric complications by Cannon, Jones, and Murray (2002) concluded that, among the different types of obstetric complication, complications of pregnancy (bleeding, preeclampsia, diabetes, and rhesus factor incompatibility) were the most strongly linked with later schizophrenia, followed by abnormal fetal growth and development and complications of delivery. In the National Collaborative Perinatal Project, which involved over 9,000 children followed from birth through adulthood, the odds of developing adult onset schizophrenia increased linearly with an increasing number of hypoxia-related complications (resulting from lack of oxygen at birth; Cannon, 1998; Zornberg, Buka, & Tsuang, 2000).

Another prenatal event that has been linked with increased risk for schizophrenia is maternal viral infection. The risk rate for schizophrenia is elevated for individuals born shortly after an influenza epidemic (Barr, Mednick, & Munk-Jorgensen, 1990; Brown et al., 2004; Limosin, Rouillon, Payen, Cohen, & Strub, 2003; Murray, Jones, O'Callaghan, & Takei, 1992), or after being prenatally exposed to rubella (Brown, Cohen, Harkavy-Friedman, & Babulas, 2001). For example, research suggests that there may be a threefold increase in risk for schizophrenia if a fetus is exposed to influenza in the first half of gestation and sevenfold increase if exposed in the first trimester (Brown & Derkits, 2010). The findings from research on prenatal maternal infection might be connected to the "season-of-birth" effect in schizophrenia. A meta-analysis has shown that a disproportionate number of patients with schizophrenia are born during the winter months (Davies, Welham, Chant, Torrey, & McGrath, 2003). This timing may reflect seasonal exposure to viral infections, which are most common in late fall and early winter. Thus, the fetus would have been exposed to the infection during the second trimester. The second trimester is an important time for brain development, and disruptions during this stage may lead to developmental abnormalities.

Studies of rodents and non-human primates have shown that prenatal maternal stress can interfere with fetal brain development, and is associated with elevated glucocorticoid release and hippocampal abnormalities in the offspring (Charil, Laplante, Vaillancourt, & King, 2010; Coe et al., 2003). Along the same lines, in humans there is evidence that stressful events during pregnancy are associated with greater risk for schizophrenia and other psychiatric disorders in adult offspring. Researchers have linked the incidence of schizophrenia to various maternal stressors during pregnancy, including bereavement (Huttunen, 1989), famine (Susser & Lin, 1992; St Clair et al., 2005), military invasion (van Os & Selten, 1998), war (Malaspina et al., 2008), flood (Selten, Graaf, van Duursen, Gispen-de Wied, & Kahn, 1999) and earthquake (Watson, Mednick, Huttunen, & Wang, 1999). It is likely that prenatal stress triggers the release of maternal stress hormones, which have been found to disturb fetal neurodevelopment and subsequent functioning of the hypothalamic–pituitary–adrenal (HPA) axis, which, in turn influences behavior and cognition (Seckl & Holmes, 2007). In addition, maternal stress may affect maternal proinflammatory cytokines, which have been associated with increased risk of offspring with schizophrenia (Brown & Derkits, 2010).

One of the chief questions confronting researchers is whether obstetric complications act independently to increase risk for schizophrenia or have their effect in conjunction with a genetic vulnerability (Mittal, Ellman, & Cannon, 2008). One possibility is that the genetic vulnerability for schizophrenia involves an increased sensitivity to prenatal factors that interfere with fetal neurodevelopment (Cannon, 1998; Preti, 2005; Walshe et al., 2005). It is also plausible that obstetric events act independently of genetic vulnerabilities, although such effects would likely entail complex interactions among factors (Susser, Brown, & Gorman, 1999). For example, to produce the neurodevelopmental abnormalities that confer risk for schizophrenia, it may be necessary for a specific obstetric complication to occur during a critical period of cellular migration and/or in conjunction with other factors such as maternal fever or immune response. Research shows evidence that the presence of serious obstetric complications may interact with certain genes, those that are regulated by hypoxia or involved in neurovascular function, to increase risk for schizophrenia (Nicodemus et al., 2008). More recently, there is some evidence to suggest that the former hypothesis is true—that genetic vulnerability heightens susceptibility for the development of brain abnormalities following obstetric complications (Forsyth et al., 2013).

Course and Prognosis

Assuming that genetic and obstetrical factors confer the vulnerability for schizophrenia, the diathesis must be present at birth. Yet, schizophrenia is typically diagnosed in late adolescence or early adulthood, with the average age of diagnosis in males about 4 years earlier than for females (Riecher-Rossler & Hafner, 2000). This raises intriguing questions about the developmental course prior to the clinical onset.

Premorbid Development There is compelling evidence that there are signs of schizophrenia long before the illness is diagnosed. Most of these signs are subtle and do not reach the severity of clinical disorder. Nonetheless, when compared with children with healthy adult outcomes, children who later develop schizophrenia manifest deficits in multiple domains. In some of these domains, the deficits are apparent as early as infancy.

In the area of cognitive functioning, children who later develop schizophrenia tend to perform below their healthy siblings and classmates. These cognitive deficits are reflected in lower scores on measures of achievement, poorer grades in school, and a lower childhood IQ compared with peers who do not go on to develop schizophrenia (Aylward, Walker, & Bettes, 1984; Dickinson, 2014; Jones, Rodgers, Murray, & Marmot, 1994). However, results are mixed and a recent meta-analysis shows no significant difference in performance on general academic achievement tests or mathematic achievement tests between those who go on to develop schizophrenia and those who do not (Dickinson et al., 2012). Specifically, research suggests early impairment in verbal knowledge, visual knowledge, simple reasoning skills, and a worsening trajectory of speeded performance, working memory, and complex problem solving (Dickinson, 2014; Richenber et al., 2010). Children who later are diagnosed with schizophrenia also show abnormalities in social behavior. They are less responsive in social situations, show less positive emotion (Walker & Lewine, 1990; Walker, Grimes, Davis, & Smith, 1993), and have poorer social adjustment than children with healthy adult outcomes (Done, Crow, Johnstone, & Sacker, 1994). In our studies of the childhood home movies of patients with schizophrenia, we found that the children who develop schizophrenia later in life showed more negative facial expression of emotion than did their siblings as early as the first year of life, indicating that the vulnerability for schizophrenia is subtly manifested in the earliest interpersonal interactions (Walker et al., 1993).

Vulnerability to schizophrenia is also apparent in motor functions. When compared with their siblings with healthy adult outcomes, children who develop schizophrenia show more delays and abnormalities in motor development, including deficits in the acquisition of early motor milestones, such as bimanual manipulation and walking (Walker, Savoie, & Davis, 1994). Deficits in motor function extend throughout the premorbid period (Walker, Lewis, Loewy, & Palyo, 1999), and persist after the onset of the clinical illness (McNeil, Cantor-Graae, & Weinberger, 2000). Furthermore, abnormal gesture behavior has been observed in both premorbid (Mittal, Tessner et al., 2006; Mittal, Walker et al., 2010) and unmedicated individuals with schizophrenia (Troisi, Spalletta, & Pasini, 1998). These data imply that the movement abnormalities recognized in schizophrenia are likely to have complex interactions with language and motor planning centers.

It is important to note that neuromotor abnormalities are not pathognomonic for schizophrenia, in that they are observed in children at risk for a variety of disorders, including learning disabilities, and conduct and mood disorders. But they are one of several important clues pointing to the involvement of brain dysfunction in schizophrenia. Further, although medication-induced movement abnormalities, such as tardive dyskinesia, involve characteristic motor signs, these are not to be confused with involuntary movements which have been demonstrated to be present in drug-free groups such as at-risk infants (Fish, 1987), at-risk adolescents (Walker, Lewis et al., 1999), and never medically treated schizophrenia patients (Khot & Wyatt, 1991).

Despite the subtle signs of abnormality that have been identified in children at risk for schizophrenia, most of these children do not manifest diagnosable mental disorders in childhood. Thus, while their parents may recall some irregularities in their development, most children who eventually develop schizophrenia were not viewed as clinically disturbed in childhood. But the picture often changes in adolescence. Many adolescents who go on to develop schizophrenia show a pattern of escalating adjustment problems (Walker & Baum, 1998). They show a gradual increase in feelings of depression, social withdrawal, irritability, and noncompliance. This developmental pattern is not unique to schizophrenia: adolescence is also the critical period for the expression of the first signs of mood disorders, substance abuse, and other mental disorders. As a result, researchers view adolescence as a critical period for the emergence of various kinds of behavioral dysfunction (Corcoran et al., 2003; Walker, 2002).

Among the behavioral risk indicators sometimes observed in "pre-schizophrenic" adolescents are "subclinical" signs of psychotic symptoms. These signs comprise the risk state that is now referred to as "ultra-high risk" or APS in the DSM-5 research section (American Psychiatric Association, 2013; described in the section on "Classification," above) and is considered to represent the putative prodromal stage of psychosis. Specifically, these symptoms are termed attenuated positive symptoms, and in research clinics typically fall under one of the following five subgroups: unusual thought content, suspiciousness/paranoia, grandiosity, perceptual abnormalities, or disorganized communication (Gee and Cannon, 2012). For APS criteria, the individual must experience the presence of symptoms at least once per week in the last month and the onset of the symptoms must be in the last 12 months or symptoms must have worsened in the last 12 months (Tsuang et al., 2013). These individuals tend to exhibit declining social and role functioning along with the subthreshold psychotic symptoms. Furthermore, research suggests that the neurocognitive and social-cognitive performance of youth with APS is somewhat between that of healthy controls and patients with schizophrenia. Although this risk period requires further study, the most recent meta-analysis suggests that approximately 18% of those identified as APS will convert to a psychotic disorder within the first 6 months and 36% after 3 years (Fusar-Poli et al., 2012). Research indicates that with each year, there is a reduction in this risk of conversion to a psychotic disorder, but it remains unclear whether seeking help/treatment is responsible for this decrease in psychosis transition or whether a certain portion of these individuals identified as at-risk would never have converted (i.e. false positives; Yung et al., 2007).

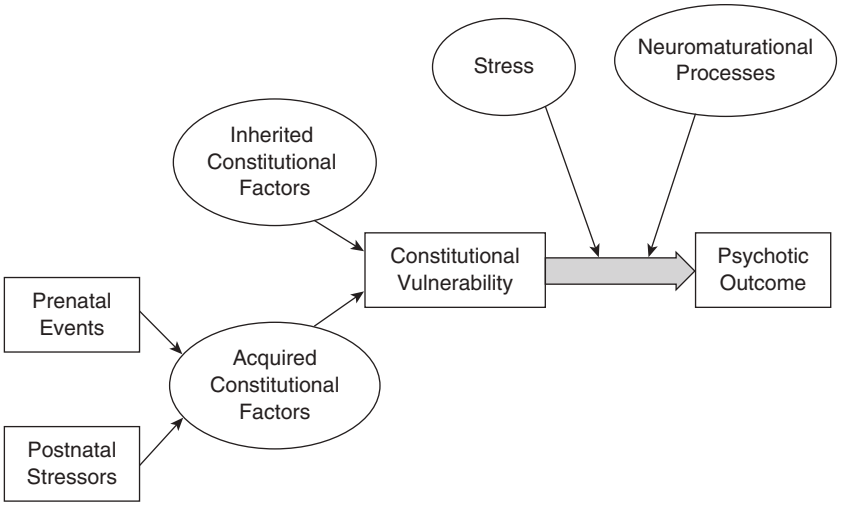

Figure 17.1 A diathesis-stress model of the etiology of schizophrenia.

Illness Onset The picture that has emerged to describe illness onset is best described in the framework of the diathesis-stress model that has dominated the field for several decades (Walker & Diforio, 1997; Walker, Mittal & Tessner, 2008).

Figure 17.1 illustrates a contemporary version of the diathesis-stress model. This particular model postulates that constitutional vulnerability (i.e., the diathesis) emanates from both inherited and acquired constitutional factors. The inherited factors are genetically determined characteristics of the brain that influence its structure and function. Acquired vulnerabilities arise mainly from prenatal events that compromise fetal neurodevelopment.

Whether the constitutional vulnerability is a consequence of genetic factors or environmental factors, or a combination of both, the model assumes that vulnerability is, in most cases, congenital. But the assumption that vulnerability is present at birth does not imply that it will be clinically expressed at any point in the life span. Rather, the model posits that two sets of factors determine the postnatal course of the vulnerable individual. First, external stressors influence the expression of the vulnerability. Although this is a long-standing assumption among theorists, it is important to clarify it. Empirical research has provided evidence that episodes of schizophrenia follow periods of increased life stress (Horan et al., 2005; Ventura, Nuechterlein, Hardesty, & Gitlin, 1992). Nonetheless, there is no evidence that individuals affected by schizophrenia experience more stressful events, perhaps with the exception of childhood trauma, than individuals without schizophrenia, but rather that they are more sensitive to stress when it occurs (Holtzman et al., 2013). This assumption is the essence of the model; the interaction between vulnerability and stress is critical.

Diathesis-stress models have incorporated mechanisms to account for the adverse impact of stress on brain function (Walker & Diforio, 1997; Walker, Mittal, & Tessner, 2008). The HPA axis, which is responsible for the release of cortisol and other stress hormones, has been examined as one of the primary neural systems triggered by stress exposure, leading to the expression of vulnerability for schizophrenia (Walder, Walker, & Lewine, 2000). Results from this research indicate that psychotic disorders are associated with elevated baseline and challenge-induced HPA activity, that antipsychotic medications reduce HPA activation, and that agents that augment stress hormone release exacerbate psychotic symptoms (Walker, Mittal et al., 2008).

In addition, the model assumes that neuromaturation is a key element. In particular, adolescence/early adulthood appears to be a critical period for the expression of the vulnerability for schizophrenia. Thus, some aspects(s) of brain maturational processes during the post-pubertal period are likely playing an important role in triggering the clinical expression of latent liabilities (Corcoran et al., 2003; Insel, 2010; Walker, Kestler, Bollini, & Hochman, 2004).

The onset of the first episode of schizophrenia may be sudden or gradual. There is some evidence that longer untreated psychotic episodes may be harmful for patients with schizophrenia and may result in a worse course of illness (Davidson & McGlashan, 1997; Harris et al., 2005; Perkins et al., 2004). However, this conclusion is controversial, and some researchers suggest that the relation between longer duration of untreated psychosis and worse prognosis may be a product of poorer premorbid functioning and an insidious onset (Larsen et al. 2001). Nonetheless, early intervention is important, regardless of the specific causal factors, as recent evidence suggests that duration of untreated psychosis is indeed a significant predictor of outcome and that the relationship between duration and functioning may be mediated by the presence of negative symptoms (Hill et al., 2012).

People with schizophrenia vary in their course of illness and prognosis. Being male, having a gradual onset, an early age of onset, poor premorbid functioning, and

a family history of schizophrenia are all associated with poorer prognosis (Gottesman, 1991). In addition, some environmental factors contribute to a worse outcome. For example, patients with schizophrenia who live in homes where family members express more negative emotion are more likely than those with supportive families to have more frequent relapses (Rosenfarb, Bellack, & Aziz, 2006; Butzlaff & Hooley, 1998).

Exposure to stress can exacerbate schizophrenia symptoms. Researchers have found an increase in the number of stressful events in the months immediately preceding a schizophrenia relapse (Horan et al., 2005; Ventura et al., 1992). Finally, there is a rapidly accumulating body of research indicating that heavy, consistent cannabis use is associated with a threefold increase in risk of schizophrenia, earlier onset of disorder in vulnerable individuals, and exacerbation of psychotic symptoms (Manrique-Garcia et al., 2012; Rey, Martin, & Krabman, 2004;). There is some evidence showing that patients with schizophrenia who started using cannabis early and chronically in life had better neurocognitive performance than patients who used later on, showing that this early cannabis use group may have a specific, less-impaired neurocognitive profile (Yücel et al., 2012).

As outlined, the prognosis for many schizophrenia patients is poor. Around 20% of individuals with schizophrenia may become homeless within the first year of diagnosis (Folsom et al., 2005). Within the first 5 years, 13.7% are able to achieve full remission of symptoms along with adequate social/role functioning, and within 25 years around 30% are able to achieve a favorable long-term outcome (Harrison et al., 2001; Robinson et al., 2004). Further, patients with schizophrenia often suffer from comorbid (i.e., co-occurring) conditions. For example, the rate of substance abuse among patients with schizophrenia is very high, with as many as 50% of all patients with schizophrenia and 90% in prison settings meeting lifetime DSM-IV criteria for substance abuse or dependence (Regier et al., 1990; Thoma and Daum, 2013).

Suicide is the leading cause of death among people with schizophrenia. It has been estimated that 50% of patients with schizophrenia attempt suicide and 4–5% successfully commit suicide (Donker et al., 2013). Risk factors associated with suicide in this population include previous attempts, more severe depressive symptoms, being male, having an earlier onset, suffering recent traumatic events, and recent hospitalization (Schwartz & Cohen, 2001; Donker et al., 2013). Further, the risk of suicide for individuals with schizophrenia is increased in the earlier stages of illness, and particularly within the first year of diagnosis (Donker et al., 2013).

Evidence-Based Interventions

Researchers have not yet identified any biological or psychological cures for schizophrenia. However, significant progress has been made in treatments that greatly improve the prognosis of the illness. As a result of this research progress, the quality of life for individuals with schizophrenia is dramatically better than it was at the turn of the 20th century.

The first issue to be addressed in the evaluation and treatment of schizophrenia is safety. The risk of self-harm and potential for violence must be assessed (McGirr et al., 2006; Siris, 2001). A medical examination is typically conducted to rule out other illnesses that can cause or exacerbate psychotic symptoms. This examination includes a review of the medical history, a physical examination, and laboratory tests. Many patients with schizophrenia have untreated or undertreated medical conditions such as nutritional deficiencies and infections that are a result of their psychological and/or socio-economic limitations (Goff, Heckers, & Freudenreich, 2001).

If a patient is not at acute risk to self or others, the next consideration becomes the type of treatment that would be most beneficial. There are several factors to consider. These include the person's living situation (many patients with schizophrenia are homeless), level of insight, willingness to accept treatment, past treatment history, financial resources, including health insurance, and family and other available social support. To increase chances of success, the patient with schizophrenia should be encouraged to talk openly about their treatment preferences, beliefs about medication, and concerns about side effects or changes.

The treatment of schizophrenia can be divided into three phases: the acute, stabilization, and maintenance phases (Sadock & Sadock, 2000). In the acute phase, the goal of treatment is to reduce the severity of symptoms. This phase is usually 4–8 weeks in duration. In the stabilization phase, the goal is to consolidate treatment gains. This usually takes about 6 months. Finally, during the maintenance phase, the symptoms are in remission (partial or complete). At this point, the goal of treatment is to prevent relapse and improve functioning.

Biological/Pharmacological Interventions The mainstay of the biological treatment of schizophrenia is antipsychotic medication. First developed in the 1950s, these medications had an enormous impact on the lives of people afflicted with schizophrenia. Their psychotic symptoms improved and many were able to leave psychiatric hospitals (deinstitutionalization). The first effective biological treatment for schizophrenia, chlorpromazine (Thorazine®), was the first in a line of medications now referred to as the "typical" antipsychotics or "neuroleptics." All of these medications act by blocking activity in the dopamine system. The typical antipsychotic medications are classified as high, medium, and low potency, and differ from each other in adverse-effect profiles (Table 17.2). High-potency neuroleptics tend to carry a higher risk of extrapyramidal effects (e.g., motor abnormalities), and are prescribed in low dosages. Some examples of high-potency agents are fluphenazine (Prolixin®),

trifluoperazine (Stelazine®), and haloperidol (Haldol®). Low-potency neuroleptics are prescribed in higher milligram doses and have lower risk of motor effects, but a higher risk of inducing seizures, antihistaminic effects (including sedation and weight gain), anticholinergic effects (including cognitive dulling, dry mouth, blurry vision, urinary hesitancy, and constipation), and antiadrenergic effects (including postural hypotension and sexual dysfunction). Examples of low-potency neuroleptics include chlorpromazine, and thioridazine (Mellaril®). Medium-potency agents tend to have adverse effects intermediate between the low- and high-potency drugs. Examples of these include: perphenazine (Trilafon®), and loxapine (Loxitane®).

In the 1990s, a new generation of antipsychotic medications became available for therapeutic use in Europe and North America. The new class of medication is commonly referred to as "atypical" or "second-generation" antipsychotics. Medications in this class share a lower risk of both the early occurring and the late emerging (or tardive) movement disorders, although recent evidence suggests there is some variation within the atypical antipsychotics as to their ability to cause these extrapyramidal effects (Rummel-Kulge et al., 2012). The atypical antipsychotics include risperidone (Risperdal®), olanzapine (Zyprexa®), olanzapine/fluoxetine (Symbyax®), quetiapine (Seroquel®), ziprasidone (Geodon®), aripiprazole (Abilify®), paliperidone (Invega®), asenapine (Saphris®), iloperidone (Fanapt®), lurasidone (Latuda®), and clozapine (Clozaril®). The individual medications differ significantly from one another in the neurotransmitter receptors that they occupy. Although all block dopamine neurotransmission to some extent, they vary in the extent to which they affect serotonin, glutamate and other neurotransmitters. These atypical antipsychotics have become the first line of treatment for schizophrenia. The efficacy of the atypical antipsychotics for the treatment of positive symptoms is at least equivalent to that of the typical antipsychotics. Some studies suggest that they are more effective for negative symptoms and the cognitive impairments associated with the disorder, although findings are mixed and inconclusive (Forster, Buckley, & Phelps, 1999; Kane & Correll, 2010; Sadock & Sadock, 2000). Of practical clinical significance, however, is the substantial risk of developing a "metabolic syndrome" related to the use of this class of medicines. Recent research has revealed that the atypical antipsychotics carry an elevated risk of substantial weight gain, new onset or worsening diabetes mellitus, and lipid abnormalities (Newcomer, 2005).

Antipsychotic medications are usually administered orally. For patients who are not compliant with oral medication, injectable, long-lasting (depot) antipsychotic medication may be administered (usually every 2–4 weeks). Six depot neuroleptics are commercially available in the United States. Two are first-generation or "typical" antipsychotics and four are second-generation or "atypical" antipsychotics. Benefits of depot neuroleptics include the ease of use for the patient, and the fact that compliance is easily monitored by the clinician. The risks are similar to the risks of all of the "typical" antipsychotics. The only additional risk is that of localized pain or swelling at the injection site.

Drug-induced movement disorders can be divided into acute and late onset syndromes. Acute, or early emerging motor symptoms include pseudo-Parkinsonism, bradykinesias (decreased movement), rigidity, and dystonic reactions (sudden onset of sustained intense, uncontrollable muscle contraction commonly occurring in the facial and neck muscles). Tardive dyskinesia is a late emerging syndrome that includes irregular choreiform (twisting, or worm-like) movements that usually involve the facial muscles, but can involve any voluntary muscle group. It is fortunate that the rate of tardive dyskinesia has declined since the introduction of atypical neuroleptics. It is important to note the these drug-induced movement abnormalities are a distinct and separate entity from the spontaneous movement abnormalities noted earlier, which occur as a natural correlate of schizophrenia.

Mention should also be made of the neuroleptic malignant syndrome. This is a rare, idiopathic, life-threatening

TABLE 17. 2
Selected Antipsychotic Drugs (from Sadock and Sadock, 2000 p. 1204)

Drug	Route of administration	Usual daily oral dose	Sedation	Autonomic	Extrapyramidal adverse effects
Chlorpromazine	Oral, IM	200–600	+++	+++	++
Fluphenazine	Oral, IM, depot	2–20	+	+	+++
Trifluoperazine	Oral, IM	5–30	++	+	+++
Perphenazine	Oral, IM	8–64	++	+	+++
Haloperidol	Oral, IM, depot	5–20	+	+	+++
Loxapine	Oral, IM	20–100	++	+	++
Olanzapine	Oral	7.5–25	+	++	0?
Quetiapine	Oral	150–750	++	++	0?
Risperidone	Oral	2–16	+	++	+
Clozapine	Oral	150–900	+++	+++	0?

IM = intramuscular

complication of neuroleptic medication. It is characterized by mental status changes (delirium), immobility, rigidity, tremulousness, staring, fever, sweating, and autonomic instability (labile blood pressure and tachycardia). Laboratory investigations often reveal an elevated white blood cell count (in the absence of infection), and an elevated creatine phosphokinase level. Treatment involves discontinuation of neuroleptic medication, supportive medical treatment, a peripheral muscle relaxant, and bromocriptine (a D2 receptor agonist; Rosebush & Mazurek, 2001).

Psychosocial Treatments of Schizophrenia Although antipsychotic medication is the crucial first step in the treatment of schizophrenia, there is substantial evidence that psychosocial interventions can also be beneficial for both the patient and the family. It is unfortunate that such treatments are not always available because of limited mental health resources. Nonetheless, it is generally agreed that the optimal treatment approach is one that integrates pharmacologic and psychosocial interventions.

Research supports the use of family therapy, which includes psychoeducational and behavioral components, in treatment programs for schizophrenia (Bustillo, Lauriello, Horan, & Keith, 2001). Family therapy has been shown to reduce the risk of relapse, reduce family burden, and improve family members' knowledge of and coping with schizophrenia. Briefly, treatment includes psychoeducation about symptoms, diagnosis, and prognosis, along with therapeutic modules focusing on communication and problem solving skills.

Comprehensive programs for supporting the patient's transition back into the community have been effective in enhancing recovery and reducing relapse. One such program, called assertive community treatment, was originally developed in the 1970s by researchers in Madison, Wisconsin (Udechuku et al., 2005; Bustillo et al., 2001; Saddock & Sadock, 2000). Acceptance and commitment therapy (ACT) is a comprehensive treatment approach for the seriously mentally ill living in the community. Patients are assigned to a multidisciplinary team (nurse, case manager, general physician, and psychiatrist) that has a fixed caseload and a high staff/patient ratio (1:12). The team delivers all services to the patient when and where he or she needs it, and is available to the patient at all times. Services include home delivery of medication, monitoring of physical and mental health status, *in vivo* social skills training and frequent contact with family members. Studies suggest that assertive community treatment can reduce time spent in hospital, improve housing stability, and increase patient and family satisfaction. Research shows that such treatment can aid in the stable living in a community, but has little impact in other areas such as social functioning and employment (Mueser, Deavers, Penn, & Cassisi, 2013).

Social skills training seeks to improve the overall functioning of patients by teaching the skills necessary to improve performance of activities of daily living, employment related skills, and interaction with others. Research indicates that social skills training can improve social competence in the laboratory and in the clinic, along with impacting social/daily living skills, community functioning and negative symptoms (Bustillo et al., 2001; Kurtz & Mueser, 2008; Penn & Mueser, 1996). There is also some evidence to suggest that combining social skills training with attention training can enhance outcomes (Silverstein et al., 2008).

The rate of competitive employment for the severely mentally ill has been estimated at less than 20% (Lehman, 1995; Marwaha & Johnson, 2004); thus, vocational rehabilitation has been a major focus of many treatment programs. Some evidence suggests that "supported employment programs" produce better results than traditional vocational rehabilitation programs as measured by patients' ability to obtain competitive, independent employment and increased wages earned (Bond, Drake & Becker, 2012; Mueser et al., 2013).

Cognitive-behavior therapy for schizophrenia draws on the tenets of cognitive therapy that were originally developed by Beck and Ellis (Beck, 1976; Ellis, 1986). The theory is that normal psychological processes can help maintain or reduce specific psychotic symptoms. Cognitive-behavioral therapy (CBT) for psychosis challenges the notion of a discontinuity between psychotic and normal thinking. The normal cognitive mechanisms that are already being used in the non-psychotic aspects of the patient's thinking can be used to help the psychotic individuals deal directly with their symptoms (Kingdon & Turkington, 2005). The choice of target symptoms is based on the patient's preference and/or severity of the problems created by the psychotic symptom in question. Psychotic beliefs are never directly confronted, although specific psychotic symptoms such as hallucinations, delusions, and related problems are targeted for intervention by means of education and cognitive restructuring skills around the symptoms, their onset, along with providing insight into how the behavioral framework of antecedents, beliefs, and consequences functions in psychosis (ABC model; Dickerson, 2000). There have been somewhere near 40 randomized controlled trials evaluating the efficacy of CBT for psychosis. A recent review (Mueser et al., 2013) noted that CBT was linked to decreases in psychotic symptoms, negative symptoms, and mood problems, as well as better social functioning. However, findings comparing CBT with other active treatments are mixed and currently somewhat inconclusive, as most studies of the efficacy of CBT where compared to treatment as usual (Mueser et al., 2013). Further, one meta-analysis suggests that CBT only has a small therapeutic effect for psychosis, which is made even smaller when acknowledging biases in research methodology (Jauhar et al., 2014).

Within this cognitive-behavioral framework, another therapeutic modality, ACT, has shown some promise in treating psychosis. ACT works within the context of CBT and emphasizes increased awareness and openness, psychological flexibility, living in-line with one's values,

and finding actions that are workable. The original studies found that receiving ACT was associated with lower rates of hospitalization and decreases in psychotic symptoms (Bach & Hayes, 2002; Gaudiano & Herbert, 2006). More recent findings indicate long-term decreases in hospitalization following a trial of ACT, although findings using this approach are still new, limited, and warrant future attention (Bach, Hayes, & Gallop, 2011).

Biological/Pharmacological and Psychosocial Treatment for Prodromal Populations Several reports indicate that antipsychotics may be effective in reducing the progression of prodromal syndromes into psychotic disorders. A review of several recently completed randomized clinical trials with antipsychotic medication reports that following the end of treatment, antipsychotic medication as a pre-psychotic intervention may delay the onset of psychosis or ameliorate pre-psychotic symptoms (de Koning et al., 2009). However, most studies find no significant differences at the end of follow up 1–4 years later. Although the onset may be delayed, there is no indication that psychosis is prevented.

It is also important to acknowledge the potential risks of antipsychotic medications when used as a preventative intervention (Corcoran et al., 2010). The adverse effects of second-generation antipsychotics include extrapyramidal symptoms, weight gain, and metabolic complications. With no proven long-term effects, the benefit/risk ratio of using antipsychotics as a pre-psychotic intervention must be weighed carefully. We also do not yet know the effects of these medications on prodromal adolescents. As our knowledge of the effects of psychotropic medication on adolescent growth and development increases, we will be in a better position to weigh any adverse effects against potential benefits, both short term and long term (Jensen et al., 1999).

There are considerably fewer randomized controlled trials evaluating psychosocial treatments for the prodromal population compared with patients with schizophrenia. The limited research base has primarily evaluated CBT, supportive therapy, integrative therapy, and cognitive remediation. The findings from these treatment studies suggest that while short-term benefits may be found, lasting effects past 12 months were not apparent in most studies. The exception was from one study evaluating integrated therapy (combining CBT, skills training, cognitive remediation, and family education), where benefits lasted up to 24 months, and another study where trending effects were noted at 18 months for CBT (Addington et al., 2011; Bechdolf et al., 2012; Morrison et al., 2007; van der Gaag et al., 2012). There is also a recent emphasis on highlighting the family as a means of intervention in the prodromal phase. Research is currently underway to examine whether family focused treatment (Miklowitz, George, Richards, Simoneau, & Suddath, 2003) may help to delay or prevent the onset of psychosis (Schlosser et al., 2012).

Cognitive Remediation and Other Promising New Interventions Additionally, cognitive remediation therapy has also received attention with over 40 randomized controlled trials published evaluating its use in treating psychosis. Cognitive remediation generally takes the form of computerized tasks aimed at enhancing specific cognitive skills such as attention, working memory, or planning. The origins of cognitive remediation for schizophrenia come from research showing the brain to be plastic (i.e., able to be changed by behavior and experience). Results find that cognitive remediation significantly improves cognition, with mixed findings on whether functioning is affected (Mueser et al., 2013; Wykes et al., 2005). There is some evidence to suggest that cognitive remediation may allow for the acquisition of new skills and improved functioning by combining it with other empirically supported treatments (Mueser et al. 2013). There has been a focus on usage of omega-3 fatty acids to prevent transition to psychosis, following a randomized controlled trial showing significantly less conversion to psychosis in individuals who received omega-3 treatment (Amminger et al., 2010). Finally, exercise is also being investigated as an intervention in prodromal populations and individuals diagnosed with schizophrenia, owing to research linking aerobic activity with the reversal of brain abnormalities commonly seen in schizophrenia (i.e., increases in hippocampal gray matter following exercise; Mittal, Gupta et al., 2013; Pajonk et al., 2010).

Summary

This chapter has reviewed a broad range of scientific research on the nature and origins of schizophrenia. Spanning over a century, the efforts of investigators have yielded, piece by piece, a clearer view of the illness. The puzzle is not solved, but we can certainly claim progress toward a solution.

In summary, although we have not found all the pieces of the puzzle, we have made significant progress in moving toward a comprehensive account of the etiology of schizophrenia. Among the mental disorders, schizophrenia remains a clear illustration of the complex interactions taking place between the individual and the environment. In the coming years, we can expect research to yield important information about the precise nature of the brain vulnerabilities associated with schizophrenia, and the mechanisms involved in the interaction of congenital vulnerability with subsequent life stress and neuromaturation. Genetic data and research into gene expression will provide insight into etiology, as well as further our understanding of the role neurotransmitter abnormalities in schizophrenia. Longitudinal studies conducted during the prodromal period hold strong promise of elucidating the complicated interactions between development (e.g., hormones, neural maturation), and latent constitutional vulnerabilities. Furthermore, research during this period holds strong potential to inform the next

generation of psychosocial and pharmacological preventive interventions.

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18

Mental Health and Aging

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The largest cohort in U.S. history is beginning to enter late life, driving an unprecedented growth in the older adult population. The leading edge of the "baby boom" generation (born between 1946 and 1964) reached the age of 65 in 2011. By the year 2030, nearly 20% of the U.S. population will be 65 or older, a sharp rise from the 13% recorded in 2010 (Howden & Meyer, 2011). Alongside this dramatic growth in the older adult population is projected an increase in the number of older adults with mental health problems (Hinrichsen, 2010), resulting from both larger numbers of older adults in the population and an elevated propensity for psychopathology in the baby boom cohort (e.g., Hasin, Goodwin, Stinson & Grant, 2005). Furthermore, the coming generation of older adults is expected to seek mental health treatment at higher rates than past generations (Qualls, Segal, Norman, Niederehe, & Gallagher-Thompson, 2002). These changes bring into sharp focus the importance of investigating the presentation, etiology and treatment of psychopathology in late life.

Psychopathology in late life can best be understood in the context of lifespan development. Developmental psychopathology has been a useful paradigm for studying abnormal behavior situated within a developmental context (Sroufe & Rutter, 1984), but the work within this field has generally focused on early development (Cicchetti & Toth, 2009). In contrast, a large body of literature within the lifespan developmental psychology tradition offers theoretical and empirical explanations for normal development across the entire lifespan and into old age (e.g., Baltes, P. B., & Baltes, M. M., 1990; Carstensen, Isaacowitz, & Charles, 1999), but less often focuses on psychopathology. In this chapter, we adopt the developmental psychopathology perspective to examine abnormal behavior in late life as a function of development across the lifespan (see also Zeman & Suveg, Chapter 2 in this volume).

Several aspects of the developmental psychopathology perspective have been particularly helpful in organizing our thinking within this chapter. Consistent with this perspective, in which psychopathology is viewed as deviation from normal development (Sroufe & Rutter, 1984; see also Chapter 2 in this volume), we begin with an overview of normal cognitive and socioemotional development in late life. In addition, within the developmental psychopathology perspective, emphasis is placed on understanding not only the presence of psychopathology at any point in time, but also the trajectory over time and even the absence of psychopathology in a person at risk. In this chapter, we differentiate, where possible, between individuals with early onset of disorder that persists into older adulthood and individuals who experience disorder for the first time in late life, referred to as "late onset." It should be noted, however, that age of onset is not specified in much of the research on late-life psychopathology. There has been relatively little research examining the absence of psychopathology in late life among individuals at risk, including those with early-onset disorder who experienced remission. Future research on resilience would make an important contribution to our understanding of lifespan developmental psychopathology. The developmental psychopathology approach also highlights the possibility that psychopathology may manifest itself differently over time, a concept that has been termed "heterotypic continuity" (Cicchetti & Toth, 2009). This issue is especially relevant to the study of late life

psychopathology because diagnostic rubrics have been based primarily on symptom presentation typically seen earlier in the lifespan. As a result, some forms of psychopathology may be underdetected among older adults. We discuss this issue further in relation to several of the disorders presented below. A final issue to consider is research methodology; both developmental psychopathology and lifespan developmental psychology traditions emphasize the need for longitudinal research before drawing conclusions about age changes. We base our conclusions on longitudinal research wherever possible.

We begin this chapter with a brief overview of biological, cognitive, social and emotional development in late life. We then examine some of the most prominent forms of psychopathology with respect to epidemiology, presentation, etiology and treatment. Where the disorder appears to increase or decrease in prevalence compared with earlier points in the lifespan, we evaluate possible explanations for these age-related changes. The disorders we cover include anxiety disorders, mood disorders and suicide, schizophrenia, substance use disorders, personality disorders, sleep disorders and dementia. For disorders not included in the current chapter, the interested reader is referred to Segal, Qualls and Smyer (2011).

Normal Development in Late Life

Normal development in late life is characterized by both stability and change in biological, cognitive, social and emotional domains. Biological aging involves changes in the central nervous system and most other organ systems, as well as a concomitant increase in the likelihood of physical illness and disability. Despite these age-related changes, however, most older adults remain independent. Only 4.5% of adults age 65 and older live in skilled nursing facilities (Zarit, S. H., & Zarit, J. M., 2007). Age-related slowing of the central nervous system and other neurological changes are associated with normative declines in cognitive abilities characterized as fluid intelligence. In contrast, crystallized intelligence, which involves fund of knowledge, remains stable or even improves into late life. Social changes with age include the possibility of bereavement, retirement, caregiving and other forms of role change. Nevertheless, 75% of older men (65 and older) and 43% of older women are married.

An influential meta-theory that purports to explain adaptation to these age-related changes is the selective optimization and compensation model (Baltes, P. B., & Baltes, M. M., 1990). According to this model, aging is associated with a narrowing of opportunities, referred to as *selection*. Selection may result from losses, as described above, or may be elective, as when a person chooses to focus on a particular occupation or other objective. *Optimization* is the process of enhancing existing abilities, such as practicing a skill. *Compensation* entails use of alternative methods of meeting a goal, such as seeking help or using an assistive device. Individuals age successfully, according to the selective optimization and compensation model, to the extent that they select goals wisely, optimize their preserved abilities and compensate for their losses. The model provides a framework for understanding some forms of psychopathology in late life.

Another useful theory for understanding normal development in late life is the socioemotional selectivity theory (Carstensen et al., 1999). According to this theory, social goals change across the lifespan. In youth (or whenever time is perceived as expansive), individuals pursue more information-oriented social goals, such as learning about social norms or evaluating oneself in relation to others. In contrast, in late life (or whenever the future is perceived to be foreshortened), the focus is on emotion-oriented social goals, such as regulating one's emotional state through contact with others. Consistent with predictions of this theory, older adults typically have smaller social networks than younger adults, but are more likely to derive emotional satisfaction from them (Carstensen et al., 1999). Older adults also exhibit improved emotion regulation compared with their younger counterparts (Carstensen et al., 1999; Charles, Reynolds, & Gatz, 2001). These aspects of the theory are particularly relevant for the understanding of psychopathology in late life. Against this backdrop of normal aging, we next consider major types of disorders in late life.

Anxiety

Anxiety disorders are relatively common in late life (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). They are associated with significant negative consequences on the individual and societal level, including increased rates of physical disability and reduced levels of wellbeing (e.g., Wolitzky-Taylor, Castriotta, Lenze, Stanley, & Craske, 2010). Drawing from data collected in the National Comorbidity Survey Replication, Kessler and colleagues (2012) estimated a lifetime prevalence of any anxiety disorder at 14.9% among adults aged 65 and older. Specific and social phobias rank among the most common anxiety disorders in late life, with a lifetime prevalence of 6.8% and 6.3%, respectively. Generalized anxiety disorder is also relatively common, with a lifetime prevalence of 3.3% (Kessler et al., 2012). Other anxiety disorders, including panic disorder (2.1%), separation anxiety disorder (1.6%), and agoraphobia (1.2%) have a lower lifetime prevalence in later life (Kessler et al., 2012). Still, researchers suggest that these numbers likely underestimate the prevalence of anxiety disorders in late life (Mohlman et al., 2012). As the population ages, the importance of identifying and treating anxiety disorders among older adults becomes more salient.

Changes were made in the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5) to diagnostic criteria for certain anxiety disorders to improve identification and diagnosis of anxiety disorders in older adults. One notable difference is the removal of the

requirement of awareness of excessive anxiety for the diagnoses of specific phobia, social phobia, and agoraphobia. Because older adults are less likely to describe anxiety as excessive (Mohlman et al., 2012), this change may improve detection of anxiety disorders in older adults. DSM-5 includes a review of age-related differences in presentation and special considerations for diagnosing anxiety disorders in older adults.

Description and Course To adequately capture anxiety disorders in later life, it is important to recognize differences in symptom presentation. Older adults generally report less frequent negative emotions in comparison to younger adults (Kryla-Lighthall & Mather, 2008), and they experience less physiological arousal in the context of anxiety inducing stressors (e.g., Lau, Edelstein, & Larkin, 2001). According to Wolitzky-Taylor and colleagues (2010), older adults also may be more apt to use exact language to describe emotional states (e.g., "concerned," but not "worried"), and endorse anxiety targets that are different from those of than younger adults. Further, researchers note that clinically relevant, but subthreshold (not meeting DSM-5 diagnostic criteria), levels of anxiety may be common among older adults (Bryant, Jackson, & Ames, 2008).

Researchers have also identified age differences in presentation of specific anxiety disorders. For example, phobias and panic attacks commonly co-occur with physical illnesses (El-Gabalawy, Mackenzie, Shooshtari, & Sareen, 2011), and older adults may be more likely than young adults to attribute their symptoms to a health problem or situational factors (Mohlman et al., 2012). Similarly, changes in physical health and role adaptation (e.g., retirement, widowhood) may complicate diagnosis of social anxiety disorder in older adults by making it difficult to differentiate developmentally appropriate activity changes from behavioral avoidance (Mohlman et al., 2012). Researchers also have noted age-related differences in generalized anxiety disorder (GAD) with regard to worry content. Older adults with GAD are more likely to worry about health than are younger adults with GAD (Diefenbach, Stanley, & Beck, 2001). While some worries are common among many older adults (e.g., concern about family), worries about health, finances, trivial issues, and social matters differentiate those with a diagnosis of GAD from those without a GAD diagnosis (Wetherell et al., 2003).

Research suggests that anxiety disorders often begin in childhood or early adulthood (Wolitzky-Taylor et. al, 2010). Although most studies have found that relatively few people have a first onset episode of an anxiety disorder in late life (e.g., Kessler et al., 2012), others have found that late-life new onset anxiety may not be uncommon for certain disorders, like GAD (e.g., Le Roux, Gatz, & Wetherell, 2005). While some anxiety disorders, such as GAD, are thought to be chronic (Roemer, Orsillo, & Barlow, 2002), others, like separation anxiety disorder and selective mutism, often resolve in childhood (Kessler et al., 2012).

Etiology The etiology of anxiety varies by anxiety disorder. Certain anxiety disorders, like specific phobias, are clearly rooted in conditioning (Barlow, 2002). Other variables, including genetic factors, parenting style, predictability or control over life events, and attributional style, increase vulnerability to other anxiety disorders (Barlow, 2002). Researchers identified biological (e.g., greater number of chronic health conditions, hypertension), psychological (e.g., external locus of control, neuroticism), and social (e.g., quality of social support) factors associated with increased risk of anxiety in later life (Vink, Aartsen, & Schoevers, 2008). Zarit, S. H., and Zarit, J. M. (2007) outline the role of other important factors, like neurotransmitter activity, physiological reactivity, cognitive distortion and behavioral avoidance, in anxiety disorders in older adults.

Explanation for Age Differences Frequent comorbid medical and psychiatric conditions, age-related differences in symptom presentation, higher rates of cognitive impairment, and the relative paucity of developmentally appropriate assessment instruments make the diagnosis of anxiety disorders in later life challenging (Wolitzky-Taylor et al., 2010). Developmental changes in social, familial, and occupational roles also may impact the manifestation of anxiety symptoms. Moreover, providers may mistakenly attribute anxiety symptoms and associated avoidance behavior to normal aging (Wolitzky-Taylor et al., 2010). These barriers may make the accurate identification and diagnosis of anxiety disorders difficult and may result in underreporting and misdiagnosis of anxiety in older adults (Brenes et al., 2005; Palmer, Jeste, & Sheikh, 1997).

Research also shows that older adults may actually experience less anxiety later in life. Some attribute the declines in negative affect to improvements in emotion regulation skills, as proposed by socioemotional selectivity (Carstensen et al., 1999), making them more adept at coping with stressors.

Assessment and Treatment Because of the diagnostic challenges discussed above, assessment of anxiety in late life can be challenging. Several measures, including the Geriatric Anxiety Inventory (Pachana et al., 2007) and the Geriatric Anxiety Scale (Segal, June, Payne, Coolidge, & Yochim, 2010) have been developed specifically for identifying and quantifying anxiety symptoms in older adults. Still, there is a need for more developmentally appropriate measures to assess for a wider range of anxiety disorders, across different populations of older adults (e.g., those with cognitive impairment).

Relatively few older adults seek treatment for anxiety disorders from mental health professionals (de Beurs et al., 1999). Instead, many present for treatment in primary care. Despite significant potential negative consequences for older adults, benzodiazepines are the most commonly prescribed treatment (Sheikh & Cassidy, 2000). Studies have shown that antidepressant medications are effective for treating certain anxiety disorders, like GAD (e.g., Lenze et al., 2009). In addition to pharmacotherapeutic interventions, a number of studies have found psychosocial interventions, particularly cognitive behavioral based interventions, to be effective in the treatment of late-life anxiety disorders (Thorp et al., 2009).

Conclusions Age-related changes in health, symptom presentation, and comorbid conditions complicate the picture of anxiety in older adults. DSM-5 attempts to address some of the challenges associated with identifying and treating anxiety in late life, though more research is warranted, in light of medical multimorbidity.

Mood Disorders

Depression is one of the most common disorders of late life and one of the most consequential. It is a leading cause of disability (Institute of Medicine, 2012) and is associated with an elevated risk of early mortality (Gallo et al., 2013). As such, it is a major public health problem.

Mood disorders listed in DSM-5 include major depressive disorder, persistent depressive disorder, and bipolar disorder (see also Alloy et al., Chapter 11 in this volume). Major depressive disorder is the primary focus of this section. Persistent depressive disorder is a new diagnostic category in DSM-5 that incorporates recurrent major depression and dysthymia. The relevance of this disorder in late life and its prevalence in older adults is unknown.

Bipolar disorder is relatively rare in older adults, with a prevalence of 0.5–1% (Sajatovic & Chen, 2011), for reasons that are not well understood. There is evidence of midlife remission in a substantial proportion of individuals (Cicero, Epler and Scher, 2009), and selective mortality from suicide or other causes has been well documented (Angst, F., Stassen, Clayton, & Angst, J., 2002; Jamison, 2000). On the other hand, the emergence of mania in late life may be more common than previously thought (Dols et al., 2014). Bipolar disorder is not discussed in detail in this chapter owing to space constraints; the interested reader is referred to Sajatovic and Chen (2011).

Depressive symptoms that do not meet criteria for a mood disorder, often described as "sub-syndromal," "subthreshold," or minor depression, are especially frequent in late life and are associated with substantial adverse outcomes in older adults (Meeks, Vahia, Lavretsky, Kulkarni, & Jeste, 2011). As such, they are also a focus of this section.

Depressive disorders are less common in late life than earlier in the lifespan, but clinically significant depressive symptoms are more common (Lee, Hasche, Choi, Proctor, & Morrow-Howell, 2013; Sutin et al., 2013). The 1-year prevalence of major depression among older adults is approximately 3% (Chou & Cheung, 2013). In contrast, across 12 studies reporting point prevalence of subthreshold depression in the community, the median was 9.8% (Meeks et al., 2011). Both depressive disorders and clinically significant depressive symptoms are found at the lowest rates in community settings and increase in prevalence in primary care, medical inpatient and long-term care settings (reviewed in Fiske, Wetherell, & Gatz, 2009).

Description and Course Major depressive disorder is characterized by pervasive dysphoria or anhedonia for at least 2 weeks, accompanied by additional symptoms, totaling at least five, from the following list: Sleep disturbance; eating disturbance; psychomotor agitation or retardation; difficulty concentrating or indecisiveness; low energy; excessive guilt; thoughts of death or suicide. The diagnosis also requires significant impairment in social or occupational functioning, and excludes symptoms attributable to a drug or other disorder.

Diagnostic criteria for major depressive disorder have changed from DSM-IV to DSM-5 in a way that may be particularly relevant to older adults: depression following bereavement is no longer excluded from the diagnosis. Zisook and colleagues (2012) concluded, based on a review of recent research (not limited to older adults), that bereavement-related depression does not differ from non-bereavement-related depression on antecedents (genetic influence, personal and family history of depression, personality characteristics), concurrent factors (comorbidity), or predictive factors (course, treatment response). The majority of bereaved older adults would not meet criteria for a major depressive episode, but some increase in prevalence of depressive disorders among older adults may be seen.

Depression is a chronic, episodic disorder. Depressed adults 65 or older in the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) reported a mean of 4.4 lifetime episodes with a mean duration of 3 years (Chou & Cheung, 2013). Existing evidence suggests that at least half of older adults with depression experienced the first episode at age 60 or later (Brodaty et al., 2001; Bruce et al., 2002). Among adults aged 65 and older in the NESARC sample, the mean age at depression onset was 51 (Chou & Cheung, 2013). Late-onset depression is characterized by vascular risk factors, neuroimaging evidence of lesions in the deep white matter of the brain, known as "deep white matter hyperintensities," and executive functioning deficits, as well as the poor health and other life stressors, whereas early-onset depression is associated with neuroticism (Oldehinkel, Ormel, Brilman, & Van den Berg, 2003; Sneed, Kasen, & Cohen, 2007). These findings suggest differential etiology for late versus early-onset depression.

Etiology The onset and maintenance of depression in older adults can be conceptualized in terms of a complex interplay between biological, psychological or social vulnerabilities that may be longstanding or may change with age, and stressors that arise in late life (Fiske et al., 2009). Fiske and colleagues proposed that a reduction in meaningful and rewarding activities following age-related stressors may be a common pathway to depression among vulnerable older adults, and that the depressive state may be exacerbated by self-critical cognitions (Fiske et al.). Biological vulnerabilities include genetic influence (Kendler, Gatz, Gardner, & Pedersen, 2006) as well as physical illness and disability (Cui, Lyness, Tang, Tu, & Conwell, 2008; Fauth, Gerstorf, Ram, & Malmberg, 2012) and cognitive impairment (Cui et al., 2008). Psychological diatheses include neuroticism (Oddone, Hybels, McQuoide, & Steffans, 2011), rumination (Andrew & Dulin, 2007) and avoidance (Garnefski & Kraaij, 2006). Social factors include stressful life events and role changes, such as caregiving (Bookwala, 2014) and widowhood (Sikorski et al., 2014). In contrast, perceived social support is protective (e.g., Fauth et al., 2012).

Explanation for Age Differences Age differences in depression may result from changes in frequency or impact of risk and protective factors over time. Although biological factors that confer risk for depression appear to increase with age, psychological and social factors that are protective in nature also appear to increase with age, including improved emotion regulation (Carstensen et al., 1999; Charles et al., 2001). The apparent reduction in the prevalence of depression in late life may also be due to diagnostic rubrics that do not fully capture depression as manifested in old age. Older adults are less likely than younger or middle-aged adults to endorse dysphoria or anhedonia (Gallo, Anthony, & Muthén, 1994), one of which is required to diagnose major depressive disorder.

It is not clear why older adults report elevated levels of depressive symptoms, and are at greater risk of sub-syndromal depression, compared with younger or middle-aged adults, but it is not likely to be an artifact of symptom reporting. Older adults are more likely than their younger counterparts to endorse somatic symptoms of depression, such as low energy, sleep disturbance and appetite disturbance, but these symptoms cannot be fully explained by comorbid physical illness (Nguyen & Zonderman, 2006).

Assessment and Treatment As noted earlier, depression is underdetected in late life. In one study, physicians failed to detect significant depressive symptoms in older adults unless the person specifically endorsed feeling depressed (Gregg, Fiske, & Gatz, 2013). Depression is also more likely to be untreated in older adults compared with their younger counterparts (Burnett-Zeigler et al., 2012), although rates of diagnosis and treatment of depression among older adults has been increasing in recent years (Akincigil et al., 2011).

Several types of interventions are efficacious for treating depression in older adults, including some that have been empirically supported in younger and mixed age samples. These include cognitive behavioral therapy, cognitive bibliotherapy, problem-solving therapy, brief psychodynamic therapy, and reminiscence therapy (Shah, Scogin, & Floyd, 2012). Evidence supports the use of innovative methods of delivering psychotherapy to depressed older adults, including telehealth (e.g., Lichstein et al., 2013). Internet-delivered cognitive behavioral therapy for depression is equally acceptable and effective in older adults as in younger and middle-aged individuals (Mewton, Sachdev, & Andrews, 2013). A growing evidence base also supports the efficacy of psychotherapy to prevent the onset of depression among older adults with sub-threshold symptoms (Lee et al., 2012).

Conclusions Depression is a relatively common disorder of late life that has potentially serious consequences. Behavioral interventions have demonstrated efficacy in treating depression in older adults. Unfortunately, depression is often undetected and untreated in this age group. Additional research is needed to examine symptom presentation in late-life depression, and to evaluate risk and protective factors that may lead to preventive interventions.

Suicide

Any discussion of pathology and mood disorders in older adults must include a discussion of suicide in late life. National statistics in the United States, as well as for several countries around the globe, indicate that older adults, particularly older men (McIntosh & Drapeau, 2014), have elevated rates of death by suicide. In 2013, the overall national suicide rate was 13 of 100,000 for all ages. However, the rate in adults 65 years and older was 16.1, and this rate increased by age, with a rate of 18.6 for those age 85 and older (Drapeau & McIntosh, 2015). Across age groups, White males have the highest risk of suicide (Drapeau & McIntosh, 2015). Another concerning facet of this phenomenon in older adults is that suicidal acts and attempts in late life are exponentially more likely to be fatal than suicidal acts in younger adults (Friedmann & Kohn, 2008). These high rates point to a need for any review of psychopathology in older adults to include a review of suicide and its relation to aging.

Definitions Suicidal behavior is often categorized into suicidal ideation, suicide attempts, and suicide (for clarity in this chapter, this is also called “death by suicide”). Suicidal ideation refers to an individual’s thoughts of engaging in suicidal behavior, which may be associated with any level of intent. A suicide attempt is a

"self-inflicted, potentially injurious behavior with nonfatal outcome for which there is evidence . . . of intent to die" (Silverman, Berman, Sanddal, O'Carroll, & Joiner, 2007, p. 273). A suicide attempt that results in death is referred to as a death by suicide. An important area of consideration for older adults is the investigation of what factors increase the likelihood that suicidal ideation will lead to an attempt or death by suicide.

Etiology A variety of theories have been proposed to explain suicidal behavior. Some of these take into account lifespan developmental factors, and therefore may specifically address suicide in late life as well. For the purpose of this chapter, we highlight some of the risk factors for suicide as they relate to one of the more recent, prominent theories in suicide research.

The interpersonal theory of suicide (Joiner, 2005) explains suicide as the result of an interaction between two broad factors—an individual's desire to die and an individual's acquired capability for suicide. The desire to die does not necessarily lead to suicidal behaviors, attempts, or death. What Joiner proposes sets individuals who perform suicidal behaviors or die by suicide apart from others who have the desire for death is an acquired capability for suicide, which refers to a habituation to the more repellent aspects of suicide such as pain or fear, which results from previous exposure to such provocative experiences.

Factors that have been suggested by research to be related to suicidal ideation, or the desire for death, in older adults include depression, difficulties in social relationships or poor social support, dysfunctional coping strategies, childhood abuse, perception of oneself as a burden on others, personality characteristics such as high neuroticism and low extraversion, and poor self-rated physical health (Almeida, Draper et al., 2012; Cukrowicz, Cheavens, Van Orden, Ragain, & Cook, 2011; Marty, Segal, & Coolidge, 2010; Segal, Marty, Meyer, & Coolidge, 2012). Those that have been found to be related to suicide attempts or death by suicide include past suicide attempts, living alone, and neurocognitive deficits in specific types of reasoning (De Leo, Draper, Snowdon, & Kolves, 2013; McGirr, Dombrovski, Butters, Clark, & Szanto, 2012). When considering these risk factors in older adults, it is notable that some are lifelong factors (e.g., personality characteristics listed above), some are factors that occur earlier in life (such as childhood abuse), some are likely to exist throughout life but may increase in intensity as one ages (for example, dysfunctional coping strategies), and others may be specific to older age (such as neurocognitive impairments). Therefore, it is important to take a lifespan developmental approach to determining risk for suicide in older adults, taking into consideration a broad range of factors. Additionally, the factors that are associated with suicidal ideation theoretically fall under the category of being related to a desire for death, while those associated with suicide attempts or death are those that may influence the capability for suicide (such as past attempts).

Assessment and Prevention There have been efforts to implement interventions to reduce the occurrence of suicide. Older adults are less likely than younger adults to contact mental health care providers within 1 year before dying by suicide, but are much more likely than younger adults to be in contact with primary health care professionals within 1 month before dying by suicide (Luoma, Martin, & Pearson, 2002), and therefore a particularly important area for intervention could be in primary care settings. However, primary care physicians may not be aware of the most effective assessment and intervention techniques to use when confronted with a patient who expresses suicidal ideation (Vannoy, Tai-Seale, Duberstein, Eaton, & Cook, 2011).

Various instruments have been developed to measure suicide ideation or risk in older adults. These include the Geriatric Suicide Ideation Scale (Heisel & Flett, 2006), the Reasons for Living–Older Adults Scale (Edelstein et al., 2009), and the Suicidal Older Adult Protocol (Fremouw, McCoy, Tyner, & Musick, 2009). Most interventions that have been researched in regard to reducing suicidal ideation or suicide target depression. Treatments and interventions that have evidence supporting an effect on suicidal ideation in older adults include elements such as physician education about geriatric depression and suicide, practitioners (such as social workers, nurses, and psychologists) who manage depression care in collaboration with physicians, and cognitive-behavior therapy (Almeida, Pirkis et al., 2012; Bhar & Brown, 2012; Bruce et al., 2004). Another type of program being tested in older adults uses social connectedness and peer companions to intervene early in the development of suicide risk (Van Orden et al., 2013). Other interventions may target the risk factors discussed above in this section, but there is little research on the effectiveness of interventions on suicide risk specifically for older adults (Lapierre et al., 2011).

Conclusions Although older adults make up the age group with the highest suicide rates, relatively little is known about risk factors and interventions to prevent death by suicide in this age group. Continued research and attention in this area is crucial. In addition, it is important to distinguish between suicidal ideation, attempts, and death by suicide, in order to understand factors that may lead to increased desire to die, as well as the factors that lead specifically to an increased capability for suicide and therefore more lethal attempts or death. Another critical consideration in this area is how lifespan developmental factors, including those that present earlier in life, affect suicidal thoughts and behavior. Finally, more needs to be done to test interventions for their effectiveness in preventing suicide or reducing suicidal ideation, including

particularly those that may be implemented by psychologists in primary care settings.

Schizophrenia

Schizophrenia is a lifespan neurodevelopmental disorder. The prevalence is 1.3% in the population aged 18 to 54 and 0.6% among individuals aged 65 and older (Jeste & Nasrallah, 2003). Schizophrenia with onset in late life has been the focus of recent research and is discussed specifically below.

Description and Course Diagnostic criteria (DSM-5) specify at least two of the following symptoms: delusions, hallucinations, disorganized speech, grossly disorganized or catatonic behavior, or negative symptoms (i.e., diminished emotional expression or avolition). New in DSM-5 is the requirement that at least one of the symptoms must be delusions, hallucinations, or disorganized speech.

Several different trajectories characterize the course of schizophrenia across the lifespan. Most commonly, schizophrenia has an onset in late adolescence or early adulthood, but in a substantial proportion of older adults with schizophrenia, onset occurred later in life (36% reported in Meesters et al., 2012). Regardless of the age at which the disease is diagnosed, evidence suggests that the disease process begins earlier, as preclinical signs such as cognitive, motor and social deficits are observed in children, adolescents and adults who later develop schizophrenia (e.g., Walker & Lewine, 1990). For 25–35% of patients, the course of schizophrenia is chronic with continuing symptoms, whereas for more than 50% of patients the course is episodic (Jobe & Harrow, 2010). A subgroup of 20–35% of patients functions well after discontinuing antipsychotic medication (Jobe & Harrow, 2010).

Etiology Leading theories attribute the etiology of schizophrenia to genetic or other biological vulnerability in combination with pre- and perinatal environmental stressors that interfere with normal neurological development (Bearden, Meyer, Loewy, Niendam, & Cannon, 2006). Evidence shows substantial genetic influence (Gottesman & Hanson, 2005). Environmental stressors that have been implicated include birth complications, such as those involving fetal hypoxia (Bearden et al., 2006) and maternal viral infection during the second trimester (e.g., Barr, Mednick, & Munk-Jörgensen, 1990).

Explanation for Age Differences There are several possible explanations for the lower prevalence of schizophrenia in late life compared with early adulthood or middle age, which is particularly notable given the frequency of late-onset cases. Lower rates of schizophrenia in late life may result, in part, from remission of the disease. Although there are varying estimates of the rate of remission, longitudinal research demonstrates convincingly that remission is possible, even among older adults (Barak & Swartz, 2012). Furthermore, the pattern of symptoms of schizophrenia appears to be characterized by stability or a decline in severity across the lifespan (Iglewicz, Meeks, & Jeste, 2011). Older individuals with schizophrenia appear to have better coping skills than their younger counterparts (Iglewicz et al., 2011). Wellbeing and functioning among older adults with early-onset schizophrenia has been shown to be stable or even reflect an increased level at older ages (Iglewicz et al., 2011). Similarly, in longitudinal research, cognitive performance has been shown to remain stable over time in older-adult outpatients with schizophrenia (Savla et al., 2006). Compared with age-matched normal controls, however, older adults with schizophrenia show greater symptomatology, poorer functioning and lower quality of life (Jobe & Harrow, 2010). Notably, stability in symptoms and functioning is only found among non-institutionalized individuals. Institutionalized older adults with early onset schizophrenia experience more severe symptoms than both younger adults and non-institutionalized older adults as well as declines in cognitive functioning (Iglewicz et al., 2011; Savla et al., 2006). An alternative explanation for the lower rates of schizophrenia in late life is selective mortality (Jobe & Harrow, 2010).

Late- and Very Late-Onset Schizophrenia "Late-onset" schizophrenia refers to onset after age 40 and "very-late-onset schizophrenia-like psychosis" refers to onset after age 60 (Howard, Rabins, Seeman, Jeste, & International Late-Onset Schizophrenia Group, 2000). Given the theorized etiology of schizophrenia as a deviation from normal brain development, onset of the disorder in late life would seem difficult to explain. Some investigators (Howard et al., 2000) have proposed that late-onset schizophrenia is a distinct disorder from early-onset schizophrenia, with differing epidemiology, presentation and risk or protective factors. Evidence is mixed.

Cross-sectional research suggests that symptom presentation is similar for early- versus late-onset schizophrenia (see Iglewicz et al., 2011), but this finding is mostly restricted to those younger than 60. For older individuals with very late onset, the expression of symptoms is less severe with more non-specific symptomatology compared to those with early onset (Häfner et al., 1998). Cognitive impairment is a symptom of schizophrenia for both early and late onset, but for those with late onset, the impairment is milder than for those with early onset (see Iglewicz et al., 2011). Some investigators have suggested that late-onset schizophrenia is better understood as a form of dementia, but longitudinal evidence from two studies suggests that this is not the case (Palmer et al., 2003; Rabins & Lavrisba, 2003).

Epidemiologic research suggests that risk factors may differ for early- versus late-onset schizophrenia.

Individuals with onset of schizophrenia in later life are less likely to have a family history of schizophrenia and more likely to experience sensory impairments than those with early onset (Howard, Almeida & Raymond, 1994; Iglewicz et al., 2011). Other correlates include being female and being unmarried, but it is unclear whether individuals with late-onset schizophrenia have better or worse social, educational, and occupational functioning compared to those with earlier onset schizophrenia (Iglewicz et al., 2011). Thus, additional research is needed to better understand whether early-onset and late- (or even very late) onset schizophrenia are the same disorder with different manifestations or distinct disorders.

Assessment and Treatment Several treatments have demonstrated efficacy in the management of schizophrenia in late life. Antipsychotic medications are effective in controlling positive symptoms, as in younger populations (Auslander & Jeste, 2004). Cognitive behavioral social skills training is associated with better social functioning, coping skills and insight among older adults with schizophrenia (Granholm et al., 2005). Supported employment has demonstrated similar success in improving outcomes in middle-aged and older adults as in younger samples (Twamley & Narvaez, 2008).

Conclusions Schizophrenia is relatively rare among older adults, but new onset is not uncommon, even after age 60. Overall, older adults with onset of schizophrenia after age 40 have the same rates of cognitive decline as those with early onset. With some exceptions, either stability in symptomatology and functioning or decreases in deficits or impairments are apparent for non-institutionalized individuals with schizophrenia. Evidence is inconclusive with respect to whether very-late-onset schizophrenia represents the same or a different disorder than early-onset schizophrenia, but it does not appear to predict dementia, and individuals with late-onset schizophrenia tend to have a better prognosis and quality of life than those with early onset.

Alcohol Use Disorders

Alcohol use disorders are associated with severe mental and physical health consequences and increased risk of mortality (Thun et al., 1997). These disorders are less common in late life than earlier in the lifespan, with the prevalence of problematic alcohol use among older adults between 1% and 22% for community populations (Johnson, 2000; Lin et al., 2011; Oslin, 2004). However, several methodological, assessment and etiological factors complicate the study of alcohol use in older age. Despite the fact that excessive drinking declines as individuals enter their 70s and 80s, many older adults still drink more than suggested consumption guidelines, especially those in the cohort born after 1920 (Moos et al., 2009).

Description and Course For a DSM-5 diagnosis of alcohol use disorder, an individual must meet two of 11 criteria, resulting in significant distress or impairment. These criteria represent a combination of the DSM-IV criteria for alcohol dependence and for alcohol abuse, with one criterion removed (recurrent legal problems) and one criterion added (craving). DSM-IV diagnostic criteria, which were not developed or validated in older adult populations, were thought to underestimate the prevalence of abuse among older adults (Patterson & Jeste, 1999). For example, diagnostic criteria specify age-dependent consequences (e.g. school or work) that may not be relevant in late life due to age-related role changes (Patterson & Jeste, 1999). Age-dependent consequences were not removed from the DSM-5 criteria, and other criteria continue to reflect standards based on younger adult samples that may not apply to older adults (e.g., classification of binge or heavy drinking). Evidence suggests that under-identification of alcohol-related problems in older adults will persist with DSM-5 (Kuerbis, Hagman, & Sacco, 2013). An alternative to the use of age-biased diagnostic criteria to identify problematic alcohol use in older adults is to measure frequency of drinking behaviors.

The major risk period for alcohol initiation is over by the age of 20 (DeWit, Adlaf, Offord, & Ogbrone, 2000), and the odds of a lifetime alcohol use disorder drops significantly for each increasing year of age at initiation (Grant & Dawson, 1997). Nonetheless, some older adults start problem drinking after age 50, often referred to as late adult onset or "reactive" drinking (Sattar, Petty, & Burke, 2003). Older adults who begin drinking later in life may do so in response to age-related stressors like traumatic losses, issues related to retirement, or major illness (Johnson, 2000; Sattar et al., 2003; Oslin, 2004). Although alcohol use disorders tend to follow a chronic course (Kerr, Fillmore, & Bostrom, 2002), drinking cessation occurs throughout the lifespan, for reasons that vary with age, including role transitions and health problems (Dawson, Goldstein, & Grant, 2013).

Etiology There are multiple possible causes of problematic alcohol use in late life. Alcohol use disorders arise from an interaction of genetic risk and environmental factors that may vary across the lifespan (Vanyukov & Targer, 2000). Risk factors for late-life drinking include pain (Brennan, Schutte, & Moos, 2005), chronic medical conditions (Ryan et al., 2013), sleep difficulties, and depression (Sattar et al., 2003; Schonfeld & Dupree, 1991). Lack of social support in late life may lead to use of alcohol as a negative coping strategy and is associated with risk of relapse (Moos, Finney, & Cronkite, 1990; Jason, Davis, Ferrari, & Bishop, 2001).

Explanations for Age Differences There are several possible explanations for the reduced prevalence of alcohol use disorders in late life. Longitudinal research shows

that excessive drinking behavior declines with age (Moos et al., 2009). Although prevalence and frequency of alcohol consumption remains stable over time among older adults, the average amount consumed declines with age (McEvoy et al., 2013). Younger alcohol users have been shown to "mature out" of problem drinking well before they reach old age as a consequence of both personality development and role transitions (Littlefield, Sher, & Wood, 2009). Physiological changes may contribute to older adults' increased sensitivity to alcohol, which may be associated with changes in alcohol use behaviors. For example, lean body mass and total body water to fat ratio decreases with age, resulting in increased serum concentrations of alcohol in the body (Oslin, 2004). Although some older adults may reduce consumption of alcohol as a consequence of physiological changes associated with aging, those older adults who continue lifelong drinking patterns unaware of their age-heightened risk may encounter drinking-related problems for the first time in late life (Merrick et al., 2008). An alternative explanation for reduced rates of alcohol use disorders in late life is selective mortality. Individuals with early-onset alcohol abuse or dependence die approximately 10 years earlier than age-matched controls (Thun et al., 1997; Fried et al., 1998), and even in late-onset cases, alcohol abuse has been found to be associated with excess mortality, especially in men (Moore et al., 2006). In addition, problems with detection may contribute to the low apparent rates of alcohol disorders in late life.

Assessment and Treatment Accurate assessment of alcohol use disorders is critical for their treatment. Unfortunately, underdetection and underreporting occur in primary care settings, which are often considered the front-line approach to older adult mental health care. D'Amico, Paddock, Burnam and King (2005) found that problem drinkers were less likely to visit general medical providers than non-problem drinkers. Additionally, medical professionals were less likely to ask about alcohol use in persons aged 50 or older (D'Amico et al., 2005).

Several other barriers have been identified in the detection of older adult alcohol misuse (St. John, Snow, & Tyas, 2010). The symptoms of alcohol use are often the same symptoms portrayed under different older adult disorders. Symptoms like falls, accidental injuries, urinary incontinence, and depression are geriatric problems that share common symptom presentations with those seen in alcohol abusers (Johnson, 2000; Sattar et al., 2003; Han, Gfroerer, Colliver, & Penne, 2009). Furthermore, the harmful effects of drinking also exacerbate problems with physical and mental health functioning. Cognitive impairment, in conjunction with or separate from alcohol disorders, may diminish the likelihood of detection, because of self-report difficulties, bias, and symptom misidentification (Blazer & Wu, 2009). In addition, because social patterns change with age (Carstensen et al., 1999), many of the social consequences of problem drinking go unnoticed (Sattar et al., 2003). Further complicating this dilemma is the fact that individuals at risk for alcohol problems or binge-drinking are less likely to report overt stress (Blazer & Wu, 2009). These factors should be considered when assessing for alcohol use problems in an older adult.

Older adults are amenable to treatment, and outcomes are especially beneficial when elder-specific treatments are used (Blow, Walton, Chermack, Mudd, & Brower, 2000; Oslin, 2004). Late-onset drinkers may even be more responsive and receptive to treatment than early-onset drinkers (Oslin et al., 2002), especially since early-onset patients may be more likely than late-onset patients to drop out of treatment (Atkinson, Tolson, & Turner 1993). Older adults attain positive outcomes across a range of measures (e.g. alcohol, emotional, social) after receiving and completing substance treatment. Older adults may also respond well to brief interventions (Oslin, 2004). However, few intervention studies use randomized controlled designs in older adults, making it difficult to suggest which types of treatment interventions are most effective (Sattar et al., 2003; Oslin, 2004).

Conclusions Cross-sectional and longitudinal research suggests that alcohol use disorders are less prevalent in old age than earlier in the lifespan. Research suggests that these findings may be explained by reduced drinking associated with age-related changes in physiological, personality and social factors as well as selective mortality. However, the lack of consensus on what is considered problem drinking, as well as difficulty detecting and screening for alcohol use disorders in late life, complicate the picture.

Although alcohol use disorders decrease with age, they still present a significant public health challenge. The number of older adults who will meet criteria for substance use disorder is projected to double from 2.8 million to 5.7 million in 2020 (Han et al., 2009). Now that the baby boom generation is moving into old age, researchers will have the unique ability to witness how alcohol use changes in a generation that has historically consumed more alcohol and been more open to psychological treatment than previous generations.

Hoarding

Description and Course Hoarding disorder is a debilitating condition that is associated with functional impairment, increased disability, fire, and incident falls during late life (Ayers et al., 2014). Hoarding disorder was recently accepted into DSM-5 and is defined as: 1) Persistent difficulty in discarding possessions that appear (to others) to be useless or of limited value; 2) the accumulation of possessions cluttering active living spaces that substantially compromises their intended use; and 3) the hoarding causes significant distress and impairment in social, occupational, or other important areas of functioning.

Hoarding onset is typically during adolescence with 68% of cases starting before age 20 (Ayers et al., 2010, Samuels et al., 2008). The course of hoarding disorder appears to be progressive and chronic across the lifespan, with very few cases of remission (Grisham, Frost, Steketee, Kim, & Hood, 2006). Many patients with hoarding disorder have comorbid mood and psychiatric disorders, including obsessive-compulsive disorder, major depressive disorder, and generalized anxiety disorder, among others (Frost, Steketee, & Tolin, 2011). Older adults who are socially isolated, never married, and have a history of childhood trauma are often more likely to hoard. Late-onset hoarding is rare, yet cases of hoarding disorder often come to attention during late life, when health departments and senior service agencies are called to intervene. Older adults appear to hoard the same items as younger adults, such as paper, clothing, and books (Kim, Steketee, & Frost, 2001). Hoarding disorder is particularly dangerous for older adults, who may also have physical and cognitive limitations. Although limited, research has begun to show that older adults with hoarding disorder have executive functioning deficits and display worse executive functioning, memory, and attention/concentration than matched controls (Ayers et al., 2013). Therefore, researchers suspect that those who develop hoarding behaviors for the first time in late life may have an underlying cognitive impairment.

Epidemiology Hoarding is relatively common during late life, with prevalence rates estimated between 2% and 5% of community-dwelling older adults (Samuels et al., 2008). Earlier research suggests that hoarding symptoms are displayed by 25% of elderly community daycare residents and 15% in nursing homes (Marx & Cohen-Mansfield, 2003). Although some research suggests that hoarding disorder is more common in older adults than younger adults (Samuels et al., 2008), it is unclear whether prevalence rates increase with age.

Many studies document the negative consequences of hoarding disorder in older adults. Hoarding disorder is associated with medication and dietary mismanagement, which may lead to accelerated medical illness (Ayers et al., 2010). Given patients' inability to move around in their home, hoarding disorder is associated with significant impairments in basic and instrumental activities of daily living (Diefenbach, DiMauro, Frost, Steketee, & Tolin, 2013). Hoarding disorder may also lead to premature relocation to senior housing, oftentimes by eviction (Whitfield, Daniels, Flesaker, & Simmons, 2012).

Etiology Hoarding disorder has been conceptualized as a result of information-processing deficits, beliefs about and emotional attachment to possessions, and avoidance behaviors (Steketee & Frost, 2007). There is evidence of familial aggregation of hoarding symptoms (Samuels et al., 2007). Indecision, which is more prevalent in relatives of obsessive-compulsive disorder probands with hoarding symptoms compared with relatives of obsessive-compulsive disorder probands with no hoarding symptoms, may be a risk factor for hoarding (Samuels et al., 2007).

Assessment and Treatment Hoarding problems often go undetected and untreated among older adults (Ayers et al., 2010). Nonetheless, several assessment measures have been validated for use in older adults. The Hoarding Rating Scale (Tolin, Frost, & Steketee, 2010) screens for presence of hoarding symptoms. The Savings Inventory, Revised (Frost, Steketee, & Grisham, 2004) provides self-report of hoarding severity, whereas the Clutter Image Rating Scale (Frost, Steketee, Tolin, & Renaud, 2008) provides self-report of level of clutter in the home. The University of California, Los Angeles, Hoarding Severity Scale (Saxena, Brody, Maidment, & Baxter, 2007) is a clinician-rating measure of hoarding severity.

Research on treatments for hoarding disorder in older adults is in its early stages. Removal of clutter is not consistently effective, as most participants do not improve, some experience worsened symptoms, and others demonstrate improvement followed by relapse (Kim et al., 2001). A form of cognitive behavioral therapy that emphasizes cognitive restructuring has been used successfully in middle-aged patients with hoarding problems (Steketee and Frost, 2007), but was not effective in older adults with compulsive hoarding disorder (Ayers, Wetherell, Golshan, & Saxena, 2011).

A newly developed behavioral treatment for compulsive hoarding disorder in older adults shows promise (Ayers et al., 2014). It incorporates cognitive rehabilitation to address executive functioning deficits and exposure techniques to address discarding/not-acquiring behaviors, and it de-emphasizes cognitive restructuring techniques. The cognitive rehabilitation includes training to strengthen prospective memory (e.g., use of to-do lists), categorization/organization, problem solving and cognitive flexibility. The treatment also emphasizes homework, as numerous studies in middle-aged and older adults with hoarding problems have confirmed that homework compliance is strongly related to decreased hoarding severity post-treatment (Ayers et al., 2011). An open trial demonstrated large improvements in both self-reported and clinician-rated hoarding behaviors.

Conclusions Hoarding disorder is a disabling form of psychopathology that affects a large number of older adults. Research on this newly identified disorder is in its early stages. Previous approaches to intervention have not been effective in older adults with hoarding disorder, but a novel behavioral treatment has already shown promise.

Personality Disorders

Personality disorders are defined as pervasive and inflexible patterns of behavior associated with distress or impairment.

A more detailed discussion of diagnostic criteria for personality disorders can be found in Chapter 12 in this volume. Although personality disorders are defined by their longstanding patterns of dysfunctional behavior, some may manifest differently as individuals age. Personality disorders are found in 10% of community-dwelling older adults (Widiger & Seidlitz, 2002), which is slightly lower than the prevalence of 10–13% for younger adults (Weissman, 1993). DSM-5 describes ten personality disorders, but only four with evident age-related differences are reviewed in detail here. Cross-sectional research suggests that antisocial personality disorder (ASPD) and borderline personality disorder (BPD) are less prevalent in older adults than in younger adults (Segal, Hook, & Coolidge, 2001), whereas schizoid personality disorder (SPD) and obsessive-compulsive personality disorder (OCPD) appear to be more prevalent (Segal et al., 2001). Among individuals aged 65–98 years old, ASPD was diagnosed in 0.2–0.3%, SPD in 0.9–1.9%, and OCPD in 1.6–2.5% (Balsis, Woods, Gleason, & Oltmanns, 2007).

Description and Course SPD is characterized by a lack of interest in interpersonal relationships and limited expression of emotion, ASPD by disregard for and violation of the rights of others, BPD by instability in relationships, affect and identity, and OCPD by preoccupation with order and control (American Psychiatric Association, 2013).

Evidence on the presentation of personality disorders over the lifespan points to changes in specific personality features as people age (Oltmanns & Balsis, 2011). Older adults with ASPD are more likely to endorse lying or deception than younger adults with ASPD (Balsis, Gleason, Woods, & Oltmanns, 2007). This age difference is consistent with the interpretation that violation of the rights of others (e.g., physical fighting and aggression) requires physical strength and agility (Kroessler, 1990), which may diminish with age. With respect to the presentation of BPD, older adults with BPD may have fewer close relationships than their younger counterparts; thus, the pattern of unstable relationships may be less frequently endorsed (Agronin & Maletta, 2000). In addition, older adults are less likely to exhibit impulsive behaviors and are more likely to experience the cognitive symptoms of BPD, such as identity disturbance (Clarkin, Spielman, & Klausner, 1999). Older adults with BPD also report fewer issues with substance use and less affect instability and self-harm behavior compared with younger adults with BPD, but report greater social impairment and chronic feelings of emptiness (Morgan, Chelminski, Young, Dalrymple, & Zimmerman, 2013). Selective mortality has not been addressed as a possible contributor to lower substance use and self-harm in older adults with BPD.

Etiology Personality disorders can be conceptualized as maladaptive and extreme variants of normal personality traits. As such, they are likely to arise from an interaction of genetic predisposition and early life environment exacerbated by current stressors. The predisposition hypothesis (Sadavoy & Leszcz, 1987, as cited in Zweig & Hillman, 1999) suggests that middle-aged and older adults with personality disorders are more susceptible to age-related stressors (e.g., loss, physical illness, forced dependency) than their counterparts. Encountered stressors may lead to poorer prognosis, increased disability, and mortality.

Explanations for Age Differences There are a few explanations for the age differences in prevalence of specific personality disorders. The maturation hypothesis posits that immature personality types (e.g., antisocial, borderline) are more likely to improve with age as impulsivity decreases (Solomon, 1981, cited in Zweig & Hillman, 1999). On the other hand, mature personality disorder types (OCPD, schizoid, and paranoid personality disorders) are thought to worsen with age as a result of mild cognitive impairment (e.g., hoarding in OCPD) or other factors.

Longitudinal research findings are broadly consistent with these explanations. At present, there are several well-constructed and promising longitudinal studies of personality disorders (e.g., McLean Study of Adult Development: Zanarini, et al., 2014; Collaborative Longitudinal Personality Disorders Study: Skodol et al., 2005). However, the cohorts are young and yield little information about individuals older than 50 years. Despite this limitation, there is longitudinal evidence that impulsive behaviors decrease over time for both ASPD and BPD. The course of ASPD varies with high mortality rates (23.9%), low remission (17.6%), and continued engagement in antisocial behavior for almost 50% of individuals (48.5%; Black, Baumgarten, & Bell, 1995). In contrast, about 60% of patients with BPD had two years of remission at 16-year follow-up (Zanarini et al., 2014). Although remission is common, some symptoms of affective instability still remain (Zanarini, Frankenburg, Hennen, & Silk, 2003). Thus, distress associated with personality disorders is stable across the life span, while symptom presentation changes.

Age differences may also be reflective of biased diagnostic criteria, with one study finding evidence for measurement bias by age group in 29% of diagnostic criteria for personality disorders (Balsis, Gleason et al., 2007). For example, the criteria for SPD may falsely identify as schizoid older adults with social isolation due to disability or decreased functional status. Some of the criteria for OCPD, which is frequently identified in older adults, may be biased towards younger age groups as the criteria focus on behaviors occurring in a work setting (Agronin & Maletta, 2000). Notwithstanding the potential bias, three other criteria were more frequently endorsed by older adults with OCPD: (1) Inflexible about moral, ethical, or values issues; (2) unable to discard worthless objects;

and (3) assumes a miserly spending style (Balsis, Gleason et al., 2007).

Additionally, selective mortality may play a role in the apparent decrease in personality disorders that involve impulsive and potentially harmful behaviors, specifically ASPD and BPD. Individuals with ASPD and BPD are more likely to die unnatural deaths, such as suicide or other violent deaths, compared with the average population (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). Older adults with personality disorder features are also more likely to experience suicidal ideation than those with no or lower levels of personality disorder features (Segal et al., 2012).

Assessment and Treatment Assessment measures used to diagnose personality disorders have been created using young adult samples and may be biased when used with older adults (Balsis, Gleason et al., 2007). In particular, when assessing older adults it is essential to differentiate behaviors consistent with personality disorders from behavior influenced by medical or neurological disorders (e.g., dementia), role changes (e.g., recently widowed), and other changes in life context (e.g., moving to long-term care facility; Zweig, 2008). One measure has been specifically developed to screen for general personality disorders in older adults (Gerontological Personality Disorders Scale; van Alphen, Engelen, Kuin, Hoijtink, & Derksen, 2006).

Among older individuals with comorbid personality disorders and other psychiatric disorders, the course of treatment may be slowed, complicated, and possibly impeded compared with individuals without personality disorders (Gradman, Thompson & Gallagher-Thompson, 1999). As there are few treatment studies to begin with, it is not clear whether certain types of psychotherapy are more beneficial than others for individuals with personality disorders. In recent years, dialectical behavior therapy has gained support in treating young adults with BPD (for a review, see Lieb et al., 2004), but has not been widely examined as treatment for personality psychopathology among older adults. Dialectical behavior therapy was found to be effective in conjunction with medication to treat depression in older adults with comorbid personality disorders (Lynch et al., 2007).

Conclusions Personality disorders reflect heterotypic continuity, in that certain symptoms (e.g., impulsivity) vary across the lifespan, while there are some stable, underlying personality characteristics. The stability in the underlying distress of personality disorders is evident as older adults with personality disorders experience more distress and are more difficult to treat compared with older adults without personality disorders. Contrary to the assumptions embedded in the diagnostic system, longitudinal studies suggest that BPD is more likely than not to remit across the life span. Additionally, individuals with BPD and ASPD may experience selective mortality, which is reflected by some cross-sectional research. Some researchers suggest that schizoid personality disorder and OCPD are more prevalent in late life. More longitudinal research is needed to elucidate the course of personality disorders across the lifespan and into old age.

Sleep Disorders

One-third of one's life is spent sleeping, yet the important function that sleep plays goes largely unnoticed until one's sleep becomes disturbed in some way. Older adults who suffer from sleep difficulties are at greater risk for falling because of fatigue, slower reaction times, and impaired daytime functioning associated with sleep impairment (Ancoli-Israel, Ayalono, & Salzman, 2008). In animal models of Alzheimer's disease, sleep deprivation exacerbated memory problems and reduced postsynaptic density, suggesting that sleep plays a role in the biological basis and manifestation of Alzheimer's disease and might be an intervention point to improve the prognosis of patients with Alzheimer's (Di Meco, Joshi, & Pratico, 2014). Sleep disturbance has even been linked to elevated risk of mortality (Hardy & Studenski, 2008).

The most common sleep disturbance that clinicians encounter is insomnia. Insomnia is both an independent DSM-5 diagnosis and a diagnostic component of many other psychological and psychiatric disorders. Diagnoses such as mood and anxiety disorders have been linked to persistent poor sleep quality in longitudinal studies of older adults (Morgan, Healey, & Healey, 1989). While insomnia can occur at any point in the life span, rates increase by 200% around the seventh and eighth decade, and insomnia is present in 41% of women aged 80–89 (Lichstein, Stone, Nau, McCrae, & Payne, 2006). In a large-scale study of adults age 65 and older, up to 34% reported symptoms of insomnia, and 50% of women reported such symptomatology (Foley et al., 1995). Women are more likely to report symptoms of insomnia across the life span, but this difference becomes more marked in late life when women are 73% more likely to experience insomnia than men (Zhang & Wing, 2006).

Description and Course The DSM-5 criteria for insomnia disorder require difficulty sleeping at least three nights a week for a minimum of three months, with some type of daytime dysfunction not due to drug or medication use or fully explained by some other psychological, medical, or sleep disorder (APA, 2014). Diagnostic criteria ruling out insomnia if other conditions are present, found in previous versions of the DSM, have been removed to allow diagnosis of comorbidities (American Psychiatric Association, 2014). In contrast, the *International Classification of Sleep Disorders*, second edition (ICSD-2), a specialized system for classifying sleep disorders, specifies diagnostic criteria for insomnia that require difficulty sleeping

when provided ample opportunity to do so and some type of daytime dysfunction (American Academy of Sleep Medicine, 2005). Subtypes of insomnia in the ICSD-2 are classified according to the time of the night when the problem occurs. Sleep onset insomnia is broadly defined by difficulty falling asleep, whereas maintenance insomnia is defined by an inability to stay asleep or by waking earlier than desired (Lichstein et al., 2006). Insomnia is often comorbid with other psychological and physical health problems such as depression, anxiety, diabetes, and heart disease (Ancoli-Israel et al., 2008; Cooke & Ancoli-Israel, 2006; Lichstein et al., 2006).

Etiology The predominant conceptual model of insomnia is Spielman's behavioral model (Spielman, Caruso, & Glovinsky, 1987). Predisposing factors, which may be biological, psychological or social, are thought to increase vulnerability to insomnia and interact with precipitating factors, such as acute stressors, in producing the acute phase of insomnia. Behavioral responses, such as extending sleep opportunity and using alcohol to induce sleep, tend to perpetuate sleep difficulties, leading to chronic insomnia. Predisposing, precipitating and perpetuating factors vary in frequency across the lifespan.

Explanations for Age Differences Changes in sleep structure over the course of a night that occur with normal aging may predispose older adults to insomnia. Sleep latency, or the time it takes one to fall asleep once in bed, increases with age, becoming most pronounced after age 65 (for a meta-analysis, see Ohayon, Carskadon, Guilleminault, & Vitello, 2004). Total sleep time decreases by approximately 10 minutes for each decade of the life span (Ohayon et al., 2004), and time awake after sleep onset also increases approximately 10 minutes each decade beginning at age 30 (Ohayon et al., 2004).

When examining sleep timing across the lifespan, a normative shift to earlier sleep onset is seen among older adults (Ancoli-Israel, 2005), as demonstrated by biochemical markers of sleep (Yoon et al., 2003). This phase shift has been associated with maintenance insomnia, such that older adults often attempt to remain awake until "normal" bed times, which are later than their biologically driven sleep onset time. In tandem, the sleep period is shortened, causing daytime fatigue. Compared with earlier lifespan periods, older adults have more flexible schedules, allowing for "make up" sleep via daytime naps, which serve to further complicate the association of advance sleep phase shift and insomnia (Yoon et al., 2003).

Assessment and Treatment Assessment of insomnia often begins with complaints of trouble falling asleep, early awakening, or daytime fatigue at least three times per week, which can be documented using a sleep diary. Confirmation of sleep and wake activity levels can be done most cost efficiently by using an actigraph, which is worn on the wrist and measures motion. Effective interventions for insomnia include pharmacological treatment with hypnotics such as benzodiazepine receptor agonists or melatonin receptor agonists, which are highly prescribed among older adults and are helpful in the short term, melatonin, and sedating antidepressants, for which evidence is mixed (Lichstein et al., 2006; Roehrs & Roth, 2012). It should be noted that use of benzodiazepine receptor agonists has been linked to increased risk of falls for all populations, which is particularly concerning for older adults (Dillon, Wetzler, & Lichstein, 2012). Yet, some studies have suggested that risk of falls among older adults using hypnotics is no higher than among older adults with untreated insomnia (Roehrs & Roth, 2012). More concerning is current research suggesting that anxiolytic and hypnotic drugs are linked to higher mortality rates among users than nonusers (Weich et al., 2014). There is also longitudinal evidence that shows that long-term use of hypnotics is ineffective and can exacerbate insomnia when patients cease taking them (Hohagen et al., 1993). Moreover, melatonin receptor agonist efficacy among older adults might be limited—owing to its short half life it does not alleviate maintenance insomnia (Roehrs & Roth, 2012). Behavioral techniques that have demonstrated efficacy in treating insomnia, separately or as part of a multicomponent treatment, include stimulus control, which strengthens the association of the bed with sleep, and sleep restriction, which limits time in bed (Perlis, Jungquist, Smith, & Posner, 2005). Research has demonstrated that the most efficacious long-term treatment of insomnia is early combined treatment with behavioral and pharmacological treatment with a phasing-out of pharmacological aids (Morin et al., 2009). In fact, older adults with comorbid diagnoses such as osteoarthritis pain may experience longer-term (18 months or more) benefits from cognitive behavioral treatment for insomnia than those with a singular insomnia diagnosis (McCurry et al., 2014).

Conclusions Older adults are at elevated risk for insomnia (Ancoli-Israel, 2005). Insomnia has been linked to daytime sleepiness which, if frequent enough, has been linked to serious health outcomes, including increased mortality rates (Morgan et al., 1989). Because treatment of sleep problems has been related to alleviation of comorbid symptomology, effective sleep treatments, such as behavioral treatments for insomnia, provide hope not only for those suffering from sleep disorders, but also those suffering from other psychiatric diagnoses.

Dementia

In 2012, approximately 25.6 million people worldwide were suffering from some form of dementia (World Health Organization, 2014). Prevalence of dementia increases with age; rates for adults in their 60s are approximately 1.5%, with rates increasing to 15–25% for

adults in their 80s. Although underlying mechanisms are not fully understood, dementia is more common among women than men (Shumaker et al., 2003). Life expectancy decreases with severity of dementia, especially for those 85 years and older, among whom dementia is a predictor of approximately 30% of all deaths in men and 50% of all deaths in women (Aevarsson, Svanborg, & Skoog, 1998). Alzheimer's disease, the most common subtype of dementia, accounts for approximately 60–70% of all dementia cases (Alzheimer's Association, 2015) and is the focus of this section. Vascular dementia is the second most common subtype and is estimated at 8–30% of all dementia cases (DiCarlo et al., 2002).

Description and Course The DSM-5 diagnosis of dementia is now included under the newly named major neurocognitive disorder. DSM-5 also recognizes a less severe form of cognitive impairment, mild neurocognitive disorder. Although the threshold between major and minor is arbitrary, this new diagnosis is consistent with several medical fields (e.g., geriatric psychiatry, neurology, rehabilitation psychology), where mild neurocognitive disorder is a focus of research and clinical care (American Psychiatric Association, 2014). The criteria for major neurocognitive disorder include memory impairments and progressive cognitive declines in one or more areas of intellectual functioning: Aphasia (language deterioration), apraxia (impaired ability to complete motor activities), agnosia (failure to identify objects), or a disturbance in executive functioning (ability to think abstractly, execute a plan; American Psychiatric Association, 2013). Cognitive declines cannot be due to preexisting central nervous system disorder or other disorder known to cause dementia. For individuals with major neurocognitive disorder, cognitive decline must be severe enough to interfere with independence, whereas individuals with mild neurocognitive disorder may have the ability to remain independent. In DSM-5, diagnostic criteria for the Alzheimer's disease and vascular subtypes have been retained (American Psychiatric Association, 2014). Neurocognitive disorders caused by other medical conditions (Lewy bodies, HIV infection, Parkinson's disease) are also included as diagnoses.

For major or mild neurocognitive disorder due to Alzheimer's disease, onset is typically after age 65 and cognitive decline is slow and progressive. Survival time ranges from approximately 3 years (if diagnosed in their 80s or 90s) to 7–10 years (if diagnosed in their 60s and 70s; Cosentino, Scarmeas, Albert, & Stern, 2006). Defining neurological features of Alzheimer's disease are the presence of amyloid plaques, neurofibrillary tangles, and brain atrophy.

Etiology Alzheimer's disease involves the progressive deterioration of the cerebral cortex and the hippocampus, which is expressed clinically through impairment in memory and other cognitive abilities. Cognitive reserve capacity, conceptualized as the brain's ability to sustain the effects of injury or disease, varies across individuals and is thought to influence the extent and timing of the clinical expression of Alzheimer's disease-related neuropathology (Borenstein, Copenhaver & Mortimer, 2006). As a result, it may be particularly helpful to distinguish between risk factors for AD neuropathology and risk factors for the clinical expression of Alzheimer's disease.

Among risk factors thought to influence Alzheimer's disease neuropathology, genetic vulnerability is the most prominent (Gatz et al., 2006). The e4 allele of the apolipoprotein (APOE) gene is well established as a risk factor for late-onset Alzheimer's disease (most cases of Alzheimer's disease are late onset; Farrer et al., 1997). APOE4 is a susceptibility gene; that is, carrying one or two APOE e4 alleles does not mean an adult will, without a doubt, develop Alzheimer's disease. In addition to APOE, two brain abnormalities are considered the hallmarks of Alzheimer's disease: beta-amyloid plaques and tangles of a protein called tau, both of which are associated with brain cell death (Reitz, 2012).

Risk factors thought to influence the clinical expression of Alzheimer's disease include education and premorbid intelligence. The association between years of education and decreased risk of Alzheimer's disease is well established (DiCarlo et al., 2002; Meng & D'Arcy, 2012). For example, Gatz and colleagues (2007) examined this relation in a population of adult twins and found that it was independent of genetic influences. Whalley, Deary, Appleton, and Starr (2004) also demonstrated that premorbid, childhood IQ predicts slower cognitive decline in older adults. Cognitive reserve may even have an influence on cognition after a diagnosis of Alzheimer's disease has been made (Starr & Lonie, 2008).

Some investigators propose the "use it or lose it" hypothesis, proposing that mental activity and stimulation early in life may increase synaptic density in the brain and thereby enhance cognitive reserve (Orrell & Sahakian, 1995). The hypothesis suggests that individuals with greater cognitive reserves experience fewer cognitive deficits during the aging process (Whalley et al., 2004), but the impact on later protection from dementia remains to be fully evaluated.

Explanations for Age Differences Alzheimer's disease is a progressive neurodegenerative disease. As such, risk for Alzheimer's disease increases directly with advancing age. In addition, aging-related conditions such as hypertension and cardiovascular diseases may play a role in Alzheimer's disease via damage to the vascular system.

Assessment and Treatment The assessment of Alzheimer's disease currently involves neuropsychological testing to evaluate memory and cognitive impairments, as well as a somatic examination and neuroimaging to rule out alternative explanations for the impairment. Autopsy studies

validate that this method can be highly accurate (Gatz et al., 2006). Advances in neuroimaging technology, which have recently made it possible to view Alzheimer's disease-related neuropathology (amyloid plaques), have made it possible to evaluate factors that affect the clinical expression of Alzheimer's disease independent of the neuropathology (Roe et al., 2010). Neuroimaging techniques include functional magnetic resonance imaging, advanced magnetic resonance imaging techniques, and positron emission tomography with fluorodeoxyglucose, among others (Risacher & Saykin, 2013). Although this type of neuroimaging may be helpful in diagnosing Alzheimer's disease, diagnostic accuracy is improved by including measures associated with the clinical expression of the disease (Roe et al., 2010).

There are no published interventions for the primary prevention of dementia and no available treatments to slow or reverse the progression of brain deterioration in Alzheimer's disease. However, the U.S. Food and Drug Administration has approved several drugs that can ameliorate symptoms for up to 12 months (Massoud & Gauthier, 2010). The acetyl cholinesterase inhibitors, such as donezepil (brand name Aricept®) and galantamine (brand name Razadyne®), have been shown to improve cognition and functioning in mild to moderate Alzheimer's disease, whereas the N-methyl-d-aspartate receptor antagonist memantine (brand name Namenda®) has demonstrated similar outcomes in mild to severely impaired patients (Massoud & Gauthier, 2010). Over 90 clinical trials are currently underway to examine ways to detect, slow, treat, and prevent Alzheimer's disease; they include treatment trials, diagnostic studies, prevention trials, screening studies, and quality of life studies (Alzheimer's Association, 2014).

Although preventive treatments do not exist, clinicians typically focus on preventive treatments of cardiovascular disease, such as controlling modifiable risk factors like cholesterol levels, blood pressure, and diabetes (Rockwood, 2002). Prevention of these conditions can be achieved through regular exercise, and lowering both blood pressure and cholesterol levels.

Conclusions Alzheimer's disease is a progressive neurodegenerative disorder that affects almost exclusively older adults. Highly accurate assessment and diagnosis is possible, and new methods for treatment and prevention are on the horizon. Substantial progress has been made in neuroimaging, biomarker signaling methods, and genetics research (Alzheimer's Association, 2014). Although there is no cure for Alzheimer's disease, current treatments are able to reduce symptom severity.

Psychopathology in Late Life

There is evidence of both similarity and difference in psychopathology in late life relative to earlier ages. Contrary to popular stereotype, late life is associated with lower prevalence of many types of psychopathology, including anxiety disorders, major depressive disorder, bipolar disorder, schizophrenia, alcohol use disorders and some types of personality disorders. There is longitudinal evidence of attenuation of symptom severity or even remission for most of these disorders. These outcomes are consistent with the improved emotion regulation that has been documented in late life. It is also possible, however, that lower rates of disorder in late life may be a methodological artifact rather than reflecting a true age-related decline. Symptom presentation of many of the disorders differs in older adults compared to younger adults, such that diagnostic rubrics established to describe disorders in younger populations may not capture these disorders when they appear in older populations. In some cases, symptoms may attenuate slightly, such that diagnostic criteria are no longer met, but remain problematic for the patient. For example, sub-threshold depressive and anxiety symptoms are common in late life. Further, selective mortality may explain some of the apparent decline in prevalence of some of these disorders (bipolar, major depressive disorder, substance use disorders, schizophrenia). More research, including longitudinal designs, is needed to tease apart alternative explanations.

In contrast to the disorders that appear to decline in prevalence with age, there are several disorders that increase in prevalence with age, including neurocognitive disorders, sleep disorders, select personality disorders and suicide. Biological changes associated with aging, which are associated with increased likelihood of executive dysfunction and other cognitive deficits, may explain not only neurocognitive disorder but also other disorders that are linked to cognitive functioning, such as hoarding. Age-related changes in the circadian rhythm have been implicated in increased rates of insomnia. Of note, however, is that biological aging is not always associated with increases in disorder; e.g., changes in metabolism with age can lead to reductions in problem drinking. In addition, behavioral and environmental factors may contribute to increased prevalence of certain disorders in late life. For example, schedule flexibility in old age and sleep-related behaviors may perpetuate insomnia; habituation to painful or provocative experiences may increase risk of high lethality suicidal behavior; and social role changes may precipitate change in alcohol use.

The assessment of psychopathology in older adults can be challenging, owing to differences in symptom presentation and comorbid physical illnesses. Age-specific measures are available for some but not all disorders. Many of the same treatments that have demonstrated efficacy in adult populations have also been shown to work in older adults, and several treatments have been developed specifically for older adults. Nonetheless, outcome research within this age group is scarce.

Increasingly, longitudinal research has begun to reshape our understanding of psychopathology in late life.

Additional longitudinal research is needed to elucidate the trajectories of psychopathology across the lifespan. Future research focused on older adults who do not manifest psychopathology in spite of risk factors could be particularly helpful in uncovering protective factors. Finally, assessment and treatment outcome research with a focus on older adults is needed. Considering the impending expansion of the older adult population, this type of research could not be more timely.

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Part III

Common Problems of Childhood and Adolescence

19

Externalizing Disorders of Childhood and Adolescence

Eva R. Kimonis and Paul J. Frick

Symptoms of common mental disorders in children and adolescents have been conceptually organized into two broad dimensions. One dimension, which is the focus of the current chapter, has been labeled as *undercontrolled* or *externalizing* and includes various acting out, disruptive, delinquent, hyperactive, and aggressive behaviors. The second dimension has been labeled as *overcontrolled* or *internalizing* and includes such behaviors as social withdrawal, anxiety, and depression (see Sander & Ollendick, Chapter 20 in this volume). The distinction between internalizing and externalizing problems is well supported by a number of factor analytic studies (Achenbach, 1995; Lahey et al., 2008).

Within the externalizing dimension, there are two major categories of behavior problems: (1) Problems of inattention, impulsivity, and hyperactivity associated with a diagnosis of attention-deficit hyperactivity disorder (ADHD); and (2) conduct problems and aggressive behavior associated with a diagnosis of oppositional defiant disorder (ODD) or conduct disorder. These two domains of externalizing problems can be separated in factor analyses and exhibit different correlates (Lahey et al., 2008; Waschbusch, 2002). For example, ADHD is specifically linked with poor academic achievement, problems in executive functioning, and parental inattention and impulsivity, whereas conduct problems are specifically associated with socioeconomic disadvantage, dysfunctional family backgrounds, and parental criminality and antisocial behavior. However, twin research suggests that shared environmental risk factors, such as parent–child conflict, may act as a common vulnerability for the development of both ADHD and conduct problems (Burt, Krueger, McGue, & Iacono, 2003). The importance of these correlates for understanding the two types of externalizing disorders is discussed in later sections of this chapter.

While research supports distinguishing between these two forms of externalizing disorders, and the following sections provide somewhat separate reviews of them, they do overlap considerably. For example, approximately 50% of children with ADHD have a co-occurring conduct problem diagnosis (Waschbusch, 2002) and between 65% and 90% of children with either ODD or conduct disorder have a co-occurring ADHD diagnosis (Abikoff & Klein, 1992). Awareness of the overlap between these disorders is important for understanding and interpreting research in this area because many studies fail to control for their co-occurrence. As a result, it is often unclear whether the correlates documented in research are due to one or the other type of externalizing disorders or both (Lilienfeld & Waldman, 1990). Further, children with both ADHD and conduct disorders show a more persistent course of ADHD (Broidy et al., 2003; Frick & Loney, 1999). Finally, the presence or absence of comorbidity also influences which type or treatment will be most effective (Arnold et al., 2004; Frick, 2012).

Attention-Deficit Hyperactivity Disorder

Definition and Description

Diagnosis The primary feature of ADHD is extreme and maladaptive levels of motor activity. It is the most common neurobehavioral disorder of childhood; the worldwide prevalence of ADHD is estimated at 2.2% for boys and 0.7% for girls, using *Diagnostic and Statistical Manual of Mental Disorders,* (DSM) or *International Classification of Diseases and Related Health Problems* (ICD) diagnostic criteria (Erskine et al., 2013). ADHD has been recognized in the psychiatric literature since the mid-19th century, although there has been long-standing

debate concerning its primary cause and the best method for diagnosing ADHD in children. This debate is reflected in numerous changes in its clinical designation (e.g., minimal brain dysfunction, hyperactive child syndrome) and in the diagnostic criteria used over the years.

In the most recent revision of the DSM, the fifth edition (DSM-5; American Psychiatric Association, 2013), ADHD is categorized as a neurodevelopmental disorder alongside intellectual disabilities, communication disorders, autism spectrum disorder, and learning and motor disorders. ADHD is characterized by two core symptom dimensions, an inattention cluster (e.g., not listening when spoken to directly, disorganization, easily distracted by extraneous stimuli) and a hyperactivity–impulsivity cluster (e.g. excessive talking or activity, difficulty awaiting turn). These two dimensions are consistent with the previous revision (DSM-IV-TR; American Psychiatric Association, 2000) and were identified through many factor analytic studies conducted with both clinic-referred and community-based samples (Frick & Nigg, 2012). As with all childhood disorders, some degree of ADHD symptoms is normal in childhood; ADHD may be best viewed as an extreme variation of an underlying trait dimension (Larsson, Anckarsater, Råstam, Chang, & Lichtenstein, 2012; Willcutt, Pennington, & DeFries, 2000). According to DSM-5, the following criteria are required to distinguish between normal variations of these behaviors and those needed to make a diagnosis (APA, 2013):

- Severity (six or more behavioral symptoms for children; five or more symptoms for individuals ages 17 and older)
- Duration (problems are evident before age 12 and persist for at least six months)
- Pervasiveness (impairment present in two or more settings, e.g., at home, school, or work; with friends or relatives; in other activities)
- Impairment (clear evidence of impaired social, academic, or occupational functioning).

For DSM-5, the Disruptive Behavior Disorders Workgroup considered several changes to the DSM-IV-TR diagnostic criteria for ADHD in order to: (a) Make the diagnosis more valid for adults (Barkley, Murphy, & Fischer, 2008); (b) raise the age of noticeable symptom presence from before age 7 to before age 12 (Kieling et al., 2010); and (c) allow the diagnosis when the child also meets diagnostic criteria for pervasive developmental disorder (Frick & Nigg, 2012).

First, with respect to making the diagnosis in adults, changes adopted in DSM-5 include lowering the symptom threshold for older adolescents and adults from six symptoms to five, and including examples to illustrate the types of behaviors adolescents and adults with ADHD might exhibit. For instance, the symptom "often runs about or climbs in situations where it is inappropriate" may be manifested as feelings of restlessness in individuals ages 17 and older. This change was supported by decades of research on adult ADHD, suggesting that the DSM-IV-TR symptom set failed to efficiently capture the adult manifestation in a developmentally appropriate way and the cut point of six symptoms under-identified adults with impairment and in need of treatment (Frick & Nigg, 2012).

Second, raising the age of noticeable symptom presence was supported by a review of the literature suggesting that: (a) Only about 50% of adults with ADHD recalled an onset before age 7 but 95% recalled an onset before age 12; by contrast, 95% recalled an onset before age 12 and 99% before age 16; and (b) children identified as having symptoms that emerge after age 7 have the same profile, course, impairment, severity, treatment response, and neurobiological findings as those identified as having onset prior to age 7 (Kieling et al., 2010). Moreover, prospective studies suggest that nearly all persons identified with ADHD over the lifespan could have been identified by age 12–14, based on the symptoms they showed at that time (Kieling et al., 2010).

Subtypes The three subtypes of ADHD included in DSM-5 were originally created in DSM-IV. The most common subtype, comprising about 55% of all children referred for treatment, is the *combined type* (ADHD-CT; Lahey et al., 1994). These children exhibit at least six symptoms of both inattention-disorganization and impulsivity–hyperactivity (five or more symptoms for adolescents and adults). Not surprisingly, given the prevalence rate, much of ADHD research focuses on this subtype.

The second subtype is the *predominantly inattentive type* (ADHD-PI; APA, 2013). Approximately 27% of children in treatment for ADHD exhibit this subtype (Lahey, Applegate, McBurnett, & Biederman, 1994). These children show six or more problems with inattention (five or more for adolescents and adults), but fewer than six symptoms of impulsivity–hyperactivity. Compared with those diagnosed with the combined type, children with ADHD-PI: (a) Demonstrate fewer conduct problems and less aggression; (b) experience less rejection from peers; (c) experience higher rates of anxiety and depression; and (d) respond better to lower doses of stimulant medication (Carlson & Mann, 2000; Lahey, Carlson, & Frick, 1997). This subtype often exhibits different cognitive and attentional difficulties from other children with ADHD, such as slow retrieval and information processing (as opposed to fast and impulsive processing of information) and low levels of alertness (Hartman, Willcutt, Rhee, & Pennington, 2004; McBurnett, Pfiffner, & Frick, 2001).

The third and least common ADHD subtype is the *predominantly hyperactive-impulsive type* (ADHD-PHI). Accounting for about 18% of children diagnosed with ADHD, those in this category show six or more symptoms of hyperactivity-impulsivity (five or more symptoms

for adolescents and adults) without problems of inattention-disorganization (Lahey, Applegate, Barkley et al., 1994). Barkley (1997) proposed that this subtype is a developmental precursor to the ADHD-CT subtype for children who have not yet experienced demands for sustained attention. Consistent with this hypothesis, a study of children diagnosed with ADHD found that the average age of the ADHD-PHI subgroup was 5.68 years, compared with 8.52 years for the ADHD-CT and 9.80 years for the ADHD-PI type (Lahey, Applegate, Barkley et al., 1994). Youth with ADHD-PHI whose symptoms persist over development tend to shift to ADHD-CT following entry into school, likely because of the increased demands placed on them (Lahey, Pelham, Loney, Lee, & Wilcutt, 2005). Additionally, when controlling for conduct disorders, a diagnosis of ADHD-PHI earlier in development appears to predict ODD symptoms later in development (Burke, Loeber, Lahey, & Rathouz, 2005).

There are several criticisms of this method of subtyping. The most common criticism is the instability of subtype membership across time, with most children showing subtype changes when followed longitudinally (Lahey et al., 2005; Willcutt et al., 2012). Because of these findings, DSM-5 differentiates these subtypes using specifiers for "current presentation" to more clearly indicate that they lack any longitudinal predictive validity. A second criticism is that the magnitude of differences among the subtypes, particularly between the ADHD-PI subtype and the other two subtypes, warrant designation as separate disorders (Milich, Balentine, & Lynam, 2001). A third criticism is that the subtypes should be defined by different cognitive styles rather than the degree of alignment with different symptom clusters. For example, some researchers argue that the ADHD-PI subtype should be defined by a sluggish cognitive tempo (e.g., drowsiness, lethargy, hypoactivity; Barkley, 2013).

The research has failed to find unique neuropsychological or cognitive problems associated with ADHD-PI versus ADHD-CT, however. Rather, it has found that cognitive problems accrue to ADHD-PI and ADHD-CT, but less so to ADHD-PHI (Willcutt et al., 2012). Moreover, when subtypes do differ, it has almost invariably been the case that ADHD-CT has worse cognitive problems than ADHD-PI, consistent with a simple severity heuristic. That is, by definition, ADHD-CT features more symptoms on average than ADHD-PI, so one should expect more problems in any correlated domain that is not specific to a subtype configuration. Thus, a parsimonious conclusion might be that ADHD-CT is simply a more severe condition than ADHD-PI, rather than a qualitatively different type of manifestation as reflected by fixed, stable types.

Developmental Course Research suggests that ADHD symptoms emerge early in development, with symptoms of overactivity emerging as early as 3–4 years of age (Loeber, Green, Lahey, Christ, & Frick, 1992). Early cognitive and behavioral markers of later ADHD symptomatology may be evident as early as 24 months of age (Arnett, MacDonald, & Pennington, 2013). Although many children swiftly learn to control such behavior upon entry into school, about half of the most hyperactive and disruptive preschoolers are diagnosed with ADHD by age 6 (Campbell, Ewing, Breaux, & Szumowski, 1986). This is reflected by a sharp increase in the worldwide prevalence of ADHD from ages 4 to 9 that is subsequently followed by a relative and rapid decrease across the lifespan (Erskine et al., 2013). For years the prevailing scientific view was that these children "outgrow" the disorder by adolescence or early adulthood (Lambert, 1988). This assumption has been challenged by several prospective longitudinal studies that followed children diagnosed with ADHD into adulthood (Biederman, Petty, Evans, Small, & Faraone, 2010; Faraone, Biederman, & Mick, 2006). These studies suggest that ADHD in childhood confers risk for long-term problematic outcomes, despite only 15% of those originally diagnosed with ADHD still meeting full diagnostic criteria at 25 years of age (Faraone et al., 2006).

Several general statements can be made about the current understanding of the developmental course of ADHD. First, for children diagnosed with ADHD, between 50% and 70% experience clinically significant symptoms into adolescence, and between 30% and 50% into adulthood (Langley et al., 2010; Lara et al., 2009). Childhood predictors of problem persistence include ADHD-CT subtype, symptom severity, comorbid psychopathology, high levels of academic and social impairment, and parental psychopathology including conduct disorder/antisocial personality disorder or mood disorders (Biederman et al., 2010; Langley et al., 2010; Molina et al., 2009). Second, even when core symptomatology has lessened to the point of no longer meeting diagnostic criteria, many adults diagnosed with ADHD as children continue to experience significant impairments in their social, occupational, financial, psychological, and/or neurocognitive functioning (Balint et al., 2009; Faraone et al., 2006). Third, the manifestation of ADHD symptoms varies across development. For example, the presentation of inattention-disorganization symptoms appears more consistent from childhood to adolescence and adulthood than hyperactive-impulsive symptoms (Hart et al., 1995). This may be partly due, however, to developmental variations in the expression of hyperactive-impulsive symptoms, such that they manifest as driving problems in adolescence (Barkley & Cox, 2007) or relationship instability in adulthood (Biederman et al., 2006).

Comorbidity and Co-occurring Problems Many children with ADHD present with comorbid disorders and co-occurring adjustment problems (although not always severe enough to warrant a separate diagnosis), the presence of which strongly predicts later maladjustment (Connor, Steeber, & McBurnett, 2010). As described above, ADHD overlaps substantially with conduct disorder and ODD, particularly when one emphasizes the

hyperactive/impulsive component of ADHD (Larsson, Dilshad, Lichtenstein, & Barker, 2011). Children with comorbid ADHD and conduct disorder/ODD are at heightened risk for tobacco, alcohol, and illicit drug use in adolescence compared with same-aged youth without ADHD, particularly when symptoms of ADHD and conduct disorder are persistent (Molina & Pelham, 2003; Wilens et al., 2011). Specifically, tobacco use and other substance abuses in later development are more common among ADHD than non-ADHD children, even after controlling for ODD and conduct disorder (Burke, Loeber, White, Stouthamer-Loeber, & Pardini, 2007). However, with respect to alcohol use disorders, a national Dutch survey found that conduct problems fully mediated their relationship with ADHD (Tuithof, ten Have, van den Brink, Vollebergh, & de Graaf, 2012).

The syndrome of ADHD overall, and the inattention dimension in particular, appear to overlap with developmental problems (Frick & Nigg, 2012). One study found that ADHD clustered together with autistic symptoms, motor co-ordination, and reading problems, in addition to ODD, and that sibling cross correlations loaded better for autistic, motor, and reading problems than behavior problems (Couto et al., 2009). Half of children with autism show symptoms consistent with ADHD, and about 20% of children with ADHD have sufficient social oddity to raise questions about overlap with autism (Reiersen et al., 2008). Between 30% and 50% of children with ADHD have significant academic problems such as learning disabilities and early school dropout (Jensen, Martin, & Cantwell, 1997). Compounding academic challenges, a high percentage of these children also experience social difficulties such as peer rejection (Miller-Johnson, Coie, Maumary-Gremaud, & Bierman, 2002).

Comorbid emotional disorders, such as clinical anxiety or depression, are evident in between 30% and 50% of children and adolescents diagnosed with ADHD (Jensen et al., 1997; Schatz & Rostain, 2006), and many show an anxiety disorder as an adult (Barkley & Brown, 2008). It is critical to determine whether this is due to common underlying processes (e.g., problems in emotional dysregulation) that may be manifested differently at different ages or whether anxiety and other signs of emotional distress are a secondary complication of stressors associated with ADHD. Other frequently co-occurring conditions include tic, eating, and sleep disorders (Gau et al., 2010; Yoshimasu et al., 2012). Taken together, the above findings suggest that comorbid diagnoses are the rule rather than the exception for children diagnosed with ADHD.

Several differences exist between families of children with and without ADHD. In a review of studies examining family functioning, Johnston and Mash (2001) found that having a child with ADHD in the home is associated with poorer parenting; marital and parent–child conflicts; and increased parental stress, self-blame, social isolation, and psychopathology. Such difficulties are exacerbated by a child's comorbid oppositional and conduct problems (see also Deault, 2010). It is unclear whether these familial problems precede the child's development of ADHD or vice versa—or whether the relationship is, instead, reciprocal. That is, constant household stressors and challenges involved in parenting a child with ADHD may increase the likelihood that parents will engage in dysfunctional and ineffective parenting strategies (Fischer, 1990). Some researchers suggest that familial factors are less responsible for the onset of ADHD than its maintenance (Johnston & Mash, 2001). Other researchers hypothesize that negative family dynamics more strongly predict the development and maintenance of co-occurring problems in children with ADHD (e.g., conduct problems; Langley et al., 2010; Pálmason et al., 2010).

Cultural and Gender Issues Historically, ADHD research has been dominated by researchers from the United States, leading to the impression that it is largely an American disorder. To test this assumption, Faraone, Sergeant, Gillberg, and Biederman (2003) reviewed studies reporting on the prevalence of ADHD within countries and cultures across the world according to DSM-III, DSM-III-R, or DSM-IV criteria. They concluded that ADHD prevalence was roughly equivalent in U.S. and non-U.S. study populations. Erskine and colleagues (2013) found that prevalence rates were similar between Europe and North America, while ADHD was more common in North Africa/Middle East than in North America. However, divergent rates in non-U.S. samples may reflect methodological differences in: (a) Diagnostic criteria used to establish disorder, with the highest prevalence rates reported for DSM-IV criteria (Faraone et al., 2003); (b) informants used to assess symptoms (e.g., parents vs. teachers, with overestimation when diagnoses are based on single informants); or (c) the population surveyed (e.g., age range, gender composition), rather than true geographic variations in ADHD. Within the United States, studies do not find consistent differences in prevalence rates across different cultural groups (Barkley, 1996).

Across the globe, boys are almost four times more likely than girls to be diagnosed with ADHD (Erskine et al., 2013). Male-to-female ratios are greater within clinic-referred versus community samples (Barkley, 1996). There is emerging evidence that the ratio differs across ADHD subtypes, with more equal gender ratios for the ADHD-PI presentation (Biederman et al., 2002; Lahey, Applegate, Barkley et al., 1994). Among children diagnosed with ADHD, there are no significant gender differences in symptom presentation, co-occurring adjustment problems, or clinical course (Gershon, 2002). However, some research suggests that gender differences in symptom presentation may vary as a function of age with more severe inattention among boys during childhood and among girls during adolescence (Kan et al., 2012). The most consistent gender differences are a stronger family history of ADHD for boys, particularly with the father,

and less severe hyperactivity and fewer conduct problems for girls, although they exhibit greater verbal intellectual impairment (Rucklidge & Tannock, 2001). Interestingly, comorbid psychopathology persists into adolescence more often for girls than boys (Monuteaux, Mick, Faraone, & Biederman, 2009).

Etiology The core symptoms of ADHD have been associated with a neurological deficit that, as noted above, led to its placement in DSM-5 in the chapter with other neurodevelopmental disorders. However, it is important to note that documentation of such a deficit is neither sufficient nor necessary for a diagnosis, since the diagnostic criteria for ADHD are purely behavioral. The child's psychosocial context (e.g., family environment, quality of educational services)—while not critical to many etiological theories—largely influences the severity and level of impairment of the child's symptoms and the development of co-occurring problems. As a result, intervening in the child's psychosocial context (e.g., improving parenting skills, improving the child's educational environment) to limit the impairment associated with ADHD and prevent the development of secondary problems are critical components for effectively treating children with ADHD, a point that is discussed later in this chapter.

Core Deficit There are a plethora of hypotheses about the core dysfunctions at the root of ADHD, including impulsivity (temporal discounting of reward), attention (particularly when framed as executive functioning and vigilance), variability of responding, and others. Models have emerged proposing multiple core problems, putting emphasis on the two-dimensional structure (for a review see Nigg, 2006). Over the period from 1994 to 2010, a fundamental finding was that the two core symptom dimensions worked well in that they differentially predicted types of impairment, comorbidities, and neuropsychological findings (for a review see Willcutt et al., 2012). Thus, a strong database established that ADHD was at least a two-component syndrome, including both "inattention" (including problems in behavioral organization and poor executive functioning) and hyperactivity-impulsivity (including discounting the value of delayed reward, social disinhibition and intrusiveness, and emotional dysregulation). The first dimension tends to overlap with learning problems, whereas the second dimension tends to overlap with oppositional and conduct problems.

Two extensive, well-articulated, and influential theories on the core cognitive deficit leading to ADHD symptoms are those of Barkley (1997) and Nigg (2006). These authors posit that ADHD results from a core deficit in behavioral inhibition—defined as the ability to inhibit a prepotent response long enough to consider the consequences of the response; to stop an ongoing response in reaction to environmental feedback; and to suppress stimuli that might interfere with a primary response (i.e., interference control; see also Nigg, 2006). According to Barkley (1997), this deficit in behavioral inhibition leads to secondary impairments in several executive functions—cognitive actions that are self-directed and allow for self-regulation. Executive functions depend on behavioral inhibition for their adequate performance and include: (a) Working memory (e.g., maintaining events in the mind, hindsight, forethought); (b) self-regulation of affect, motivation, and arousal (e.g., self-control of emotions, social perspective taking); (c) internalization of speech (e.g., problem-solving, moral reasoning); and (d) reconstitution (e.g., verbal and behavioral fluency).

These impairments explain a number of the self-regulation problems that are exhibited by children with ADHD. That is, the deficit in behavioral inhibition and the resulting impairments in executive functions cause the behavior of individuals with ADHD to be controlled by the immediate context rather than internal representations of information (Nigg, 2006). Characteristics of the child's immediate context, such as the novelty of the task, the intrinsic interest of the child in the activity, the child's level of fatigue, and the degree of immediate reinforcement present, can have a strong influence on the child's behavior (Barkley, 1996, 1997). These deficits also explain both the core symptoms of inattention-disorganization and hyperactivity-impulsivity as well as several secondary features, such as impaired sense of time and high emotional reactivity. Neurological deficits are theorized to be a mediating process rather than the cause behind the child's behavioral symptom profile (Nigg, 2006).

Neurological Substrates Neuroimaging studies suggest several important differences in the brains of children with ADHD. Structurally, those with ADHD show on average a 5% reduction in total brain volume, with greater reductions in the prefrontal cortices, corpus callosum, basal ganglia, and cerebellum (Giedd, Blumenthal, Molloy, & Castellanos, 2001; Swanson & Castellanos, 2002). Although the prefrontal cortex is generally one of the last areas of the brain to fully mature, it appears to mature at an even slower rate for children with ADHD (Shaw et al., 2007). This same study found that the motor cortex reaches its peak thickness slightly faster in children with ADHD than controls. Neuroimaging studies in persons with ADHD report dysfunction of prefrontal-striatal circuitry, and large-scale neural networks, including frontal-to-parietal cortical connections (Castellanos & Proal, 2012; see Nigg, 2006). These findings are strongest for children with ADHD-CT (Loo & Barkley, 2005). Importantly, the implicated brain areas have been linked with deficits in emotion regulation, inhibition, and executive functions and, thus, could explain the core deficits described previously (Barkley, 1997; Nigg, 2006). Stimulant medication, which is effective at reducing symptoms of ADHD, also increases activity in the prefrontal regions of the brain, thus providing a rationale for the method of action of this

medication in reducing core symptoms (Lou, Henrikson, Bruhn, Borner, & Nielson, 1989).

Despite these promising findings, research on the neurological underpinnings of ADHD is limited in several ways. First, as in earlier research, it is still evident that many persons with ADHD do not show clear deficits in brain structure or function. Second, findings are not always consistent across studies. For example, some studies find more defuse abnormalities in cerebral blood flow that is not limited to prefrontal regions of the brain in adults with ADHD (Zametkin et al., 1990); other studies find that diminished blood flow is limited to adolescent girls, and not boys, with ADHD (Ernst et al., 1994; Zametkin et al., 1993). However, some of these inconsistencies may reflect differences in the sample studied (e.g., variations in which subtypes and comorbid conditions are included). Few neurobiological studies compare subtypes; however, one large study found atypical functional connectivity across multiple brain systems in children with ADHD-CT, but in the bilateral dorsolateral prefrontal cortex regions and the cerebellum specifically for ADHD-PI (Fair et al., 2012). Third, these studies do not indicate what causes the neurological deficits.

Causes of the Neurological Abnormalities There are several different pathways through which a neurological deficit can develop in the brain systems involved in inhibition. One of these pathways may be via an inherited deficit. Family and adoption studies find evidence for the familial transmission of ADHD symptoms (see Faraone & Biederman, 2000; Sprich, Biederman, Crawford, Mundy, & Faraone, 2000). A review of twin studies estimated that approximately 76% of the variance in measures of ADHD can be attributed to genetic influences, making it one of the most heritable forms of childhood psychopathology (Faraone et al., 2005). Familial transmission of ADHD may be subtype specific. A meta-analysis of family inheritance studies, found that relatives of children with ADHD-PI tended to have both ADHD-PI and ADHD-CT in greater than expected rates, whereas relatives of children with ADHD-CT had only ADHD-CT at elevated rates, suggesting that ADHD-PI includes two distinct genetic types (Stawicki, Nigg, & von Eye, 2006).

Genetic research suggests a polygenetic contribution to susceptibility to ADHD. Molecular genetics studies implicate several viable candidate genes, each of which may have a small effect in increasing the risk for developing ADHD (Faraone & Mick, 2010). Other research finds evidence for an increased rate of large, rare submicroscopic chromosomal structural abnormalities, often referred to as copy number variants, among children with ADHD, after controlling for intellectual disability, schizophrenia, and autism (Williams et al., 2010). To date, genetic association studies have not identified any genes definitely conferring major risk for ADHD, consistent with the idea that the genetic vulnerability to ADHD is mediated by many genes of small effect (Neale et al., 2010).

The influence of various genes may differ according to the subtype of ADHD, gender, and comorbidity, explaining some of the inconsistencies in molecular genetic findings. For example, the dopamine active transporter 1 gene (DAT1) may be specifically associated with ADHD without comorbid conduct problems (Sharp, McQuillin, & Gurling, 2009). Other research suggests that common genes may confer risk for ADHD as well as other psychiatric disorders. One study found significant overlap between genetic factors for ADHD and major depressive disorder, interpreted as reflecting a common genetic pathway to both disorders (Cole, Ball, Martin, Scourfield, & McGuffin, 2009).

Environmental factors may also play a role in the development of ADHD (Benerjee, Middleton, & Faraone, 2007). There is evidence that biological trauma may influence the development of ADHD in some children. For example, children with ADHD are more likely to have been born premature, with obstetric complications, and with low birth weight relative to children without ADHD (Johnson et al., 2010). Additionally, prenatal exposure to environmental toxins, such as alcohol and tobacco, lead, manganese, mercury, and polychlorinated biphenyls are linked to ADHD symptoms (Benerjee et al., 2007; Stein, Schettler, Wallinga, & Valenti, 2002). At least some research suggests that associations (e.g., for maternal smoking during pregnancy) may be attributed to unmeasured confounding familial factors (Skoglund, Chen, Lichtenstein, & Larsson, 2014). Genetic vulnerabilities may also interact with environmental factors to predict ADHD symptomatology (Neuman et al., 2007). For example, Lahey and colleagues (2011) found that 4–6-year-old children meeting diagnostic criteria for ADHD, who were exposed to more negative and less positive parenting practices in early childhood, showed greater conduct disorder symptoms several years later when they possessed a certain polymorphism of the dopamine transporter gene, compared with children without this polymorphism. Like other forms of psychopathology, there probably are multiple developmental pathways to ADHD and related neurological deficits (Nigg, 2006).

Empirically Validated Treatments Many treatment approaches have been attempted with ADHD, most of which have gained only limited support for their efficacy (Barkley, 2006). Only two treatments consistently demonstrate efficacy in controlled treatment outcomes studies. These evidence-based treatments include pharmacological intervention and behavioral therapy focusing on developing structured contingency management systems within the child's home and school environments (Pelham & Fabiano, 2008).

The use of stimulant medication as a pharmacological intervention has robust support in the literature with

over 30 years of research indicating that it leads to significant improvements in ADHD symptoms in 70–80% of children (see Swanson, McBurnett, Christian, & Wigal, 1995, for a review). Stimulant medication not only reduces the core symptoms of ADHD but also alleviates many secondary problems, such as conduct problems, aggression, social and academic problems (Richters et al., 1995; Swanson et al., 1995). With appropriate and carefully monitored trials, the adverse effects of stimulant medication are fairly mild. The most common adverse effects are appetite suppression and delayed onset of sleep (Feldman & Reiff, 2014). Although much of the treatment research has been conducted with preadolescent children with ADHD, there is growing support for the efficacy of stimulant medication for reducing symptoms of ADHD in preschool children (Greenhill et al., 2006) and adults (Dowson, 2006). Only a small proportion of affected adults are actually treated, however (Biederman, Spencer, & Wilens, 2004).

The majority of research on pharmacological intervention focuses on one particular stimulant medication, methylphenidate (Ritalin®). However, controlled treatment outcome studies find similar response profiles for other stimulant medications. Also, while stimulants are the pharmacological evidence-based treatments for ADHD, a number of non-stimulant medications have also been used, including noradrenergic reuptake inhibitors, tricyclic antidepressants, and antihypertensive agents (Biederman et al., 2004). The limited research on these non-stimulant medications suggests that they are superior to placebo in reducing symptoms of ADHD, although it is unclear whether they are superior in efficacy to the more extensively researched stimulant medications (Biederman et al., 2004). Nevertheless, when parents oppose treatment with stimulants, when stimulants are contraindicated or have adverse effects, or when there is a high likelihood or history of addiction or recreational use of medication, non-stimulant medications may be an important alternative for managing ADHD symptoms.

Despite robust findings for the efficacy of medication (Swanson et al., 1995), pharmacological interventions for children with ADHD are limited in several important ways. First, many parents report negative feelings about the use of medications, reflected in poor and inconsistent adherence to treatment regimens or even refusal of this form of treatment (Pappadopulos et al., 2009; Richters et al., 1995). Second, a considerable proportion (10–20%) of children with ADHD are left with no significant improvement in symptoms with pharmacological intervention and, even for those who do show improvement, their behavior is often not brought within what would be considered a normal range (Pelham, 1993; Wells et al., 2000). Third, and most importantly, stimulants are most beneficial in the short-term, and there is little evidence that they have a long-term benefit for improving outcomes in adolescence or adulthood or once they are discontinued (Pelham et al., 2000; Swanson et al., 1995). Considering that the largest controlled treatment study to date found that only a minority of youth (38%) continued taking medication post-treatment and into adolescence (Molina et al., 2009), symptoms will persist unless other intervention approaches are considered.

Several studies have reported superior performance for pharmacological intervention over behavioral interventions (Abikoff et al., 2004; Hinshaw, Klein, & Abikoff, 2007). These findings have had a significant influence on some guidelines for the treatment of ADHD and the preference for pharmacological treatment over behavioral interventions (Leslie & Wolraich, 2007). For example, the American Academy of Child and Adolescent Psychiatry (2007) promotes pharmacological treatment as the initial and only intervention for ADHD, except when symptoms are mild and minimally impairing or when parents reject medication. Preliminary research, however, suggests that when behavioral interventions are used first they may be effective at treating a majority of children with ADHD, and that most children first treated with stimulant medication will require an additional behavioral intervention (Dopfner et al., 2004). As a result, the American Academy of Pediatrics (2011) promotes behavior therapy as the first line of treatment for ADHD in preschool children, followed by pharmacological intervention if symptoms do not significantly improve.

Of the psychosocial interventions available for the treatment of ADHD, behavioral parent training and behavioral classroom management have garnered the most empirical support (Pelham & Fabiano, 2008). Behavioral parent training involves teaching parents intensive behavior management skills and related concepts (e.g., effective commands, token economies in the home, effective discipline) and may also include teaching parents strategies for working effectively with teachers and advocating for their child with school personnel, as well as methods for managing stress, anger, and negative moods to enhance parenting abilities. Behavioral classroom management involves training teachers on how to establish behavioral classroom management techniques (e.g., daily report cards, classroom token economies). Intensive peer-focused behavioral interventions that are typically implemented in recreational settings, such as in summer camp programs (e.g., MTA Cooperative Group, 1999a, 1999b), have also been identified as a well-established treatment (Pelham & Fabiano, 2008). It is also common for studies to combine these behavioral interventions to create more comprehensive and individualized behavioral management programs since interventions will only be effective in those settings to which they are applied.

The most prominent and widely cited treatment study is the Collaborative Multimodal Treatment Study of Children with ADHD, which was a longitudinal multisite treatment study of 579 children aged 7–9.9 years diagnosed with ADHD-CT (excluding ADHD-PI and ADHD-HI) funded by the National Institute of Mental

Health. The controlled treatment trial compared the efficacy of pharmacological and behavioral interventions across four randomly assigned treatment groups: (1) Psychosocial treatment only; (2) stimulant medication only; (3) combined psychosocial and stimulant medication treatment; and (4) standard community treatment (being whatever treatment was typically delivered in that specific community). At the end of the 14-month intervention period, all groups showed improvement relative to the baseline assessment that was maintained 8 years post-treatment. However, youth with ADHD continued to function more poorly than their non-ADHD counterparts across development, despite intensive treatment (Molina et al., 2009). Medication and combined treatment groups initially showed greater reduction in ADHD and ODD symptoms and greater improvement in overall adjustment (Conners et al., 2001; Swanson et al., 2001). Combined treatment was also found to be more effective for children with coexisting anxiety problems (Jensen et al., 2001; March et al., 2000) and children of ethnic minority backgrounds (Arnold et al., 2003). However, any differences in treatment efficacy dissipated by 22 months post-treatment (Jensen et al., 2007). Instead, the severity of the child's clinical presentation of ADHD symptoms in the first 3 years of the study, and not treatment group assignment, best predicted outcomes 8 years post-treatment (Molina et al., 2009).

Summary of Treatment for ADHD The intervention approach used in the Multimodal Treatment of Attention Deficit Hyperactivity Disorder (MTA) study provides an excellent example of applying evidence-based practices for treating children with ADHD (although see Barkley, 2000; Greene & Ablon, 2001; Pelham, 1999, for critiques of the intervention). Despite limited findings for the long-term efficacy of empirically supported treatment, the initial findings of this large-scale treatment trial, combined with findings from other treatment studies, support the efficacy of both stimulant medication and behavioral interventions. That is, pharmacological and behavioral interventions are currently the state-of-the-art in treating children with ADHD, with some support for combining them to optimize the effects for some children (Pelham et al., 2000). This is reflected in the clinical practice guidelines of the American Academy of Pediatrics (2011), which recommends combined treatment for school-aged children and adolescents. Pharmacological intervention may be superior to behavioral intervention for treating the behavioral symptoms described in the DSM, while behavioral treatments may be more effective at treating general functioning and adaptive skills (Pelham & Fabiano, 2008; Wells et al., 2006). There is mixed support for the greater cost-effectiveness of one form of intervention over the other (Jensen et al., 2005; Pelham, Foster, & Robb, 2007). At the least, early intervention is critical for preventing the later development of defiant, aggressive, and rule-breaking behaviors (e.g., McCain & Kelley, 1993; McGoey & DuPaul, 2000).

Unfortunately, despite the well-documented efficacy of these two treatment modalities, a proliferation of unproven therapies with limited evidence for their effectiveness continues. These have ranged from biofeedback programs to specialized diets (Lilienfeld, 2005; Loo & Barkley, 2005; Waschbusch & Hill, 2003). The popularity of these unproven approaches to intervention is likely, owing to strong biases against the two evidence-based treatments. Many parents, and even many professionals, have strong negative feelings about the use of medication for controlling children's behavioral problems. Further, most parents report having tried a system of rewards and punishments in an effort to control their children's behavior, albeit not at the level of consistency and intensity needed to bring about meaningful changes in the child's behavior. As a result, they are often reluctant to agree to interventions focusing on behavior management strategies. These biases often lead to the unfortunate outcome of many children with ADHD not receiving the optimal treatment: a combination of a carefully controlled trial of stimulant medication and an intensive behavior management program.

Oppositional Defiant Disorder and Conduct Disorder

Definition and Description

Diagnosis Conduct problems are the most common reason for referral of children and adolescents to outpatient mental health clinics and residential treatment centers (Frick & Silverthorn, 2001). The worldwide prevalence is estimated at 3.3% for ODD and 3.2% for conduct disorder, using DSM or ICD diagnostic criteria (Canino, Polanczyk, Bauermeister, Rohde, & Frick, 2010). Much research has focused on understanding and treating this externalizing psychopathology, particularly as severe conduct problems often lead to delinquency and violence (Broidy et al., 2003; Moffitt, 2003). The potential value of saving a single high-risk youth from a criminal career is estimated to range between $3.2 and $5.5 million (Cohen & Piquero, 2009).

Factor-analytic research supports the distinction between the angry and defiant behaviors forming the diagnostic criteria for ODD and the antisocial and aggressive behaviors forming the criteria for conduct disorder (Lahey et al., 2008). However, all disorders within the externalizing domain appear to share substantial genetic influences, suggesting at least some common causal factors between them (Lahey et al., 2011). The current distinction between ODD and conduct disorder that is specified in DSM-5 was first introduced in DSM-III (American Psychiatric Association, 1980). In DSM-5, ODD and conduct disorder are subsumed under the rubric of the disruptive, impulse control, and conduct disorders, along with intermittent explosive disorder, pyromania, and kleptomania.

Disorders in this category involve problems in the self-control of emotions and behaviors. Whereas many other psychiatric disorders may also involve problems in emotional and/or behavioral regulation, these disruptive disorders are unique in that the problems associated with them are manifested in behaviors that violate the rights of others (e.g., aggression, destruction of property) and/or that bring the individual into significant conflict with societal norms or authority figures (American Psychiatric Association, 2013).

ODD is defined as:

- a recurrent pattern of angry, irritable, argumentative, defiant, or vindictive behavior;
- that persists for at least 6 months; and
- is characterized by the frequent occurrence of at least four of the following behaviors: Losing temper, being touchy or easily annoyed, being angry and resentful, arguing with authority figures, actively defying or refusing to comply with requests or rules of adults, deliberately annoying others, blaming others for his or her own mistakes or misbehavior, or being spiteful or vindictive (American Psychiatric Association, 2013).

ODD symptoms are organized into three clusters in DSM-5: angry/irritable mood (e.g., loses temper, angry/resentful), argumentative/defiant behavior (e.g., argues with adults, defiant/noncompliant), and vindictiveness. This organization was heavily influenced by factor-analytic studies converging on a three-dimensional conceptualization of the ODD criteria (Burke, 2012; Rowe, Costello, Angold, Copeland, & Maughan, 2010). Although item loadings vary somewhat across samples, the angry–irritable mood dimension forms a separate factor from the defiant–headstrong behavior dimension. What is less clear from these analyses is the appropriate placement of the spiteful/vindictive symptom, which has been included on its own to comprise the hurtful dimension since it does not consistently load with the other two symptom dimensions and may be more related to the severe conduct problems of conduct disorder (Rowe et al., 2010; Stingaris & Goodman, 2009). Despite these findings of separate factors, it is also important to note that the three dimensions are highly correlated, with *r* ranging from .62 to .78, suggesting that a large number of youth scoring high on one dimension would also score high on another (Stingaris & Goodman, 2009).

Conduct disorder is defined as:

- a repetitive and persistent pattern of behavior which violates the rights of others or major age appropriate societal norms or rules;
- these behaviors fall into four main groupings: aggressive conduct that threatens physical harm to other people and animals, nonaggressive conduct that causes property loss or damage, deceitfulness or theft, and serious violations of rules; and
- three or more characteristic behaviors must have been present during the past 12 months, with at least one behavior present in the past 6 months (American Psychiatric Association, 2013).

Developmental Course Considerable evidence suggests that ODD often precedes the development of conduct disorder in children (e.g., Burke, Waldman, & Lahey, 2010). As a result, many researchers consider ODD and conduct disorder to be age-related manifestations of a common syndrome, with conduct disorder representing a more severe developmental progression of disruptive behavior. ODD typically begins between the ages of 3 and 8, and, for many of these children, the behaviors gradually escalate into increasingly frequent and severe patterns of conduct problems (Shaw, Gilliom, Ingoldsby, & Nagin, 2003). Youth who do progress from ODD to conduct disorder do not *change* the types of behaviors they display but instead *add* the more severe conduct problem behaviors to their existing behavioral repertoire (Kim-Cohen et al., 2009) and, typically, ODD symptoms are retained (Loeber, Lahey, & Thomas, 1991).This is reflected by a sharp increase in conduct disorder prevalence rates from 4 years of age that continues to 18 years of age (Erskine et al., 2013). Beauchaine, Hinshaw, and Pang (2010) propose that the typical developmental course for delinquent behavior in boys begins with severe hyperactive–impulsive behaviors in early childhood, followed by ODD at preschool age, childhood-onset conduct disorder at elementary school age, substance-related disorders in adolescence, and antisocial personality disorder in adulthood. It must be noted, however, that approximately 40% of children with ODD do not later exhibit more severe conduct problems (Shaw et al., 2003).

Conduct problems are relatively stable across development (Broidy et al., 2003; Shaw et al., 2003). In one study, approximately half of children diagnosed with conduct disorder at an initial assessment were rediagnosed with the disorder at a follow-up assessment up to 5 years later (Frick & Loney, 1999). Severe conduct problems that persist into adulthood are often diagnosed as antisocial personality disorder. Antisocial personality disorder is diagnosed when an individual age 18 or older displays a chronic pattern of recklessness, impulsivity and irresponsibility, deceitfulness, criminal behavior, and lack of remorse. Robins (1966) found that 31% of boys and 17% of girls referred to a mental health clinic for conduct disorder symptoms were diagnosed with an antisocial disorder as adults. In addition, 43% of the boys with conduct disorder and 12% of girls were later imprisoned at least once as an adult. Further, antisocial personality disorder in adulthood is more common among children with co-occurring of conduct disorder and ADHD (Hofvander, Ossowski, Lundström, S., & Anckarsäter, 2009).

Many children with early-starting conduct problems remit from these behaviors over the course of development

(Raine et al., 2005; Robins, 1966), particularly those diagnosed as preschoolers (Kim-Cohen et al., 2009). However, even when conduct disorder symptoms remit, these children continue to show adjustment problems later in development (Kim-Cohen et al., 2009). Although they have received less empirical attention, factors related to desistance from antisocial behavior include decreased exposure to risk factors known to contribute to conduct problems and increased exposure to positive factors that protect against them (Loeber, Pardini, Stouthamer-Loeber, & Raine, 2007). Positive factors most often identified as contributing to desistance are normative developmental processes such as increased physical and mental maturity (Glueck & Glueck, 1940), identity formation (Neugarten, B. L., & Neugarten, D. A., 1996), formation of social controls, such as getting a job or developing bonds with others, including romantic relationships (Laub & Sampson, 2001; Moffitt, 1993), and development of internal controls that deter criminal involvement, such as greater perceived likelihood of detection and sanctions (Loeber et al., 2007; Mulvey et al., 2004; Stouthamer-Loeber, Wei, Loeber, & Masten, 2004).

Co-occurring Problems Conduct problems co-occur with several psychiatric disorders other than ADHD. ODD in particular is frequently comorbid with a host of other disorders, including emotional disorders (Biederman, Petty, Dolan et al., 2008; Biederman, Petty, Monuteaux et al., 2008; Burke, Waldman, & Lahey, 2010; Nock, Kazdin, Hiripi, & Kessler, 2007). For example, 10–20% of children with ODD develop internalizing disorders as preschoolers, with somewhat higher rates for older youth (among community samples, 15–46% present with comorbid major depression and 7–14% present with a comorbid anxiety disorder), particularly those with persistent ODD (Boylan, Vaillancourt, Boyle, & Szatmari, 2007). Research indicates that it is the angry–irritable dimension of ODD that is most strongly related to emotional disorders, whereas the defiant–headstrong dimension is related to ADHD, and the spiteful–vindictive symptom is related to conduct disorder (see also Drabick & Gadow, 2012; Ezpeleta, Granero, de la Osa, Penelo, & Domènech, 2012). Similar divergent predictions from the different ODD dimensions have been found longitudinally, with most studies reporting that all three dimensions predict risk for later conduct disorder, but that only the angry–irritable dimension also predicts risk for later emotional disorders (Burke, Hipwell, & Loeber, 2010; Rowe et al., 2010; Stingaris & Goodman, 2009).

Conduct disorder also often co-occurs with anxiety and mood disorders (Boylan et al., 2007). It is estimated that one-third of children in the community and three-quarters of those who are clinic-referred with conduct disorder meet diagnostic criteria for a comorbid depressive and/or anxiety disorder (Russo & Beidel, 1994; Zoccolillo, 1993). The development of internalizing problems, particularly depression, among youth with conduct problems has been attributed to their frequent interpersonal conflicts (e.g., with parents, peers, teachers, and police) and to other stressors (e.g., family dysfunction, school failure) that often result from the youth's problematic behavior (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999). Much of the overlap between conduct disorder and internalizing problems may be attributed to co-occurring ODD (Loeber, Burke, & Pardini, 2009). Specifically, the presence of angry–irritable ODD symptoms may help to designate a group of children with conduct disorder who have problems with emotional regulation (Frick & Morris, 2004; Lahey & Waldman, 2003), which may place them at particular risk of developing emotional disorders (Burke, Hipwell, & Loeber, 2010; Drabick & Gadow, 2012; Ezpeleta et al., 2012; Rowe et al., 2010). Much of the overlap between conduct disorder, social skills problems, peer rejection, academic problems, and depression or anxiety has been attributed to co-occurring ODD (Burke et al., 2005; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004) or ADHD (Fergusson & Horwood, 1995; Miller-Johnson et al., 2002).

Subtypes of Conduct Disorders Much of the stability in conduct disorders found in large samples is accounted for by a small group of children who show particularly severe and stable conduct problems (Broidy et al., 2003; Moffitt, 2003). There have been many attempts to uncover characteristics that distinguish between more severe and chronic forms of conduct problems and more benign and transient forms. Similar to ADHD, one of the more consistent predictors of poor outcome is the initial severity of the disorder. The frequency and intensity of the behavior exhibited, the variety of different types of symptoms displayed, and the presence of symptoms in more than one setting are all linked with a more severe and persistent form of conduct disorder (Loeber, 1991). Children with comorbid ADHD and intellectual deficits also have worse outcomes than children with either disorder alone (Farrington, 1991; Waschbusch, 2002). Family dysfunction and socioeconomic adversity have also been linked with poorer outcomes for children with conduct problems (Frick & Loney, 1999; Moffitt, 1990).

One of the most consistent predictors of poor outcome is the age at which the child begins to show serious conduct problems (see Frick & Loney, 1999; Moffitt, 1990, for reviews). For example, a prospective study of the adult outcomes of a birth cohort in New Zealand compared two groups of adults who had severe conduct problems as youth (Moffitt, Caspi, Harrington, & Milne, 2002). One group who began showing serious problems prior to puberty made up only 10% of the birth cohort but accounted for 43% of violent convictions, 40% of the drug convictions, and 62% of the convictions for violence against women in the sample. The second group whose conduct problems emerged in adolescence (26% of the

sample) showed less temperamental and psychosocial adversity than the childhood-onset group (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Moffitt et al., 2002). The remaining youth showed antisocial behaviors that were "childhood-limited" (8%), normative level relative to the full cohort (51%), or showed no antisocial behavior across childhood (5%, called "abstainers"). The adolescent-onset youth were also 50% to 60% less likely to be convicted of an offense than the childhood-onset group and their offenses tended to be less serious (e.g., minor theft, public drunkenness) and less violent (e.g., accounting for 50% of the convictions for property offences). These findings have led DSM-5 to distinguish between childhood-onset and adolescent-onset subtypes of conduct disorder, depending on whether severe behavior problems begin before or after the age of 10.

One problem with this subtyping approach is that there appear to be important distinctions that can be made within the childhood-onset group. First, not all youth with childhood-onset conduct disorder continue to show problems into adulthood; at least some show problems that are limited to childhood (Odgers et al., 2008; Tremblay, 2003). For example, in a birth cohort of children in New Zealand, 24.3% of the sample showed serious conduct problems that were limited to childhood, and these children were found to experience few physical or mental health problems as adults, with the possible exception of internalizing problems among men in middle adulthood (Odgers et al., 2008). Second, although the childhood-onset group generally tends to show more dispositional risk factors than the adolescent-onset group, the type of dispositional risk factors may vary for subgroups of children and adolescents within the childhood-onset group (Frick & Viding, 2009). This latter finding has led to research exploring methods for distinguishing subgroups within the broader category of childhood-onset conduct disorder.

More recent research considers the presence of a "callous" (e.g., lack of empathy, absence of guilt, uncaring attitudes) and "unemotional" (e.g., shallow or deficient emotional responses) interpersonal style to designate a distinct group of children with severe and chronic conduct problems and aggressive behaviors (Frick, Ray, Thornton, & Kahn, 2014). This approach is based on a long history of clinical research showing that psychopathic traits characterize an important subgroup of antisocial adults (Cleckley, 1941; Hare, 1993; Lykken, 1995). A significant body of research has emerged refining how the key features associated with psychopathy may be expressed in children and adolescents, and demonstrating the clinical and etiological importance of using these features to define a distinct subgroup of antisocial youth. Callous–unemotional traits are more common in the childhood-onset relative to the adolescent-onset group (Moffitt et al., 2002) and are present in 20–50% of children with conduct disorder (Kahn, Youngstrom, Findling, & Youngstrom, 2012; Pardini, Stepp, Hipwell, Stouthamer-Loeber, & Loeber, R.2012).

Research has revealed several other emotional, cognitive, personality, and social differences between antisocial youth with and without callous–unemotional traits (Frick et al., 2014). For example, clinic-referred children with conduct problems and callous–unemotional traits show a greater number and variety of conduct problems, more police contacts, and stronger family histories of antisocial personality disorder than their non-callous–unemotional counterparts (Christian et al., 1997). The utility of callous–unemotional traits for predicting a more severe pattern of conduct problems has been found in samples of preschool children (Kimonis, Frick, Boris et al., 2006), elementary school-aged children (Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005), and adolescents (Kruh, Frick, & Clements, 2005). It has also been found for both boys and girls (Marsee, Silverthorn, & Frick, 2005). Callous–unemotional traits have been associated with severity of antisocial behavior in several different countries such as Australia (Dadds, Fraser, Frost, & Hawes, 2005), the United Kingdom (Moran, Ford, Butler, & Goodman, 2008; Viding, Simmonds, Petrides, & Frederickson, 2009), Belgium (Roose, Bijttebier, Decoene, Claes, & Frick, 2010), Germany (Essau, Sasagawa, & Frick, 2006), Cyprus (Fanti, Frick, & Georgiou, 2009), and Israel (Somech & Elizur, 2009). Importantly, research has also shown that youth with callous–unemotional traits respond more poorly to traditional treatment approaches compared with other antisocial youth (Hawes & Dadds, 2005; Waschbusch, Carrey, Willoughby, King, & Andrade, 2007).

The importance of recognizing the utility of callous–unemotional traits for defining a distinct subgroup of youth is reflected by the incorporation of these traits into the diagnosis of conduct disorder in DSM-5. DSM-5 includes a specifier to conduct disorder for children "with limited prosocial emotions" (American Psychiatric Association, 2013). Specifically, for children who meet criteria for conduct disorder, the specifier would be given if the child persistently shows two or more of the following characteristics over at least 12 months and in multiple relationships and settings:

- lack of remorse or guilt;
- callous—lack of empathy;
- lack of concern about performance (at school, at work, or in other important activities);
- shallow or deficient affect.

One of the driving concerns that led to referring to this constellation of traits as "with limited prosocial emotions" was the pejorative connotation associated with the term "callous–unemotional" (see Frick & Nigg, 2012). Although there is no research directly testing the effects of the label "callous–unemotional traits," there is research on the negative effects of the use of the term "psychopathy" when applied to children and adolescents (for a review, see Murrie, Boccaccini, McCoy, & Cornell, 2007). The

findings indicate that the term "psychopathy" does affect the decisions made by professionals (e.g., clinicians' estimation of treatability), but it does not have any more negative effects than using the term "conduct disorder" itself. Thus it appears that any term used to describe individuals with antisocial behavior or traits will acquire negative connotations.

Cultural and Gender Issues The evidence for cultural variations in ODD and conduct disorder is mixed. Two meta-analytic studies examining the worldwide prevalence of conduct disorders found no variation between geographical regions of the United States, as a marker for culture (Canino et al., 2010; Erskine et al., 2013). There is evidence that aggressive and violent behavior varies in rate across cultures. For example, the United States has 4–73 times the rates of violence found in other industrialized nations (Fingerhut & Kleinman, 1990). This high rate of violence in the United States has been linked to various factors, such as greater exposure in childhood to violence within the home, neighborhood, and through media portrayals (Anderson et al., 2010; Huesmann, Moise-Titus, Podolski, & Eron, 2003), cultural glorification of violence, and the availability of handguns (O'Donnell, 1995).

Within the United States, higher rates of conduct problems in African American youth have been found in some samples (Fabrega, Ulrich, & Mezzich, 1993) but not in others (McCoy, Frick, Loney, & Ellis, 2000). Also, lower rates of conduct disorder have been reported for Americans of Asian descent than for European Americans and African Americans (Compton, Conway, Stinson, Colliver, & Grant, 2005). With the growing immigrant populations in the United States, risk for conduct disorder appears to vary according to migration status and level of exposure to American culture. For example, comparing different immigrant groups, one study found that the risk for developing conduct disorder was more than seven times higher among Mexican American children of U.S.-born parents, four times higher among Mexican-born immigrants raised in the United States (odds ratio = 4.12) and half as likely among the general population of Mexico (Breslau, Saito, Tancredi, Nock, & Gilman, 2012). It is unclear whether any association between minority status and conduct problems is independent of the fact that some ethnic minorities are more likely to experience economic hardships and live in urban neighborhoods with higher concentrations of crime than nonminority individuals (Lahey, Waldman, & McBurnett, 1999).

Although there do not appear to be clear differences in conduct problems across countries, there are clear sex differences, with most studies showing that boys are more likely to show conduct problems than girls (see Silverthorn & Frick, 1999, for a review). The worldwide prevalence of conduct disorder is 3.6% for boys and 1.5% for girls (Erskine et al., 2013), although gender differences vary somewhat across development. For example, for preschool children, gender differences are small and sometimes non-existent (Keenan & Shaw, 1997), whereas throughout childhood there is a male:female ratio of about 4:1 (Silverthorn & Frick, 1999). This ratio closes to about 2:1 in adolescence when both boys and girls show a dramatic increase in rates of ODD and conduct disorder (Loeber, Burke, Lahey, Winters, & Zera, 2000). It is unclear whether developmental changes in prevalence rates are real differences or an artifact of diagnostic criteria that are not sensitive to sex differences in the expression of conduct problems. For example, some argue that girls are less often diagnosed with severe conduct problems than boys because they manifest more indirect or relational aggression (i.e., spreading rumors, hurting others in the context of a relationship) than physical aggression (Crick & Grotpeter, 1995; Underwood, 2003). Others argue that girls manifest similar types of behaviors as boys, but that they should be diagnosed using more lenient criteria that compares girls to other girls rather than to mixed samples of girls and boys (Zoccolillo, 1993). Still others argue that girls are less likely to experience the necessary pathogenic processes (e.g., impulsivity; deficits in conscience) that can lead to the development of antisocial behavior (Moffitt & Caspi, 2001; Silverthorn & Frick, 1999).

Boys and girls show similar risk factors for their conduct problems (Fergusson & Horwood, 2002; Lahey et al., 2000), although there has been significantly less research on the applicability of conduct disorder subtypes to girls. Consistently, childhood-onset conduct problems are less common in girls than boys. For example, in an entire birth cohort of New Zealand children, only six girls with childhood-onset conduct disorder were identified compared to larger groups of adolescent-onset girls ($N = 78$), childhood-onset boys ($N = 47$), and adolescent-onset boys ($N = 122$) (Moffitt & Caspi, 2001). Similarly, in an adjudicated sample of adolescent boys and girls, an almost equal number of boys had a childhood-onset (46%) or adolescent-onset (54%) of severe antisocial behavior, whereas 94% of girls had an adolescent-onset to their antisocial behavior (Silverthorn, Frick, & Reynolds, 2001).

Despite the predominance of the adolescent-onset subtype in antisocial girls, girls show a large number of dispositional and contextual risk factors for their severe conduct problems, similar to childhood-onset boys. They also show poor outcomes in adulthood, such as high rates of criminality, violence, antisocial personality, and other psychiatric disorders (Zoccolillo, 1993). To explain these findings, Silverthorn and Frick (1999) proposed a delayed-onset pathway to serious conduct problems in girls. That is, girls who develop serious conduct problems in adolescence often show many risk factors that may have been present in childhood and predate their behavioral problems. However, the onset of conduct problems may be delayed until adolescence, coinciding with biological (e.g., hormonal changes associated with puberty) and psychosocial (e.g., less parental monitoring and supervision; greater contact with deviant peers) changes that

encourage antisocial behavior in girls with predisposing vulnerabilities (e.g., callous–unemotional traits; problems in emotional regulation).

In an initial test of this theory, adjudicated adolescent girls with an adolescent onset to their antisocial behavior also showed high levels of callous–unemotional traits and problems with impulse control which was more similar to childhood-onset boys than to adolescent-onset boys (Silverthorn et al., 2001). Despite this initial positive finding, additional tests of this model have been more mixed (Lahey et al., 2006; Odgers et al., 2008; White & Piquero, 2004).

Etiology: Developmental Pathways to Severe Conduct Problems A large number of factors have been identified that increase a child's risk for developing conduct problems. These range from individual risk factors (e.g., poor impulse control, poor emotional regulation, low intelligence, lack of social skills), to problems in the child's immediate psychosocial context (e.g., exposure to toxins, poverty, parental psychopathology, inadequate parental discipline, association with a deviant peer group, parental prenatal smoking), to problems in the child's broader psychosocial context (e.g., living in a high-crime neighborhood, exposure to violence, lack of educational and vocational opportunities; see Dodge & Pettit, 2003; Frick & Viding, 2009, for reviews). It has been difficult to weave this broad array of risk factors into a coherent, yet comprehensive, causal model for conduct problems both because of the sheer number of factors and because they involve so many different types of causal processes. Risk factors are also typically not independent of each other and likely operate in a transactional (e.g., one risk factor having an influence on another risk factor) or multiplicative fashion (Dodge & Pettit, 2003). For example, Jaffee and colleagues (2005) found that childhood-onset conduct problems were more common in children with a genetic vulnerability to problem behavior who were also exposed to maltreatment. Altogether, this body of research suggests that youth with conduct problems are a highly heterogeneous group, for whom there are multiple developmental pathways (Frick & Viding, 2009; Moffitt, 2003; see Zeman & Suveg, Chapter 2 in this volume, for a more general discussion of developmental psychopathology).

As a result, the development of conduct problems for any given child is likely the result of a number of different risk factors that interact to increase that child's risk level. Another complicating factor is the possibility that the causal mechanisms leading to conduct problems differ across subgroups of youth exhibiting antisocial behavior. For example, the distinction between childhood- and adolescent-onset conduct disorder not only has important predictive utility, as mentioned previously, but the two groups also show different correlates that could suggest divergent etiologies. Childhood-onset conduct disorder is linked with a host of co-occurring problems, including aggression, cognitive and neuropsychological disturbances (e.g., executive functioning deficits, autonomic nervous system irregularities), impulsivity, social alienation, and more dysfunctional family backgrounds compared with adolescent-onset conduct disorder (Dandreaux & Frick, 2009; Moffitt, 2006), although, as noted previously, these findings may not be as consistent for girls. There also appears to be a strong genetic contribution to the childhood-onset subtype of conduct disorder (Moffitt, 2003, 2006). For youth with conduct problems and comorbid disorders (e.g., ADHD), research suggests that common genetic factors often explain much of the comorbidity (Bornovalova, Hicks, Iacono, & McGue, 2010).

A greater emphasis is placed on social causes for the development of the adolescent-onset subtype (Moffitt, 2003, 2006). The onset and course of this type of conduct disorder appears highly influenced by associations with delinquent peers. Youth with adolescent-onset conduct disorder show fewer risk factors, although they tend to score higher on measures of rebelliousness and rejection of conventional values than those in the childhood-onset group (Dandreaux & Frick, 2009; Moffitt et al., 1996). Although it was long thought that adolescent-onset conduct disorder reflected an exaggeration of normative adolescent behavior and was limited to the adolescent period, the picture now appears far less optimistic. Specifically, individuals with adolescent-onset conduct disorder continue to show significant levels of antisocial activity into their mid-20s and early 30s, as well as various other problems in life adjustment (e.g., impulsivity, substance-related problems, financial difficulties, physical health problems; Moffitt et al., 2002; Odgers et al., 2008). Furthermore, access to opportunities (e.g., gainful employment, higher education) may be limited for these individuals through their encounter of "snares" related to early delinquent involvement, such as substance use disorders, a criminal record, or teen parenthood.

As mentioned previously, the presence of callous–unemotional traits designates a more severe group of children within the childhood-onset group. These traits may also distinguish a group of children with different causal processes leading to their behavioral problems (see Frick & Viding, 2009; Frick et al., 2014, for reviews). For example, some research suggests that the genetic influence to childhood-onset conduct disorder is largely accounted for by youth with significant levels of callous–unemotional traits (Larsson, Andershed, & Lichtenstein, 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003). A large twin study found that the genetic influences on conduct problems were over twice as great for children high on callous–unemotional traits as for children low on callous–unemotional traits (Viding, Blair, Moffitt, & Plomin, 2005). Differences in heritability could not be attributed to differences in the severity of conduct problems or levels of impulsivity–hyperactivity (Viding, Jones, Frick, Moffitt, & Plomin, 2008). Viding and colleagues (2005) further reported that the influence of shared environment was substantial for the group low on callous–unemotional

traits but negligible for youth high on callous–unemotional traits.

Twin studies suggest that there is considerable overlap in the genes that influence callous–unemotional traits and conduct problems, but that there are also unique genetic influences on callous–unemotional traits (Bezdjian, Raine, Baker, & Lynam, 2011; Forsman, Larsson, Andershed, & Lichtenstein, 2007; Viding, Frick & Plomin, 2007). This is consistent with the finding that high levels of callous–unemotional traits have been observed in the absence of clinical levels of conduct problems (Frick, Cornell et al., 2003; Kumsta, Sonuga-Barke, & Rutter, 2012). Twin studies also suggest that stability in callous–unemotional traits is driven by genetic influences (Fontaine, Rijsdijk, McCrory, & Viding, 2010; Forsman, Lichtenstein, Andershed, & Larsson, 2008).

Differences in heritability are theorized to be a function of deficits in amygdala functioning in youth with callous–unemotional conduct disorder (Blair, R. J. R. Mitchell, & Blair, K., 2005), which has been supported by recent brain imaging studies (Jones, Laurens, Herba, Barker, & Viding, 2009; Marsh et al., 2008). Together, research employing magnetic resonance imaging techniques indicate that youth with conduct problems and callous–unemotional traits demonstrate atypical patterns of brain structure and function, particularly in areas critical for affective processing, affective decision making, and moral emotions (see Viding and McCrory, 2015). Broadly speaking, these findings are in line with those reported in studies of adult psychopathy.

Candidate gene association studies tentatively implicate variants in the serotonin and oxytocin genes as genetic risk factors for callous–unemotional traits (e.g. Beitchman et al., 2012; Dadds, Moul, Cauchi, Hawes, & Brennan, 2013; Dadds, Moul, Cauchi, Dobson-Stone et al., 2013; Fowler et al., 2009; Malik, Zai, Abu, Nowrouzi, & Beitchman, 2012). As with conduct problems more generally, callous–unemotional traits are likely to manifest as a result of genetic risk in the presence of environ mental risk. This is supported by research conducted by Sadeh and colleagues (2010), who found that the long allele of a serotonin transporter polymorphism, which is linked with low amygdala reactivity, was associated with callous–unemotional traits among adolescents from low socioeconomic backgrounds. Similarly, Willoughby, Mills-Koonce, Propper, and Waschbusch (2013) found evidence of a genotype by environment interaction in predicting ODD and callous–unemotional traits among preschool-aged children. Children with conduct problems who display callous–unemotional traits tend to be more fearless (Barker, Oliver, Viding, Salekin, & Maughan, 2011; Pardini, 2006) and more thrill and adventure seeking (Frick, Kimonis, Dandreaux, & Farell, 2003; Frick, Lilienfeld et al., 1999), and show less trait anxiety than other youth with the same level of conduct problems (Essau et al., 2006; Frick, Cornell et al., 2003). They also show a decreased sensitivity to punishment cues in laboratory and social settings (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006; Dadds & Hawes, 2006), lower resting and stress-induced cortisol levels (Loney, Butler, Lima, Counts, & Eckel, 2006; O'Leary, Loney, & Eckel, 2007), and are less reactive to threatening and emotionally distressing stimuli than other children with conduct problems (Blair, 1999; Kimonis, Frick, Fazekas, & Loney, 2006; Loney, Frick, Clements, Ellis, & Kerlin, 2003). Of note, a study by Willoughby, Waschbusch, Propper, and Moore (2011) suggested that such differences in emotional processing may be evident very early in life. Specifically, 5-year-old children (N = 178) with high levels of parent-reported callous–unemotional traits and symptoms of ODD were rated as less soothable and showed less negative reactivity to the "still-face" paradigm (i.e., a parental face showing no emotion or interaction with an infant) at 6 months of age, compared to those with symptoms of ODD but without a callous–unemotional presentation.

This temperamental style, characterized by low fear and low emotional reactivity to aversive stimuli, can place a child at risk for missing some of the early precursors to empathic concern, which require emotional arousal evoked by the misfortune and distress of others (Blair, 1995). It could also lead a child to be relatively insensitive to the prohibitions and sanctions of parents and other socializing agents (Frick & Morris, 2004; Kochanska, 1993). Finally, it could create an interpersonal style in which the child becomes so focused on the potential rewards and gains of aggression or other antisocial means of solving interpersonal conflicts that he or she ignores the potentially harmful effects of this behavior on him- or herself and others. Thus, low fearfulness and low emotional reactivity to aversive stimuli may be fundamental characteristics that lead to the development of callous–unemotional traits (Frick et al., 2014). In support of these hypotheses, antisocial and delinquent youth with callous–unemotional traits are less distressed by the negative effects of their behavior on others (Blair, Jones, Clark, & Smith, 1997; Pardini, Lochman, & Frick, 2003), are more impaired in their moral reasoning and empathic concern toward others (Blair, 1999; Pardini et al., 2003), expect more instrumental gain (e.g., obtaining goods or social goals) from their aggressive actions (Pardini et al., 2003), and are more predatory in their violence than antisocial youth without these traits (Caputo, Frick, & Brodsky, 1999; Kruh et al., 2005). On a laboratory task measuring altruistic behavior, adolescents scoring high on conduct problems and callous–unemotional traits were more likely than controls were to make decisions that benefited themselves while harming others (Sakai, Dalwani, Gelhorn, Mikulich-Gilbertson, & Crowley, 2012).

In contrast, children with childhood-onset conduct problems without callous–unemotional traits are more highly reactive to emotional and threatening stimuli (Kimonis, Frick, Fazekas et al., 2006; Loney et al., 2003), more distressed by the negative effects of their behavior on themselves and others (Frick et al., 2003; Pardini

et al., 2003), and are more likely to attribute hostile intent to the actions of peers, compared with those high on callous–unemotional traits (Frick et al., 2003). These findings suggest that children with conduct problems without callous–unemotional traits may have greater difficulties with behavioral and emotional regulation that are related to high levels of emotional reactivity. Studies find that the aggressive and antisocial behavior of children with conduct problems without callous–unemotional traits is more strongly associated with dysfunctional parenting practices (Hipwell et al., 2007; Oxford, Cavell, & Hughes, 2003; Wootton, Frick, Shelton, & Silverthorn, 1997) and deficits in intelligence (Loney, Frick, Ellis, & McCoy, 1998) than for children high on callous–unemotional traits. Emotional regulation problems can lead to impulsive and unplanned aggressive acts for which the child may be remorseful afterwards, but over which he or she still has little control. They can also lead to a higher susceptibility to anger, owing to perceived provocations from peers leading to violent and aggressive acts within the context of high emotional arousal (Kruh et al., 2005).

Empirically Validated Treatments Given the link between conduct problems and later violence and delinquency, it is not surprising that the treatment of antisocial and aggressive behavior in youth has been the focus of a large number of controlled treatment outcome studies. Several reviews of the literature have identified four treatments that have proven effective in controlled outcome studies (see Comer, Chow, Chan, Cooper-Vince, & Wilson, 2013; Eyberg, Nelson, & Boggs, 2008; Frick, 2012, for more extended discussions).

The first intervention involves the use of contingency management programs. The basic components of contingency management programs are: (a) Establishing clear behavioral goals that gradually shape a child's behavior in those areas of specific concern for the child; (b) monitoring the child's progress toward these goals; (c) reinforcing appropriate steps toward reaching these goals; and (d) providing consequences for inappropriate behavior. These programs can bring about behavioral changes for children with conduct problems in a number of different settings, such as at home (Ross, 1981), school (Abramowitz & O'Leary, 1991), and in residential treatment centers (Lyman & Campbell, 1996).

The second effective treatment is parent management training (PMT). PMT is sometimes also called "behavioral parent training", as it was described above as an effective psychosocial intervention for ADHD. A critical goal of PMT programs is to teach parents how to develop and implement structured contingency management programs to alter their child's behavior in the home (Kazdin, 2005; Kazdin & Whitley, 2003). However, these programs also focus on: a) Improving the quality of parent–child interactions (e.g., having parents more involved in their children's activities, improving parent–child communication, increasing parental warmth and responsiveness); b) changing antecedents to behavior that enhance the likelihood that positive prosocial behaviors will be displayed by children (e.g., how to time and present requests, providing clear and explicit rules and expectations); c) improving parents' ability to monitor and supervise their children; and d) using more effective discipline strategies (e.g., being more consistent in discipline, using a variety of approaches to discipline). The effectiveness of this type of intervention has been the most consistently documented of any technique used to treat children with severe conduct problems (Kazdin, 1995; Kazdin & Whitley, 2003). PMT programs include "Helping the Noncompliant Child" (McMahon & Forehand, 2003); "Parent-Child Interaction Therapy" (Brinkmeyer & Eyberg 2003); "The Incredible Years: Early Childhood BASIC Parent Training Program" (Webster-Stratton, 2005); "Triple P-Positive Parenting Program" (Sanders, 1999); and "Parent Management Training-Oregon" (Patterson, Reid, Jones, & Conger, 1975).

The third type of intervention that has proven effective in treating children with conduct problems is a cognitive-behavioral approach designed to overcome deficits in social cognition and social problem-solving experienced by many children and adolescents with conduct problems. For example, as mentioned previously, some children with conduct problems tend to attribute hostile intent to ambiguous interactions with peers that make them more likely to act aggressively toward peers. Other aggressive children overestimate the likelihood that their aggressive behavior will lead to positive results (e.g., enhanced status, obtaining a desired toy) and this cognitive bias makes them more likely to select aggressive alternatives when solving peer conflict (Dodge, Lochman, Harnish, Bates, & Pettit, 1997). Most cognitive-behavioral skills-building programs teach the child to inhibit impulsive or angry responding. This allows the child to go through a series of problem-solving steps (e.g., how to recognize problems, consider alternative responses and select the most adaptive one) and overcome deficits in the way they process social information (Larson & Lochman, 2003).

For the large proportion of children with conduct problems and comorbid ADHD, the final intervention that has proven to have some effectiveness in reducing conduct problems is the use of stimulant medication. The impulsivity associated with ADHD may directly lead to some of the aggressive and other poorly regulated behaviors of children with severe conduct problems. The presence of ADHD may also indirectly contribute to the development of conduct problems through its effect on: (a) Children's interactions with peers and significant others (e.g., parents and teachers); (b) parents' ability to use effective socialization strategies; or (c) a child's ability to perform academically. Therefore, for many children and adolescents with conduct problems, reducing ADHD symptoms is an important treatment goal. The effectiveness of stimulants for reducing conduct problems in children with ADHD has been demonstrated in several controlled medication

trials (Hinshaw, Heller, & McHale, 1992; Pelham et al., 1993).

Limitations Each of these interventions have substantial limitations (Brestan & Eyberg, 1998; Kazdin, 1995). First, a significant proportion of children with severe conduct problems do not show a positive response to these interventions and, for those that do respond positively, their behavior problems are often not reduced to a normal level. Second, treatment is most effective with younger children (before age 8) with less severe behavioral disturbances. Third, the generalizability of treatment effects across settings tends to be poor. That is, treatments that are effective in changing a child's behavior in one setting (e.g., mental health clinics) often do not bring about changes in the child's behavior in other settings (e.g., schools). Fourth, improvements in behavior are often difficult to maintain over time.

Given these limitations, there has been an increasing focus on trying to improve treatments by integrating our knowledge about the causes of conduct problems with the development of innovative approaches to treatment (Frick, 2012). Each of the four treatments described previously targets basic processes that research has shown to be important in the development of conduct problems (e.g., family dysfunction, problems in impulse control). However, these treatments have ignored the fact that severe conduct problems are generally caused by many different and interacting processes. As a result, any single intervention is not likely to be effective for all children with ODD or conduct disorder.

Empirical Supported Principles Based on this research, there is not likely to be any single "best" treatment for severe conduct problems. Instead, interventions must be tailored to the individual needs of children with ODD or conduct disorder and these needs will likely differ depending on the specific mechanisms underlying the child's behavioral disturbance. Research on the different developmental pathways to conduct problems has great potential for informing these individualized approaches to treatment (Frick, 2012). For example, interventions for children in the adolescent-onset pathway will likely be somewhat different from interventions for children in the childhood-onset pathway. Even within the childhood-onset pathway, the focus of intervention may be different depending on the presence or absence of callous–unemotional traits (see Frick, 2012, for examples).

Youth with callous–unemotional conduct disorder may be in greatest need of intensive interventions because of their chronic and severe pattern of antisocial behavior across development. However, growing evidence suggests that children with callous–unemotional traits benefit significantly from standard interventions, although they are likely to enter and finish treatment with more pronounced behavioral and social difficulties (e.g., Hawes, Dadds, Brennan, Rhodes, & Cauchi, 2013; Kimonis et al., 2014; for a review see Waller, Gardner, & Hyde, 2013). Preliminary research suggests some particularly promising interventions for these youth. For example, exposing antisocial children to warm parenting reduced callous–unemotional traits and antisocial behaviors in later development (Kroneman, Hipwell, Loeber, Koot, & Pardini, 2011; Pasalich et al., 2012; see Waller et al., 2013 for a review). Also, a parenting intervention with clinic-referred boys with a primary diagnosis of ODD that taught methods for using positive reinforcement, rather than punishment-oriented behavior modification, to encourage prosocial behaviors found a significant reduction in disruptive behaviors for children high on callous–unemotional traits (Hawes & Dadds, 2005). Similarly, an intensive treatment program administered to incarcerated adolescents high on callous–unemotional traits that utilized reward-oriented approaches, taught empathy skills, and targeted the youth's interests led to reductions in recidivism over a 2-year follow-up period (Caldwell, Skeem, Salekin, & Van Rybroek, 2006).

One approach to treatment that incorporates research on developmental models of conduct problems is multisystemic therapy (MST). MST was originally developed as a general approach to intervention for psychopathological conditions (Henggeler & Borduin, 1990) but has been applied extensively to the treatment of severe antisocial behavior in children and adolescents (Henggeler, Schoenwald, Borduin, Rowland, & Cunningham, 2009), and for the treatment of juveniles who sexually offend (Henggeler, Letourneau et al., 2009). The orientation of MST is an expansion of a systems orientation to family therapy. In systemic family therapy, problems in children's adjustment are viewed as being embedded within the larger family context. MST expands this notion to include other contexts, such as the child's peer, school and neighborhood contexts. Most importantly, MST is an intervention framework that emphasizes a comprehensive and individualized approach to intervention that is consistent with the treatment principles outlined above and is delivered in the youth's natural environment.

MST involves an initial comprehensive assessment that seeks to understand the level and severity of the child or adolescent's presenting problems and to understand how these problems may be related to factors in the child's familial, peer, and cultural environment. The information from this assessment is used to outline an individualized treatment plan based on the specific needs of the child and his or her family. Unlike the individual interventions described previously, MST does not emphasize the use of specific techniques. Instead, it emphasizes several principles that follow from its orientation to intervention. These principles include:

- The identified problems in the child are understood within the child's familial, peer, and cultural context.
- Therapeutic contacts emphasize positive (strength-oriented) levers for change.

- Interventions promote responsible behavior among family members.
- Interventions are present focused and action oriented, targeting specific and well defined problems.
- Interventions target sequences of behavior within and between multiple systems.
- Interventions must be developmentally appropriate.
- Interventions are designed to require daily or weekly effort by family members.
- Intervention effectiveness must be evaluated continuously from multiple perspectives.
- Interventions are designed to promote maintenance of therapeutic change by empowering caregivers.

A critical component of MST is a system of intensive supervision for the therapists implementing the treatment, to determine how these principles should be implemented to meet the needs of each individual case and to ensure that the principles are followed throughout the intervention (Henggeler, Schoenwald et al., 2009). One of the important contributions of MST to the treatment outcome literature is its demonstration that individualized interventions can be rigorously evaluated through controlled treatment outcome studies.

Several randomized trials have found that adolescent offenders who receive MST exhibit lower rates of delinquent behavior, substance use, recidivism, and incarceration following treatment compared to youth receiving standard community care (Henggeler & Sheidow, 2012). A follow-up study found lower rates of recidivism 22 years after treatment initiation among seriously delinquent juveniles who received MST relative to those assigned to individual therapy (Sawyer & Borduin, 2011). The positive effect of MST on even severe antisocial behavior appears to be due in part to the program's ability to improve family relations, increase the use of positive discipline techniques, and reduce deviant peer group affiliation (Henggeler, 2011; Henggeler & Sheidow, 2012). Despite the evident promise of MST for treating youth with serious conduct problems, preliminary research suggests that even this highly individualized and comprehensive treatment does not reduce the antisocial behaviors of children with callous–unemotional traits to levels equivalent to youth without callous–unemotional traits (Manders, Deković, Asscher, van der Laan, & Prins, 2013; see also Masi, et al, 2013). Moreover, results are mixed with respect to its ability to significantly reduce callous–unemotional traits relative to treatment as usual (Butler, Baruch, Hickey, & Fonagy, 2011; Manders et al., 2013).

Overall Summary

Externalizing disorders are one of the most common reasons children are referred to mental health clinics for treatment. Children with disorders such as ADHD, ODD, and conduct disorder often cause significant disruption to those around them, particularly parents and teachers, who are most likely to refer them for treatment. Further, children with these problems are at increased risk for a number of longer-term problems in adjustment, such as delinquency, substance abuse, and depression. Fortunately, there is a large body of research on externalizing disorders, which has led to great advances in our understanding of their causes and the development of effective interventions to treat them in youth. Unfortunately, this research is often not translated well into practice and, as a result, many children with these disorders do not receive state-of-the-art treatment. This research-to-practice gap may be the result of practitioners not remaining current on this research or not being trained in the most current theories and approaches to treatment, or of research not being conducted or presented in a way that is useful to practicing psychologists, or a combination of the two. In any case, the quality of services provided to children with disruptive behaviors depends heavily on advances in research and our ability to translate these findings into widely used applications. The focus of this chapter has been to summarize research on children with externalizing disorders in a way that promotes such a translation.

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20

Internalizing Disorders of Childhood and Adolescence

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Introduction

Internalizing disorders in childhood and adolescence include the anxiety and affective disorders. As such, they consist of problems related to worry, fear, shyness, low self-esteem, sadness, and depression. Prevalence of anxiety or depressive disorders in children and adolescents in the general population may be between 8% (Orton, Riggs & Libby, 2009), with clinically elevated symptoms between 9% in boys and 15% in girls (Ghandour, Kogan, Blumberg, Jones & Perrin, 2012), and somewhat higher in high-risk samples, such as children in the welfare system (Letcher, Sanson, Smart & Toumbourou, 2012). These "emotional" problems have frequently been found to be interrelated in clinical settings and to be associated statistically with one another in factor analytic studies. Internalizing problems are contrasted with externalizing problems—problems frequently associated with inattention, conduct problems, opposition and defiance. At the same time, internalizing disorders may include irritability and mood dysregulation, which can resemble symptoms of disruptive externalizing disorders (American Psychiatric Association (2013). It is of historic interest to note that these two broad dimensions of anxiety and depression in children and adolescents have been recognized for many years. Horney (1945), for example, spoke of children who "move against the world" (i.e., externalizing disorder children) and those who "move away from the world" (i.e., internalizing disorder children).

Connection Between Anxiety and Depression Although there is little question about the existence of internalizing problems in childhood and adolescence and their detrimental effects on the growing child and adolescent (Grills-Taquechel & Ollendick, 2012; Kendall, Settipani, & Cummings, 2013), there is considerable controversy about whether they constitute a single broadband internalizing set of problems or whether they constitute multiple, narrow-band psychiatric disorders. At the heart of this issue is the frequent observation that anxiety disorders and affective disorders frequently co-occur with one another (i.e., they are comorbid disorders) and that they are rarely observed in their "pure" forms, at least in childhood and adolescence (Seligman & Ollendick, 1998). A recent integrative review suggests that the inconsistent findings about the relationship between anxiety and depression may be because the relationship varies across important individual and contextual factors. The overlap in anxiety and depression, for example, may vary by diagnosis, age, and sex, as well as other community, psychosocial considerations, and environmental sources of stress (Cummings, Caporino & Kendall, 2014).

Diagnoses of Internalizing Disorders Although the major anxiety and affective disorders in childhood and adolescence overlap, we present them as separate diagnostic entities in this chapter, consistent with nosological approaches, such as the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV; American Psychiatric Association, 1994) and 5th edition, (DSM-5; American Psychiatric Association, 2013). In DSM-5, several diagnostic criteria for anxiety and depressive disorders were revised. The major changes are discussed in more detail below.

Anxiety Disorders in Children and Adolescents

Phenomenology For children and adults alike, anxiety is a normal and common emotional response to a perceived

threat to one's physical or emotional wellbeing. Feeling fearful and fleeing from a genuinely dangerous situation is adaptive. However, if the anxiety response is elicited by a situation or object that is not truly dangerous, then the anxiety and the avoidance associated with it are not adaptive. A diagnosis of an anxiety disorder may be warranted if the anxiety response is excessive in frequency, intensity and/or duration, and if it results in significant impairment in functioning (Rapee, Bogels, van der Sluis, Craske, & Ollendick, 2012). Excessive anxiety is distressing to children and adolescents, and the associated avoidance interferes with their ability to engage in developmentally appropriate tasks and activities. Alarmingly, anxiety disorders are some of the most common psychological difficulties experienced by children and adolescents, and these disorders tend to persist into late adolescence and adulthood unless effective treatment is received (Ollendick & March, 2004; Grills-Taquechel & Ollendick, 2012).

Consistent with Lang's (1979) tripartite model, anxiety is best viewed as a multidimensional construct involving *physiological* features such as increased heart rate and respiration, *cognitive* ideation including catastrophic and unhelpful thoughts (e.g. "I can't do this," "What if something bad or terrible happens to me?"), and *behavioral* responses such as avoidance of the anxiety provoking object or situation. Furthermore, there are developmental differences in the expression of anxiety. For example, young children may avoid objects or situations that scare them; however, they may have difficulty identifying and even verbalizing the exact cognitions associated with the feared situation. Young children may also have trouble relating or connecting their physiological symptoms to their anxiety. For example, a child with separation anxiety might insist that the reason she is sick with a headache and stomachache when she has to go to school is that she has come down with a physical illness such as the flu, not because she is fearful about separation from her caretaker and going to school or spending the night with a friend.

Developmental Considerations As noted, for most children and adolescents, fears and worries are a normal part of development. Accordingly, clinicians assessing anxiety in children need to be aware of what constitutes "normal" fears at each level of development. For example, young infants and toddlers tend to fear aspects of their immediate environment such as loud noises, unfamiliar people, separation from caregivers, and heights. Fears of animals, being alone, and of the dark begin to emerge during the preschool years. As cognitive abilities continue to develop during the early school years, children's fears begin to include abstract, imaginary, or anticipatory fears such as fear of failure or evaluation, death, bodily injury, and supernatural phenomena. Finally, concerns about death, danger, social comparison, personal conduct, and physical appearance extend from adolescence to adulthood (Gullone 2000; Ollendick, King, & Muris, 2002).

Developmentally normal fears are, by definition, age appropriate and transitory in nature. In contrast, a diagnosis of an anxiety disorder is warranted when a child experiences anxiety that is not typical of a child his or her age, and when a child experiences anxiety that is severe and causes considerable distress, and/or which impairs a child's functioning at home, school, or in peer and family relationships.

Gender Differences Research examining the prevalence of anxiety disorders in boys and girls has produced mixed results. Studies using community samples have found that girls are more likely to report anxiety than boys (Essau, Conradt, & Petermann, 2000; McGee et al., 1990). In contrast, gender differences are usually not found in clinic samples (Strauss & Last, 1993; Grills-Taquechel & Ollendick, 2012). There are at least two explanations for this discrepancy. First, societal expectations of gender appropriate behavior for boys and girls may mean that girls are more likely to report anxiety symptoms than boys. Alternatively, anxiety symptoms may be more common in girls, and the equal ratio of males to females in clinic samples may indicate that boys experiencing anxiety are more likely to be referred for treatment than girls with similar symptoms.

The direction and size of the gender difference is also dependent on the diagnostic category being considered. Generalized anxiety disorder (GAD) appears to be similarly prevalent in boys and girls during childhood, although in adolescence it is more common among females than males (Cohen et al., 1993). Research concerning gender differences for separation anxiety disorder (SAD) has been mixed. Although some studies find no gender differences for SAD (Cohen et al., 1993; Last, Perrin, S., Hersen, M., & Kazdin, 1992), most studies find that girls outnumber boys (e.g., Kashani, Orvaschel, Rosenberg, & Reid, 1989). The research on social phobia is less clear, although few gender differences have been noted (Ollendick & Ingman, 2001).

Prevalence Estimated prevalence rates for the childhood anxiety disorders using DSM criteria have been found to vary considerably but typically range between 7% and 12% (Kashani et al., 1989; Kessler et al., 2005). For example, 7.4% of 792 children in a New Zealand longitudinal study met criteria for an anxiety disorder when they were 11 years of age (Anderson, Williams, McGee & Silva, 1987). McGee and colleagues (1990) reported that, at 15 years of age, 10.7% of adolescents in their general sample met criteria for an anxiety disorder. A 3.3% increase in the anxiety disorders was evident over the 4-year period. Similar prevalence rates for the anxiety disorders have been found by a host of other researchers, with many indicating that the prevalence of some anxiety disorders increase with age (e.g., GAD, social phobia, panic disorder), whereas others tend to decrease with age (e.g., SAD,

specific phobia). Thus, overall prevalence rates vary by gender and age. They also vary by the diagnostic category being considered, which we discuss next.

Diagnostic Categories: DSM-5

Separation Anxiety Disorder DSM-IV describes SAD as developmentally inappropriate and excessive anxiety associated with separation from home or from those to whom the individual is attached (American Psychiatric Association, 1994). DSM-5 (American Psychiatric Association, 2013) continues with this earlier broad description of SAD, although it now indicates that this disorder can and does occur into adulthood. DSM-5 indicates that the fear, anxiety and avoidance associated with SAD must last at least 4 weeks in children and adolescents; however, it needs to last 6 months or longer to meet the criteria in adults. Often, children with SAD worry about danger or harm coming to themselves (e.g., being kidnapped) or their loved ones (e.g., becoming ill or having a car wreck) when they are separated from them. Children with SAD exhibit distress when they are separated from their attachment figures and will undertake steps to avoid being apart from them. This may result in children refusing to attend school, go to day camps, or sleep away from home. The evidence indicates that as children become older, the prevalence of SAD declines. For example, the 12-month prevalence rate for SAD was 3.5% for 11-year-old children but 2.0% for 15-year-old adolescents. In adolescence, this disorder seems to become less common compared with other disorders such as GAD, social phobia, and panic disorder (Mattis & Ollendick, 2002; Grills-Taquechel & Ollendick, 2012).

Generalized Anxiety Disorder GAD is characterized as excessive anxiety and worry, which occurs more days than not for at least 6 months. These children find it difficult to control their worries about a number of events or activities. To meet the criteria for GAD, children need to experience at least one associated physiological symptom, although typically these children report multiple physical symptoms. Physical symptoms include stomach aches or nausea, headaches, muscle tension, restlessness, irritability, fatigue and sleep disturbance (Whitmore, Kim-Spoon, & Ollendick, 2014). The DSM-IV category of GAD in children replaced overanxious disorder in the revised third edition (DSM-III-R; American Psychiatric Association, 1980). As noted above, there is some evidence to suggest that the prevalence of overanxious disorder/GAD increases with age (Essau & Ollendick, 2013; Strauss, 1994). No significant changes in GAD were made in DSM-5.

Specific Phobia Specific phobia is defined as persistent fear of a specific object or situation that is excessive or unreasonable. Phobias of certain animals or insects, the dark, heights, storms, and medical procedures are among the most common in children. There are four major types of specific phobia in DSM-IV and DSM-5: animal, environmental, situational, and blood/injury/injection. A study conducted some years ago in Germany reported a prevalence rate of 2.5% in 12–17-year-olds (Essau et al., 2000). Studies in the United States have reported prevalence rates ranging from 3.6% to 9% (Costello et al., 1988; Kessler et al., 2005). Taken together, studies suggest a prevalence rate of about 5% for specific phobia in children and adolescents (Ollendick, Raishevich, Davis, Sirbu, & Ost, 2010).

Social Phobia Social phobia in children, as in adults, is characterized by a marked or persistent fear of social situations or performance situations (American Psychiatric Association, 1994, 2013). Typically, in these situations, the child is exposed to unfamiliar people and/or is scrutinized by others. Social phobia is a disorder with a later age of onset (usually around 11 years of age, or early adolescence), being rarely diagnosed in children younger than 10 years (Davidson, Hughes, George, & Blazer, 1993). A study using DSM-IV criteria reported that 6.3% of adolescents in a community sample would meet criteria to be diagnosed with social phobia (Schniering, Hudson, & Rapee, 2000), a pattern that likely continues with DSM-5 criteria, as they are relatively unchanged.

Differential Diagnosis and Comorbidity In clinical populations, the most common comorbidity with any specific anxiety disorder is another type of anxiety disorder (Kendall, Brady, & Verduin, 2001). Children with comorbid disorders tend to have a greater severity and persistence of symptoms, more interpersonal problems, and may be more refractory to change and clinically challenging for therapists (Manassis & Monga, 2001). In addition to comorbidity with other anxiety disorders, anxious children may also exhibit high rates of depression (Kendall et al., 2013), as noted previously. Using a large cohort of 1,710 adolescents, the Oregon Adolescent Depression Project found that 49% of the adolescents with an anxiety disorder also had comorbid depressive disorders using DSM criteria (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Similarly, high rates of comorbidity were found in the early Dunedin, New Zealand, sample of youths (Anderson et al., 1987). This series of studies also found that the rates of comorbid mood disorders increased as the children became older. Rates of comorbidity of anxiety and depressive disorders were higher among adolescents than among younger children.

Rates of comorbidity between anxiety and externalizing problems such as attention-deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) are also high (Caron & Rutter, 1991; Cunningham & Ollendick, 2010). Studies examining this relationship have found that between 13% and 24% of children with an anxiety disorder also have ADHD, ODD, or conduct disorder (Last et al.,

1987; Keller et al., 1992). Consistent with these findings, Kendall and colleagues (2001) reported that 25% of their clinical sample of anxious children also met DSM criteria for one of these three disruptive behavior disorders.

Developmental Course and Prognosis A common misconception is that children and adolescents will 'outgrow' their worries and fears. Although this may be true of developmentally normal fears and everyday concerns, research suggests that anxiety disorders tend to persist unless treated (Ollendick & King, 1994). For example, Pfeffer, Lipkins, Plutchik, and Mizruchi (1988) found that for children aged 6–12 years who were diagnosed with what was known as overanxious disorder in an earlier version of the DSM, 70.6% still met this diagnosis 2 years later. Similarly, in another study, longitudinal results showed that girls diagnosed with an anxiety or depressive disorder at one age were more likely to continue to meet diagnostic criteria in subsequent years (McGee, Feehan, Williams, & Anderson, 1992).

Anxiety disorders have been shown to persist not only over time but also to be associated with the development of more severe symptomatology (Albano, Chorpita, & Barlow, 1996). Older children tend to report more severe anxiety and comorbid symptomatology than do younger children with the same diagnosis (Strauss, Lease, Last, & Francis, 1988). Similarly, many adults with anxiety disorders report a lifelong history of anxiety symptoms beginning in childhood (Markowitz, Weissman, Ouellette, Lish, & Klerman, 1989; Ollendick, Lease, & Cooper, 1993). For example, a longitudinal study conducted in the United States found that children with an anxiety disorder were at greater risk for future anxiety disorders, and they were also at increased risk of developing dysthymic disorders (Lewinsohn et al., 1993; Orvaschel, Lewinsohn, & Seeley, 1995).

Etiological Theories of Childhood Anxiety Understanding the development of anxiety and its disorders requires us to consider the potential for complex, reciprocal interactions and transactions between many etiological factors over the course of a child's development, consistent with a developmental psychopathology approach. Empirical efforts to explain the development and maintenance of anxiety have focused on the interaction between personal characteristics of the child (e.g., genetic vulnerability, behavioral inhibition, cognitive processes) and interpersonal factors such as attachment to caregivers and learning processes that occur within the family. Some of these etiological factors are addressed briefly below.

Biological and Familial Factors Family studies using both referred and non-referred samples show that the parents of anxious children, and the children of anxious parents, are more likely to experience anxiety problems than non-clinic controls (McClure, Brennan, Hammen, & Le Brocque, 2001). Although family studies have demonstrated that anxiety disorders tend to run in families, the mechanism of transmission remains unanswered. Twin studies help to distinguish whether this mechanism is genetic, environmental or a combination of the two. Although specific heritability estimates vary across studies, almost all twin studies support the conclusion that there is a heritable genetic risk for anxiety disorders (see Thapar & McGuffin, 1995, for a review). There is preliminary evidence that separation anxiety may have a higher heritability than other anxiety disorders, at 73% parent–child overlap in diagnosis (Bolton et al., 2006). However, rather than a risk towards developing a specific anxiety disorder, the broad base of research is strongest for a genetic vulnerability toward either an anxiety or a depressive disorder (Eley & Stevenson, 1999).

One of the proposed mechanisms by which a predisposition for anxiety is transmitted genetically is via inherited temperamental characteristics such as behavioral inhibition. Kagan Reznick, and Snidman (1987) found that behavioral inhibition occurs in about 15–20% of children and that children with behavioral inhibition tend to react with withdrawal, avoidance, or distress when confronted with unfamiliar people, situations, or objects. Behavioral inhibition has also been shown to be a risk factor for later childhood anxiety problems (Biederman et al., 2001). However, it is important to remember that not all behaviorally inhibited children develop anxiety disorders, and it is best conceptualized as one possible predisposing factor that may lead to anxiety given the "right" set of other contextual conditions (Ollendick & Benoit, 2012).

Learning Influences The theory of classical conditioning can be invoked to help explain the development of certain fears and phobias, as well as anxiety disorders in general. For example, a child might develop a dog phobia after the previously neutral stimulus (a dog) was associated with the pain of being bitten (unconditioned stimulus) resulting in a conditioned fear response. However, classical conditioning theory alone cannot explain why some individuals who experience a pairing of trauma or pain with a particular object or situation do not develop a phobia (Ollendick & Cerny, 1981). In more recent years, theorists have integrated information-processing theories with classical conditioning theory, suggesting that an individual's internal representation and subsequent evaluation of the unconditioned stimuli mediates the strength of the conditioned response (Davey, 1992). For example, Reiss (1980) suggested that a person's anticipation of social or physical danger, or their expectation of anxiety, might make a person more susceptible to traumatic conditioning.

Operant conditioning theory suggests that behaviors that are reinforced are more likely to occur again and behaviors that are followed by punishment are less likely to occur again (Skinner, 1938). Furthermore, the concept of negative reinforcement is frequently used to explain

how avoidant behavior maintains anxiety symptoms over time. Specifically, when an anxious child avoids a feared situation, the child is reinforced by the resultant reduction in anxiety symptoms. The child therefore learns that to prevent experiencing the unpleasantness of anxiety, he or she needs to avoid the feared situation or object.

Operant conditioning principles can also be used to explain the process by which a child's non-anxious behavior gradually decreases in frequency, while the anxious behavior increases (Ollendick, Vasey, & King, 2000). For example, Barrett, Rapee, Dadds, and Ryan (1996) found that parents of clinically anxious children attended more to the anxious and avoidant behaviors of their children than to their brave coping behaviors. Although this study was unable to clarify whether this pattern of parenting behavior existed prior to or after the development of their child's anxiety, it does indicate that positive reinforcement from parents may serve to maintain the problem.

Childhood anxiety has also been shown to develop as a result of vicarious learning and modeling. Bandura (1999) suggested that behaviors might be acquired, facilitated, reduced or eliminated by observing the behavior of others. Accordingly, fears may appear after the child has observed their parents or peers reacting fearfully to certain objects or situations. Silverman, Cerny, Nelles, and Burke (1988) studied a group of anxious parents and their children and found that children's anxious behaviors were associated with heightened levels of avoidance evidenced by their parents. Interestingly, these researchers noted that anxiety disorders showing the most pronounced avoidance behaviors (social phobia, specific phobia and agoraphobia) seem to have the highest rates of familial risk.

Cognitive and Information-Processing Biases Cognitive theorists suggest that when processing information, anxious people tend to overestimate the threat of danger, and underestimate their abilities to cope with that threat (Beck, 1991). Using a variety of methodologies, attentional biases toward threat-related stimuli have been demonstrated for clinically and non-clinically anxious adults (e.g., Pury & Mineka, 2001; Wood, Mathews, & Dalgleish, 2001). Similar research has found evidence of attentional bias toward threat-related cues in both clinically anxious (Taghavi, Neshat-Doost, Moradi, Yule & Dalgleish, 1999) and non-clinically anxious children (Kindt, Brosschot, & Everaerd, 1997). Reviews by Bar-Haim, Lamy, Pergamin, Bakermans-Kraneburg, and van Ijzendoorn (2007) and Waters, Bradley, and Mogg (2013) examine the theoretical underpinnings of these attentional mechanisms and explore their implications for clinical research and practice.

Findings of biased attention in anxious children have been complemented by research examining the process of threat interpretation (Cowart & Ollendick, 2010). For example, a series of studies by Barrett and colleagues (Barrett et al., 1996; Dadds & Barrett, 1996; Dadds, Barrett, Rapee, & Ryan, 1996) demonstrated that clinically anxious children tended to interpret ambiguous vignettes of social and physical situations as threatening compared with non-clinic controls.

The Parent–Child Relationship An increasing number of studies have examined the relations between parenting behavior and anxiety in children. Parents of clinic-referred anxious children have been found to be more controlling (Dumas, LaFreniere, & Serketich, 1995), more restrictive (Krohne & Hock, 1991), more over-involved emotionally (Hirshfeld, Biederman, Brody, Faraone, & Rosenbaum, 1997), less accepting (Ollendick & Horsch, 2007), and less granting of psychological autonomy (Siqueland, Kendall & Steinberg, 1996) than are parents of non-referred children. Because these studies are cross-sectional, however, it is impossible to draw firm conclusions about whether relations between parent behavior and anxiety represent cause or effect, or, for that matter, whether it is more reciprocal in nature. This latter possibility is more consistent with a developmental psychopathology perspective (for extended discussion of these issues, see Dadds & Roth, 2001).

Assessment Only a brief overview of assessment practices is provided here; for a more complete review of assessment practices with anxious youths, see McLeod, Jensen-Doss, and Ollendick (2013). Anxiety, of course, is recognized as a multidimensional construct and as such a multi-informant and multi-method approach to the assessment of childhood anxiety is recommended. Diagnostic interviews with the child and the child's parents are one of the best methods for distinguishing normal, developmentally appropriate fears and anxieties from problematic anxiety disorders. The most commonly used diagnostic interview to assess youth with anxiety disorders is the Anxiety Disorders Interview Schedule for Children (Silverman & Albano, 1996). This interview can be used to solicit detailed information about a range of individual anxiety symptoms, interference in daily functioning, school refusal behavior, interpersonal functioning, and avoided situations. In addition to the interviews, the child and her or his parents are usually asked to complete self-report questionnaires, and responses on these questionnaires can be compared to available normative data.

The Multidimensional Anxiety Scale for Children (March, Parker, Sullivan, Stallings, & Connor, 1997), the Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1985), and the Fear Survey Schedule for Children–Revised (Ollendick, 1983) are examples of questionnaires that are frequently used in this regard. Parents and teachers can complete ratings scales such as the Child Behavior Checklist and Teacher Report Form (Achenbach & Rescorla, 2001). These scales have the advantage of measuring anxiety/depression symptoms as well as externalizing and other related problems. In

addition to these measures, it is recommended that behavioral observation, cognitive assessment and physiological assessment be considered (McLeod et al., 2013).

Interventions

Pharmacological Interventions There are a limited number of controlled pharmacological treatment trials for anxiety in children and adolescents. Several reviews of psychopharmacological treatments for anxiety disorders in children and suggest that selective serotonin reuptake inhibitors (SSRIs) represent the first line of medical treatment for childhood anxiety disorders (as well as affective disorders; Ollendick & March, 2004). Serotonegic and tricyclic antidepressants are second-line anxiety agents, and buspirone may be used as a second or third-line anxiety treatment. Medication alone is rarely the treatment of choice for children with anxiety disorders, and typically medication is used in combination with psychological treatment (Ollendick & March, 2004).

Psychosocial Interventions Cognitive-behavioral therapy (CBT) for childhood anxiety has the strongest empirical support (Ollendick & King, 2012). CBT consists of four main strategies. First, exposure requires the child to approach the object or situation she or he fears or worries about, either directly (*in vivo*) or imaginally. Exposure is typically conducted in a graduated and progressive manner. Exposure may also take the form of systematic desensitization in which the child receives relaxation training, and then practices relaxation while facing his or her feared situations *in vivo* or imaginally. *In vivo* and imaginal desensitization are both effective treatments for childhood anxiety disorders, although *in vivo* procedures are generally viewed as more effective than imaginal ones (Seligman & Ollendick, 2011).

The second strategy implemented to treat childhood anxiety is modeling. Modeling involves a person demonstrating approach behavior in situations that the child finds anxiety provoking. Variants of modeling include filmed modeling where the child watches a video, live modeling where the person modeling is in the presence of the anxious child, and participant modeling where a model interacts with the child and guides him or her to approach the feared situation. This approach may be particularly well suited for younger children (Puliafico, Comer & Albano, 2013).

Progressive exposure and modeling assume that fear must be reduced before approach behavior will occur. In contrast, the third CBT strategy—contingency management—based on the principles of operant conditioning, encourages increases in approach behavior by altering the consequences of a child's behavior in the anxiety provoking situations. Contingency management involves modifying the antecedents of anxiety or the consequences of anxious behavior through positive reinforcement, punishment, extinction, and shaping. For example, positive reinforcement for "courageous" or approach behaviors to the feared or dreaded stimulus are frequently used in such programs. These procedures are implemented by the therapist during the therapy session, and are often taught to parents and teachers for use in the home, school, and other community settings. Treatments involving reinforced practice have been shown to be superior to no-treatment or waitlist control conditions, and other treatments (verbal coping skills, modeling) in reducing phobic symptoms. As such, reinforced practice is considered to be a well-established treatment for childhood fears and phobias (see Ollendick & King, 2012).

The fourth strategy used in cognitive-behavioral therapy is a primarily a cognitive or information-processing approach. Cognitive interventions include techniques such as identifying self-talk, cognitive restructuring, and problem solving, and they are usually taught in combination with one or more of the behavioral strategies reviewed above. Consequently, most published studies have examined treatments combining cognitive and behavioral interventions, and very few have examined the effectiveness of cognitive strategies in isolation. These integrated CBT programs are the most widely used treatments for children suffering from the most common anxiety disorders.

Kendall, Settipani, and Cummings (2013), summarized an integrated cognitive-behavioral treatment program for anxiety in children called "Coping Cat". The four coping strategies taught to anxious children were summarized in an acronym—FEAR—which helped children remember the steps to take when they felt anxious: "Feeling frightened, Expect good things to happen, Actions and attitudes to take, and Reward yourself." A host of randomized controlled clinical trials provide strong evidence that individual CBT programs such as Coping Cat and its variants are more effective than a waitlist and credible placebo conditions for reducing anxiety related to SAD, social phobia, GAD, and school refusal (see reviews by Kendall et al., 2013; Ollendick & King, 2012). In general, these studies suggest that approximately two-thirds of youth recover from their anxiety diagnosis at the end of treatment. Moreover, long-term outcome studies suggest that these gains are maintained over time and, in at least some instances, continued improvement is seen.

Studies have also examined the impact of incorporating parents in the therapeutic process. For example, teaching parents better strategies to manage their own anxiety and informing parents about strategies their children are learning to manage their anxiety may lead to better treatment outcomes than interventions which focus solely on the child (Choate, Pincus, Eyberg & Barlow, 2005). Kendall and colleagues (2013) also support using a family-based supportive approach. Although both individual and family-based approaches were equally effective in reducing symptoms of anxiety, the latter approach also resulted in changes in family functioning that might maintain the gains made in therapy.

Despite these positive findings, not all studies have shown enhanced outcomes associated with parental involvement (e.g., Simon, Bogels, & Voncken, 2011; van der Sluis, van der Bruggen, Brechman-Toussaint, Thissen, & Bogels, 2012). We are mindful of, and agree with, the cautionary statement by Barmish and Kendall (2005), that "as alluring as it might be to include parents as co-clients for multiple theoretical reasons, this belief cannot be mistaken as evidence" (p. 578). Considerably more research is required to understand the conditions under which parental involvement might be most beneficial to the child client.

Group interventions for anxiety in children have been used for some time (Ollendick & King, 2012), but it is only recently that group CBT has been investigated in controlled clinical trials. A series of studies by different research teams suggests that group format CBT is more effective in reducing anxiety than waitlist and placebo conditions (Flannery-Schroeder & Kendall, 2000; Shortt, Barrett, & Fox, 2001; Silverman et al., 1999). The question as to whether group CBT is more, less, or equally as effective as individual CBT awaits large-scale studies.

Summary Anxiety is a common problem in childhood and adolescence. Anxiety problems are frequently comorbid with other anxiety disorders, depression, or externalizing behaviors, and have a poor prognosis if not treated. Several etiological theories have been proposed to explain the development and maintenance of anxiety. Although research indicates a familial risk of anxiety, most anxiety problems can be conceptualized as an interaction between temperament and environmental and contextual factors. Anxiety in children and adolescents can be reliably assessed, and promising pharmacological interventions as well as evidence-based cognitive-behavioral treatments are available.

Depressive Disorders

Phenomenology Depressive disorders also affect a significant number of children and adolescents, and there are important developmental factors to consider when making a diagnosis and planning for treatment with youths who have these disorders (Duggal, Carlson, Sroufe, & Egeland, 2001). A thorough understanding of risk factors, development, family factors, and individual cognitive variables, as well as comorbid disorders, assists in both assessment and treatment of depression in youth (Stark, Sander, Yancy, Bronik, & Hoke, 2000). The literature base spans approximately 30 years, describing symptoms of depression in children and adolescents and empirical evaluations of its treatment (Costello, Erkanli, & Angold, 2006; Weisz, McCarty, & Valeri, 2006).

Developmental Considerations In general, many children and some adolescents who are depressed do not report feeling sad or "depressed," and developmentally appropriate language is needed to understand and communicate with them about their experiences. Children may not always look "depressed" either, so with mild or moderate levels of clinical symptoms, an untrained observer, including parents or teachers, may not recognize signs of depression. In particular, children tend to express their negative emotions as irritability or anhedonia (i.e., things that once were fun or enjoyable no longer are). In DSM-5, a new diagnostic category highlights the irritability that can accompany depressive disorders in children, "disruptive mood dysregulation disorder" (American Psychiatric Association, 2013).

Children may also lack the emotional vocabulary to describe their irritable or flat affect and other depressive symptoms in terms of feelings, and even if they know how to voice their experiences, dysthymia poses unique problems for children. A child who has dysthymia, or chronic mild depression, captured in the persistent depressive disorder diagnosis in DSM-5, a milder but more chronic set of symptoms over a period of at least 12 months, may not have the cognitive-developmental awareness that their depressive symptoms are different from the emotions of other children, or even their own pre-depression life. Although some researchers and clinicians suggest that the observed developmental differences in symptoms warrant different diagnostic categories (not unlike developmental differences between ODD and conduct disorder), the field appears to have accepted these differences in symptom patterns across development and to embrace them.

Children and adolescents also differ in their perceptions of personal control and resultant depression. A model put forth by Weisz, Southam-Gerow, and McCarty (2001) articulated developmental differences in perceived contingency, control, and competence related to depression in children and adolescents. Data were collected in several mental health outpatient centers with 360 child and adolescent participants. The contingency, control, and competence model defined perceived control as one's power to influence or engineer a desired outcome. Contingency was determined by how much one's efforts caused the outcome, considering other possible contributing factors. Competence was defined as the degree to which one actually carried out the necessary behaviors to produce the desired outcomes. Both children and adolescents appeared negatively affected by low perceived control and low perceived competence, but adolescents were also sensitive to the contingency or "fairness" of the circumstances in a more global sense. Such findings may have important implications for current treatments for depression in children versus adolescents.

As in many types of clinical distress and disease, certain factors increase the likelihood that a youth will develop the disorder. In a prospective, longitudinal study by Duggal and colleagues (2001), significant differences emerged for factors associated with childhood-onset and adolescent-onset depression. Abuse at an early age, higher maternal stress, and less supportive early care

differentiated childhood-onset from adolescent-onset depression. In general, adverse family relationships were associated with childhood-onset depression but not adolescent-onset depression. As reported in more recent studies, family functioning is a concern but it could be family conflict, specifically, that relates to prognosis (Feeny et al., 2009).

Gender Differences Prevalence rates of depressive disorders are different for boys and girls. The differences change systematically with development, adding an additional factor to consider. Prior to adolescence, there are approximately equal proportions of depressive disorders among boys and girls, hovering near 4–6% of boys and girls at any given time (Costello et al., 2006). However, beginning in adolescence and continuing into adulthood, depressive disorders occur more frequently among females than males (Kessler, Avenevoli, & Merikangas, 2001), with ratios upward of 2:1 (Axelson & Birmaher, 2001). One theory espoused by Mezulis, Hyde, Simonson and Charbonneau (2011) elaborates on the affective, biological, and cognitive risks that, when combined, may predispose females, specifically, to depression over males. The exact reasons for these gender differences remain unclear. Still, these gender differences have been found to be robust across cultures. For example, in a large study of Mexican youth, Benjet and Hernandez-Guzman (2001) reported that prevalence of depression were similar for boys and girls pre-puberty, and increased for females post-menarche but not for males post-puberty.

In a thorough review, Beardslee and Gladstone (2001) provided an overview of risk factors for the development of depression in boys and girls. These factors included having a biological relative with a mood disorder, presence of severe stressor, low self-esteem or hopelessness, being female, and low socioeconomic status (poverty). Males and females were characterized, at least partially, by a unique set of risk factors. For boys, but not girls, neonatal and subsequent health problems posed risk for depression; in girls, but not boys, death of a parent by age 9 years, poor academic performance, and family dysfunction were related to risk for depression. In another review, Duggal and colleagues (2001) indicated that early caregiving patterns, such as household stress and quality of early childhood care, predicted depression for adolescent males, but not adolescent females, whereas presence of maternal depression predicted depression in adolescent females, but not adolescent males. The patterns of gender differences in prevalence and risk are reasonably well established. Yet, the mechanisms contributing to gender differences remain elusive and the field is ripe for new ideas and investigations.

Prevalence Prevalence and incidence rates of depressive disorders vary across studies, and large-scale research is currently available via a meta-analysis. Gathering data from a period of 30 years from 1965 to 1996, Costello and colleagues (2006) synthesized data from over 60,000 children that used clinician interviews as the measure of depressive symptoms. Rates were generally consistent, with 5% of children meeting criteria for clinical depression, and this has been replicated in other studies (Rohde et al., 2012). The lifetime prevalence rates for the spectrum of depressive disorders in adolescents are between 5% and 21% in community samples (Diamantopoulou, Verhulst & van der Ende, 2011). Of course, rates increase for sub-clinical levels of depressive symptoms—depressive symptoms are reported by 50% of children and adolescents at one time or another in their lives (Costello et al., 2006).

Depression is a cyclic, often recurrent, disorder. Emslie and Mayes (2001) reported that 90% of youngsters recovered from a depressive episode within 2 years. Yet, within 6–7 years, 25–50% of them re-experienced significant symptoms and distress. In addition, within 8 years, 54–72% had a recurrent episode of depression. These relapse rates hold true for the treatment studies on depression in youth as well, which we describe in a later section.

Diagnostic Categories: DSM-5 In DSM-5, the general symptoms are consistent with earlier research about depression. There are four hallmark depressive disorders in DSM-5, with additional substance-related and unspecified depressive disorders also listed.

Disruptive Mood Dysregulation Disorder Disruptive mood dysregulation disorder is a new diagnostic category in DSM-5. It is only diagnosed in children between ages 6 and 18 years of age, and onset must be before age 10. The primary feature is chronic irritability of at least 6 months, accompanied by developmentally inappropriate temper outbursts. DMDD is distinct from ODD or irritability seen with bipolar disorder. DMDD should not be diagnosed concurrently with either ODD or bipolar disorder, and differential diagnosis is warranted. Symptoms may overlap with intermittent explosive disorder or autism spectrum disorder, and ODD. If criteria for DMDD are met, that should be the diagnosis. The differential diagnosis should reflect either DMDD, intermittent explosive disorder, ODD, or bipolar. Differential diagnosis is important, owing to the distinguishing features of DMDD relative to the other disorders with a similar presentation but different etiology and developmental trajectory.

Major Depressive Disorder The primary features of this disorder are at least 2 weeks of an interfering level of depressed/sad mood or loss of interest/pleasure in activities. In children and adolescents, irritability may be present instead of sadness, but not to the extent that would warrant DMDD. As noted in a prospective, large study, it is not typical to see irritability without the sad or flat

affect in children and adolescents with MDD (Stringaris, Maughan, Copeland, Costell & Angold, 2013). Additional symptoms include: weight/appetite change, sleep difficulties, psychomotor disturbances, fatigue, difficulties concentrating, thoughts about death/suicide, and feelings of worthlessness or guilt. In DSM-5, the severity of the depressive episode, as well additional features (i.e., anxious, melancholic, mood-congruent psychotic, etc.) is necessary in the diagnosis.

Persistent Depressive Disorder (Dysthymia) Persistent depressive disorder reflects a chronic, mild form of depression. It is only slightly revised in DSM-5. The condition is similar to MDD, but of lesser intensity or severity, and is longer in duration (2 years in adults, 1 year in children and adolescents). There are also specifiers to address additional features, similar to MDD, but also to note the presence of concurrent MDD, and severity.

Premenstrual Dysphoric Disorder Symptoms include extreme lability in mood, irritability, depressed mood or hopelessness, anxiety or tension within the week before a menstrual cycle, which improve during or after menstrual onset. This is a new category in DSM-5. It is a more severe set of emotional symptoms that accompany premenstrual syndrome. Premenstrual dysphoric disorder may occur in adolescent girls who have reached menarche, but may have higher prevalence or severity in women who approach menopause.

Differential Diagnosis and Comorbidity Another important (if not vexing) factor in the diagnosis and treatment of depression is the existence of comorbid disorders. Comorbidity is particularly relevant in assessment of suicide risk. Youths with depression and anxiety or disruptive disorders are at higher risk for suicidality (Foley, Goldston, Costello & Angold, 2006). Rates of comorbid diagnoses with depression are high, around 40%. Comorbid disorders most often include an anxiety disorder, conduct disorder, and substance abuse (Esbjørn, Hoeyer, Dyrborg, Leth & Kendall, 2010). Although depression may be the first-onset with some comorbid disorders (especially with substance abuse disorders), in general, anxiety disorders such as panic disorder and GAD frequently precede depression (Kessler et al., 2001). In externalizing disorders, such as conduct disorder, a model of comorbidity was presented by Capaldi and colleagues (Capaldi, 1992; Wiesner, Kim & Capaldi, 2005). The model elaborates on how early childhood behavioral challenges, school and social failure, and more serious conduct problems in adolescence eventually lead to depression after many repeated failures over time.

Although anxiety disorders are common comorbid disorders with depression, the two classes of disorders appear to be distinct in children, but related through a common construct of negative affectivity (Axelson & Birmaher, 2001; Seligman & Ollendick, 1998). In depressed youth, up to 75% may have a lifetime prevalence of a comorbid anxiety disorder, and between 45% and 50% may have a disruptive behavior disorder or substance use disorder (Kessler et al., 2001). Point prevalence rates for children and adolescents with depression and a comorbid anxiety disorder range from 25% to 50%; in contrast, point prevalence rates for anxious youths with depression range between 15% and 20%. In other words, it is more common for depressed children and adolescents to have a comorbid anxiety disorder than for anxious children and adolescents to have a comorbid depressive disorder. Presence of a comorbid disorder appears to relate to severity of symptoms, such that youngsters with major depression and comorbid anxiety possess more severe depressive symptoms (Axelson & Birmaher, 2001).

Etiological Considerations There is no single pathway to predict youth depression. Instead, multiple considerations are important. The literature has not yet reached a consensus about the etiology of youth depression in a way that clearly articulates the individual, biological, environmental, cognitive and social variables that predict vulnerability (Hankin, 2012).

Biological Factors Specific neurotransmitters have been implicated in the onset and course of depression in children and adolescents. One prominent theory suggests that select neurotransmitters such as the monoamines, norepinephrine, serotonin, and dopamine are not available at receptor sites in sufficient supply (Wagner & Ambrosini, 2001). Other theories, which have not yet been demonstrated with children, implicate poor growth hormone stimulation, as well as hypothalamic–pituitary–adrenal axis regulation difficulties (Axelson & Birmaher, 2001). There is also emerging research about the interaction between specific genetic vulnerabilities related to serotonin and other neurotransmitters and environmental stress (Hankin, 2012).

Family and Interpersonal Factors Family risk factors have historically focused on maternal factors, such as maternal depression or low maternal warmth and availability. More recent models include a more complex conceptualization of family risks for youth depression, including the contribution of fathers and mothers, genetic risk, and other environmental considerations, such as poverty and stressful life events. Goodman and Gotlib's (1999) integrative model of risk for transmission of depression from mother to child considers heritability, depressive maternal affect and symptoms, stress within a household with a depressed parent, and neuroregulatory consequences from that environmental circumstance. In addition, they

propose that the timing, severity, and duration of depressive symptoms interact with the developmental tasks and challenges for children (Goodman & Gotlib, 1999).

The Oregon Adolescent Depression Project (Allen, Lewinsohn, & Seeley, 1998) examined prenatal, neonatal, developmental, and family relationship factors in psychopathology. They reported several factors that were correlated with increased risk of adolescent depression, including maternal depression. In other studies, such as one based on the Ontario Health Survey data, childhood depression was related to parental mental health and experience of physical abuse in childhood; the higher the number of adverse events or chronic health issues, the higher the risk for depression during childhood (Gonzales et al., 2012).

Yet, relationship quality with parents may not be the most important consideration across developmental stages. In a study by Williams, Connolly and Segal (2001), the level of intimacy in romantic relationships predicted risk for depression in adolescents beyond other parental and friendship relationship factors. Sander and McCarty (2005) conducted a comprehensive review of family factors and interventions addressing family factors in depressed youth. Their conclusions were that many factors, including cognitive style of parents, parental psychopathology, emotional availability of parents, coping styles, and family conflict all contribute in important ways to risk for youth depression. Importantly, both mothers and fathers, along with other contextual factors, are important in conceptualizing risk (Sander & McCarty, 2005).

The causal effect of early relationship quality on later depression in children and adolescents is unclear, as is the contribution made by youngsters with a predisposition for depression to the negative quality of their interpersonal interactions. However, depressed youths do appear to have poorer social competence, and there does appear to be an intergenerational pattern of relational difficulties from mothers to adolescents (Katz, Hammen & Brennan, 2013). Professionals need to acknowledge and integrate interpersonal deficits or struggles, including peer and parental relationships, when conceptualizing and implementing treatment programs for depressed youngsters (Mufson, Weissman, Moreau, & Garfinkel, 1999; Stark et al., 2000). In addition, depression and its associated cognitive distortions appear to be related to interpersonal factors and, therefore, can or should be treated within an interpersonal context (Joiner, Coyne, Blalock, 1999).

Consistent with research on the role of interpersonal and cognitive variables in depression in youth, a cognitive-interpersonal theory of depression proposes that cognitive style and interpersonal relationship patterns combine to result in depression (Stark et al., 2000). From this perspective, Bowlby's (1980) attachment theory plays an important role. In brief, the early or primary interpersonal relationships are proposed to either buffer or increase risk for cognitive style associated with depression (Stark et al., 2000). According to this theory, early relationship patterns and the caregiver's responsiveness to the child shape the child's expectations of how they will be treated by others and how responsive others will be to their needs. An unresponsive caregiver, such as a depressed parent, could inadvertently communicate the message that the child's needs are unimportant, thus predisposing the child to adopt a negative self-schema (Duggal et al., 2001; Stark et al., 2000). Several scholars highlighted a model including cognitive-interpersonal pathways in a theoretical integration, emphasizing the importance of interpersonal, developmental and cognitive factors (Gotlib & Hammen, 1992; Rudolph, Flynn & Abaied, 2008). Additional research is necessary before a final model can be clarified to explain and predict the interpersonal context in connection with the other important risks.

Individual and Cognitive Variables Among individual variables, cognitive style, including attributional style, has received the most consistent empirical support. In brief, cognitive vulnerability to depression includes several components. It includes the concepts of negative thought patterns, depressive self-schema, and pessimistic attributional style about events (Beck, 2011; Stark, Schmidt, & Joiner, 1996). According to cognitive theory of depression, the self-schema guides information processing and may produce errors in perception that are consistent with a depressive self-schema - typically that the self is unlovable or incompetent (Beck, 2011). Attributional style refers to the patterns of causality assigned to events, such as how stable, global, and internal those events are perceived to be (Nolen-Hoeksema, Girgus, & Seligman 1992).

Depressed children and adolescents, in contrast to non-depressed children and adolescents, distort events and make information-processing errors that confirm negatively biased assumptions about the self (Stark et al., 1996; Jacobs, Reinecke, Gollan, & Kane, 2008). The negative attributional style, referring to the internal, stable, and global nature of negative events, is also a clear risk factor for youth depression (Rohde, Stice & Gau, 2012). Developmentally, children as young as 8–9 years of age make attributions about causality of events; before that age, attributional style in terms of cognitive vulnerability for depression may be unclear (Jacobs et al., 2008) and could be related to developmentally limited understanding of causality as well as the means to articulate the abstract ideas and emotional experiences.

Information-processing errors, such as a bias for depressive information, is evident in children as young as 5 years of age. At the same time, there are differences in how children present with cognitive and information-processing errors relative to adolescents. Children may display an inability to recall positive events, rehearse negative information about events, and have lower rates of positive self-descriptions, but, in some cases, the measures available are less sensitive to these aspects of children's information-processing errors (Jacobs et al., 2008).

Adolescents are typically able to articulate and report their cognitive errors, information processing patterns, and indicate their cognitive vulnerabilities to depression on established measures (Jacobs et al., 2008), which are described below.

Assessment The scope of this chapter does not allow for a thorough review of the assessment process. However, brief recommendations are put forth (see Maag & Swearer, 2005; and D'Angelo & Augenstein, 2012, for a thorough discussion of this topic). With depression in particular, it is important to include youth self-report, whether in interview or via self-report rating forms. In children in particular, the symptoms of depression may not be obvious based on behavioral observations or parent report alone (Weisz et al., 2006).

Within the field of depression, a diagnostic interview is helpful in assessing clinical depression. One option is the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Ambrosini, 2000; Ambrosini & Dixon, 2000). The K-SADS is a semi-structured diagnostic interview designed to be used by clinically trained interviewers, and can be administered to the youth and parent separately. The complete interview is cumbersome and was designed for research. However, the specific prompts and questions can be incorporated into an interview for clinical practice (Stark et al., 2000). The DSM-5 online assessment measures are another option; these include disorder-specific prompts and are consistent with the revised depressive disorder criteria and categories. The DSM-5 prompts would be appropriate to administer in a manner similar to the K-SADS.[1]

Other questionnaires and self-report measures can be administered efficiently and used in community or clinic populations, such as the depression scale of the Behavior Assessment Scale for Children (Reynolds & Kamphaus, 2004), the Beck Youth Inventory Depression Scales (Beck, Beck & Jolly, 2005), or the Children's Depression Inventory (Kovacs, 2010), but these are recommended only when used in conjunction with a clinician's interview and other sources of information to determine accurate diagnosis. Some these measures are available for no charge to download, such as the Mood and Feelings Questionnaire (Angold, Costello, Messer & Pickles, 1995; Duke University Health System, 2008).

Assessment of suicide risk and behavior is important. In adolescents, there may be a precipitating stressor that has compromised developmental tasks such as establishing autonomy, acceptance by their desired peer group, or conflict with important peers or with family members. The clinician must remain attuned to those potential events in relation to increased risk for suicide in youths (Rudd & Joiner, 1998), as well as assessing for concurrent anxiety, substance use and antisocial behavior, as discussed previously. Several self-report measures listed above include items and clusters of items that address suicidal risk and should be reviewed immediately following administration. There are also specific measures of suicidality that could be considered (Gutierrez, 2006).

Interventions Treatment of depression in youth is a somewhat daunting undertaking and relapse rates are typically high (40–50%) for clinically depressed youths (Asarnow, Jaycox, & Tompson, 2001). There have been several large-scale studies within the past 10–15 years, as well as a few thorough research synthesis studies on the topic, that are instrumental in advancing treatment for child and adolescent depression (Ollendick & Jarrett, 2009). Even so, the results and conclusions are not decisively clear, and practice guidelines and wide spread use of empirically supported interventions remain sparse. There are three main approaches in the treatment of child and adolescent depression—pharmacological interventions, family or interpersonal psychosocial treatment, and cognitive-behavioral interventions.

Pharmacological Interventions As with the anxiety disorders, there have been a limited number of controlled, randomized clinical trials of treatment of depression in children and adolescents, either pharmacological or psychosocial. Pharmacologically, the most promising results have been obtained with use of SSRIs (e.g., fluoxetine, marketed as Prozac®) to reduce depressive symptoms. However, rates of improvement have been 40% (Wagner & Ambrosini, 2001) to 60% at post-treatment (Reinecke et al., 2009). As a class of medications, the SSRIs are most likely to quickly reduce depressive symptoms and have fewer and less detrimental side effects than other antidepressants (Emslie & Mayes, 2001).

In the most extensive multi-site medication and psychosocial intervention study with adolescents to date, which included 439 adolescents from 2000 to 2003, the Treatment of Adolescent Depression Study (TADS; Curry et al., 2005) team reported that fluoxetine medication plus cognitive-behavioral treatment was the most effective intervention to alleviate depression in adolescents, with improvement rates at 81% at 3 months post-treatment (March et al., 2004). Fluoxetine was superior to cognitive-behavioral treatment alone in terms of providing earlier response to treatment and reduction of symptoms (March et al., 2004). Although the cognitive-behavioral interventions incorporated for the TADS study were criticized for being less rigorous than those used in cognitive-behavioral treatment studies (Hollon, Garber & Shelton, 2005), the conclusions from the large study do offer cautiously positive indications that medication, for some children and adolescents with depression, may be useful as a treatment component (Reinecke et al., 2009). It should be noted that even when medication was effective, particularly early on, in follow-up studies the relapse rate for medication alone was higher than that of the cognitive-behavioral approach (McCarty & Weisz, 2007).

Psychosocial Interventions In a synthesis of psychosocial interventions for depressed children and adolescents, Weisz and colleagues (2006) included 35 studies from 1980 to 2004 to examine efficacy of psychosocial treatment of childhood and adolescent depression. The research synthesis consolidated many smaller-scale studies and reported outcomes using a similar measure of effect size across studies. An effect-size calculation takes into account the means and standard deviations of the treatment and comparison groups, as well as sample size. The overall finding from the synthesis of all psychosocial interventions for depression that were available is a cautiously positive endorsement. Psychosocial interventions as a group certainly seem helpful in the short term (post-treatment). Investigations do not commonly include long-term outcomes, and follow-up effect sizes are not always available, but some results are promising, as discussed below.

Taking a closer look at the effective interventions at post-treatment, more immediately at the conclusion of treatment, not necessarily several months later, several studies had moderate effects. McCarty and Weisz (2007) defined a moderate effect size as .5 or greater in the meta-analysis, and they identified nine studies that stood out as successful. They provided a table of each of these studies, together with specific components within each treatment approach. All treatments had multiple components, and there were some components common across several interventions. The approaches to treatment in these effective interventions included cognitive-behavioral therapy, interpersonal therapy, cognitive therapy, and family therapy. One point to note is that these were all clinical trials, and the studies with the best treatment outcomes were those with a waitlist condition, rather than a comparison treatment condition (McCarty & Weisz, 2007). It would be helpful to have data comparing one intervention to another, but that information is not yet available. Nonetheless, results from these specific studies, when synthesized, indicate that the currently available psychosocial treatments using some common elements are efficacious.

Cognitive-Behavioral Therapy CBT has received the most empirical support among the psychosocial treatments (Curry, 2001). Components of CBT include affective education, planning positive activities, proactive problem solving, social skills training, coping strategies, and cognitive restructuring. There are several manualized CBT-based interventions for youth depression. The Coping with Depression for Adolescents (CWD-A) program (Clarke, Lewinsohn, & Hops, 1990; Rohde, Lewinsohn, Clarke, Hops, & Seeley, 2005), the ACTION program (Stark, Streusand, Krumholz, & Patel, 2010), and the Primary and Secondary Control Enhancement Training (Weisz, Gray, Bearman, Southam-Gerow, & Stark, 2005) are promising interventions. Family and interpersonal components may also be included. CBT has been implemented with children as young as 8–9 years of age. Among 15 CBT studies reviewed by Curry (2001), treatment duration ranged from 5 to 16 weeks. Some therapy protocols involved twice-weekly sessions, with an average of 11 sessions in total. New treatment investigations include an emphasis on family components, combined with cognitive-behavioral approaches (Asarnow et al., 2001; Curry, 2001; Ollendick & Jarrett, 2009).

There are two controlled clinical trials of high scientific rigor that address depression in youth from a cognitive-behavioral perspective. In addition, these studies included samples that are typical of real-world practice with complex, multiple diagnoses and clinical symptoms, the CWD-A group treatment (Lewinsohn, Clarke, Hops, & Andrews, 1990) and the ACTION program (Stark et al., 2006).

First, the CWD-A group treatment, which was included in the Weisz and colleagues (2006) research synthesis, is based on cognitive-behavioral principles and has been effective in reducing depressive symptoms in youths with a primary diagnosis of depression (Lewinsohn et al., 1990). The most recent empirical investigation of CWD-A is with a sample of 93 girls and boys (age 13–17 years) who had comorbid depression and conduct disorder (Rohde, Clarke, Mace, Jorgensen, & Seeley, 2004). Components of treatment included altering cognitions and behaviors, use of a structured intervention session, teaching mood monitoring, social skills, and pleasant activity scheduling. The CWD-A treatment did not appear to address the comorbid conduct disorder symptoms, but it did reduce depressive symptoms in this highly challenging and distressed group of youths at post-treatment (Rohde et al., 2004). At post-treatment, the CBT group had a 39% recovery rate for depression, compared with 19% in the life skills condition. The difference between conditions was not maintained over time, but the life skills and CBT groups all had reduced symptoms of depression over a 1-year follow-up. Given the overall challenges of working with this complex clinical picture, these results are notable at post-treatment in terms of some psychosocial interventions that reduced depression.

Another CBT approach is the ACTION program. This program (Stark et al., 2010) is a cognitive-behavioral group treatment for young adolescent girls (age 9–13 years). It has not been included in a research synthesis, but it is nonetheless a well-designed study with two active treatment groups and one waitlist condition. The ACTION program included 158 girls who had a primary diagnosis of depression, and the majority (60%) had dual diagnoses, most often an anxiety disorder (Sander, 2004). The ACTION program is delivered over 11 weeks, with a total of 20 group meetings and two individual meetings with the girls. There is a child workbook and therapist manual. An individual case conceptualization is created for each girl in treatment so that the therapist incorporates the girl's individual needs throughout the group intervention. There

is also a manualized parent component to treatment in the ACTION program, with focus on parental support for the skills and tools the girls learn during the group treatment (Stark et al., 2004). Based on post-treatment diagnostic interviews, 84% and 81% of girls who received CBT or CBT plus parent training, respectively, no longer met diagnostic criteria for any depressive disorder. This was compared to 46% of girls in a minimal contact control condition. One of the key elements of treatment for girls at this particular age emphasized by Stark and colleagues is the behavioral activation component of CBT. This component includes active problem-solving, coping strategies, and pleasant activity scheduling. Although the outcomes have not yet been included in another research synthesis or in terms of treatment effects that would allow for examination of effect sizes, the overall clinical findings are promising.

Interpersonal Therapy Interpersonal therapy for depressed adolescents (IPT-A) has also received promising empirical support. In IPT-A, focus is on resolving conflicts in current, important, interpersonal relationships and improving communication and relationship skills (Mufson, Moreau, Weissman, & Klerman, 1993; Brunstein-Klomek, Zalsman & Mufson, 2007). The goals of IPT-A are to reduce depressive symptoms via improving interpersonal functioning. The main areas to consider for improvement in interpersonal areas are: grief, role disputes, role transitions, and interpersonal deficits (Mufson, Dorta, Olfson, Weissman, & Hoagwood, 2004). The empirical support for this intervention from one major clinical trial (Mufson et al., 1999) was included in McCarty and Weisz's (2007) summary. In a more recent study, IPT-A was delivered in school clinics located within urban, low-income neighborhoods to a sample (N = 57) of predominantly Hispanic children, 12–18 years of age. Results indicated IPT-A was beneficial and feasible within these school-based clinics as well as in more controlled settings from earlier clinical trials (Mufson et al., 2004).

Family Therapy The most promising family therapy approach for youth depression is currently attachment-based family therapy (Diamond, G. S., Reis, Diamond, G. M., Siqueland, & Isaacs, 2002). This intervention was included in the McCarty and Weisz (2007) synthesis of the nine most effective interventions. The treatment objectives of attachment-based family therapy are not solely to reduce depressive symptoms, but to improve relationships, repair relationally based wounds such as abandonment, and increase empathy among family members. The reduction of family conflict and discord is also important, as is fostering trust among family members.

Suicidality Managing suicidal risk is very important to include early in treatment. Integrating appropriate intervention to reduce suicidality is a necessary skill when working with depressed youngsters, particularly as outpatients. Managing this risk involves several straightforward strategies (Rudd & Joiner, 1998). The first is to have a proactive stance and a plan for potential hospitalization, to be revised or revisited frequently during suicide risk periods. In addition, treatment progress and goals should be revisited and updated often, particularly noting the changes that co-occur with decreased suicidal ideation. Sessions may need to occur more frequently, and the family's involvement and a phone contact list, as well as availability of emergency 24-hour support, is necessary. Furthermore, full consideration of medication assessment and monitoring, as well as consultation with medical professionals to manage this aspect of treatment, is recommended. In the large-scale TADS study, the combination of CBT and medication was found to be the most effective in reducing suicidality. The specific addition of CBT with the medication seemed particularly helpful in attaining a quicker reduction in suicidal thoughts (Reinecke et al., 2009).

In summary, although the available treatments for depressed and suicidal children and adolescents show promise, several important caveats should be kept in mind. Overall, recovery rates across broadly defined interventions are generally at or below 50% (Asarnow et al., 2001; Curry, 2001; Ollendick & Jarrett, 2009). The most successful interventions, at least in clinical trials, achieve recovery rates at 80% or better at post-treatment, but long-term follow-up rates are rarely available. Moreover, relapse rates are high. Also, comorbidity has been rarely addressed in treatment efficacy studies, and it remains an important factor to consider in day-to-day clinical practice as well as in major clinical outcome trials (Curry, 2001). Recent studies are including comorbidity as part of the design, rather than focusing on single-diagnosis participants. Psychosocial interventions, particularly the CBT-based approaches, are moving in new directions to reflect a more integrative treatment, incorporating individual, cognitive, relational, and interpersonal factors. One possible resilience factor is higher overall cognitive ability (Weeks et al., 2014), which could relate to better prognosis and response to cognitive-based interventions. Consideration of the individual's risk factors, developmental level, and ability to implement skills addressed in the evidence-based interventions, will all guide selection of intervention approaches.

Summary

The research on child and adolescent depression offers a number of exciting and promising findings. Treatment research is ongoing, including new directions involving biological and interpersonal factors. Although efficacious treatments exist, both pharmacological and psychosocial (primarily CBT, IPT-A, and attachment-based family therapy), not all youth are being helped by these interventions. Furthermore, the issue of comorbidity has not been adequately addressed nor has the role of culture and ethnicity. The protective factors and risk factors associated with

different cultural and ethnic practices have yet to be examined in standard research and clinical practice. These could be pivotal factors in understanding and treating childhood depression in our increasingly diverse society.

Future Directions

The internalizing disorders of children and adolescents represent a major challenge to practicing clinicians and researchers alike. As we have seen, the anxiety and affective disorders are highly prevalent in childhood and adolescence. Their effects are distressing in the short run and can be long lasting.

Although we have attempted to illustrate a developmental psychopathology perspective when examining these disorders, it is evident that more research is needed to fully understand these problems. Many studies continue to look at single causal pathways and direct, linear outcomes. As we have suggested, it may be more productive to posit that several very different pathways can lead to any one outcome such as depression or anxiety (i.e., the developmental principle of equifinality) and that any one pathway can lead to diverse outcomes (i.e., the principle of multifinality). Thus, a risk factor such as child sexual abuse can and frequently does lead to multiple outcomes, whether they are an internalizing disorder or one of the externalizing disorders. Tracking this developmental process is undoubtedly a complex and challenging undertaking, but a necessary one. This effort will benefit greatly from carefully planned, multi-site, longitudinal studies (Weisz, Sandler, Durlak, & Anton, 2005).

Our treatment and prevention programs must move away from a "one size fits all" mentality to address the complexity of each child or adolescent, and the bio-psychosocial context. Children and adolescents become depressed or anxious through a variety of pathways, and they express these disorders in a variety of ways. We need to take this complexity into consideration in designing, implementing, and evaluating our interventions. Although current empirically supported or evidence-based interventions work with most (i.e., 50–70%) of the children and adolescents and families who come into our practices and research clinics, we must do better. An approach that is individualized and prescriptive, and one that is based in developmental theory and grounded in established principles of behavior change (e.g., exposure, cognitive change, interpersonal relationships), is likely to be most effective, although such remains to be carefully documented.

Note

1. These and other online measures are available from the American Psychiatric Association website: http://www.psychiatry.org/practice/dsm/dsm5/online-assessment-measures (accessed May 7, 2015).

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21

Autism Spectrum Disorders

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Autism spectrum disorder (ASD) is a chronic and highly debilitating neurodevelopmental disorder. Like all neurodevelopmental disorders (e.g., intellectual disabilities, communication disorders), ASD begins early in life and is characterized by deficits that impair the individual's functioning across multiple life domains (e.g., academic and social). ASD is usually identified and diagnosed during childhood, and most affected individuals retain the diagnosis through adulthood (Cederlund, Hagberg, Billstedt, Gillberg, & Gillberg, 2008). The major diagnostic features of ASD are *social communication/interaction deficits* (i.e., impaired reciprocity, absent or delayed nonverbal interpersonal communication, and impoverished social relationships) and *presence of restricted, repetitive behaviors or interests* (e.g., insistence on keeping rigid routines, hyper/hyporeactivity to sensory stimuli) See McPartland and Dawson (2014) for a review of the history of the diagnosis and changes to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 2013) and the International Classification of Diseases (ICD; World Health Organization, 1992).

Diagnosis of Autism Spectrum Disorder

ASD is more common than childhood diabetes—affecting approximately one in 88 children (Centers for Disease Control and Prevention, 2012). Some of the earliest markers of the disorder include behavioral regression or loss of language skills; lack of cooing, babbling, or other socially meaningful gestures by around 12 months of age; no single-word speech by 16 months; and a lack of two-word spontaneous phrase speech by 24 months of age (Johnson & Myers, 2007). Screening for possible ASD usually involves behavioral questionnaires completed by the caregiver, such as the Social Communication Questionnaire (Rutter, Bailey, & Lord, 2005) or Social Responsiveness Scale-2 (Constantino & Gruber, 2012). A full diagnostic evaluation for ASD should include measures specifically designed and validated for ASD assessment, such as the Autism Diagnostic Observation Schedule (Lord et al., 2012), as well as cognitive testing and assessment of adaptive behavior. Ideally, a full diagnostic assessment will be conducted (e.g., with semi-structured clinical interview, teacher reports of behavior) to adequately consider the possibility of other disorders that may be comorbid or that might better explain the presenting concerns, rather than only an ASD-specific evaluation. It is also recommended that other professionals (e.g., speech therapist, genetics specialist) be involved to the extent possible, as ASD often co-occurs with other conditions, such as fragile X.

Challenges Ahead

An in-depth account of the history of ASD is beyond the scope of this chapter. For detailed commentaries on the history of ASD and debate about nosology, the reader is referred to Davis, White, and Ollendick (2014). What we now label "ASD" has been recognized, in assorted forms and by various terms, since at least 1943, with Kanner's (1943) account of 11 children who demonstrated idiosyncratic language, impaired socialization, and repetitive behaviors. The label "infantile autism" first became part of our clinical nosology 40 years later with the publication of the third edition of the DSM (DSM-III; American Psychiatric Association, 1980) and, since this initial introduction, the scope of the diagnosis has broadened and

its nuances have been elaborated—largely as a function of ever-growing research and public interest, as well as increased clinical recognition and diagnosis.

The most fundamental, and hotly debated, change in the diagnostic criteria for autism from the fourth edition of the DSM (DSM-IV; American Psychiatric Association, 1994a) to DSM-5 was the elimination of all sub-diagnoses (i.e., pervasive developmental disorder–not otherwise specified; Asperger's disorder; childhood disintegrative disorder; autistic disorder) and development of one umbrella diagnostic label of "ASD." The diagnosis of ASD thus replaced the category of pervasive developmental disorders, under which autistic disorder, pervasive developmental disorder–not otherwise specified, and Asperger's disorder fell. Second, the criteria for the diagnosis were restructured. Since the 1970s (e.g., Rutter, 1978), ASD comprises a triad of impairments in the domains of social impairment, communicative deficits, and restricted interests and repetitive behaviors. In DSM-5, these three domains are reduced into two broad domains: social communication features and restricted interests features. Language delay is no longer diagnostic; it is identified as an *associated* condition. Third, for diagnosis, an individual must present persistent deficits in every subdomain of the first domain (social communication impairment). In DSM-IV, there were no broad criteria in which each sub-domain had to be present, making the predecessor a more polythetic approach, which has undoubtedly contributed to some of the vast clinical heterogeneity seen in ASD. Although there are other changes in DSM-5, such as greater recognition of the possibility that problems might not be apparent until later childhood or even adulthood, these three represent the most substantive modifications.

The clinical and scientific consequences of these changes are, as yet, unknown. Clinically, individuals with diagnoses now subsumed under ASD (e.g., Asperger's disorder) who were evaluated using DSM-IV criteria, are to be 'grandfathered' in, meaning that the DSM-5 diagnosis of ASD applies without need for reevaluation. Newly diagnosed individuals who would have previously been diagnosed with Asperger's disorder now would likely be diagnosed with ASD. Nonetheless, there are likely to be some challenges ahead with respect to resource allocation and services. The composition of study samples over time will likely differ considerably, assuming inclusion criteria for new studies are based on current nosology. This will affect our ability to make comparisons over time across the samples in studies. This issue, of course, has been faced in the past with DSM revisions. However, the impact might be greater and harder to navigate with ASD because of the scope of the diagnostic changes seen in DSM-5 and the quick pace with which new research is being conducted and produced now and over the past 5 years in this field (e.g., Reichow & Volkmar, 2011). There is much greater attention given to possible comorbidities with ASD (e.g., social anxiety disorder, attention-deficit/hyperactivity disorder), which should ease differential diagnostic decisions when both disorders truly do present in the same person. There is also more consideration for the severity of the disorder related to daily adaptive behavior. This should yield more clinically informative data than the "sub-diagnosis" approach that may have obfuscated diagnosis with adaptive behavior (e.g., high-functioning being synonymous with either pervasive developmental disorder–not otherwise specified or Asperger's disorder). The more unified the criteria, the more likely the diagnosis will yield more homogeneous samples and will thus increase our ability to identify disease biomarkers and establish efficacious treatments with discernable underlying change mechanisms. In summary, although there are several weighty concerns (e.g., under-identification and poor sensitivity), the changes seen in DSM-5 are empirically based and are consistent with changes seen for other psychiatric disorders—moving toward a more consistent, individualized, and dimensional approach to diagnostic categorization.

Etiology

Historically, autism was first thought to be the result of highly educated parents, especially mothers, who lacked warmth in their parenting. These parents were described as "refrigerator mothers" (Kanner, 1954; Rapin, 2011). Advances in the field, including development in fields such as psychopharmacology and neuroscience, led researchers to examine autism with a biological lens (Rapin, 2011). Owing to increased prevalence and awareness of ASD, research on its etiology has advanced considerably over the previous 40 years. Among the initial forays of genetic research in ASD in the 1970s and 1980s was research on associated genetic conditions, such as fragile X syndrome, epilepsy, and tuberous sclerosis (Freitag, 2007; Rapin, 2011), as well as twin studies and family heritability studies (Rapin, 2011). As new genetic, neuroscience, and epigenetic and environmental methods emerged, additional information concerning ASD etiology has continued to accumulate.

Biogenetic Factors Research concerning familial aggregation of ASD and its symptomatology has provided indicators of the nature of its etiology. Twin studies of heritability in ASD, in which monozygotic and dizygotic twins are compared, have found that up to 90% of ASD has a genetic cause (see Rutter, 2005, for a review). Owing to the likely genetic underpinnings of ASD, much research has used a sample of younger siblings of children diagnosed with ASD to investigate the developmental trajectories in ASD. This research on siblings of children diagnosed with ASD has shown that approximately 15–20% of siblings of a child with ASD are also diagnosed with the disorder, and an additional segment of siblings not diagnosed with ASD at age 3 years nonetheless present with delays in motor and language functioning (Messinger et al., 2013; Rogers, 2009). Research on first-degree relatives of individuals with ASD, mostly with adult samples, has also shed light on the existence

of subclinical traits of ASD in what has been termed the broader autism phenotype (Klusek, Losh, & Martin, 2014; Sasson, Lam, Parlier, Daniels, & Piven, 2013). Social difficulties, difficulties with sociocommunicative interactions, restricted and repetitive interests and behaviors, and personality traits such as rigidity comprise the broader autism phenotype, a term used to describe genetic relatives of individuals with ASD who may have characteristics associated with ASD but who nonetheless do not have ASD diagnoses. First-degree and extended relatives of individuals with ASD have been found to score higher on measures of broader autism phenotype symptomatology, lending evidence to the genetic basis of ASD (Pickles et al., 2000; Piven & Palmer, 1999; Sasson et al., 2013). In addition to research on family members, broader autism phenotype characteristics also present in subclinical levels among the general population (Hurley, Losh, Parlier, Reznick, & Piven, 2007).

Overall, research on the genetic basis of ASD has highlighted the heterogeneous nature of the disorder. Only a minority of cases of ASD are due to a single gene mutation; the majority of cases appear to be due to multiple genetic mutations, including duplications and deletions, which can be inherited from parents' genetic information or due to mutations at the individual's conception (Geschwind, 2008). Thus far, studies have had divergent findings in genetic linkage studies, where individual genes have only accounted for up to 1–2% of cases of ASD (Geschwind, 2008).

Brain-Based Factors Much of the previous research on the etiology of ASD has focused upon neurological methodologies, such as functional and functional connectivity magnetic resonance imaging (fMRI and fcMRI; McFadden, Minshew, & Scherf, 2011). Overall, imaging data suggest that ASD affects no single area of the brain, but instead is pervasive in altering functioning. Structural differences are seen as well; while there is no uniform pattern of structural difference, a majority of individuals with ASD show abnormally increased gray and white matter growth in the first several years of life, which is then followed by a dampened growth trajectory that results in average brain volume, gray matter, in adolescence and adulthood (McFadden et al., 2011). fcMRI studies, in particular, have demonstrated that individuals with ASD have less white matter connectivity, especially in the frontal regions and connecting across the brain, and greater connectivity within the parietal, visual, and temporal cortices (Just, Cherkassky, Keller, & Minshew, 2004). This pattern of under- and overconnectivity may serve to explain some commonly reported of ASD phenotypic expression—specifically superior visual or auditory processing, alongside impairment of executive functioning and other higher-order processes (McFadden et al., 2011; Philip et al., 2012). Many of the domains of functioning most impaired in ASD, such as social cognition, theory of mind, pragmatic and idiomatic language, and conceptual thinking, are those which require coordination of several distinct neural regions; conversely, intra-regional skills such as simple motor movements, formal and rule-based language, and memory are sometimes areas of strength for people with ASD (McFadden et al., 2011). A review of studies comparing individuals with ASD and neurotypical individuals found differences in neural areas associated with motor processing, visual processing, executive functioning, both simple and complex social processing tasks, cortical connectivity, and a lack of preference for social stimuli, as indicated in under-activation of the fusiform face area during tasks which require participants to attend to faces and eyes (Philip et al., 2012; Russo et al., 2007; Vissers, Cohen, & Geurts, 2012).

Psychosocial/Environmental Factors Although the current consensus is that ASD is predominantly genetic in origin, increasing attention has been paid to the identification of potential environmental factors that may contribute to ASD, largely due to the increased prevalence and awareness of ASD among the general population.

Another growing area of research in the environmental risk factors for ASD is epigenetics, the study of how gene expression is altered. Many factors, including parental age, infection during pregnancy, and exposure to environmental toxins during pregnancy or postnatally, have been examined in large-scale epidemiology studies as factors that could potentiate expression of underlying genetic vulnerability. Overall, studies suggest that no single environmental factor is strongly associated with later ASD diagnosis, and instead each environmental agent may contribute synergistically to existing genetic vulnerabilities to result in ASD (Hertz-Picciotto, 2011; Persico & Bourgeron, 2006).

The most well-known, and hotly debated, environmental agent linked to ASD has been vaccines, in particular the preservative used in the measles, mumps and rubella vaccine, thimerosol. Thimersol, which contains mercury, was studied as a potential environmental factor associated with the increase in ASD diagnoses (Gerber & Offit, 2009; Stehr-Green, Tull, Stellfeld, Mortenson, & Simpson, 2003). However, research on a global scale, including regions where usage of preservatives in vaccines differs, has shown that vaccines and thimerosol in particular do not have an effect on ASD prevalence (Stehr-Green et al., 2003). Nevertheless, some parents have chosen to forego vaccinating their children because of continuing concerns about the association of vaccines to ASD, which has contributed to the reemergence of preventable diseases (Poland, 2011).

Culture and Gender There is some evidence for the effects of gender and cultural factors on identification of ASD. For example, age at diagnosis has been found to vary on several culturally relevant factors, including socioeconomic

status and geographic location. Worldwide, while recognition of ASD has continued to increase, there remains scant large-scale data concerning prevalence or research on ASD in many areas outside of North America, Western Europe, and some Asian nations, and the similarity of core symptoms and onset of ASD worldwide remains an assumption (Grinker, Yeargin-Allsopp, & Boyle, 2011).

Within the nations where ASD is more intensively studied, cultural factors are still a significant indicator of when an individual is diagnosed with ASD. Initially, ASD was thought to be limited to children of highly educated parents and some researchers posit that ASD is more common among children of engineers and scientists (Baron-Cohen, 2004). Socioeconomic status does appear to effect identification (specifically *when* an individual is initially identified and diagnosed), though not actual occurrence of ASD. A study of Pennsylvania families in which a child was diagnosed with ASD found that the average age at diagnosis was higher for children with fewer language difficulties and less severe symptom presentations, but that children located in rural areas and children of families in poverty or near-poverty levels were significantly older (0.4 and 0.9 years, respectively) when they received their diagnosis (Mandell, Novak, & Zubritsky, 2005). Racial and ethnic identity have been found to be associated with age of initial diagnosis, with individuals of ethnic minorities receiving their diagnosis at least 1 year later than White children, although this finding has not been demonstrated consistently in the literature (Mandell, Listerud, Levy, & Pinto-Martin, 2002; Mandell et al., 2005). Factors such as geographic location and socioeconomic status may interact with race and ethnicity (Mandell et al., 2002). Given the importance of early identification and treatment in achieving optimal outcomes among individuals with ASD, these cultural differences in access to services and obtaining an initial diagnosis can have considerable consequences.

Since Kanner's (1943) first observations of children diagnosed with autism, ASD has been found to occur more frequently in males than females. The ratio of males to females diagnosed has ranged from 3:1 to 7:1, with some studies finding that fewer females are diagnosed, especially at the higher-functioning edge of the spectrum. Females with ASD are also more likely to be diagnosed later in life than males (see Kreiser & White, 2013, for a review). However, researchers have also argued that this discrepancy in diagnosis across gender may reflect methodological biases in our assessment practices and lack of sensitivity in the diagnostic criteria to gender-based differences in manifest expression, in addition to actual sex-based etiological differences (Kreiser & White, 2013; van Wijngaarden-Cremers et al., 2014). These arguments center on a different presentation of ASD in females, particularly those who are higher functioning. This "female prototype" of ASD is characterized by better mask sociocommunicative deficits, alongside fewer stereotyped behaviors and routines and restricted interests that appear less unusual than is often seen in males with ASD; ambivalent social motivation; and increased internalizing (e.g., poor emotion regulation) symptoms (Kreiser & White, 2013; Trubanova, Donlon, Kreiser, Ollendick, & White, 2014).

Treatment of ASD

We must consider evidence-based treatment for ASD a bit differently than we might normally for most other mental health disorders, owing to the pervasiveness of impairment and the multi-faceted impairments and symptoms for which a person with ASD seeks (or, more often, is referred for) treatment. More to the point, treatment is usually sought for problems that are co-occurring (e.g., aggression) rather than core ASD symptoms (Joshi et al., 2010). Given the chronicity of the disorder, interventions are not intended to cure ASD or treat to remission. Yet another factor that complicates identification of evidence-based treatments for ASD is the tremendous phenotypic heterogeneity present among people diagnosed with ASD (e.g., in verbal and cognitive ability, in severity of core and secondary symptoms). As such, identification of evidence-based treatments for ASD is a complicated and nuanced endeavor. Herein, we describe the most commonly used pharmacological and psychosocial interventions, and also summarize interventions that are contraindicated.

Biological/Pharmacological Interventions It is estimated that over half of children and adults diagnosed with ASD are treated pharmacologically (Mandell et al., 2008). Once medicated, young people with ASD tend to remain on psychotropic medication during adolescence and into adulthood (Esbensen, Greenberg, Seltzer, & Aman, 2009). Additionally, pharmacological intervention for ASD tends to target specific secondary problems (e.g., anxiety, irritability) rather than ASD core symptoms such as social disability (Malone, Maislin, Choudhury, Gifford, & Delaney, 2002).

Most pharmacological treatment for people with ASD is done off-label, as the only medications approved by the U.S. Food and Drug Administration for individuals with ASD are risperidone and aripiprazole, both of which are indicated for reduction of irritability (including extreme aggression). The most commonly prescribed drug classes are neuroleptics (atypical antipsychotics), antidepressants, and stimulants (Mandell et al., 2008). Repetitive and stereotyped behaviors are often treated with serotonin-reuptake inhibitors, although careful monitoring of adverse effects is encouraged (Vahabzadeh, Buxton, McDougle, & Stigler, 2013). There is general agreement that severe irritability (e.g., temper tantrums, physical aggression) can be treated effectively pharmacologically, although not without considerable adverse effects such as increased appetite and weight gain, tremors, and fatigue (e.g., Owen et al., 2009; Scahill, Koenig, Carroll, & Pachler, 2007). Supplementation of pharmacological treatment with a

psychosocial approach (e.g., parent training) may be most effective (Aman et al., 2009), although there has been little research on the use of such integrated approaches. Most of the clinical research in this field has been done with children and adolescents and, as a result, we know fairly little about pharmacological treatment for adults with ASD. There is active research on other compounds, and it is likely that the next decade will see considerable improvements with respect to our knowledge of pharmacological interventions. For example, recent research on the use of oxytocin for social impairments in people with ASD has produced very promising findings (Sikich et al., 2013; Wudarczyk, Earp, Guastella, & Savulescu, 2013).

Psychosocial Interventions There are many thorough, informative reviews on evidence-based treatments in this area, of which the most recently published (Wong et al., 2013) used five databases and generated over 29,000 articles published in just a 21-year period (1990–2011). In the most recent report from the National Professional Development Center on ASD, 27 evidence-based practices (defined as those to be shown effective through high-quality scientific research) were identified (Wong et al., 2013). The National Autism Center (2009), likewise, has developed empirically informed guidelines for treatment of ASD in the schools, dividing approaches into those that are "emerging" (22 treatments identified as such) and those that are "established" (11 treatments identified as such; e.g., antecedent packages, modeling). These large-scale reviews have applied stringent criteria by which to rate the quality of the reviewed treatment research, and are extremely useful resources. The *Encyclopedia of Autism Spectrum Disorders* (Volkmar, 2013) and the fourth edition of the *Handbook of Autism and Pervasive Developmental Disorders* (Volkmar, Paul, Rogers, & Pelphrey, 2014) are also valuable resources, offering summaries of empirically based interventions, and critical analyses of the extant research. In this chapter, we do not cover all of the comprehensive and focused psychosocial treatments for ASD. Rather, we concentrate on approaches that can be used to target a range of clinical foci: behavioral/cognitive-behavioral interventions, skills training interventions, and educational interventions. Several intervention approaches that have been well-researched and supported for treatment of core deficits in ASD, such as augmentative communication systems (e.g., Picture Exchange Communication System; Sulzer-Azaroff, Hoffman, Horton, Bondy, & Frost, 2009) and comprehensive educational and treatment programs (e.g., Treatment and Education of Autistic and related Communication handicapped Children; Panerai, Ferrante, & Zingale, 2002) are not covered.

Behavioral/Cognitive-Behavioral Interventions Of the approaches used with people who have ASD, applied behavior analysis is the most widely used and empirically supported treatment (e.g., Lovaas, 2003). Common targets of applied behavior analysis include teaching new skills such as verbal speech, mathematics, and daily living skills, and reducing undesirable or dangerous behaviors such as self-injurious behaviors, tantrums, and disruptive behaviors such as leaving the classroom or screaming when instruction is taking place. Intensive applied behavior analysis (i.e., up to 40 hours per week) is usually recommended for children under the age of 4 years who are diagnosed with ASD (e.g., Eikeseth, Smith, Jahr, & Eldevik, 2007; Harris & Handleman, 2000; National Research Council, 2001), and there is evidence that the effect of applied behavior analysis is strongest when dosage is high and the client is very young (Smith, 2010). Applied behavior analysis is based on the principles of operant condition, or stimulus-response learning (Skinner, 1938). The particular technique of discrete trial training, often used with young children with ASD, relies on structured prompting and reinforcing of specific, targeted skills and behaviors. Applied behavior analysis is a highly structured, data-based approach. Individual (one-on-one) instruction is the mainstay and, as such, the approach is costly.

Behavior therapy and cognitive-behavioral therapy (CBT) are often used to treat secondary (non-core) problems in people with ASD as well (see Scarpa, White, & Attwood, 2013). Approximately 40% of higher-functioning people with ASD have co-occurring problems with anxiety (van Steensel, Bogels, & Perrin, 2011). CBT is a supported treatment for anxiety in children and adolescents with ASD (e.g., Chalfant, Rapee, & Carroll, 2007; White et al., 2013). Although depression is also common in ASD, estimated to effect up to 40% of children and adolescents with ASD (Strang et al., 2012), there is insufficient research to know if CBT is effective for treatment of depression in ASD. Many children with ASD are referred because of problems with aggressive behavior (Johnson & Myers, 2007). For such cases, a functional assessment is first used to identify the factors that maintain or exacerbate the behaviors (Matson, 2009), followed by behaviorally based interventions such as parent training (e.g., teaching the parent to consistently deliver consequences and use visual schedules to ease transitions) and environmental modifications (e.g., reducing noise level to decrease baseline arousal) (e.g., Johnson & Myers, 2007; Research Units on Pediatric Psychopharmacology, 2007).

In implementing a CBT treatment program with a client with ASD, modifications in both content and approach are often made to improve response. Although it has not been scientifically established which (if any) modifications are necessary for CBT to be helpful for people with ASD, we review some of the most common modifications. Behavioral experiments and practices (e.g., practice, self-talk) tend to be emphasized over the more cognitively oriented tasks (e.g., emotion recognition, thought challenging) of CBT (Lang, Regester, Lauderdale, Ashbaugh, & Haring, 2010), although there is evidence that children

with ASD do not uniformly show deficiency in cognitive skills likely to influence response to CBT intervention. Lickel and colleagues (2012) showed that children with ASD performed similar to typically developing peers on tasks of thought/feeling/behavior discrimination and cognitive-affective inference, but significantly poorer on tasks of emotion recognition. With young clients, parents are often more involved in treatment than what might be typical of individual CBT for a child without ASD. Given the chronicity of ASD and the pervasiveness of impairments, it can be helpful to educate the family about the disorder and the treatment, encourage familial support in the intervention, and have them involved in outside-session practices (e.g., Sofronoff, Attwood, & Hinton, 2005; White et al., 2010). Consistent, directive feedback as opposed to indirect or subtle feedback, and intensive practice (e.g., of new skills) are often emphasized as well. Finally, incorporation of teaching aides that involve the client's interests or address some aspect of his learning style (e.g., using visual supports with a client who processes auditory information slowly) can help maintain interest and motivation (White et al., 2010).

Skills Training Interventions The hallmark characteristic of ASD is pervasive social disability (American Psychiatric Association, 2013). Interventions are often aimed at improving general social competence in children, adolescents, and adults with ASD. Although interventions to improve social functioning in youth with ASD have generally demonstrated promising results (Reichow & Volkmar, 2011; Wang & Spillane, 2009), the supporting evidence has not been consistently strong, and common methodological limitations (e.g., primarily parent report measures and use of wait-list comparison conditions) have hampered comparative evaluation across programs. There are, however, many intervention models to target social disability in ASD, with an emerging base of empirical support including applied behavior analysis-based behavior modification, peer as interventionist and tutor models, social stories, computer-based training games, and video-modeling, many of which we discuss below. There are also resources available on how to design individualized programming to address the social problems of children with ASD (e.g., White, 2011).

Social skills training in a group format is common and, although research indicates such an approach is feasible and often helpful (effective), there has been little research comparing active (i.e., not just a wait-list control) programs and very few controlled trials (White, Keonig, & Scahill, 2007). There are many commercially available curricula developed specifically for young people with ASD (e.g., Baker, 2003; Bellini, 2008; Laugeson & Frankel, 2010; McAfee, 2002). Regardless of the approach or curriculum taken, it is generally suggested that parents (or other family members) be involved and that practice be frequent across contexts, to help promote generalization of learned skills (White et al., 2007).

Video-based and computer-delivered interventions to improve social functioning have also been evaluated empirically (e.g., Hopkins et al., 2011; Tanaka et al., 2010). Such technology is appealing as an intervention modality for many reasons, including capacity to increase "dose" of intervention without necessarily adding cost, allowing tighter controls over the intervention parameters in a "safe" environment, and an-oft reported affinity for technology and computers among many individuals affected by ASD. Although this field is still early in its development, there is considerable promise of feasibility (Wainer & Ingersoll, 2011). We are sure to see much more research on the efficacy of such programs in the next few years.

Educational Interventions Educational interventions aim to improve learning, and decrease behaviors that interfere with learning and functioning, within the typical school (general education) setting. A full review of the scope of educational interventions is beyond the scope of this chapter. We do not, for instance, review available comprehensive special education programs for learners with ASD (e.g., TEACCH; Mesibov, Shea, & Schopler, 2005; Learning Experiences and Alternative Program for Preschoolers and their Parents; Strain & Hoyson, 2000). Instead, we focus on more targeted intervention approaches typically implemented within the regular education classroom.

When a behavior is identified as detrimental to the student's progress, the first step is usually a functional assessment of the behavior to identify the goal(s) of the behavior, as well as precipitants (antecedents) and maintaining processes (consequences; Gresham, Beebe-Frankenberger, & MacMillan, 1999; Gresham et al., 2004). The functional assessment begins with data gathered from teachers and support staff (e.g., via interview) and careful, direct observation of the learner. Once hypotheses are formulated to try to explain the occurrence of the behavior, target variables (e.g., amount of stimulation in environment, teacher's choice of response to the behavior) are systematically altered while the behavior is closely tracked. Through this systematic, experimental process one can reduce (or increase, if that is the intent) the behavior. Functional assessments, within an inclusive school setting, can be useful for a range of difficulties, such as aggression, elopement, and work refusal (e.g., Williams, Johnson, & Sukhodolsky, 2012).

Antecedent-based approaches are implemented prior to the occurrence of a target problem behavior (National Autism Center, 2009). Such approaches, sometimes referred to as stimulus control or environmental modification, are often sufficient to address the behavior. Within this broad class of intervention approaches we include use of visual cues and prompts, chaining procedures (teaching skills in small steps hierarchically), and errorless learning (prompting the behavior, followed by reinforcement, before error or non-response can occur).

Consequence-based strategies modify what happens after the behavior. For example, if it is determined that escape from task demands is what is maintaining and reinforcing a target behavior (e.g., screaming during class), the student may be sent to a different room with his or her work to complete, rather than being sent out of the classroom empty-handed. Academically, embedded instruction is often used, in which strategies to address the child's identified skill deficiencies are integrated into the general education routine (e.g., during transitions between activities; see, e.g., Johnson, McDonnell, Holzwarth, & Hunter, 2004). Peer-mediated approaches, such as peer tutoring, have been found to be effective in improving the academic skills of students with ASD (e.g., Kamps, Barbetta, Leonard, & Delquadri, 1994). Peer support interventions can also more generally promote academic engagement of students with ASD (McCurdy & Cole, 2014; for more comprehensive summaries of educational interventions for students with ASD, see Odom, Boyd, Hall, & Hume, 2014; Martins, Harris, & Handleman, 2014).

Interventions That Are Contraindicated by Research The myriad controversial intervention approaches for ASD can be grouped into two broad categories: Those that lack sufficient scientific validation or have weak/equivocal support; and those that are not supported (i.e., contraindicated). Auditory integration therapy, vitamin supplements, and special diets fall in the former camp, in that they lack empirical support to suggest efficacy (Sinha, Silove, Wheeler, & Williams, 2005; Smith, 2008). In other words, such approaches may be effective for some individuals, but either the research to date has been highly equivocal in its support or there has been insufficient research to make a determination on impact. In addition to the limited empirical support for efficacy, the considerable cost of many of these interventions should be considered and discussed with clients and their families as a potential risk during the informed consent and education prior to commencing treatment, given that the resources could be used for more effective or efficient treatments. Facilitated communication, where another individual physically assists someone without verbal communication in using a keyboard or assistive technology device to communicate, and secretin therapy fall in the latter camp, among those treatments that have been disproven, pose risk and are, as such, contraindicated (American Psychological Association, 1994; Jacobson, Foxx, & Mulick, 2005). Secretin, a hormone that aids with digestion, is probably one of the best-studied agents in ASD treatment research, but it not effective in treating ASD (Smith, 2008; Williams, Wray, & Wheeler, 2012).

The field of ASD treatment research is developing at a rapid pace, but there is still much to learn about for whom supported treatments work best (e.g., moderators and individual differences) and through what mechanisms interventions exert influence. Clinicians are encouraged to keep an open mind because many families do considerable online research and want to learn about treatment alternatives, but they should also be cautious consumers. When an evidence-based treatment is not viable or not available, or when the client has not responded to the primary evidence-based treatment or is otherwise not able to participate, fully informed consent is critical at the outset of the therapeutic relationship and the clinician must balance costs and risks of the approach with careful data-based monitoring of treatment outcomes.

Summary

Given the complexities of the presentation of ASD and its treatment, a case example may be instructional. "Tyler", a 9-year-old male, is a third grade student. He presented as a bright boy who reportedly enjoys reading and playing video games. His parents were concerned with his performance in school. He reports that his classes are "easy," but his grades were lower than would be expected based on assessed intellectual ability. He was referred to a community-based practitioner for an evaluation for attention-deficit hyperactivity disorder.

During the clinical interview, Tyler's mother reported that he does not have any close friends, and that he spends most of his time reading about military history, his favorite topic, or playing video games with his younger brother. His mother reported that his knowledge of the American Civil War is immense, and that he has enjoyed visiting battle sites and discussing details of the battle with anyone who shows interest. Throughout the assessment, Tyler displayed compliant behavior, but little eye contact with the examiner. Additionally, he appeared to have difficulty engaging in conversation with the examiner. Results from cognitive and school-based achievement measures indicate that Tyler's functioning is above average in most domains, although his processing speed score on the Wechsler Intelligence Scale for Children IV was significantly lower than his other scores. His academic achievement scores were even with his cognitive scores.

As a result of these findings and clinical observations of social difficulties, the examiner administered the Autism Diagnostic Observation Schedule-2, Module 3 (Lord et al., 2012) to Tyler. This is a semi-structured clinical observation task; in Module 3, used with verbally fluent children, the examiner asks the child questions and has the child complete tasks. Tyler's obtained scores fell above the autism spectrum cutoffs. The Autism Diagnostic Interview-Revised (Lord, Rutter, & Le Couteur, 1994) was administered to Tyler's mother, to assess his current behavior and developmental history, which indicated continuing difficulties in social, communication, and restricted interest and repetitive behavior domains since early childhood. Based upon these findings, Tyler was diagnosed with autism spectrum disorder without accompanying intellectual and language impairment.

Following his diagnosis, intervention concerning his school performance and social skills was recommended.

Treatment included individual therapy centering on developing social skills and managing anxiety concerning interacting with his peers. Specifically, the therapist helped Tyler identify classmates who had some of the same interests in military history and video games, and didactic instruction and practice on how to initiate and participate in reciprocal conversations. In school, Tyler qualified for access to extra supports in the form of one class period of direct tutoring each day, which allowed for instruction on time management and organization of his homework assignments so that homework completion was increased.

This case example highlights some of the pertinent issues commonly seen among individuals with ASD. Tyler's symptomatological presentation is one of a high-functioning individual on the spectrum; while his cognitive and academic achievement abilities were unimpaired with respect to global domains of functioning, he demonstrated difficulties in maintaining social reciprocity in conversation, including a lack of effective eye contact, establishing and maintaining typical friendships with peers, and a restricted interest in military history. Tyler's initial referral for attention-deficit hyperactivity disorder and older age at initial diagnosis are also common among individuals who are higher functioning on the spectrum, as their early developmental delays, if present at all, can be slight (such as a slight delay in speech or motor skills) and thus can go unnoticed until the social milieu exceeds their abilities. Comorbid difficulties in attention and anxiety when interacting with peers are also commonly seen among children and adolescents with ASD, and are imperative to evaluate when designing effective interventions.

The pace of research being produced related to etiology, assessment, and treatment of ASD is strong. As such, we can anticipate tremendous advancements made in our understanding of the disorder as well as clinical care and policy. ASD is a heterogeneous neurodevelopmental disorder. Although genetic in origin, there is no single identifying pathway to diagnosis. Treatment research to date has largely focused on educational, comprehensive programming for very young children, but recently more attention has been given to treatment options for both core and secondary problems in adolescents and adults with ASD. This is critical, as ASD is both pervasive and chronic.

Resources

Books

Amaral, D. G., Dawson, G., & Geschwind, D. G. (2011). *Autism spectrum disorders*. New York, NY: Oxford University Press.

O'Brien, M., & Daggett, J. (2006). *Beyond the autism diagnosis: A professional's guide to helping families*. Baltimore, MD: Brooks.

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White, S. W. (2011). Practitioner's guide to social skills training in children with asperger's syndrome and high functioning autism. New York, NY: Guilford.

Online Resources

- National Institute of Mental Health. Autism spectrum disorder: http://www.nimh.nih.gov/health/topics/autism-spectrum-disorders-pervasive-developmental-disorders/index.shtml?utm_source=rss_readers&utm_medium=rss&utm_campaign=rss_full (accessed May 7, 2015).
- Centers for Disease Control and Prevention. Autism spectrum disorder: http://www.cdc.gov/ncbddd/autism/research.html (accessed May 7, 2015).
- Organization for Autism Research: http://www.researchautism.org (accessed May 7, 2015).
- Autism Speaks: http://www autismspeaks.org (accessed May 7, 2015).
- Global and Regional Asperger Syndrome Partnership (GRASP): http://grasp.org (accessed May 7, 2015).

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22

Learning Disorders of Childhood and Adolescence

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Learning disorders represent an assorted category of generalized neurodevelopmental challenges that interfere with the ability to learn academic and/or social skills (Pennington, 2009). Broadly, learning disorders originate from a combination of congenital, acquired and/or environmental conditions or circumstances. Congenital learning disorders include specific learning disabilities (e.g., dyslexia, dyscalculia), communication disorders (e.g., stuttering) and developmental disabilities (e.g., intellectual disability, autism). They are present at birth, and manifest throughout the lifespan as children and adolescents engage in increasingly challenging cognitive and academic tasks. Learning disorders also can be acquired at any point in the lifespan following acute health conditions or severe injury (e.g., traumatic brain injury). Despite their organic etiology, risk and protective factors shape the developmental trajectory of both congenital and acquired learning disorders. Poor environmental conditions (e.g., not reading aloud to young children and implementing teaching methods that are not evidence-based) can bring about and exacerbate learning difficulties and disorders, even when there is not a biological predisposition for them.

The current chapter focuses on the three most ubiquitous learning disorders: specific learning disabilities, speech or language impairments, and intellectual disability. Based on special education eligibility criteria (2012–13 data), approximately 6.4 million (12.9%) of the total school-age population has an identified disability and receives public school special education services (National Center for Education Statistics, 2015). Roughly 2.3 million children and adolescents (4.6% of the school-age population) are identified by special education legal standards as having specific learning disabilities, representing almost half of all students who receive special education services. After specific learning disabilities, the most frequently occurring learning disorders are speech or language impairments (1.3 million or 2.7% of the school-age population) followed by intellectual disabilities, representing (430,000 or 0.9% of the population). Although true learning disorders are present at birth, considerable space in the current chapter is dedicated to discussing malleable environmental conditions (e.g., importance of implementing excellent, research-based teaching methods) that can alter the trajectory of organic learning disorders. Protective factors that foster student resilience, such as teaching basic academic skills until students achieve fluency or mastery, can prevent acquired learning disorders from manifesting in the first place. Although endogenous learning disorders have a biological origin, children and adolescents exposed to environmental risk factors (e.g., teachers who fail to teach phonics to struggling readers) may "learn to be learning disabled" (Clay, 1987, cited in Gresham, VanDerHeyden, & Witt, 2005) and struggle academically in ways that are indistinguishable from students with endogenous learning disorders.

In the current chapter, we provide information about the three most common varieties of learning disorders. Specifically, the first section of this chapter provides an in-depth discussion of specific learning disabilities, including a description of the changing definition and identification criteria. Next, we describe a relatively recent educational paradigm applied to the prevention and early intervention of learning disorders that is rooted in the public health model commonly applied to the prevention and intervention of sickness and disease. This

model, Response to Intervention (RTI), has been dubbed one of the most revolutionary manifestations in education today. RTI approaches all learning disorders, regardless of their etiology or severity, from a solution-focused perspective of prevention and intervention. Proponents of RTI are less likely to focus on learning disorders as a within-student problem (i.e., non-malleable condition) and more likely to concentrate on the instructional and environmental conditions that fail to support student learning and the ways they can be altered to help students learn (i.e., learning disorders as malleable conditions). Next, we describe the assessment, etiology, and prevalence of communication disorders, focusing on speech and language impairments. Specifically, we provide information on the more common speech and language impairments: Phonological disorders and stuttering. In the third section, we introduce intellectual disabilities (previously known as mental retardation), a less common condition but nonetheless pervasive developmental disorder that results in difficulties with learning.

The chapter concludes with specific strategies the reader can use to help clients with learning disorders and their families navigate the school system, particularly within special education. We also provide suggestions for clinical psychologists and social workers who desire to work closely with staff at their client's school. Specifically, we recommend ways clinicians can (a) promote prevention of learning failure; (b) participate in school-wide efforts to support children and adolescents with learning disorders; and (c) partner effectively with parents and schools to improve the schooling experience of students with learning disorders.

Specific Learning Disabilities

A disability classification, such as specific learning disabilities, provides a mechanism to organize a heterogeneous group of people into smaller subgroups of individuals with similar attributes who differ from other subgroups in important and observable ways. Ideally, disability classifications facilitate communication among professionals and the individuals themselves. Classification systems help inform programming and treatment efforts.

Valid disability classifications and appropriate diagnoses require clear operational definitions and identification criteria. Historically, there has been a lack of agreement about the best way to identify specific learning disabilities.[1] Despite being the most prevalent of the learning disorders, specific learning disabilities are among the least understood and most passionately debated disability categories (Bradley, Danielson, & Hallahan, 2002). Inconsistent application of identification criteria in school and clinical practice has contributed to contradictory and unstable classification, identification and remediation procedures. Understanding the current state of the field requires a brief historical overview of the field, which we present in the next section.

Historical Overview and Evolving Definition Scientific, social, and political changes over the last century have shaped the field of learning disabilities (see Turnbull, 2009, for an analysis). The desire and need to identify individual differences in learning and performance have dominated scientific and clinical efforts aimed at understanding specific learning disabilities. Observations and reports of individuals with learning disabilities in one area (e.g., reading) while having otherwise average or typical performance in other areas (e.g., mathematics) first emerged in the early 19th century (Fletcher, Lyon, Fuchs, & Barnes, 2007). A hallmark of the earliest definition of learning disabilities is that it discussed what learning disabilities were *not* rather than what they *were*. For example, Kirk (1963) first coined the term "learning disabilities" to describe a "group of children who have disorders in the development of language, speech, reading, and associated communication skills needed for social interaction . . . not includ[ing] children who have sensory handicaps such as blindness . . . [or] who have generalized mental retardation" (pp. 2–3). Specific exclusionary or "rule out" considerations remain in the most recent (2004) federal definition of a specific learning disability.

Research conducted up to and through the early 1960s had a significant impact on our modern conceptualization of learning disabilities. Collectively, evidence about individual learning disorders contributed the following to our understanding: (a) Learning differences could be understood by observing how children approach tasks and process information; (b) instruction and programming should be designed in recognition of a child's specific strengths and weaknesses; (c) specific learning disabilities are associated with neurological functioning but are not a primary result of sensory impairments; and (d) specific learning disabilities occur in the context of overall average cognitive functioning (Kavale & Forness, 1985; Torgesen, 1991). Also in the 1960s, researchers, educators and policy makers began advocating fervently and effectively for adequate educational conditions on behalf of all children with disabilities. In response, the U.S. Department of Education began the process of operationally defining the condition, specific learning disability, for legislative purposes. In 1968, the following definition, which included exclusionary factors, was adopted:

> The term 'specific learning disability' means a disorder in one or more of the basic psychological processes involved in understanding or in using language, spoken or written, which may manifest itself in an imperfect ability to listen, speak, read, write, spell, or to do mathematical calculations. The term includes such conditions

> as perceptual handicaps, brain injury, minimal brain dysfunction, dyslexia, and developmental aphasia. The term does not include children who have learning disabilities, which are primarily the result of visual, hearing, or motor handicaps, or mental retardation, or emotional disturbance, or of environmental, culturally, or economic disadvantage.
>
> (U.S. Department of Education, 1968, p. 34)

The definition above drew from the field's collective understanding of learning disabilities in terms of its biological etiology (Orton, 1937) and in relation to exclusionary factors (e.g., Kirk, 1963). The 1968 definition also marked the first time the term, "specific learning disabilities" was used. Consequently, the 1968 definition was included in the Learning Disabilities Act of 1969 and as the statutory definition of specific learning disabilities in the landmark Education for All Handicapped Children Act of 1975 (Public Law [PL] 94-142), which guaranteed, for the first time in history, a free, appropriate public education to all children with a disabilities, including those identified with learning disorders. The 1968 definition continues to be, with a few minor wording adjustments, the definition of specific learning disabilities in the most recent federal special education regulations, the Individuals with Disabilities Education Act (IDEA) of 2004, that today guides school-based evaluation and eligibility decisions.

Although the federal definition and accompanying regulations were somewhat helpful in evaluation, identification, and special education program development, significant concerns remained regarding the procedures for *identifying* learning disabilities. Indeed, researchers, practitioners and advocates continually criticized the definition for emphasizing what learning disabilities are not but omitting a clear description and criteria of what learning disabilities *are* (Fletcher et al., 2007) and how to identify students who had specific learning disabilities. An ongoing debate that seeks clarity regarding the definition of specific learning disabilities and identification practices continues within the field and will likely continue for the foreseeable future. A full account of this debate is beyond the scope of this chapter; interested readers are directed to writings by Kavale & Forness (2000), Kavale & Spaulding (2008), and Fuchs & Fuchs (2006) for more information.

One of the factors contributing to lack of shared consensus about definition and identification procedures for specific learning disabilities lies in the various definitions of specific learning disabilities that have been formalized by organizations such as the American Psychiatric Association (i.e., the fifth edition of the *Diagnostic and statistical manual of mental disorders*, DSM-5; American Psychiatric Association, 2013) and various advocacy groups such as the National Joint Committee on Learning Disabilities, including the World Health Organization (i.e., 10th edition of the *International Classification of Diseases*; ICD-10; World Health Organization, 1992). Professionals in clinical and medical practices favor the new DSM-5 classification system and use it to *diagnose* clients with a specific learning disability. In contrast, school personnel use the federal special education definition and identification criteria to *identify* learning disorders (including specific learning disabilities, speech or language impairments and intellectual disability) for the purpose of providing special education services in schools. It is imperative to note that eligibility for special education services in the schools does not require a medical or clinical diagnosis of a learning disorder; likewise, a medical or clinical diagnosis of a learning disorder does not ensure special education eligibility. This may be confusing for parents and even for professionals who do not work in a school system. Legally, the purpose of special education eligibility procedures is to determine educational need and, therefore, eligibility for special education services. Federal law requires a multidisciplinary team consisting of qualified professionals *including the parents* to *identify* whether a student has a disability that requires special support services. The multidisciplinary team must determine whether the disorder prevents the student from learning in the general education and would benefit from specialized services offered through the local special education program.

In addition to the lack of universal consensus about the definition of a specific learning disability, an equally contentious predicament in the history of specific learning disabilities concerns identification procedures. Although PL 94-142 provided an operational definition for specific learning disabilities as noted above, the omission of clear inclusionary criteria to help identify students with specific learning disabilities immediately became problematic for multidisciplinary teams responsible for making special education eligibility decisions for children with significant learning problems.

In 1977, the U.S. Department of Education published guidelines designed to facilitate the identification of students with specific learning disabilities. The guidelines included the identification criterion of a "severe discrepancy" between intelligence and academic achievement. School professionals interpreted the criterion literally to mean a significant discrepancy or difference between ability, as measured by a standardized IQ score and academic achievement, as measured by a standardized achievement test score. Despite the widespread acceptance of the discrepancy criterion, there was limited evidence of its validity (Fletcher et al., 2002). Nevertheless school-based professionals in the fields of special education and school psychology came to consider this IQ-achievement discrepancy as *the* diagnostic criterion for the presence (or absence) of a specific learning disability.

Concerns About the IQ-Achievement Discrepancy Since the passage of the All Handicapped Children Act of

1975, educators have expressed concern about the lack of emphasis on prevention for students with specific learning disabilities (National Association of State Directors of Special Education, 2005). Reschly (2003) criticized the discrepancy approach to identifying specific learning disabilities as being: (a) Unreliable and unstable; (b) invalid (e.g., poor readers with high IQs do not substantially differ from poor readers with lower IQs); (c) neglectful of providing the necessary focus on exclusionary criteria such as mental retardation, and environmental, cultural, and/or linguistic diversity; and (d) harmful because it represents a "wait-to-fail" approach that delays identification and services. Gresham (2002) similarly criticized the discrepancy approach because it relied upon assessment information that had restricted utility in designing, implementing, or evaluating the impact of classroom instruction and intervention. In other words, the assessment data did not drive decisions about which instructional practices or interventions would be most beneficial for the student.

In the early part of the 21st century, alternate methods for addressing learning problems at the first sign of difficulty in school and identifying specific learning disabilities began to emerge in the literature. Chief among these methods was RTI. RTI is a data-driven model for identifying and responding to the needs of students who demonstrate academic and behavioral difficulties. The model includes school-wide universal screening, a continuum of interventions and supports, and ongoing monitoring of student growth and learning (Brown-Chidsey & Steege, 2005; Martínez & Nellis, 2008). Although RTI is relatively new to education, its conceptual origins lie in the public health model of preventing disease across a population. RTI is discussed in greater detail in a later section of this chapter, where we discuss effective ways of preventing and remediating learning disorders.

The Problem With Special Education In the 25 years spanning 1975 to 2000, a spike in the number of students identified as having specific learning disabilities prompted great concern at the federal level. Consequently, in 2001, the then President of the United States, George W. Bush, appointed the Commission on Excellence in Special Education to report on the condition of special education broadly and to propose policy for improving the educational performance and school experience of all students with disabilities. The resulting 2002 report, *A New Era: Revitalizing Special Education for Children and their Families*, documented the condition of special education and shed light on fundamental systemic flaws that, at best, contributed to the overrepresentation of students identified with specific learning disabilities relative to the other disability categories and, at worst, outright failed children (U.S. Department of Education Office of Special Education and Rehabilitative Services, 2002).

The authors of the report acknowledged that emphasis on the process (e.g., excessive paperwork) of providing special education services in the schools resulted too frequently in simply identifying students as eligible for special education, rather than promoting teaching practices in both general and special education that ensured effective instruction for all struggling students (regardless of an identified disability). They did not mince words when underscoring that the education system often failed struggling learners and students with learning disorders largely because educators did not readily embrace or implement evidence-based educational and intervention practices. Indeed the authors conceded that "special education should be for those who do not respond to strong and appropriate instruction and methods provided in general education" (p. 7). Lyon (2002) poignantly referred to students who were failed by the general education system as *instructional casualties* whose learning disorders manifested as a direct result of not being taught using effective educational practices. The 2002 report concluded that the special education system had become obsolete because the system inadvertently facilitated a process of literally waiting for students to fail before they received help, instead of promoting prevention, early identification and hard line, evidence-based intervention approaches to help struggling learners. Finally, methods of identifying children with disabilities, particularly with respect to the category of specific learning disabilities, were sharply criticized in the 2002 federal report cited above and by Gresham (2002) as lacking validity and misidentifying, over identifying and, at times, under identifying certain students for special education services.

Special Education Today The December 2004 reauthorization of the IDEA of 1997, the United State's special education law (originally PL 94-142), was released in August 2006, and included RTI as an alternative method (to the IQ-achievement discrepancy approach) for identifying a specific learning disability in the schools. The regulations included language specifying that, in the identification of specific learning disabilities, state educational agencies *must not require* that local education agencies use a severe discrepancy between intellectual ability and achievement and *must permit* school districts to incorporate the use of a process based on the child's response to scientific research-based intervention or other alternative research-based procedures for determining the existence of specific learning disabilities (e.g., absolute low academic functioning irrespective of IQ; §300.307).[2] The IDEA 2004 definition of specific learning disabilities was a slightly modified version of the definition included in the first iteration of

special education legislation in 1975 (i.e., then called PL 94-142) and reads as follows:

> § 300.8(c)(10) . . . A disorder in one or more of the basic psychological processes involved in understanding or in using language, spoken or written, that may manifest itself in the imperfect ability to listen, think, speak, read, write, spell, or to do mathematical calculations, including conditions such as perceptual disabilities, brain injury, minimal brain dysfunction, dyslexia, and developmental aphasia. (ii) *Disorders not included*. Specific learning disability does not include learning problems that are primarily the result of visual, hearing, or motor disabilities, of mental retardation, of emotional disturbance, or of environmental, cultural, or economic disadvantage.

The eight areas in which a specific learning disability can be identified are: (1) Oral expression; (2) listening comprehension; (3) written expression; (4) basic reading skill; (5) reading fluency skills; (6) reading comprehension; (7) mathematics calculation; and (8) mathematics problem solving.

Response to Intervention Regulations in IDEA 2004 required state educational agencies to determine whether RTI would be *mandated* or simply *permitted* as a component identifying presence of specific learning disabilities. Zirkel and Thomas (2010a) reported that 12 states required RTI but the majority of states in the United States continued to allow schools to use the discrepancy criterion to determine presence of a specific learning disabilities. Approximately 20 states also permitted schools to use other research-based models that were not explicitly defined but permitted in the IDEA 2004. Zirkel and Thomas noted that while such state regulations allow for flexibility at the district and school levels, they also create the potential for confusion and foster inconsistent practice, similar to what the discrepancy model originally engendered. Zirkel and Thomas (2010b) examined state guidelines for using RTI and described and found that all elements of RTI implementation were not equally addressed across the documents. Further, the lack of explicit state or federal criteria to guide schools in determining when a student crosses the line from being a struggling student to one with a true learning disability make it very difficult for multidisciplinary teams to make special education eligibility decisions.

What Is Response to Intervention? RTI is a continuum of services that are available to all students based on individual needs and regardless of whether a student has been identified as eligible for special education services. The core RTI philosophy mirrors the public health model of prevention and early intervention in the case of disease. RTI is the application of public health in the public schools. This RTI continuum includes universal interventions, sometimes referred to as "primary prevention," which are in place for all students, including general education students, to support positive academic, behavioral, and mental health outcomes. Tier 1 (i.e., primary prevention) in a RTI model involves high-quality school and classroom environments, scientifically sound core curriculum and instruction, and intentional instructional practices. Tier 2, or secondary prevention, consists of targeted academic and behavioral services for those students who demonstrate risk for failure and for whom universal interventions are not resulting in their success. Services at Tier 2 are more intense and are focused on the specific needs of a student or group of students. Examples of educational services at Tier 2 might include small group instruction for either academic or behavioral needs, additional tutoring support, or involvement in remediation programs such as Title I. Despite the educational services at Tiers 1 and 2, some students need more intensive intervention at the tertiary or intensive level. Tier 3 services are available for those students for whom Tiers 1 and 2 services have not been sufficient. In theory, the needs of approximately 80–90% of all students in any given school can be met with universal educational services at Tier 1. Beyond Tier 1, 5–15% of the student body population may require Tier 2 intervention and 1–5% may require Tier 3 intervention (Walker & Shinn, 2002).

Response to Intervention for Identifying Presence of a Specific Learning Disability Eligibility for special education services for students with specific learning disabilities (as described above) are made by a multidisciplinary team, as described in §300.308. The team includes a child's parents or guardians and a team of qualified professionals, including a teacher and at least one member trained to diagnostically evaluate the student's learning (such as a school psychologist or speech–language pathologist—see Box 22.1 for descriptions of these roles). As stated earlier, *eligibility* for special education services is not determined by a clinical (i.e., DSM-5; American Psychiatric Association, 2013) *diagnosis* of a disability. Thus, although a child or adolescent may have been diagnosed by a clinical practitioner with a specific learning disability, if the individual's impairment does not adversely affect his or her academic and/or functional performance as determined by the school's multidisciplinary team, the individual may not be identified as a student with a specific learning disability and deemed eligible for special education services. For this reason, it is in a client's best interest for psychologists and social workers to work collaboratively with staff and administrators at their client's school to ensure that optimal educational services are provided. We provide practical suggestions for working with schools in greater detail in a later section of this chapter.

Box 22.1 Description of School Psychologists and Speech Language Pathologists

School Psychologists

School psychologists are professionals with extensive training in both psychology and education.[3] The main goal of the school psychologist is to help children and youth to succeed academically, behaviorally, emotionally, and socially. To best achieve this goal, school psychologists collaborate with teachers, parents, administrators, and other professionals to develop healthy, safe, and supportive academic environments that emphasize and strengthen connections between school, home, and the community for all children. Some of the unique functions of the school psychologist include identifying and addressing behavior and learning problems that interfere with school success, working within a multidisciplinary team to determine student eligibility for special education services, helping teachers create positive classroom environments, and implementing school-wide prevention programs that help maintain positive school climates.

The implementation of the RTI process creates new opportunities and a greater need for school psychologists. The training that the school psychologist receives in consultation, behavior and academic interventions, research, counseling, and evaluation is called upon as districts implement new RTI procedures.[4] The roles of the school psychologist in an RTI model expand to include duties such as working with administration to obtain "buy-in" and facilitate system change, planning and conducting necessary staff training to implement RTI, developing local academic achievement norms, and consulting with both teachers and parents regarding early intervention tools for in the classroom and at home.

Speech–Language Pathologists

Speech–language pathologists, or SLPs, are specialists who receive intensive training in both atypical and typical communication development and pathology (Sunderland, 2004). SLPs are often the lead service providers for individuals with speech and language disorders (Justice, 2006). An SLP has a degree or certification in speech and language pathology and is qualified to diagnose speech and language disorders and to prescribe and implement therapeutic measures for these disorders (Nicolosi, Harryman, & Kresheck, 2004). Under IDEA 2004, speech–language pathology services include the following: The identification of children with speech or language impairments, the diagnosis and assessment of specific speech or language impairments, the referral of individuals for medical or other professional attention necessary for the treatment of speech or language impairments, the provision of speech and language services for the habilitation or prevention of communication impairments, and the counseling and guidance of children, parents, and teachers concerning speech or language impairments [§ 300.34(c)(15)]. The SLP plays a critical role in the identification and treatment of individuals with speech or language impairments.

SLPs working in districts where RTI procedures are being implemented are able to contribute to assessment and intervention through system-wide program design and collaboration, as well as individual work with students. SLPS are able to offer expertise in the language basis of learning and literacy, an understanding of the utilization of student outcome data in making instructional decisions, and experience with collaborative instruction and intervention approaches (Ehren, et al, 2007). A few of the ways SLPs contribute in the RTI model include assisting in identifying systemic patterns of student need pertaining to language skills, explaining the interconnection between spoken and written language to other school professionals, and identifying, utilizing, and disseminating evidence-based practices for RTI interventions or speech and language services (Ehren, Montgomery, Rudebusch, & Whitmire, 2007).

As part of special education eligibility determination for a specific learning disability as outlined in IDEA 2004, the school-based multidisciplinary team must consider evidence that the child has *not* made sufficient progress in response to scientific, research-based intervention (e.g., RTI) *or* exhibits a pattern of strengths and weaknesses that is determined to be relevant to the identification of a specific learning disabilities. Addressing longstanding concerns about the rigor and appropriateness of instruction in reading and math, the regulations also require the team to consider data demonstrating that appropriate instruction in general education was provided prior to, or during, the referral process (§300.309 [b]). Such data might include assessments that all students in a school are taking (e.g., universal screening of core skills), tests that teachers are using in their classrooms (e.g., teacher-made assessments), and targeted assessments (e.g., progress monitoring tests) given frequently to students who are receiving additional intervention.

Despite considerable research, debate, and legislative activity regarding specific learning disabilities, and more recently the role of RTI in eligibility decisions,

researchers and practitioners still cast doubt on the validity of RTI procedures for identifying specific learning disabilities (Fletcher, Morris, & Lyon, 2003; Kavale & Spaulding, 2008; McKenzie, 2009). In addition, Johnson and colleagues (2010) caution that RTI, even when implemented with fidelity, may provide restricted information about why a student has failed to make progress over time. Thus, an explanation or hypothesis about why the student struggles is sometimes missing which limits future education programming and planning efforts.

Speech or Language Impairment

Approximately 1.3 million school-age students in the United States have speech or language disorders (National Center for Education Statistics, 2015). Generally, children with speech or language impairments are identified in preschool although some children may not be identified until early elementary school (Schuele, 2004). IDEA 2004 defines a speech or language impairment as [§ 300.8(c)(11)] "a communication disorder, such as stuttering, impaired articulation, a language impairment, or a voice impairment, that adversely affects a child's educational performance." Under the broad umbrella of "communication disorder" are three varieties of impairments: speech, language, and hearing (Justice, 2006); this chapter focuses on speech and language disorders or impairments.

Speech, language, and hearing are three fundamental elements of spoken communication and essential to learning. A breakdown in speech, language, or hearing can manifest as a communication disorder. As with specific learning disabilities, the earlier communication difficulties are identified, the greater the chance of improving and remediating symptoms of the disorder. The RTI model described previously is also applicable to the prevention, identification, and remediation of speech and language impairments, especially in the first few years of formal schooling.

Speech Disorders A speech disorder is an impairment in the presentation of spoken language (Worster-Drought, 1968). Speech disorders are characterized by a breakdown in speech production (e.g., articulation) and include disorders of the voice. Common speech problems include stuttering or dysfluency, articulation disorders, and unusual voice quality (U.S. Preventive Services Task Force, 2006). Stuttering (i.e., stammering) is characterized by repetitions, prolongations and long pauses in the production of speech. Stuttering affects approximately 5% of the population and the incidence is twice as great in boys compared to girls (Andrews, 1984). Most children recover naturally without intervention within 3 years of initial identification (Zebrowski, 2003), although approximately 1% of the adult population stutters (Zebrowski, 2003).

Language Disorders Language involves the understanding, processing, and production of communication (U.S. Preventive Services Task Force, 2006). A language disorder is a breakdown in the linguistic system that can impact at least one of the following linguistic domains: Syntax, semantics, phonology, morphology, and pragmatics (Justice, 2006). Specific language impairments encompass the largest group of children with language disorders and affect approximately 7% of all children (Ziegler, Pech-Georgel, George, Alario, & Lorenzi, 2005). Speech or language impairments are often followed by associated problems such as difficulties in reading and spelling and may generalize to include abnormalities in interpersonal relationships, and emotional and behavioral disorders (Arkkila, Rasanen, Roine, & Vilkman, 2008).

Phonological disorders are considered a type of language disorder and among the most prevalent communication disorders present in preschool and school-age children. A phonological disorder reflects an inability to articulate speech sounds (Gierut, 1998). Phonological awareness is essential to learning how to read (Adams, 1990) and deficits in phonological awareness are inextricably linked to reading disabilities (Lyon, Shaywitz, S. E., & Shaywitz B. A., 2003). Thus, for SLPs employed in the school system, children with phonological disorders may represent as much as 91% of their average caseloads (American Speech-Language-Hearing Association, 2006).

Assessment of Speech–Language Impairments For both articulation and phonology disorders, methods of assessment usually include a hearing screening and informal and formal procedures. Informal procedures may consist of a review of existing data, examination of the structure and function of the oral mechanism, speech sampling, and direct observation. Psychometric tests administered by licensed SLPs play a significant role in the process of identifying children with speech–language impairments. These standardized tests allow professionals to observe different aspects of language function and to relate a child's performance to normative data (Bishop & McDonald, 2009). Generally, methods of assessing language disorders include the following: Hearing screening, review of existing data, questionnaires, teacher/parent/student interviews, direction observation and language sampling, student work examples, and standardized tests (Sunderland, 2004).

Intellectual Disability

The third most common learning disorder is a direct result of biological, cognitive impairments present at birth. Traditionally, individuals with intellectual disability have been grouped into one of four degrees of severity based on cognitive functioning: Mild, moderate, severe, and profound. As measured by most standardized assessments of cognitive ability, a standard IQ score falling within one standard deviation of the mean (85–100; mean = 100) is

considered in the average range. Likewise, a score of 70, or two standard deviations below the mean, is considered the upper limit of mild intellectual disability. IDEA 2004 defines intellectual disability, or mental retardation, as follows [§ 300.8(c)(6)]: "Mental retardation means significantly subaverage general intellectual functioning, existing concurrently with deficits in adaptive behavior and manifested during the developmental period, that adversely affects a child's educational performance."

In 2007, the American Association on Mental Retardation officially changed its name to the American Association on Intellectual and Developmental Disabilities (AAIDD), and has since advocated for the use of the diagnostic term "intellectual disability." Although the term "mental retardation" remains in use in current federal educational law (IDEA, 2004) as illustrated above, in the most recent iteration of the DSM (DSM-5; American Psychiatric Association, 2013), the term has been replaced by intellectual disability (intellectual developmental disorder). The parenthetical name "intellectual developmental disorder" is included to indicate cognitive deficits present from the earliest developmental period.

Assessment and Identification Intellectual disability is characterized by three core traits: (a) Significantly below average cognitive functioning; (b) limited functioning in adaptive behavior; and (c) onset prior to the age of 18. Comprehensive assessment of cognitive functioning *and* adaptive behavior is vital to an accurate diagnosis of intellectual disability, and should take into account cultural and environmental factors that may influence performance in these areas. Specifically, adaptive impairment is defined as follows:

> Deficits . . . that result in failure to meet developmental and sociocultural standards for personal independence and social responsibility [p. 33] . . . [The criterion] is met when at least one domain of adaptive functioning—conceptual, social, or practical—is sufficiently impaired that ongoing support is needed in order for the person to perform adequately in one or more life settings at school, work, home or in the community.
>
> (American Psychiatric Association, 2013, p. 38)

DSM-5 includes only one diagnosis for intellectual disability (intellectual developmental disorder), and severity is based not only on IQ,[5] but also on the degree of impairment in adaptive behavior. Deficits in adaptive functioning are evident in one's communication skills, social skills, personal independence at home or in community settings, and/or school or work functioning. Clinicians estimate severity based on clinical impressions that range from mild to moderate, severe, or profound. In DSM-5,

> the various levels of severity are defined on the basis of adaptive functioning, and not IQ scores, because it is adaptive functioning that determines the level of supports required. Moreover IQ measures are less valid in the lower end of the IQ range.
>
> (American Psychiatric Association, 2013, p. 33)

Cognitive Functioning Several norm-referenced instruments are used to assess cognitive functioning, or intelligence. Among the most commonly used, particularly with children, are the Wechsler Intelligence Scale for Children, fourth edition (WISC-IV; Wechsler, 2003) and the Stanford–Binet Intelligence Scales, fifth edition (SB-5; Roid, 2003). These instruments were created based on the theory that intelligence is a multifaceted construct that encompasses performance and ability in domains such as verbal reasoning, memory, abstract problem solving, and visual-spatial perception.

WISC-IV is an individually administered test that comprises 10 core subtests and five supplemental subtests. The test yields a full-scale IQ, as well as four index scores: Verbal comprehension, which measures verbal abilities in reasoning, comprehension and conceptualization; perceptual reasoning, which measures perceptual reasoning and organization; working memory, which measures attention, concentration, and working memory; and processing speed, which measures the speed of mental and psychomotor processing. Similarly, the SB-5 consists of 10 subtests and also yields a full-scale IQ. Each subtest in the verbal domain has a corresponding subtest in the nonverbal domain. The five domains are: fluid reasoning, knowledge, quantitative reasoning, visual-spatial processing, and working memory.

The full-scale IQ score yielded by tests such as the WISC-IV and SB-5 is used as the basis for determining impaired cognitive ability. However, as both tests yield scores in verbal and nonverbal domains, should a significant discrepancy exist in an individual's performance in these areas, clinicians should closely examine the individual's pattern of strengths and weaknesses, rather than simply relying on averaging these scores to derive a full-scale IQ (American Psychological Association, 2000).

Adaptive Behavior Adaptive behavior can be defined as "how effectively individuals cope with common life demands and how well they meet the standards of personal independence expected of someone in their particular age group, sociocultural background, and community setting" (American Psychological Association, 2000, p.42). According to the American Association on Intellectual and Developmental Disabilities (2010), adaptive behavior encompasses three types of skills: Social skills (including interpersonal skills, social responsibility, social problem solving, and the ability to follow rules and avoid victimization); conceptual skills (money, time, and numerical concepts, language and literacy, self-regulation); and practical skills (including self-care and activities of daily living, occupational skills, healthcare, travel and transportation, adhering to schedules and routines, and safety). Assessment of adaptive behavior is not only a key component of the diagnostic profile of intellectual disability, it also forms the basis from which intervention and remediation plans are typically developed.

Assessment of adaptive behavior typically consists of multi-informant rating scales completed by parents,

educators, community mental health providers and/or the individual him or herself. The most frequently used measures of adaptive behavior are the Adaptive Behavior Assessment System, second edition (Harrison & Oakland, 2003), and the Vineland Adaptive Behavior Scales, second edition (Sparrow, Cicchetti, & Balla, 2005).

Assessments of appropriate adaptive functioning are highly subjective and heavily based on social, cultural, and other environmental norms. When selecting assessment instruments and interpreting results, therefore, the evaluator should pay attention to the suitability of the assessment tool for the individual, as well as external factors that may impact his or her performance (American Psychological Association, 2000).

School Identification and Special Education Eligibility

In addition to community agencies, schools serve as one of the primary environments in which children with intellectual disability are identified and receive support. Recent statistics show that students identified using special education criteria for an intellectual disability represented 9% of all students served under IDEA, Part B (U.S. Department of Education, 2009). Educational professionals, including school psychologists, counselors and social workers, are traditionally charged with the task of conducting valid assessments and designing educational interventions for this group of students. Though standardized assessments of cognitive ability and adaptive behavior may provide a sufficient basis for an identification of intellectual disability or for determining eligibility for special education services, results from these assessments offer little to inform the process of intervention planning in educational contexts. To help fill this void, it is recommended that school teams conduct multidisciplinary, functional and ecologically valid assessments to inform educational planning for children with intellectual disability (Powell-Smith, Stoner, Bilter, & Sansosti, 2008). As a starting point, detailed information on the child's developmental, medical, and social history provides important clues regarding the onset of the child's delay and possible etiologies of the condition, allowing the team to rule out other potential causes for the child's academic and behavioral difficulties (i.e., specific learning disability). Further, information on student performance should be gathered from multiple sources in various environments, including the home, school, and community settings. Particularly important are data on functional life skills and "cues and correction procedures" that occur naturally and without planned intervention and effectively assist the individual in completing daily life tasks.

Finally, the focus of assessment and intervention should be on functional life skills that will allow for successful transition to post-school settings. Central to working with students with intellectual disability is the provision of supports that increase the individual's ability to function as independently as possible. We strongly encourage clinical professionals serving clients with intellectual disability (or specific learning disability) outside of the school system to work collaboratively with school-based identification teams and educational professionals to ensure optimal and appropriate educational and intervention supports for clients with intellectual disability.

Working With Children Who Have Learning Disorders and Their Families

In this section, we provide psychologists and social workers specific suggestions for working with clients who have specific learning disabilities, speech or language impairments, or intellectual disability. We contend that these and other practitioners provide optimum clinical services when they collaborate with teachers, administrators, and school personnel at their client's school to ensure that the full spectrum of services is provided to their clients. We believe that psychologists and social workers can be effective partners in the schooling of their clients when they: (a) Promote prevention and early intervention; (b) partner with school personnel to optimize the educational supports and services being provided; and (c) assist parents and families in supporting clients with learning disorders. Here, we describe each recommendation in detail.

Promote Prevention and Early Intervention and Encourage Family to Become Children's First Teachers

Psychologists and other clinicians may be most helpful to their clients who have learning disorders when they promote prevention strategies at home and at school. As noted previously, environmental conditions may predispose a child without an organic learning deficit to "learn to be learning disabled" (Clay, 1987 cited in Gresham, VanDerHeyden, & Witt, 2005) such that organic and acquired learning disorders are indistinguishable. One way in which clinicians can promote prevention is by conducting parent education seminars, community workshops, and/or parent counseling sessions that emphasize the critical early role parents play in ensuring their children's subsequent academic success. In *Helping your Child Become a Reader* (U.S. Department of Education, Office of Communications and Outreach, 2005), a pamphlet available free of charge on the U.S. Department of Education, Office of Communications and Outreach website, parents of young children (birth to age 6) are encouraged to: (a) Talk and listen to their young children even as babies; (b) spend at least 30 minutes daily reading aloud to their children; (c) bring attention to the letters and words in books and help them understand that they have meaning; and (d) foster children's innate curiosity to write, because reading and writing go hand in hand.

As consultants, psychologists can urge their client's teachers to focus on direct, explicit instructional strategies (Carnine, Silbert, & Kame'enui, 1997). Struggling students need intense instruction in small groups for longer duration with greater frequency compared with the instruction provided to typically achieving students (Deshler, 2005). One

of the best tools for being an effective consultant and recommending best practices in academic interventions is to be knowledgeable about available evidence-based academic and behavioral interventions. Links to helpful websites are provided at the end of this chapter. Additionally, attending national professional conferences, such as the National Association of School Psychologists and the American Speech-Language-Hearing Association Annual Conventions, can provide psychologists and other clinicians the opportunity to gain valuable knowledge about the most effective, cutting-edge instructional strategies currently available.

Participate in School Efforts to Support Clients With Learning Disorders Another way in which psychologists and other clinicians can help clients who have learning disorders is by their willingness to work collaboratively with school personnel to advocate on behalf of their clients so that they receive the best educational services in general and special education settings. Federal law requires that students with disabilities be educated in the least restrictive environment and with typically achieving peers to the maximum extent that is deemed appropriate. Therefore, practitioners want to ensure that schools are providing their clients with special education services in inclusive environments with typically achieving peers and that their clients have equal access to the curriculum and extracurricular activities. Additionally, clinicians working with children who are in special education or being considered for special education eligibility can be effective as collaborative, contributing members of the school multidisciplinary team that weighs the available evidence to make decisions about children's placement and the remedial services they receive. Collaborating with school personnel, such as school psychologists and SLPs, provides an opportunity both to give input into school programming and services, and to plan direct services that seek to reinforce and generalize the school-based efforts. For students with more severe learning disabilities (i.e., intellectual disability) practitioners can also play a vital part in ensuring their client receives the school-based services and supports they need to successfully transition into employment, supported or independent living, and/or further education after exiting the school system.

Assist Parents and Families to Support Clients With Learning Disorders Psychologists can help their clients and their clients' families develop the skills and supports needed to successfully advocate in the school system. For example, they may work directly with clients on self-monitoring study strategies, and on developing the social skills for asking for help when they need it. Psychologists can work with parents on skills and strategies to advocate for their children and communicate their needs and desires to school personnel successfully. Parents sometimes experience guilt or denial when their child is identified with one of the learning disorders described in this chapter. Helping parents to understand the importance of consistency and routine in the home, as well as following through with school recommendations for the home can help parents more effectively support their child who has a learning disorder.

Summary

This chapter has considered three specific types of learning disorders, specific learning disabilities, speech or language impairments, and intellectual disabilities. We described issues surrounding their definitions, history, identification, and intervention. Specific learning disabilities are the most prevalent of the learning disorders, occurring in 4.6% of the school-age population. Specific learning disabilities were first defined in 1968 and recognized in federal law in 1975. Inconsistencies in identification procedures for specific learning disabilities since the disorder first appeared in federal education law have resulted in a lack of consensus about how to appropriately identify students with specific learning disabilities for special education services in schools. The most promising model to date for identifying students with specific learning disabilities is RTI, which is rooted in the public health model of prevention and early intervention at the first sign of difficulty. RTI is permitted by federal law as a method for screening populations of students so that those who are not meeting important educational benchmarks can be provided intense educational interventions to remediate or reduce academic risk.

We also described speech or language impairments, which are the second most prevalent learning disorders occurring in 2.7% of the school-age population of all school-age children and include impairments in speech, language, and hearing which are essential to learning in school. Finally, we described intellectual disability, the third most common learning disorder, characterized by significantly below average cognitive functioning, limited functioning in adaptive behavior, and onset before age 18.

The chapter concluded by providing the reader with specific strategies for helping clients with learning disorders and their families navigate schools generally and the special education system specifically. In particular, we recommend strategies that psychologists in clinical practice can implement in their private practice to more effectively: (a) Promote prevention of learning failure; (b) participate in school-wide efforts to support children and adolescents with learning disorders; and (c) partner effectively with parents and schools.

Notes

1. The term "learning disability" is a broad designation for a variety of learning disorders; we use the term "specific learning disability" throughout this chapter to be consistent with legal language and practical consideration because learning disabilities generally manifest in specific areas, like reading decoding, mathematical computation, etc.
2. Under the new law, schools are still permitted to use the discrepancy approach, if their state allows it, to identify specific learning disabilities; however, clear research evidence does not support this identification method (see Gresham, 2002; Reschly, 2003).

3. Who are school psychologists? National Association of School Psychologists, Bethesda, MD: http://www.nasponline.org/about_sp/whatis.aspx (accessed May 8, 2015).
4. Response to intervention (RTI). American Speech-Language-Hearing Association. Retrieved from http://www.asha.org/slp/schools/prof-consult/RtoI.htm (accessed May 8, 2015).
5. In the previous edition of the DSM (American Psychiatric Association, 2000), the ranges of severity of intellectual disability relied heavily on scores obtained on IQ tests. Scores on IQ tests resulted in the following categories: Mild (55–70), moderate (40–54), severe (25–39), and profound (below 25).

Recommended Internet Resources

For Children

Professor Garfield: Comprehensive free website for children offering lively, interactive and free "edutainment" games and activities that help to motivate and empower kids while addressing fundamental reading skill sets: http://www.professorgarfield.org/pgf_home.html

For Parents

Reading Rockets: Teaching kids to read and helping those who struggle:

http://www.readingrockets.org

Colorín Colorado: A bilingual site for families and educators:

http://www.colorincolorado.org

Starfall: A free public service to motivate children to read with phonics:
http://www.starfall.com

For Professionals

Evidence Based Intervention Network:
http://www.ecu.edu/cs-cas/psyc/rileytillmant/EBI-Network-Homepage.cfm

Florida Center for Reading Research:
http://fcrr.org

Intervention Central:
http://www.interventioncentral.org

Vaughn-Gross Center for Reading and Language Arts:
http://www.meadowscenter.org/vgc

Advocacy Groups and Organizations

International Dyslexia Association:
http://www.interdys.org

LD OnLine:
http://www.ldonline.org

National Dissemination Center for Children with Disabilities:
http://www.nichcy.org

National Center for Learning Disabilities:
http://www.ncld.org

National Research Center on Learning Disabilities:
http://www.nrcld.org

Centers for Disease Control and Prevention: *Facts About Intellectual Disability*:
http://www.cdc.gov/ncbddd/dd/ddmr.htm

Professional Associations

American Association on Intellectual and Developmental Disabilities:
http://www.aaidd.org

American Speech-Language-Hearing Association:
http://www.asha.org

National Association of School Psychologists:
http://www.nasponline.org

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23

Eating Disorders

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Until the 1960s and 70s, few people had heard of anorexia nervosa, but it soon began to be reported with increasing frequency in Western societies. Young females from middle-and upper-class families were voluntarily starving themselves and losing weight to the point of emaciation and sometimes death. A decade later, a new eating disorder was recognized; in bulimia nervosa, young women alternate between restricting and eating large amounts of food, often followed by purging. Although these eating disorders have flourished recently in Westernized societies during periods of relative affluence and enhanced social opportunities for women (Bemporad, 1996, 1997), voluntary self-starvation and periods of binge eating and purging have been reported throughout history. Eating disorders that would be recognizable today as anorexia nervosa and bulimia nervosa have existed since ancient times (Bemporad, 1997). Binge eating disorder and residual- and mixed-symptom eating disorders (i.e., other specified feeding and eating disorder [OSFED] and unspecified feeding and eating disorder [USFED], previously known as eating disorder not otherwise specified)[1] were first included in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994), and remain in the manual's current fifth edition (DSM-5; American Psychiatric Association, 2013).

Whether the various eating disorders are fundamentally different from one another is debatable (Fairburn, Cooper, & Shafran, 2003; Vohs & Heatherton, 2000). The transdiagnostic hypothesis cites compelling evidence of consistent core psychopathology, attitudes and behaviors across disorders (e.g., dietary restraint, binge eating, compensation, over-evaluation of eating, shape, and weight and its control) and frequent migration between diagnostic categories, therefore considering all eating disorders different manifestations of a single disorder (Fairburn et al., 2003). In this chapter, we discuss the major eating disorders, using DSM-5 to describe the primary features of anorexia, bulimia, binge eating disorder and OSFED. We then review the prevalence of eating disorders and provide an overview of the main hypotheses concerning what causes them to occur in a given individual. Finally, we discuss the principal treatment options and prognosis for eating disorders.

Diagnostic Criteria and Core Pathological Features of Eating Disorders

The DSM is the most widely accepted set of diagnostic criteria for psychological/psychiatric disorders in North America. DSM-5 updated the classification of psychological/psychiatric disorders from the revised fourth edition (DSM-IV-TR; American Psychiatric Association, 2000), based on the most current research. The symptoms of eating disorders were compiled and updated by a panel of experts who treat these problems, and the symptoms were then sent to others in the field for comments and corrections. In this version of the DSM, all symptoms are supposed to be empirically supported and to reflect a consensus among those who treat and study the disorders. For eating disorders, this is a difficult proposition. Not all criteria for the disorders are easy to define (for example, exactly what behaviors constitute binge eating?); the criteria as listed have some ambiguities. Moreover, there are controversies concerning just how universal some symptoms are (e.g., overconcern with weight and shape). Finally, many psychologists object to the medicalization of abnormal behavior implied by a diagnosis. Despite these debates about the utility of the DSM, many researchers

use the DSM criteria for eating disorders to facilitate communication across research settings by ensuring that all are studying the same phenomena. For clinicians treating these problems, diagnostic criteria can point to symptoms that need to be treated and methods of treating them, and also can allow for assessment of successful change. In this chapter, we discuss the criteria for eating disorders as set out in DSM-5, including noting key revisions to the criteria from DSM-IV-TR, as well as pointing out some of the issues and concerns about the current criteria.

The DSM-5 diagnostic criteria for anorexia nervosa focus on restriction of energy intake (e.g., food restriction, excessive exercise) that results in the maintenance of a very low weight, accompanied by an intense fear of fatness or disturbed experience of one's body weight/shape, or self-esteem that is unduly influenced by weight and shape. In addition, anorexia nervosa can be subtyped into either binge-eating/purging-type anorexia, which involves regular episodes of binge eating and/or purging over the preceding 3 months, whereas restricting-type anorexia does not involve these behaviors in the past 3 months (both types may involve fasting and excessive exercising). There are two primary changes to the diagnostic criteria of anorexia nervosa in DSM-5. In DSM-IV-TR, the first criterion focused on an individual's *refusal* to maintain a minimally adequate body weight, which was defined as less than 85% of what was normal for age, height, and sex. This has been amended in DSM-5 to focus on *behaviors* that result in significantly low weight, which is now defined as less than minimally normal for age, height and sex (i.e., the 85% criterion has been deleted, as it was being used as a cutoff rather than a guideline). The term "refusal" implied intentionality, which is difficult to assess compared with concrete behaviors such as food restriction. Secondly, DSM-5 has eliminated the criterion of amenorrhea; in the previous edition, this criterion required post-menarchal females to have an absence of three consecutive menstrual cycles for her symptoms to meet diagnostic criteria for anorexia nervosa. Although amenorrhea had been a key diagnostic criterion for anorexia nervosa for some time, women with anorexia nervosa and women with all the features of anorexia nervosa except amenorrhea are otherwise psychologically indistinguishable; in fact, recent research has shown that the presence or absence menstruation in anorexia nervosa primarily reflects severity of nutritional status rather than any meaningful psychological differences (Attia & Roberto, 2009; Cachelin & Maher, 1998; Garfinkel et al., 1996). In addition, this criterion could not be applied to premenarchal females, to females taking oral contraceptives, to post-menopausal females, or to males. As such, the amenorrhea criterion has been deleted in DSM-5. In addition to these two primary revisions, severity indices have also been added for anorexia nervosa, ranging from mild (body mass index, BMI, greater than 17.0) to extreme (BMI less than 15.0). The severity index does not change the diagnosis of anorexia nervosa, but rather provides additional clinical information about the patient's level of functioning.

DSM-5 describes bulimia nervosa as recurrent episodes of binge eating (i.e., eating more food than most people would eat in a similar time period and situation and feeling out of control of one's eating during the episode) accompanied by compensatory behaviors (such as vomiting, laxative or diuretic abuse, exercising, or fasting) in an effort to prevent a corresponding weight gain. These behaviors must occur on average at least once a week for at least 3 months. The core maladaptive cognition of bulimia nervosa (and anorexia nervosa) is also reflected in the diagnostic criteria, specifically that an individual's self-evaluation relies excessively on body weight and shape. If any of the symptoms of bulimia nervosa occur in the context of an episode of anorexia nervosa, then anorexia nervosa becomes the diagnosis. The frequency criterion for binge eating and compensatory behaviors is the primary revision to bulimia nervosa criteria in DSM-5, which was reduced from twice per week for 3 months in DSM-IV-TR to once per week in the present volume. This was based on a systematic literature review, which found that the clinical characteristics of individuals reporting bingeing and compensating once per week were similar to those meeting the current criterion of twice per week (Wilson & Sysko, 2009). Another recent study showed that individuals with once-weekly symptoms meeting DSM-5 criteria for bulimia nervosa are similar to those of patients with DSM-IV anorexia nervosa on a range of clinical and psychological constructs (MacDonald, McFarlane, & Olmsted, 2014). As such, research suggests that individuals with once-weekly symptoms are likely best classified as bulimia nervosa. An additional revision to DSM-5 is that bulimia nervosa is no longer subtyped. Previously, it was subclassified as purging type, which featured self-induced vomiting or laxative, diuretic, or enema abuse, or nonpurging type, which featured fasting or excessive exercise to compensate for binges, and the absence of purging behaviors (American Psychiatric Association, 1994). The elimination of bulimia nervosa subtypes is based on a review which showed that the purging and nonpurging types did not differ qualitatively, and that the nonpurging type was infrequently diagnosed, therefore failing to provide empirical support or clinical utility for the use of subtypes (van Hoeken, Veling, Sinke, Mitchell, & Hoek, 2009). In DSM-5, subtyping has been removed and fasting and compensatory exercise are included as inappropriate compensatory behaviors of bulimia nervosa. Finally, severity specifiers have been added to DSM-5, which focus on the frequency of compensatory behaviors, ranging from mild (one to three compensatory behaviors per week) to extreme (14 or more compensatory behaviors per week).

Impulsive behaviors such as sexual promiscuity, self-harm, suicide attempts, drug abuse, and stealing or shoplifting are common among people with bulimia nervosa (e.g., Goldner, Geller, Birmingham, & Remick, 2000). Specifically, the impulsivity observed in patients with bulimia nervosa has been associated with higher frequencies of purging behavior (Matsunaga, Kiriike et al., 2000).

Binge eating disorder is a new addition to the eating disorders section of DSM-5, although it was included in the previous edition in the appendix for criteria requiring additional research. Its inclusion in the current edition was based on a comprehensive literature review which demonstrated the validity and clinical utility of the binge eating disorder category (Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009). Binge eating disorder is similar to bulimia nervosa in that it entails recurrent binge eating episodes accompanied by subjective feelings of lack of control over one's eating on average at least once a week for 3 months. The difference between bulimia nervosa and binge eating disorder is that the compensatory behaviors that occur regularly in bulimia nervosa are not present or are infrequent in binge eating disorder (American Psychiatric Association, 2013). Additionally, for a diagnosis of binge eating disorder, the individual must experience at least three of the following five symptoms related to the binges: Eating much more quickly than usual; eating until uncomfortably full; eating a large quantity of food even though one is not feeling hungry; eating alone because of feeling embarrassment how much one is eating; and feelings of depression, disgust or extreme guilt about how much is being eaten. Individuals also experience marked distress about their binge eating episodes. Severity can range from mild (one to three binge episodes per week) to extreme (14 or more binge episodes per week). The binge eating in this disorder typically begins in late adolescence or the early 20s and frequently follows dieting and weight loss. Although not part of the diagnostic criteria, individuals with binge eating disorder are typically overweight, and do exhibit weight and shape overconcern that characterize people with anorexia nervosa and bulimia nervosa (Eldredge & Agras, 1996). Finally, "Other Specified Feeding or Eating Disorder" is currently a residual category for syndromes that do not quite fit the diagnostic criteria for the any specific eating disorders, and for which the clinician is able to describe why the disorder does not fit these other categories; for example, by being consistent with subthreshold anorexia nervosa, bulimia nervosa, or binge eating disorder, purging disorder (see Keel & Striegel-Moore, 2009) or night eating syndrome (see Striegel-Moore, Franko, & Garcia, 2009). "Unspecified Feeding or Eating Disorder" is a similar residual category but for which the clinician cannot or chooses not to describe why the disorder fails to meet criteria for a more specific diagnosis (e.g., if not enough information was obtained to concretely ascertain a specific diagnosis). These two categories were collectively labeled "Eating Disorder Not Otherwise Specified" (EDNOS) in DSM-IV. Interestingly, EDNOS is the most common diagnostic category found in community, outpatient (Fairburn & Bohn, 2005) and tertiary care treatment settings (Rockert, Kaplan, & Olmsted, 2007). Clearly, there is something amiss with the current classification system if the "residual" category captures the majority of individuals with eating disorders.

One of the goals for the DSM-5 working group was to improve and expand the categories of anorexia nervosa, bulimia nervosa, and binge eating disorder to reduce the number of people diagnosed with EDNOS. Preliminary research into the reclassification of eating disorders using DSM-5 has suggested that the revised criteria has in fact reduced the proportion of eating disorders classified as EDNOS. For example, one study assessed 397 individuals with eating disorders using both DSM-IV-TR and DSM-5 criteria, and found that using the DSM-5 criteria reduced the proportion of the sample with EDNOS from 68% to 53% (Keel, Brown, Holm-Denoma, & Bodell, 2011). That is, based on the revised criteria of anorexia nervosa and bulimia nervosa, and the inclusion of binge eating disorder, 15% of the sample was reclassified to these disorders rather than EDNOS, meaning that DSM-5 criteria provided greater diagnostic specificity. Another study examined the prevalence of eating disorders in large high-school and university samples, and found that use of DSM-5 criteria reduced the prevalence of EDNOS cases by approximately 22–29% by reclassifying these cases as more specific eating disorders (Machado, Goncalves, & Hoek, 2013). In addition to greater diagnostic specificity using the revised criteria, another study showed that use of DSM-5 rather than DSM-IV-TR criteria accounted for a greater proportion of the variance of symptom-rating measures (Birgegard, Norring, & Clinton, 2012), suggesting that the updated criteria have empirical validity in addition to clinical validity.

Despite the presence of binge eating as a diagnostic feature of bulimia nervosa, binge eating disorder, and anorexia nervosa-binge-eating/purging subtype, attempts to define binge eating have been unsatisfactory, and there is considerable variability in what sorts of eating episodes patients and professionals label as binges (Johnson, Carr-Nangle, Nangle, Antony, & Zayfert, 1997). What, for instance, is a larger than normal amount of food? A systematic review examined the validity of the criteria for binge eating and found that the large majority of binges do indeed typically take place in under 2 hours (Wolfe, Wood, Smith, & Kelly-Weeder, 2009), which is consistent with the DSM criteria. The reviewed evidence also supported the loss of control criterion during binge episodes; regardless of binge size, loss of control is consistently linked to the perception of an eating episode as a binge, and is also associated with increased caloric intake, greater distress, and increased eating disorder and other psychopathology (Wolfe et al., 2009). However, with respect to defining the size of a binge, laboratory and self-report studies have indicated extreme variability in the quantity of food consumed. In DSM, the size of a binge is quantified only as an amount of food that is definitely larger than most people would eat under similar circumstances, which can be challenging to operationally define and which is undoubtedly influenced by individual clinicians' subjectivity of what is larger than most would eat. Indeed, reviewed studies of binge size in bulimia nervosa reported mean binge sizes ranging from 1300 to 3500 calories, and studies of binge eating disorder reported mean binges ranging from 750 to

2900 calories (Wolfe et al., 2009). Although 750 calories could easily comprise a normal-sized meal, 3500 calories is nearly double what is recommended for most average women to eat in an entire day: Clearly there is wide variability in terms of how binge size is defined, which presents challenges for both clinical diagnosis and research.

Commonly noted triggers for binge eating include negative emotions (including depression, stress, anxiety, irritability, loneliness, anger, boredom, or generalized negative mood), depersonalization, hunger, and dietary restraint (Wolfe et al., 2009). Additionally, the presence of attractive "forbidden food," abstinence violation (i.e., having already eaten something fattening or diet-breaking), ingestion of alcohol, and being alone have also been noted as triggers for binges (Polivy & Herman, 1993). Examination of videotaped eating episodes indicates that feelings of loss of control and violation of dietary strictures are critical in leading participants to construe a particular episode as a binge (Johnson, Boutelle, Torgrud, Davig, & Turner, 2000).

Other information related to binge eating and purging includes that patients with anorexia and bulimia nervosa typically have greater psychopathology (e.g., depression, anxiety, self-harm and suicidality, impulsivity, and substance use) and poorer outcomes compared with patients with restricting-type anorexia nervosa, and research suggests that worsening of psychopathology leads to a substantial proportion of patients with anorexia nervosa "crossing over" from restricting-type to binge eating/purging-type (Peat, Mitchell, Hoek, & Wonderlich, 2009). Additionally, laxative abuse among women with eating disorders is an indicator of greater psychopathology, irrespective of other features such as eating disorder diagnostic category, age, body weight, impulsive behaviors, or personality features (Pryor, Wiederman, & McGilley, 1996).

Course, Incidence and Prevalence of Eating Disorders

Both anorexia nervosa and bulimia nervosa often emerge during late adolescence, with a female-to-male ratio between 10:1 and 15:1 (Bramon-Bosch, Troop, & Treasure, 2000; Braun, Sunday, Huang, & Halmi, 1999). Although these disorders are more prevalent in females, the nature of the disorders is the same in the two sexes. For individuals with anorexia nervosa or bulimia nervosa, a negative body image and a variety of psychological problems appear to emerge during puberty (see e.g., Polivy & Herman, 2002, for a review). Even before the onset of their disorder, adolescents with anorexia nervosa report various problems such as weight-related concerns, attitudes of withdrawal and social isolation, and deterioration of body image, self-image, and relationships with siblings and peers. The prevalence of these problems among adolescents with bulimia nervosa suggests that early psychological distress may precede the onset of an eating disorder (Corcos et al., 2000). Binge eating disorder is radically different from anorexia nervosa and bulimia nervosa in sex distribution, with only a 3:2 ratio of women to men, compared with 10:1 and 15:1 for anorexia nervosa and bulimia nervosa, suggesting possible etiological differences for this disorder (Grilo, 2006).

Hoek (2006) reviewed epidemiological studies to determine the incidence and prevalence of anorexia nervosa and bulimia nervosa (using DSM-IV-TR criteria); incidence rates are eight per 100,000 persons per year and 13 per 100,000 persons per year, respectively. The average point prevalence rates for anorexia nervosa and bulimia nervosa among young females are 0.3% and 1%, respectively. Stice, Marti, and Rohde (2012) examined epidemiological rates using DSM-5 criteria in a sample of 496 girls and young women, over an 8-year period, and found that, by age 20, the lifetime prevalence of DSM-5 anorexia nervosa and bulimia nervosa were 0.8% and 2.6%, respectively. As previously discussed, partial syndrome eating disorders (i.e., EDNOS) are even more common than either anorexia or bulimia, with point prevalence rates of 2.37% detected in a large community sample of adolescents and young adults (Machado, P. P. P., Machado, B. C., Gonçalves & Hoek, 2007). The lifetime prevalence estimates of binge eating disorder using DSM-IV-TR research criteria are between 2% and 4%, based on community samples, and 20–30% among individuals seeking weight-loss treatment (Hudson, Hiripi, Pope, & Kessler, 2007; Jager & Powers, 2007). Similarly, lifetime prevalence of binge eating disorder using DSM-5 criteria is estimated at around 3.0% (Stice et al., 2012). With respect to ethnicity, Hoek (2006) found that anorexia nervosa is a common disorder among young White females, but is extremely rare among Black females. In contrast, bulimia nervosa and binge eating disorder occur frequently among Black women, but might also be more common among White women. Diagnostic crossover is also common, particularly with respect to moving from subthreshold bulimia nervosa or binge eating disorder (i.e., EDNOS diagnosis) to one of either bulimia nervosa or binge eating disorder (Stice et al., 2009), as well as crossover from anorexia nervosa to bluimia nervosa (Peat et al., 2009). Crossover from bulimia nervosa to anorexia nervosa also takes place but appears to be less common than in the reverse direction (Peat et al., 2009).

There have been reports of an increase in the incidence of anorexia nervosa over the past century (e.g., Eagles, Johnston, Hunter, Lobban, & Millar, 1995; Moller-Madsen & Nystrup, 1992), as well as an increase in the incidence of bulimia nervosa since it was first described in the late 1970s (Turnbull, Ward, Treasure, Jick, & Derby, 1996). However, it is unclear to what extent these observed increases in the incidence of anorexia and bulimia are a result of increased awareness and recognition (Wakeling, 1996). Nevertheless, these incidence figures may well be significant underestimates; the disorders still often escape detection or diagnosis (Rooney, McClelland, Crisp, & Sedgwick, 1995) because dieting and the pursuit of thinness are so ubiquitous and socially acceptable

in Western culture, and because of the secretive nature of the disorder (Grilo, 2006). In a review, Hoek (2006) concluded that even the studies with the most complete case-finding methods yield an underestimate of the true incidence of eating disorders. He found that the most substantial increase in incidence over the past century was among females aged 15–24 years, for whom a significant increase was observed from 1935 to 1999. General-practice studies show that the overall incidence rates of anorexia nervosa remained stable during the 1990s, compared with the 1980s. Some evidence suggests that the occurrence of bulimia nervosa is decreasing. Despite increased awareness, only about one-third of the people with anorexia nervosa and only 6% of those with bulimia nervosa receive mental health care. Hence, the majority of individuals with eating disorders do not receive appropriate treatment.

Concurrent Psychological Problems

As many as 80% of patients with eating disorders who have been diagnosed in a clinical setting exhibit one or more other psychiatric disorders at some point, with major depression being the most common diagnosis (Grilo, 2006). People with eating disorders often engage in obsessive-compulsive behaviors such as calorie counting, body preoccupation, ruminations about food, ritualism, perfectionism, and meticulousness (Kaye, 1997). The lifetime prevalence of obsessive-compulsive disorder has been estimated to be 30% among people with eating disorders but only 2.5–3% among the general population. Obsessive-compulsive disorders are three times more common among people with anorexia nervosa than among people with bulimia nervosa (Hudson, Pope, & Jonas, 1983).

Several other disorders are frequently found in conjunction with eating disorders. Alcohol abuse is commonly found in bulimia nervosa, and anxiety disorders, depression and posttraumatic stress disorder are prevalent in both anorexia nervosa and bulimia nervosa (e.g., Blinder, Cumella, & Sanathara, 2006; Dansky, Brewerton, & Kilpatrick, 2000; Tagay, Schlegl, & Senf, 2010). A follow-up of a large sample of anorexia nervosa patients found that, 10 years later, 51% still met criteria for an Axis I psychiatric disorder (especially anxiety disorder), and 23% met the criteria for a personality disorder (most often avoidant-dependent and obsessive-compulsive; Herpertz-Dahlman et al., 2001).

Indeed, Axis II personality disorders are frequently observed among people with eating disorders (e.g., Wilfley et al., 2000; Wonderlich, 1995; Wonderlich & Mitchell, 2001). Obsessive-compulsive personality disorder, like obsessive-compulsive disorder, is especially common in anorexia nervosa, whereas disorders related to impulsive personality, as well as borderline personality disorders, are more often present in bulimia nervosa and BED (e.g., Wonderlich & Mitchell, 2001). Because malnutrition can exaggerate symptoms of personality disorders, Matsunaga, Kaye, and colleagues (2000) studied individuals who had recovered from eating disorders for at least 1 year to be certain that the measurement of personality disorders was not distorted by malnutrition and eating disorder symptomatology. They found that 25% of their participants met the criteria for at least one personality disorder.

Causal Theories of Eating Disorders

Eating disorders are not uniform conditions; there is no single cause. The etiology of eating disorders is not really understood as yet, but there seem to multiple causes and pathways (Grilo, 2006).

Hilde Bruch (1975), one of the first modern theorists to discuss eating disorders, described anorexia nervosa as "a complex condition determined by many simultaneously interacting factors" (p. 159). She was the first to point out that these patients use eating or not eating to fulfill a variety of non-nutritional needs. Moreover, despite the characteristic preoccupation with food and eating accompanying eating disorders, patients are "unable to recognize hunger or distinguish it from other states of bodily tension or emotional arousal" (p. 160). Bruch posited that people with eating disorders interpret all tension states as a "need to eat" instead of identifying the correct source.

Fairburn (e.g., Fairburn, Welch, Doll, Davies, & O'Connor, 1997) hypothesizes that there are two broad classes of risk factors for eating disorders: those enhancing the general risk for psychiatric disorder and those that specifically increase the risk for dieting and eating problems. With respect to risk factors, patients with bulimia nervosa resemble patients with other psychiatric disorders more than they resemble people without diagnosable psychological problems. Patients with bulimia nervosa, however, show a distinctive pattern of exposure to factors likely to elevate the risk of dieting and negative self-evaluation, supporting the hypothesis that bulimia nervosa is the result of exposure to both general risk factors for psychiatric disorder and specific risk factors for dieting.

The addiction model (Davis & Claridge, 1998; Wilson, 1991) posits an addictive process operating in eating disorders. Conditioned physiological responses to food produce anticipatory secretion of insulin, which causes both craving and overeating, if only through increased tolerance to food (Booth, 1988; Wilson, 1991; Woods & Brief, 1988). Some argue that self-starvation accompanied by excessive exercising reflects an addiction to the body's endogenous opioids (Davis & Claridge, 1998). These authors cite high scores by patients with anorexia nervosa and bulimia nervosa on the Addiction Scale of the Eysenck Personality Questionnaire, as well as correlations between the addiction scale and both weight preoccupation and excessive exercising as evidence of this. More recently, some authors have begun to conceptualize binge eating disorder within the addiction model. Specifically, the compulsive, driven nature of eating, and loss of

control over eating in binge eating disorder are similar to characteristics observed in individuals with substance-use disorders; other similarities may include intense cravings for food or drugs, and chronic relapsing behaviors both for weight and binge eating and for drug use (Davis & Carter, 2009).

In contrast, Wilson (1991) has dismissed the addiction model for three reasons. First, he claims that compelling evidence for an addictive personality is lacking. Second, the model does not address the core clinical characteristics of eating disorders (e.g., the role of dietary restraint and abnormal attitudes about the importance of body shape) or the identified concomitant psychopathology (e.g., extremely low self-esteem, interpersonal distrust, and feelings of ineffectiveness). Third, it fails to account for psychobiological connections between dieting and eating disorders. Wilson notes that bulimic behavior does not meet the criteria for an addictive disorder (i.e., tolerance, physical dependence, or craving), and therefore he sees the addiction model as a conceptual dead end. In contrast, with respect to binge eating disorder, a 2009 review of research has provided more convincing evidence of these traditionally substance-related constructs being present in eating (Davis & Carter, 2009). With respect to tolerance, laboratory evidence indicates that sugar-enhanced diets in animals increases caloric intake as time passes, and in binge eating disorder, binge sizes progressively increase as the disorder becomes chronic. In terms of withdrawal, evidence suggests that sudden removal of sugar from the diet in both humans and animals on high-sugar diets can lead to symptoms of symptoms similar to substance withdrawal. Those with binge eating disorder report significantly higher cravings and greater preferences for high-sugar and high-fat foods compared to same-weight individuals without binge eating disorder. Additionally, several studies have also shown that the rewarding effects of substances and food are modulated by common dopamine pathways (Davis & Carter, 2009). As such, although the addiction model of eating disorders remains controversial, some evidence indicates that binge eating disorder may be a phenotype of addictive processes.

Cognitive theories of eating disorders emphasize the biases in people's beliefs, expectancies, and information processing pertaining to body size and eating. Some research has supported predictions derived from these cognitive theories (Williamson, Muller, Reas, & Thaw, 1999). As we discuss later in this chapter, these theories dominate current treatment strategies. Information-processing biases, such as a focus on food and weight to the exclusion of other information, may explain several psychopathological features of anorexia nervosa and bulimia nervosa, including denial, resistance to treatment, and misinterpretation of therapeutic interventions.

Personality factors are also thought to contribute to susceptibility to eating disorders. Some assessment instruments such as the Eating Disorders Inventory (Garner, Olmsted, & Polivy, 1983) were specifically designed to measure underlying personality dispositions theoretically linked to eating disorders. Investigations using this scale have found that personality factors such as perfectionism, feelings of ineffectiveness (or low self-esteem), reduced interoceptive awareness (or sensitivity to internal signals such as hunger and satiety), and interpersonal distrust are characteristic of those with eating disorders (e.g., Garner, Olmsted, Polivy, & Garfinkel, 1984; Leon, Fulkerson, Perry, & Early-Zald, 1995). Strober (1980) found evidence of obsessive personality traits, extraversion, and need for social approval before the development of the disorder in adolescents with anorexia nervosa who had returned to normal weight.

One investigation took the innovative step of simply asking the patients what caused the emergence of their eating disorder (Nevonen & Broberg, 2000). Interpersonal and weight-related problems were the most commonly reported causes, and dieting or dieting plus purging was the most commonly reported response to these stresses. Thus, interpersonal and weight-related distress, together with dieting behavior, are what people see as responsible for the emergence of their eating disorders. Self-report—in response to oral or written questions—is one of the main sources of data in research on eating disorders; unfortunately, self-report is notoriously unreliable (Nisbett & Wilson, 1977). Eating disorder researchers by and large are too eager to take patients' self-descriptions at face value, if only because such descriptions are easy to elicit. It is not likely that people with eating disorders would be able to identify the source of their disorder with such ease when the research of several decades has not been able to do so. A full understanding of the causes of eating disorders will require more work and more sophisticated research designs than are currently being used.

Sociocultural models suggest that the idealization of thinness and unremitting portrayals of slim role models in the media contribute to widespread body dissatisfaction, which in some susceptible individuals produces pathological dieting and ultimately eating disorders (e.g., Heatherton & Polivy, 1992; Stice, 2001; Striegel-Moore, 1993). Some research suggests that exposure to idealized models may promote dieting not by causing body dissatisfaction but rather by inspiring young women to work toward a fantasized, thinner future self (Mills, Polivy, Herman, & Tiggemann, 2002). The sociobiological position posits that eating disorders and the societal pursuit of thinness reflect sexual competition among women (Abed, 1998). Thinness may either enhance women's reproductive prospects by making them more attractive to males or, in a contrary version, delay reproduction (by impairing fertility through emaciation) until a more propitious time.

Speculation about the cause(s) of eating disorders has thus gone through many phases over the last three decades, variously favoring biological, familial, and psychosocial factors, which are hypothesized to interact in complex ways to produce the disorders (Ward, Tiller, Treasure, & Russell, 2000). For at least the past decade

(1990s to 2000s), the biopsychosocial model, positing an interplay between the organism, its past behavior, and its environment (biological, psychological, and environmental variables) has been the primary explanatory model of eating disorders (e.g., Schlundt & Johnson, 1990). For example, in binge eating, environmental (e.g., situational influences, sociorelationship systems), behavioral (e.g., previous eating or dieting, ongoing activity), cognitive (e.g., knowledge of dieting, expectations, body image), emotional (e.g., mood, psychopathology), and physiological (e.g., blood levels of nutrients, hormones, neurochemicals) antecedents affect behaviors such as binge eating, purging, and dieting, which then themselves have consequences for behavior, cognition, emotion, and physiology.

Risk Factors for Eating Disorders

Current theories of eating disorders—what causes them, why they mainly afflict women, and why there has been such an increase in recent times—thus range widely. The basic question of what goes wrong to produce eating disorders has been addressed at the broad level of sociocultural influences, at the narrow level of familial and environmental effects, and at the even narrower level of individual risk factors related to personality, cognition, or physiology (e.g., Leung, Geller, & Katzman, 1996; Polivy & Herman, 2002; Polivy, Herman, Mills, & Wheeler, 2003). One might expect that the research on specific risk factors would be derived from one or another of the causal theories, but such is not the case. The voluminous literature on risk factors is to a great extent independent of and uninformed by the causal theories outlined previously. Whereas some self-report questionnaires (such as the Eating Disorders Inventory) are based on theoretical models (such as Bruch, 1975), research on risk factors for eating disorders seems to be, for the most part, atheoretical. Many studies in this area merely report correlations among self-report inventories and make no attempt to tie the particular measures chosen to any particular theory of how eating disorders develop. In part, this situation no doubt reflects the difficulty of measuring preclinical pathological processes with paper-and-pencil self-report devices, as well as the problem of operationalizing theoretical constructs. Despite these drawbacks, literally thousands of studies have attempted to specify the risk factors for eating disorders, and numerous chapters and books have addressed these questions. It is crucial to remember, however, that the risk-factor approach is essentially correlational, and that risk factors are really just variables associated with eating disorders, usually in an unspecified fashion. Space limitations permit only a brief overview of prominent research trends examining risk factors associated with eating disorders.

Sociocultural Factors For several decades, an unrealistically thin body shape has been the cultural ideal for women in Western society. To attain this physique, women have become increasingly likely to diet and/or exercise (e.g., Polivy & Herman, 1987; Stice, 2001), but are unlikely to succeed in this quest. This societal obsession with a slim female body has been blamed for women's widespread dissatisfaction with their bodies and a concomitant rise in the prevalence of eating disorders (Stice, 2001). These sociocultural pressures have long been targeted as causes (or at least contributors) to eating disorders (e.g., Grilo, 2006; Striegel-Moore, 1993). Girls who participate in sports or professions that emphasize having a slim body, such as gymnastics or ballet, seem to be more at risk than usual, supporting the view that the sociocultural emphasis on slimness contributes to the disorder (Davison, Earnest, & Birch, 2002). The chief promoters of this sociocultural pressure to be thin are the media, sex-role expectations, and particular economic, racial, and ethnic contexts.

Peer and Media Influences To no one's surprise, it has been shown that adolescents watch more television, read more magazines, and in general appear to attend more to the media than any other age group, and they are thus bombarded with messages about thinness and dieting. These messages are directed primarily at girls, who are presented with thin, attractive models, an insistence that thinness will bring success and happiness in all spheres of life (no matter how unrelated to appearance), and a blatant derogation of fat or even normal-weight physiques (Polivy & Herman, 2002; Polivy et al., 2003). Many studies have demonstrated an increase in depression and negative self-image in young women following exposure to thin media images (Dittmar, 2005; Pinhas, Toner, Ali, Garfinkel, & Stuckless, 1999), and those with greater eating disorder symptomatology tend to expose themselves to larger doses of these media images than do most women their age (Stice & Shaw, 1994).

Some data, however, suggest that it would be premature to blame the media for the increase in eating disorder. Mills, Polivy, Herman, and Tiggemann (2002) found that looking at pictures of thin models actually made chronic dieters feel thinner and better about themselves, possibly by inspiring them to fantasize about emulating the models. This outcome should not be so surprising—after all, why would women voluntarily buy and read fashion magazines if looking at them induced depression and self-derogation? More important, eating disorders have been reliably documented for centuries, without the benefit of 20th-century media exposure. Media idealization of an unrealistically thin female shape may be a contributor to the increased prevalence of eating disorder, but it is clearly not the primary cause. After all, this "cause" is so prevalent in our culture that if it were as important as is sometimes claimed, we would be hard pressed to explain why fewer than 10% of young women have eating disorders.

Gender "Why women?" and "Why now?" are the two most frequently asked questions about the recent increase

in the prevalence of eating disorders (e.g., Polivy et al., 2003; Striegel-Moore, 1993). Around the time that eating disorders began to proliferate, the social role of women was undergoing drastic changes. Instead of enacting the role of homemaker and mother, women were expected to fulfill the "superwoman ideal," requiring them to be smart, beautiful, have a successful career, and still maintain a perfect house, perfect children, and a perfect relationship with the perfect man. The stress of trying to be all things to all people may drive some young women to focus on one thing that they think they can control, their weight. The societal preference for thin female shapes may provide a goal that seems more attainable than becoming a superwoman, or perhaps even a means of becoming a superwoman. This role change may help to explain "Why women?" and "Why now?"

In addition, the pressure to be thin makes women dissatisfied with their bodies. When dissatisfied with their bodies, men tend to turn to healthy eating (Nowak, 1998) and exercise, but women turn to dieting (Drewnowski, Kurth, & Krahn, 1995). Dieting, however, is more likely to produce eating disorder symptomatology than sustained weight loss (e.g., Polivy & Herman, 1993; Stice, 2001). The thin ideal thus produces body dissatisfaction in women, which elicits dieting, which encourages pathological eating. However, if this were the whole story, all female adolescents who are unable to achieve thinness (or at least all who diet) would be eating disordered, which is clearly not the case. Therefore we must look further for an explanation.

Race, Ethnicity, and Social Class Until about 20 years ago, eating disorders were primarily restricted primarily to Western, middle- and upper-class, White adolescent females. Now, however, they have penetrated all social classes, races, and even some non-Western venues such as Japan. Although the prevalence is still lower in most non-White groups and nonindustrial countries (Polivy et al., 2003), Black women in the United States are as likely as are White women to display eating disorder symptoms (Mulholland & Mintz, 2001). In addition, they are quickly becoming comparable to their White counterparts with respect to body-image dissatisfaction (Grant et al., 1999), vomiting, and abuse of laxatives or diuretics, and they are more likely to report binge eating (Striegel-Moore et al., 2000). Among the several potential explanations for the globalization of eating disorders, the most obvious is the influence of the media (Nasser, 1997).

Young minority-group females who are heavier, better educated, and who identify more closely with White, middle-class values have an increased risk of eating disorder. Assimilation into White culture thus carries with it an associated risk of developing eating disorders (Cachelin, Veisel, Barzegarnazari, & Striegel-Moore, 2000). After acculturation, non-White women appear to have eating attitudes similar to those of Western Whites, although some studies do not find this (e.g., Ogden & Elder, 1998). Black women in predominantly Black cultures, however, still seem much less likely to develop eating disorders, with a recent study in Curacao finding no cases at all in the majority Black population (Hoek et al., 2005).

The expression of eating disorders among non-White groups differs in some ways from what is found with White women. Minority women with eating problems are usually truly overweight, whereas White women with disordered eating may only feel overweight, but actually tend to be normal weight (Striegel-Moore et al., 2000). Body dissatisfaction and fear of fatness occur less frequently, if at all, among non-White groups, but Black adolescents are more likely to binge and use laxatives than are Whites, who are more likely to restrict their eating (Striegel-Moore et al., 2000). Black adolescent girls may experience less pressure to conform to the thin ideal plaguing White girls and seem less inclined to derive their self-esteem, identity, and perception of self-control from their weight and appearance (Polivy et al., 2003). When eating disorder symptoms do occur among non-Whites, however, they seem to be related to the same risk factors and precipitants—including low family connectedness, perfectionism, emotional distress, body dissatisfaction, and sometimes serious depression and anxiety (e.g., Davis & Katzman, 1999)—as they are among Whites. Mixed race women (in Curacao) who experienced the identity challenge of being of mixed race and trying to fit into the wealthier White subculture and distance themselves from the Black majority were also particularly likely to develop eating disorders (Katzman, Hermans, Van Hoeken, & Hoek, 2004).

Familial and Environmental Influences If societal values and pressures can have enough impact on an individual to constitute a risk factor for the development of an eating disorder, how much more influential are familial interactions? The family can be a source of cultural transmission of pathological values or a stressor on its own (through miscommunication, lack of emotional support, or internal conflict). It can also be a protective factor.

Family influences, such as how the family mediates cultural ideas about thinness and how family members convey these messages to each other, have been implicated as causes of eating disorders (Haworth-Hoeppner, 2000; Laliberte, Boland, & Leichner, 1999; Strober, Freeman, Lampert, Diamond, & Kaye, 2000). A critical family environment, coercive parental control, and a dominating discourse on weight in the household appear to increase the risk of eating disorders (Haworth-Hoeppner, 2000). Even during treatment, family influences are important. For example, it was found that mothers' expressed emotion (especially critical comments) predicted an adverse outcome for eating disorder patients (Vanfurth et al., 1996).

Both mothers and fathers can contribute to the development of eating disorders. For example, mothers who themselves have an eating disorder transmit pathological

behaviors to their daughters by the time the girls are 5 years old. Even when the mothers do not have eating disorders, one recent study identified mothers' abnormal eating attitudes as a potential risk factor for the development of an eating disorder among their adolescent daughters (Yanez, Peix, Atserias, Arnau, & Brug, 2007). Mothers of daughters with eating disorder symptoms tend to find their daughters less attractive and more in need of weight loss than do mothers whose daughters are asymptomatic. Mothers' comments about weight and shape convey their attitudes and behaviors to their daughters. Daughters of mothers who diet are more likely to do so themselves, and to use more extreme weight-loss techniques if mothers encourage them to or are dissatisfied with their own bodies (Polivy & Herman, 2002; Polivy et al., 2003). Moreover, if either parent is inaccurate in estimating their daughter's body esteem, the daughter is more likely to have greater body dissatisfaction (Geller, Srikameswaran, Zaitsoff, Cockell, & Poole, 2003).

Critical comments and expression of negative emotions by either parent predict both the development of anorexia nervosa and worse outcome for anorexia nervosa patients. Parental comments which encourage weight/size control have been found to be stronger predictors of daughters' body dissatisfaction (which predicts disordered eating) than any other types of parental comments (Kluck, 2010). Mothers' complaints about a lack of family cohesion also foretell daughters' increased eating pathology 2 years later. High parental expectations for achievement that ignore the daughter's needs and goals have also been detected in girls with eating disorders; these extrinsic goals force these girls to struggle to please others at the expense of their own autonomy. Parents of both anorexia nervosa and bulimia nervosa patients appear to discourage autonomy, negating their daughters' needs and self-expression. The patients feel that their parents are overcontrolling in an affectionless manner. The fact that these sorts of parent–adolescent conflicts over autonomy and identity tend to be more intense among girls than among boys may help to explain why eating disorders are so much more common among females than among males (Polivy et al., 2003).

Like other psychopathologies, the environment can contribute to the etiology of eating disorders. For example, weight-related teasing has been associated with unhealthy weight control behaviors, weight-related attitudes, dietary restraint, bulimic behaviors and clinical eating disorders (Eisenberg, 2008; Menzel et al., 2010). Early traumatic events are also a common theme among this population, with studies reporting around 60% of individuals with eating disorders experiencing at least one trauma in their life (Tagay et al., 2010). Examples of traumatic life events could include neglect, exposure to violence, or physical, emotional, and sexual abuse. Exploratory studies have found that elevated levels of perceived stress precede the onset of binge eating disorder, and when compared with overweight/obese controls, more reports of neglect, emotional abuse, and higher depression levels have been observed (Allison, Grilo, Masheb, & Stunkard, 2007; Striegel-Moore & Bulik, 2007). Early experience can also have an impact on treatment outcome in eating disorders. One South American study examined treatment response among 70 women who had unsatisfactory outcomes and 90 women with more favorable responses. They found that the poorest outcomes were observed among those that had been exposed to sexual abuse or violence. It was also noted that this group was 10 times more likely to drop out of treatment and three times more likely to relapse versus non-exposed patients (Rodríguez, Pérez, and García, 2005).

Again, it is important to remember that trauma is not the entire story. There is no evidence to indicate that early abuse or trauma necessarily causes eating psychopathology; not everyone who suffers a traumatic event will develop an eating disorder, and not everyone who suffers from an eating disorder has been exposed to traumatic events. In fact, once study showed that depression fully mediated the association between some forms of childhood trauma and disordered eating (Kong & Bernstein, 2009). This highlights the importance of not assuming causality in correlational studies, but also indicates that early intervention aimed at depression might help to create a buffer against the development of eating disorders among traumatized individuals.

Individual Factors If sociocultural influences on pathogenesis were very powerful, anorexia nervosa and bulimia nervosa would presumably be more common than they are. In addition, the numerous clear descriptions of anorexia nervosa from at least the middle of the 19th century, and possibly earlier, suggest that factors other than our current culture cause the disorders. Moreover, although there is some evidence that the disorders may afflict more than one offspring in a family, if the family were responsible for the development of an eating disorder, we would expect to see greater concurrence among the female offspring in a family. Thus, whereas culture, family, and the environment contribute to the development of eating disorders, they are not sufficient to explain the appearance of a disorder in a given person. In light of the causal models discussed earlier, perhaps we may find the missing piece of the puzzle in factors specific to the individual, such as biological, identity, personality, or cognitive determinants.

Genetic and Physiological Factors The reasonably stereotypic and reliable clinical presentation of anorexia nervosa and bulimia nervosa, with a consistent sex distribution and age of onset, suggests a biological substrate for the disorders. Twin studies, family studies, and recent molecular-genetic findings point to a potential genetic factor (e.g., Klump & Gobrogge, 2005), especially for anorexia nervosa. Females with a first-degree relative who has an eating disorder are two to three times more likely to develop one themselves (Polivy et al., 2003). Research has

attempted to link specific genes to specific eating disorders, suggesting that anorexia nervosa may be linked to a different gene than bulimia nervosa (Grilo, 2006; Tozzi & Bulik, 2003). One interesting twin study also revealed that the role of genetics may be more important for females and the role of the environment may be of greater significance for males in the context of body satisfaction and drive for thinness (Baker et al., 2009).

A genetic predisposition to eating disorders may operate through faults in neurotransmitters, of which serotonin is the most frequently studied, possibly because it inhibits feeding, stimulus reactivity, and sexual activity. A disturbance of serotonergic activity is consistent with many accounts of patients with anorexia, and is correlated with traits that are common among such populations including higher drive for thinness and body dissatisfaction ratings (Frieling et al., 2006). Patients with bulimia nervosa show signs of serotonin dysregulation, which may contribute to their symptoms, including binge eating (Steiger et al., 2001, 2006). Serotonin may be associated with impulsivity, which may relate to the binge eating behavior and other impulsive behaviors exhibited by bulimia nervosa patients (Steiger et al., 2001). Early research also indicates a genetic component to binge eating disorder that cannot be substantially improved by including shared environment (Javaras et al., 2008). Additionally, recent research has shown that binge eating disorder is related to two specific genotypes reflecting enhanced dopamine transmission (Davis et al., 2012), and that individuals with binge eating disorder experience increased dopamine transmission in the striatal region of the brain in response to food stimuli after receiving a dopamine-blocking agent, but not placebo (Wang et al., 2011). Both of these findings suggest that individuals with binge eating disorder may be hypersensitive to reward via the dopaminergic pathway. More research is needed to determine whether or not other neurotransmitter abnormalities, such as serotonin, also exist within patients with binge eating disorder. Because the evidence for the influence of neurotransmitter dysregulation on eating disorders is strictly correlational, it is not possible to conclude that abnormal serotonergic or dopaminergic activity causes eating disorders; indeed, it may instead be a consequence of the disorder.

A surge of intrauterine twin studies has recently shed light on the influence of hormones and susceptibility to eating disorders. Findings consistently demonstrate that same-sex female twins have the highest risk of developing eating disorders, followed by females of opposite-sex twins, males of opposite-sex twins, and finally same-sex male twins (Culbert, Breedlove, Burt, & Klump, 2008; Procopio & Marriott, 2007). What is particularly interesting is the fact that males of opposite-sex twins have a higher risk of developing an eating disorder than do other males, and this risk is not significantly different from that of female opposite-sex twins. These results support the notion that prenatal hormones can influence one's susceptibility for developing disordered eating in adulthood. Findings from studies by Klump and colleagues (2006) support this theory by showing that both lower levels of prenatal testosterone and elevated adult levels of estradiol are associated with increased eating disorder symptoms. They hypothesized that relatively low testosterone levels before birth in females predisposes their brains to respond to estrogen at puberty, when the hormones activate the genes contributing to disordered eating in vulnerable individuals.

Puberty seems to be a critical period for the development of eating disorders, owing to genetic and hormonal influences. The tracking of changes in genetic and environmental factors has revealed that the influence of genetics seems to account for roughly half of the variance in disordered eating at ages 14 and 18 among same-sex female twins, compared with only 6% at age 11 years (Klump, Burt, McGue, & Iacono, 2007). Puberty in females is also accompanied by increased body fat and a drastically different body shape—the curves that develop in pubescent girls definitely subvert the thin ideal to which girls aspire. This new, curvy shape may give rise to the increased body dissatisfaction and accompanying dieting that become normative in adolescent females and that are themselves potential risk factors for eating disorders (e.g., Polivy et al., 2003; Striegel-Moore, 1993). At the same time, hormonal development pushes girls into heterosexual interactions, which girls appear to find more stressful than do boys (Striegel-Moore, 1993). Finally, developmental physical factors such as premature birth or birth trauma, being born between April and June, early childhood eating or digestive problems, early puberty and having a high premorbid BMI all seem to increase the risk of developing an eating disorder (Touyz, Polivy, & Hay, 2008).

Self-Esteem and Identity Among dieters who do not have eating disorders, depression scores are elevated and self-esteem scores are reduced; these scores are even more extreme among those with eating disorders (e.g., Polivy & Herman, 2002; Polivy et al., 2003), as they are in certain other psychological syndromes. Negative emotions and low self-esteem are linked to disruptive eating behaviors in a cyclic or spiral pattern (e.g., Polivy et al., 2003). Negative self-evaluation is possibly the most ubiquitous risk factor among eating disorder patients (Fairburn, Cooper, Doll, & Welch, 1999; Grilo, 2006); feelings of ineffectiveness and lack of a strong sense of self were recognized as hallmarks of eating disorders as early as the 1970s (e.g., Bruch, 1975).

The lower self-esteem among those with eating disorders also seems to be connected to a lack of a cohesive identity and distrust of one's body's ability to function on its own, both of which appear to be distinctively related to eating disorders. Self-esteem deficits may also reflect negative life experiences such as sexual or emotional abuse, which may also interfere with formation of a stable identity.

Owing to already low self-esteem, individuals with eating disorders (and to a lesser extent, chronic dieters) begin to connect their self-worth to their weight and shape. This connection continues to decrease overall self-esteem, and can perpetuate eating disordered behaviors (McFarlane, McCabe, Jarry, Olmsted, & Polivy, 2001). For example, chronic dieters (restrained eaters), who resemble patients with eating disorders in many ways, have been studied in the laboratory to learn about processes in eating disorders. Restrained eaters exposed to false feedback (from an inaccurate scale) indicating that they have gained weight react by overeating (McFarlane, Polivy, & Herman, 1998). Similarly, when allowed to eat after reporting about ways in which they have failed to meet their life goals, restrained eaters responded by overeating (Wheeler, Polivy, & Herman, 2002). Threats to one's identity seem to elicit disordered eating behavior (Polivy & Herman, 2007).

Body Dissatisfaction and Dieting The negative self-perception and low self-esteem characterizing eating disorders crystallize in negative feelings about one's body and an investment in improving it as a means of self-redemption (Polivy & Herman, 2002, 2007). The dual-pathway model posits that it is the thin ideal that produces body image dissatisfaction, which then produces dieting, and eventually eating disorder symptomatology (Stice, 2001). Adolescent girls do have greater body dissatisfaction than do boys or older women, and those who place more emphasis on their bodies and are more dissatisfied with them tend to exhibit greater eating pathology (Dittmar, 2005; Polivy et al., 2003). Moreover, dieting has been implicated as a cause of binge eating and is thought to be a primary contributor to the "dieting disorders," as eating disorders are often called (Polivy & Herman, 2002). But if all the girls who embraced the thin ideal, dieted, and were dissatisfied with their bodies became eating disordered, much of the adolescent female population would be incapacitated. Thus, although most models of eating disorders include a role for body dissatisfaction and dieting, it is clear that these risk factors must interact with other factors such as extremely low self-esteem and identity deficiencies, other personality factors, and the familial, environmental and cultural issues discussed earlier.

Personality Factors Those with eating disorders exhibit a consistent pattern of personality traits before, during, and after the disordered eating phase. Converging evidence from clinical reports, psychometric studies, and family or collateral sources presents the premorbid (i.e., before the onset of the disorder) personality of patients with anorexia nervosa as perfectionistic, obsessive, socially inhibited, compliant, and emotionally restrained or fearful (e.g., Grilo, 2006; Wonderlich, 1995). Those with bulimia nervosa are not only compliant and perfectionistic, but also tend to be impulsive, emotional, lacking in interoceptive awareness, and extraverted (e.g., Lilenfeld et al., 2000; Steiger et al., 2001). Perfectionism and negative self-evaluation characterize patients with anorexia nervosa and bulimia nervosa before, during, and after recovery from the disorder (Fairburn et al., 1999). Negative affect, behavioral inhibition, and obsessiveness also continue after recovery from anorexia nervosa (Kaye, Gendall, & Strober, 1998). Moreover, perfectionism, ineffectiveness and interpersonal distrust are found in family members of eating disorder patients who do not themselves show symptoms of eating disorders (Lilenfeld et al., 2000). There are thus a number of personality traits linked to eating disorders. Although the evidence is correlational, the persistence of such traits before and after the disorder and their presence in nondisordered family members suggest that they could interact with adverse familial, environmental and social experiences and render an individual more likely to develop an eating disorder (e.g., Lilenfeld et al., 2000; Strober & Humphrey, 1987).

Cognitive Factors People with eating disorders display several cognitive aberrations such as obsessive thoughts, distortions of attention and memory, and rigid, all-or-nothing thinking (Polivy & Herman, 2002). Obsessing about weight and shape, and using these characteristics to determine one's self-worth, are central and defining features of eating disorders. Biased attention and memory related to weight, shape and food-related information reflects a preoccupation with these issues. Studies using the Stroop color-naming task have empirically demonstrated biased attention to weight, shape and food-related words in patients with bulimia nervosa, and a bias to weight and shape-related words in patients with anorexia nervosa (Dobson & Dozois, 2004). A memory bias for shape and weight-related words has recently been demonstrated in patients with binge eating disorders, such that these patients recall less positive weight and shape-related words than an overweight control group (Svaldi, Bender, & Tuschen-Caffier, 2010).

All-or-nothing, black-and-white thinking can lead to perceiving the self as a failure if one bite of a "forbidden" food is ingested, and may promote binges (Polivy & Herman, 2002). The prominent role of cognitive features in eating disorders has encouraged the use of cognitive therapies to normalize the cognitions that are presumed to underlie the eating disorder.

Combining Risk Factors Because risk-factor research is of necessity correlational, it cannot determine cause and effect. Furthermore, no single risk factor alone is capable of producing an eating disorder, and it would be simplistic to suppose that a disorder as complex as eating disorders would have a single cause. In 1987, Johnson and Connors proposed a biopsychosocial model of eating disorder development. This model posits that biological predispositions

(from genetic, hormonal, and pubertal influences), family factors, and cultural pressures to be thin and to fulfill a demanding social role interact to produce an identity-conflicted, vulnerable dieter. Those dieters with low self-esteem and affective instability are most susceptible to an eating disordered identity, particularly in response to stress or failure. Striegel-Moore (1993) has suggested that the number of simultaneous life challenges faced by an individual is related to her susceptibility to eating disorders. Girls who mature early and begin dating at the same time report more disturbed attitudes about eating and shape. Striegel-Moore also posits that early maturation is a source of adjustment difficulties, which, along with body dissatisfaction, dieting, and conflict with parents is sufficiently stressful to increase risk for an eating disorder. A related theory proposes that the many transitions that occur in adolescence (e.g., moving to junior high or high school, puberty, dating, disruptions in friendships, increased academic and sex role demands) and the restructuring of personality and behavior demanded by these transitions may overwhelm an adolescent who is already vulnerable because of familial or personal problems (e.g., Smolak & Levine, 1996).

The combination of (a) the contextual background of sociocultural expectations to look and act a certain way; (b) familial interaction patterns that negate the individual's attempts to achieve autonomy; (c) individual vulnerabilities based on genetics and personality, self-esteem, and identity deficits; and (d) the stresses required by transitional adjustments or traumatic events offers a potential starting point for identifying individuals most likely to develop an eating disorder. Although these combinatory models attempt to incorporate the complexity of eating disorders and the many identified risk factors, they have not yet provided predictive power sufficient to identify individuals most likely to succumb to the pressures and develop eating disorders (see, e.g., Jacobi, Haywood, de Zwaan, Kraemer, & Agras, 2004, for a review).

Prevention Most of the interventions for eating disorders are therapeutic rather than preventative; that is, we tend to focus our efforts on treating eating disorders rather than preventing them from arising in the first place. An effective public health effort—stopping eating disorders before they get started—probably would be more valuable than effective therapy, but preventative interventions require a better understanding of what causes the disorder.

Early research in prevention showed mixed results, often producing positive effects (such as improved attitudes and behaviors) immediately following prevention programs only to find a complete reversal of these results after 6 months and sometimes even worse effects than before participants started (Carter, Stewart, Dunn, & Fairburn, 1997). This led some to posit that prevention programs might do more harm than good; fortunately, subsequent research has helped to clarify the situation. In a review of 29 programs, Dalle Grave (2003) concluded that "school-based eating disorder prevention programs do not have harmful effects on student attitudes and behaviors" (p. 579). Negative effects were typically limited to uncontrolled programs that were run by poorly trained educators (O'Dea, 2005; O'Dea & Abraham, 2001). More recent research is much more promising including programs such as Healthy Schools—Healthy Kids (McVey, Tweed, & Blackmore, 2007), which was successful in reducing weight-loss behaviors and the internalization of media ideals among male and female students, and in reducing disordered eating among females, especially with high-risk participants. Many of these results, however, also disappeared by the 6-month follow-up. More research is needed to determine how to produce longer-lasting and larger-scale positive results. Early meta-analyses and review papers can offer some insight into how to effectively tailor such programs. Stice and Shaw (2004) found larger effects for programs targeting high-risk individuals, interactive and multisession programs, programs offered solely to females and to participants over age 15, programs without psychoeducational content, and for trials that used validated measures. Building on these findings, Yager and O'Dea (2008) confirmed that psycho-educational, information-based, and cognitive behavioral approaches were among the least effective, while media literacy- and dissonance-based educational approaches and incorporating health education activities that build self-esteem were identified as successful elements.

Treatment of Eating Disorders

This section reviews the basic rationale and techniques of the major therapies used in the treatment of eating disorders, as well as the effectiveness of each. We start with treatments targeting the most focal eating disorder symptoms and then discuss treatments that modify symptoms indirectly, by altering other internal or external processes. We then discuss factors that contribute to relapse.

For those with a serious eating disorder (i.e., individuals with very low body weight or intractable symptoms) intensive treatment is usually recommended. This can take the form of an inpatient/residential program (where patients are admitted for up to 5 or 6 months) or a day hospital, which is usually a shorter stay and allows patients to go home to practice their skills in the evening and on the weekend. Ideally, intensive treatment programs use a step-down approach whereby patients graduate to less intense programming based on their success at interrupting their eating disorder symptoms. Intensive treatments usually involve a variety of therapeutic strategies, including cognitive-behavioral therapy (CBT) and containment for normalization of eating, symptom interruption, and weight restoration for anorexia nervosa, family therapy, pharmacotherapy, and group-based psychosocial programming. Although the dropout rates can be high (especially among those who are not motivated or ready to give up their eating disorder), most studies show that

people who complete intensive treatment are able to experience a successful reduction of eating disorder symptoms (Olmsted et al., 2010). Unfortunately, the problem remains that 35–50% of people relapse within the first 2 years after intensive treatment. This speaks to the need for a long-term approach to treatment that focuses on maintenance of gains and relapse prevention.

Cognitive-Behavioral Therapy The most widely used and presumably most effective treatment for treating eating disorders continues to be CBT. The basic ideas underlying CBT were initially described by Beck and his colleagues, who applied them to depression (see Beck, Rush, Shaw, & Emery, 1979, for a review). Soon thereafter, it was modified and extended to the treatment of the eating disorders. In 1981, Fairburn was the first to outline a cognitive-behavioral approach to the management of bulimia nervosa, and in 1982 Garner and Bemis applied CBT to anorexia nervosa. This treatment has now been proposed for use with binge eating disorders (e.g., Wilson & Fairburn, 2000). Although identifying, modifying, and replacing maladaptive behaviors and thoughts are central to therapy in all of these cases, the distinctive symptomatology of each disorder and individual means that treatment must be adapted accordingly. However, Fairburn (2008) published a CBT-enhanced (CBT-E) manual consistent with his transdiagnostic theory, aimed at treating eating disorders regardless of diagnosis positing that eating disorders share more characteristics than they differ on. The manual recommends tailoring the treatment plan for each individual, and includes optional modules on interpersonal problems, clinical perfectionism, and core low self-esteem. At 60-week follow-up of one study involving CBT-E, 61% of patients with bulimia nervosa and 46% of patients with EDNOS had a level of eating disorder features less than one standard deviation above the community mean (Fairburn et al., 2009). Similarly, a study of CBT-E in patients with anorexia nervosa showed that at the 60-week follow-up, 55% of the patients had maintained their weight above a BMI of 18.5, and 78% had maintained eating disorder psychopathology within one standard deviation of community means (Fairburn et al., 2013).

Cognitive-Behavior Therapy for Anorexia Nervosa The rationale for the use of CBT for anorexia nervosa is that irrational and recalcitrant beliefs about the importance of body weight and shape support relentlessly pursued maladaptive behavior (especially weight loss). These patients also suffer from deficiencies of identity and self-worth that makes treatment even more difficult. CBT for anorexia nervosa focuses both on specific symptoms and on underlying difficulties. According to Vitousek (1995), this therapy for anorexia nervosa targets four main areas. The first is the egosyntonic nature of the disorder (i.e., seeing the disorder as positive or desirable); because individuals with anorexia nervosa prize slenderness, they must first acknowledge that the pursuit of slimness has a negative impact on their lives. Vitousek proposes asking the client to list both the positive and negative aspects of her anorexia; in turn, "each claimed advantage and disadvantage is cast as a hypothesis that can be examined for its validity and adaptiveness" (p. 326), an exercise effectively constituting the first steps toward modifying cognitions. The second focus involves setting behavioral goals for normalized eating and weight gain and implementing cognitive coping strategies to deal with these changes. For instance, the belief that certain "forbidden" foods cause drastic weight gain may be tested by having patients eat these foods in controlled exposures. The third, related focus of treatment is modifying beliefs about weight and food. Clients are taught to challenge their self-defeating thoughts by learning to examine them rationally and replace them with more realistic possibilities (e.g., "there are no forbidden foods *per se*; it is excess caloric intake that leads to weight gain"). Once these most urgent aspects of the disorder have been addressed, treatment can then focus on the underlying self-concept issues that may be maintaining the disorder; ideally, other more attainable and adaptive goals may be identified to replace slenderness as the basis of identity and self-esteem.

Fairburn, Shafran, and Cooper (1998) have proposed a more focused cognitive-behavioral program for anorexia nervosa, targeting the patient's need for control and the attempt to attain this control through dieting. Treatment involves helping the client to achieve success and fulfillment from pursuits unrelated to weight and shape. They suggest that this streamlined treatment should be used first and broadened only if other issues prove to be obstacles to change.

The data on CBT for anorexia nervosa are limited. There are seven published randomized controlled studies, and most did not provide evidence that CBT is more effective than the comparison treatments (i.e., treatment as usual, nutritional counseling, family therapy, and interpersonal therapy) for acute anorexia nervosa. The older studies were limited by a variety of methodological issues (e.g., small sample sizes, high attrition). Two more recent studies, both of CBT-E in anorexia nervosa samples, provided more positive findings. One of the studies administered CBT-E to adults with anorexia nervosa, and found that a substantial proportion of the patients (62%) were able to increase their weight to a BMI greater than 18.5 and reduce their eating disorder psychopathology to within one standard deviation of community means (88%; Fairburn et al., 2013). These gains were maintained by 55% and 78% over 1 year later (Fairburn et al., 2013). However, about one-third of patients did not complete treatment, and those who did not finish had more psychopathology at baseline (Fairburn et al., 2013). In another recent study, CBT-E was provided to adolescents with anorexia nervosa. Similar to the study in adults, about two-thirds of the patients completed treatment, and a substantial proportion

of the patients were able to increase their weight and reduce eating disorder psychopathology to within a normal range (Dalle Grave et al., 2013). At a 60-week follow-up, the mean weight had increased from post-treatment, and about 90% of the patients had eating disorder symptoms within one standard deviation of community means, suggesting little residual psychopathology (Dalle Grave et al., 2013). Although these studies cannot yet result in the conclusion that CBT is effective for anorexia nervosa, given that the research is still limited and these two studies were conducted by the same two collaborating research groups, nevertheless these findings do suggest promise in the use of CBT for treating anorexia nervosa. Future well-designed studies with strong findings will help to support these data before any firm conclusions can be drawn about the effectiveness of CBT for acute anorexia nervosa (Pike, Carter, & Olmsted, 2010). In addition to the acute treatment of anorexia nervosa, there are two published studies that do provide promising preliminary evidence for the effectiveness of CBT for preventing relapse and improving recovery rates in weight-restored patients (Carter et al., 2009; Pike, Walsh, Vitousek, Wilson, & Bauer, 2003).

Cognitive-Behavior Therapy for Bulimia Nervosa Because bulimia nervosa is less egosyntonic (i.e., less acceptable to one's self-image) than is anorexia nervosa—the binge–purge cycle is often extremely distressing to the individual—CBT for bulimia nervosa has a narrower focus, typically limited to eating behavior and thoughts about food, weight, and shape. Thus, as in anorexia nervosa, clients with bulimia nervosa are taught to establish a normalized eating pattern consisting of regularly scheduled meals including a variety of foods. Incorporating previous "forbidden foods" can help to prevent psychological or physiological deprivation that may foster a binge. In addition, clients work on the reduction or elimination of bingeing and associated compensatory behaviors through behavioral goal setting and the development of coping strategies. They are taught to identify the usual precipitants to eating disorder behaviors (often with the help of a daily food diary), and to use distraction techniques (alternatives to binges such as taking a walk, or calling a friend) or other coping mechanisms (i.e., such as self-talk, stimulus control). Compensatory behaviors after binges—such as vomiting, laxative use, excessive exercise or dietary restraint—are discouraged, in an attempt to break the binge–purge cycle; this technique is akin to the behavioral technique of exposure with response prevention. Finally, as with anorexia nervosa, excessive concern with body weight and shape is challenged through cognitive restructuring (e.g., Wilson, 1996, 1999).

CBT is considered the treatment of choice for bulimia nervosa (Wilson, Grilo, & Vitousek, 2007); it leads to reduction or remission of bingeing and purging in 50% of patients and improvements in dietary restraint, attitudes toward shape and weight, and associated psychopathology (Wilson, 1996). The use of CBT-E in patients with bulimia nervosa has also demonstrated similar outcomes (Fairburn et al., 2009). A recent meta-analysis showed that small effects favored bona fide CBT treatments for bulimia nervosa demonstrated compared to bona fide non-CBT treatments (Spielmans et al., 2013). However, despite that CBT is the treatment of choice for bulimia nervosa, it is important to be aware of viable alternatives, given that about half of patients with bulimia nervosa do not experience an improvement in bingeing and purging behaviors as a result of CBT.

Cognitive-Behavior Therapy for Binge Eating Disorder Because binge eating disorder involves bingeing without purging or any other form of compensatory behavior, individuals with this disorder are more often overweight than are those with bulimia nervosa. Thus, behavioral weight-loss strategies (e.g., an exercise plan) may be included, and the individual experience with weight prejudices and stigma must be addressed. CBT seems to be effective in reducing the behavioral and psychological features of binge eating disorders (i.e., binge eating episodes and psychological distress), but not in reducing obesity (e.g., Grilo, Masheb, & Wilson, 2005; Loeb, Wilson, Gilbert, & Labouvie, 2000; Wilson & Fairburn, 2000). Long-term outcomes for both CBT and behavioral weight loss treatments appear to be comparable with respect to reducing binge eating. In a 6-year follow-up study, participants in both treatments reduced their binge eating during treatment, although CBT resulted in more substantial reductions. Binge eating symptoms did worsen in the subsequent 6 years for both groups, although they were still substantially improved over baseline levels, indicating that both treatments may be useful in reducing binge eating in binge eating disorder (Munsch, Meyer, & Biedert, 2012). Other authors have suggested that "the treatment of choice [for binge eating disorder] would appear to be cognitive behavioral self-help" (Wilson & Fairburn, 2000, p. 352). Indeed, no difference in outcome between therapist-led versus self-help format CBT has been found, and there were significant improvements over wait-list controls in the latter, suggesting that it may be a cost-effective, accessible alternative for patients with binge eating disorder (Loeb et al., 2000).

It is important to note that these descriptions of CBT for the various eating disorders reflect either how it is carried out in experimental tests or in the ideal clinical setting, neither of which necessarily corresponds to clinical reality. Individual clinicians often tailor the technique as they see fit for individual clients, or they may combine CBT with other treatment strategies in an eclectic treatment plan.

Nutritional Counseling and Psychoeducation As its name implies, nutritional or dietary counseling involves educating the patient about normal caloric needs and physiological processes. Kahm (1994) explained that while

people with eating disorders may have a wealth of knowledge regarding the calorie content of most foods, this knowledge is usually used to achieve drastic weight loss rather than to maintain a healthy body. In fact, Beumont and Touyz (1995) suggested that failing to address nutritional issues in eating disorders "is as ridiculous as prohibiting the discussion of drinking behavior with patients with alcohol related disease" (p. 306). They also stated that this therapy is intended to change attitudes about food and eating, and then eating behavior, by arming people with accurate knowledge.

Psychoeducation provides accurate nutritional information, but also educates patients about the nature of their disorder. Olmsted and Kaplan (1995) advocated addressing four main topics beyond dietary information: the complex etiology of eating disorders, the often serious medical consequences of severe food restriction or continual bingeing and vomiting, the sociocultural idealization of the thin female body, and cognitive and behavioral strategies that one may use to modify eating problems. Nutritional counseling and psychoeducation are rarely used as standalone therapies; it has been estimated that educational information is incorporated in 75% of treatments for bulimia nervosa (Olmsted & Kaplan, 1995).

Motivational Interviewing Because CBT involves modifying problematic thoughts and behaviors related to eating disorder pathology, it is often considered an 'action-oriented' approach; that is, individuals participating in CBT are asked to take action, often within the first several days of initiating treatment, to challenge their beliefs and change their eating disordered behavior. However, individuals with eating disorders often harbor a great deal of ambivalence about overcoming their symptoms, and as a result, may be reluctant to engage in action-oriented treatment. Motivational interviewing (Miller & Rollnick, 2002) acknowledges fluctuations in motivation and readiness for change. The therapist's task is to highlight problematic aspects of symptoms and collaboratively explore and resolve ambivalence. Motivational interviewing is based on the transtheoretical model of change (Prochaska & DiClemente, 1992), which describes the process of behavioral change with reference to five "stages": precontemplation (denial of problem existence), contemplation (acknowledgment of problem and plan to change in next 6 months), preparation (intending to change within the next month and preparing to take action), action (initiating behavioral change), and maintenance (sustaining change). The transtheoretical model posits that these stages are universal, non-linear, and cyclical: that is, individuals are presumed to possess fluctuating insight into and motivation to change problem behaviors, and may relapse often before achieving sustained behavioral modification.

Research has now begun on the use of motivational interviewing with individuals who have eating disorders (see Wilson & Schlam, 2004, for a review; see Treasure & Ward, 1997, for anorexia nervosa; see Killick & Allen, 1997, for bulimia nervosa). A handful of small studies suggest its potential utility with this population, although the present body of literature is somewhat mixed. Feld, Woodside, Kaplan, Olmsted and Carter (2001) described the use of a four-session, group-based motivational interviewing program conducted prior to the initiation of CBT, during which individuals were encouraged to examine the costs and benefits of their eating disorder and its consistency with their values, among other topics. Results showed that compared with patients' own baseline levels, group participation resulted in increased motivation, confidence, and readiness to change (but did not affect eating disorder symptoms). Similarly, individuals with refractory anorexia who participated in a motivational interviewing program for 5 hours a day, twice a week over 6 months displayed increased motivation for change, representing a shift from "early" to "late" contemplation (George, Thornton, Touyz, Waller, & Beumont, 2004). Studies using "treatment as usual" control groups indicate that motivational programs foster longer engagement, increased motivation to change, and encourage participants to continue with treatment (Dean, Touyz, Rieger, & Thornton, 2008; Wade, Frayne, Edwards, Robertson, & Gilchrist, 2009).

Nevertheless, a recent review of the evidence of motivational interventions in eating disorders found that the summative body of literature is mixed. Although motivational interventions tended to improve both cognitive readiness for and actual behavioral changes to binge eating, the review did not support that motivational interviewing was helpful in changing compensatory behaviors and restrictive eating (Knowles, Anokhina, & Serpell, 2013). The authors concluded that although motivational interviewing might improve motivation for treatment, it does not appear to be superior compared to other treatment methods (Knowles et al., 2013). While motivational interviewing has not been shown to reflect a change in treatment outcome, it may lead to treatment participation for many individuals who would not have otherwise been open to treatment. Waller (2012) has suggested that perhaps verbal declarations of cognitive readiness to change is not enough when it comes to eating disorders: Perhaps increasing motivation to change as per motivational interviewing does not translate into a behavioral reality for individuals with eating disorders, and that more concrete methods to facilitate early behavior change and subsequent learning might be more useful with this population. This is an interesting hypothesis about the nature of motivation, behavior change, and eating disorders that will need to be further examined in clinical research before drawing conclusions about its usefulness.

It is also important to note that motivational interviewing is not intended as a standalone treatment approach, but rather as a potentially useful precursor, and/or adjunct to, other more action-oriented approaches. Because motivational interviewing maintains a focus on an individual's readiness for change, actual symptom change is not an

intended outcome of this intervention. Therefore, successful outcomes following motivational interviewing presume that the individual will move on to implement the changes she has decided are appropriate. Further, although motivational interviewing finds a natural niche among individuals whose symptoms are recalcitrant, its applicability does have limits. For instance, if an individual is medically compromised due to severe or very rapid weight loss (Killick & Allen, 1997), respect for her ambivalence will necessarily be weighed against the potential consequences of inaction.

Interpersonal Therapy In contrast to psychoeducation and CBT, which are focused on improving eating and symptom interruption, interpersonal therapy does not touch on these issues in the treatment of eating disorders. Instead, it targets maladaptive personal relationships and relational styles because difficulties in these areas are seen as contributing to the development and maintenance of eating disorders (Birchall, 1999). Thus, the fundamental task of the interpersonal therapist is to identify one of Birchall's "problem areas—grief, role transitions, interpersonal role disputes (or) interpersonal deficits" (p. 315), and to work to improve the client's functioning in that area. Improved interpersonal relationships might reduce eating disorder symptoms in a number of ways. Enhanced control in relationships may generalize to enhanced control of eating in bulimia nervosa or binge eating disorders. Furthermore, interpersonal therapy may reduce or eliminate common interpersonal triggers to bingeing. For example, depressed mood and interpersonal stress may be alleviated, and more frequent, positive social interactions may reduce boredom. Decreasing interpersonal stress may also improve anorectic behavior by providing a greater sense of control in areas of one's life other than eating. Despite the fact that CBT is considered the "gold standard" treatment for eating disorders, some authors view interpersonal therapy as a viable alternative first-line treatment, owing to its conceptual similarities with etiological theories of the development of these disorders (e.g., Tantleff-Dunn, Gokee-LaRose, & Peterson, 2004).

Although CBT produces more rapid positive outcomes in bulimia nervosa (Birchall, 1999; Fairburn et al., 1995), interpersonal therapy seems to be equally effective by the end of treatment and at follow-up (Wilson & Shafran, 2005). There has been some research to show that African American participants showed greater reductions in binge eating when treated with interpersonal therapy compared with CBT, suggesting that interpersonal therapy may be particularly appropriate for African American women with bulimia nervosa (Chui, Safer, Bryson, Agras, & Wilson, 2007). Further research on interpersonal therapy for eating disorders with different racial and ethnic groups is needed.

With regard to binge eating disorders, interpersonal therapy has been shown to be as effective as CBT in reducing binges (and as ineffective at producing weight loss), and the rate of improvement for binge eating disorders is equal in both therapies (Tasca et al., 2006; Wilfley et al., 2002; Wilson & Fairburn, 2000). It has also been shown that those who received interpersonal therapy for binge eating disorders maintained their reductions in binge eating and disordered cognitions for at least 5 years (Bishop, Stein, Hilbert, Swenson, & Wilfley, 2007).

Dialectical Behavior Therapy Dialectical behavior therapy (DBT; Linehan, 1993) was developed to treat borderline personality disorder and is now being considered in the treatment of eating disorder symptoms that have been refractory to "first-line" treatments (such as CBT). DBT encourages commitment to and enactment of behavioral change while validating individuals' emotions and treatment ambivalence, with a specific focus on building skills in emotional recognition and regulation. McCabe and Marcus (2002) suggest that DBT has the potential to be effective in the treatment of anorexia nervosa through its conceptualization of eating disorder symptoms as maladaptive emotional avoidance or regulation strategies that can be improved by increasing commitment to change and building the requisite skills to do so; although clinical experience suggests this may be the case, further empirical evidence is required to support this conclusion. A systematic review of DBT for eating disorders showed that DBT was effective in reducing eating disorder symptoms such as binge eating, purging behaviors, and restrictive eating in individuals with bulimia nervosa, binge eating disorders, and anorexia nervosa (Bankoff, Karpel, Forbes, & Pantalone, 2012). These studies also showed that DBT helped individuals with eating disorders reduce related features such as self-harm behaviors, as well as general psychopathology (e.g., depression and anxiety; Bankoff et al., 2012). The body of evidence has not demonstrated that DBT is superior to other empirically supported treatments such as CBT, partly because the literature to date has not yet compared these treatments head to head. As such, ongoing research comparing DBT to evidence-based eating disorder treatments will be necessary. Importantly, however, DBT has been shown to be superior to supportive psychotherapy in maintaining engagement in treatment (Safer, Robinson, & Jo, 2010). Given these preliminary findings, it appears that DBT may be a useful option for those with bulimia nervosa or binge eating disorders, in particular among individuals for whom affect dysregulation is prominent (e.g., those who also present with self-harm behaviors).

In additional, although DBT was originally developed for borderline personality disorder, the randomized controlled studies reviewed by Bankoff and colleagues (2012) all excluded patients with this diagnosis, indicating that these studies tested DBT in relatively straightforward (i.e., less complex) eating disorder samples. However, recent thinking by some authors has been that DBT might

be particularly helpful for the most complex segment of patients with eating disorders, precisely because it was developed for the complex psychopathology of borderline personality disorder. Accordingly, a recent case series showed that an intensive DBT program was helpful for complex eating disorder patients who had experienced repeated treatment failures in typical (i.e., CBT-based) intensive eating disorder treatments, and who also had complex psychopathological presentations (i.e., comorbid Axis I or II diagnoses), pervasive deficits in emotion regulation, extreme difficulty generalizing skills outside of the treatment setting, and/or who presented behaviors in therapy that significantly impeded treatment progress (Federici & Wisniewski, 2013). These are the most challenging of the patients with eating disorders, the ones who typically drop out or who are prematurely discharged from standard evidence-based treatments. These patients reduced their binge eating, purging, and restrictive behaviors, gained weight (if applicable), reduced their self-harm and suicidal behaviors, improved their medical stability, and improved their ability to stay in treatment (Federici & Wisniewski, 2013). Another recent study showed that, after receiving DBT, patients with eating disorder who had comorbid substance-use disorders (also considered a challenging clinical presentation) were able to improve eating disorder symptoms, substance-use behaviors, emotion regulation difficulties, and symptoms of depression (Courbasson, Nishikawa, & Dixon, 2012). Patients who received DBT also were more likely to stay in treatment than those who received CBT, and those in CBT experienced a worsening rather than improvement with respect to substance use (Courbasson et al., 2012). Although preliminary evidence, these findings suggest that DBT might be particularly helpful for those eating disorder patients who present complex presentations or whose histories have been presumed "treatment refractory".

Family Therapy Family therapy focuses on interpersonal relationships, emphasizing the importance of stresses within the family as a whole rather than on individuals. It postulates that change by one member affects every other member in turn (Minuchin, Rosman, & Baker, 1978; Thode, 1990). The patient with an eating disorder is seen in conjunction with her parents and siblings (or with her spouse and children, as the case may be), and the goal becomes reducing family stressors and miscommunications to bring about a reduction in eating disorder symptoms. Thus, family therapy places responsibility for recovery on both the patient and her relatives. Geist, Heinmaa, Stephens, Davis, and Katzman's (2000) description of treatment provides a good example of the specific content of family therapy: (a) Recruiting parents to actively engage in managing the patient's weight gain and eating; (b) ameliorating maladaptive communication within the family and particularly between the parents; and (c) elucidating the difference between normal methods of coping with family conflict and the symptoms the patient is currently exhibiting.

Because of its low prevalence and its additional medical complications, there are significantly fewer controlled treatment trials for anorexia nervosa than there are for bulimia nervosa and binge eating disorder. Furthermore, very few studies directly compare various forms of treatment for anorexia nervosa, making differences in efficacy somewhat difficult to gauge. Despite these limitations, there is a tenuous consensus that family therapy is the first-line treatment for most cases of anorexia nervosa (e.g., Wilson, 1999). On the basis of five randomized controlled trials of family therapy with anorexia nervosa, Eisler, Lock and le Grange (2010) concluded that family therapy is effective for anorexia nervosa with short duration. It may be particularly effective for adolescents because they tend to express greater denial and less desire for help (Fisher, Schneider, Burns, Symons, & Mandel, 2001), making family involvement potentially more important. Family therapy for bulimia nervosa has not been well studied, although Dare and Eisler (2000) reported encouraging preliminary findings for their multifamily treatment program for adolescents with bulimia nervosa. More recently two randomized controlled trials showed additional support for family therapy in adolescents with anorexia nervosa. Family therapy was shown to be superior to individual supportive psychotherapy on both behavioral and attitudinal aspects of bulimia nervosa (le Grange, Crosby, Rathouz, & Leventhal, 2007). A recent meta-analysis of family therapy for adolescents with anorexia nervosa and bulimia nervosa showed that for both diagnoses, family therapy was equivalent to individual therapy at post-treatment, but that at 6- and 12-month follow-ups, the teens who received family therapy were functioning better than those in individual treatment (Couturier, Kimber, & Szatmari, 2013). The authors concluded that family therapy specifically targeting eating disorder symptoms should be a first-line treatment for adolescents with eating disorders.

Psychoanalytic Therapy Psychoanalytic or psychodynamic therapy may encompass a wide variety of specific techniques or foci. Herzog (1995) defines it broadly as "all long-term therapies that explicitly use the relationship between the patient and therapist as the primary treatment tool and that attend to transference and countertransference reactions" (p. 330). This therapy assumes that eating disorder symptoms are merely the overt pathological manifestations of underlying conflicts that the client has been unable to resolve adaptively. The psychoanalyst's task is to determine the unique meanings of these symptoms for each client. Many theorists suggest that establishing a strong therapeutic alliance is the first (and perhaps most crucial) step (Dare & Crowther, 1995; Gonzalez, 1988; Herzog, 1995). Establishing such an alliance may be particularly difficult and take longer in anorexia nervosa,

because the patient will perceive the therapist as someone who wants her to give up her egosyntonic dietary restriction. Once trust has developed, the psychoanalyst establishes a neutral stance toward the patient, who then free associates: "the hope is that the patients will project upon this 'blank screen' their characteristic thoughts and behaviors (transference)" (Johnson, 1995, p. 351). Much of therapy is then devoted to analyzing these transference reactions in the context of a thorough developmental history. Another strategy used in identifying the roots of the eating disorder is the analysis of dreams to determine their symbolic meaning. The countertransference relationship also constitutes an important source of information about the client; therapists interpret their own reactions to the client to further elucidate the quality of the client's interpersonal relationships. A nonjudgmental relationship between the therapist and client is said to serve a further purpose, providing the patient with an opportunity to relate appropriately on an interpersonal level, allowing the patient to resolve dependent or otherwise pathological relations with others (Herzog, 1995).

Contemporary psychoanalytic writers acknowledge that psychoanalysis may not be appropriate for all eating disorder patients. Herzog (1995) maintains that the treatment is appropriate for those patients presenting with character pathology; both Herzog (1995) and Johnson (1995) recommend psychoanalysis for prior-treatment nonresponders (i.e., for those patients whose symptoms linger after a course of cognitive or interpersonal therapy). Finally, Gonzalez (1988) cautions against using psychoanalysis for suicidal patients or for those whose extremely low weight makes hospitalization necessary; in these cases, the long-term focus of psychoanalysis would leave the patient at immediate risk of harm. Indeed, although psychoanalysis for anorexia nervosa has not been well studied, Herzog (1995) stated that patients often require a minimum of 1–2 years of psychoanalysis, and maybe as much as 8–10 years for improvement to take place. One long-term outcome study (Steinhausen, Seidel, & Metzke, 2000) found that 80% of surviving patients with anorexia nervosa recovered at 11-year follow-up regardless of the type of therapy received. In light of this finding, it is difficult to make a case for the use of long-term psychoanalysis in the treatment of anorexia nervosa. Similarly, outcome data for psychodynamic treatment of bulimia nervosa are scarce, leading Johnson (1995) to recommend this approach only for those patients who do not respond to briefer forms of therapy. A study by Murphy, Russell, and Waller (2005) suggests that integrative and time-limited forms of psychodynamic therapy may be useful in reducing bulimic symptoms, but more research is needed.

Pharmacotherapy Pharmacotherapy departs significantly from other treatments by postulating some sort of organic cause for the disorder—or at least that the symptoms can be controlled pharmacologically—and attempting to treat symptoms accordingly, with medications. By far the most common drugs are antidepressants, both tricyclic and mainly selective serotonin reuptake inhibitors (SSRIs). Their use is based on the fact that depressive and anxiety symptoms are prominent in eating disorders, and that these symptoms resemble the clinical features of other disorders (i.e., major depressive disorder, anxiety disorders) that respond favorably to these medications (e.g., Kruger & Kennedy, 2000; Mayer & Walsh, 1998).

Attempts to treat anorexia nervosa with pharmacological agents have not been successful. Neither antidepressants, antipsychotics, nor any other class of drugs, has been found to lead to significant weight gain, improve distorted attitudes or beliefs, or enhance the effects of inpatient programs (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007). In fact, most researchers recommend against the use of drugs in the acute treatment of anorexia nervosa (Kruger & Kennedy, 2000; Mayer & Walsh, 1998). In addition, the evidence is equivocal regarding the use of SSRIs (i.e., fluoxetine) with weight-restored patients with anorexia nervosa and the maintenance of treatment gains. One study demonstrated better outcome related to fluoxetine versus placebo (Kaye et al., 2001); however, another larger study failed to observe any benefit of adding fluoxetine versus placebo to CBT in terms of reducing relapse and in maintaining weight (Walsh et al., 2006). In support of these findings, a recent review of individual studies, meta-analyses, and clinical recommendations by several health organizations (e.g., National Institute for Health and Clinical Excellence in the UK; American Psychiatric Association), has indicated that pharmacological interventions do not appear to have efficacy in treating anorexia nervosa, either as a standalone or adjunctive treatment, and that its evidence for relapse prevention is also limited (Flament, Bissada, & Spettigue, 2012).

In terms of bulimia nervosa, the SSRI fluoxetine is the most widely studied agent with six double-blind placebo-controlled trials (Broft, Berner & Walsh, 2010). The results of these studies indicated that fluoxetine was superior to placebo in reducing bingeing and purging, and was associated with significant improvement in other psychological symptoms (e.g., Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992; Goldstein, Wilson, Thompson, Potvin, & Rampey, 1995). These findings are supported by a review which showed that SSRIs, particularly fluoxetine, provide moderate effects with respect to improvement of bulimia nervosa symptoms (Flament et al., 2012). However, one study showed that only a small minority of patients becomes free of eating disorder symptoms on this drug and most continue to meet diagnostic criteria (Narash-Eisikovits, Dierberger, & Westen, 2002).

Overall, treatment with antidepressants appears to be inferior to CBT at reducing frequency of bingeing and purging, depression, and distorted eating-related attitudes (Whittal, Agras, & Gould, 1999). When CBT is added to antidepressant treatment, it is better than antidepressant treatment alone (Narash-Eisikovits et al., 2002), but not

better than CBT alone. Therefore it is recommended to use pharmacotherapy (i.e., fluoxetine) only if CBT is not available, or if this is the intervention that the patient prefers (Broft et al., 2010). Alternatively, fluoxetine may be a viable option if psychotherapy fails, although these results are mixed (Mitchell et al., 2002; Walsh et al., 2000).

In binge eating disorder, many drugs, including antidepressants (McElroy, Hudson, Malhotra et al., 2003; McElroy et al., 2000), antiobesity medications (Wilfley et al., 2008) and anticonvulsants (McElroy, Shapira et al., 2004) have demonstrated efficacy in reducing binge eating and weight. A review of evidence showed that SSRIs may reduce binge eating frequency and result in weight loss for individuals with binge eating disorder (Flament et al., 2012). There have also been three randomized controlled trials of sibutramine (antiobesity medication) and all three showed this drug to be superior to placebo in reducing the frequency of binge-eating episodes and facilitating weight loss for up to 3 months of treatment (Appolinario et al., 2003; Milano et al., 2005; Wilfley et al., 2008). However, weight loss was modest at best (e.g., 5% weight loss) and often did not seem to be maintained (e.g., Devlin, Goldfein, Carino, & Wolk, 2000). Importantly, marketing of sibutramine was suspended in January 2010, owing to evidence of elevated cardiovascular risks associated with use of this drug, namely increased incidence of nonfatal heart attacks and strokes, which were deemed to outweigh its potential benefits. The National Institute for Health and Clinical Excellence subsequently withdrew its recommendations for use of sibutramine in the treatment of obesity (NICE, 2006, 2014).

There is also evidence supporting the use of anticonvulsants in the treatment of binge eating disorder. Initial trials of the anticonvulsants topiramate (McElroy, Arnold et al., 2003; McElroy et al., 2007) and zonisamide (McElroy et al., 2006) resulted in significant decreases in binge eating episodes and body weight; however, these medications also appear to be associated with a high discontinuation rate as a result of adverse events and nonadherence (McElroy, Shapira et al., 2004). There is also little evidence that any of these medications improve psychological symptoms in patients with BED (Bodell & Devlin, 2010).

Reas and Grilo (2008) conducted a review of pharmacotherapy studies for binge eating disorder, and concluded that pharmacological treatments are superior to placebo in the short term for reducing binge eating and weight. These findings are supported by a more recent review of evidence for pharmacotherapy in eating disorders, including binge eating disorder (Flament et al., 2012). There is no evidence to suggest that medication is superior to CBT, or that medication enhances the effects of psychotherapy on primary outcome measures of binge-eating and weight (Grilo et al., 2005). Therefore, CBT should be considered the treatment of choice for binge eating disorder, whereas pharmacotherapy could be chosen for reasons of availability, cost or preference (Bodell & Devlin, 2010).

Recent Treatment Developments There have been a couple of new and exciting developments in treatment for eating disorders (Grilo & Mitchell, 2010). One development is cognitive remediation therapy for anorexia nervosa (Tchanturia & Hambrook, 2010). This therapy draws on neuropsychology and targets the maladaptive thinking processes (not content) in anorexia nervosa. The rationale is that patients with anorexia nervosa have global impairments in cognitive processes related to thinking flexibly and holistically, and that these thinking patterns contribute to the rigidity and obsessional thinking that is typical to those with anorexia nervosa and which is related to many of their symptoms. In cognitive remediation therapy, patients practice cognitive flexibility, holistic processing, and metacognition in session, and are then encouraged to apply these new skills to their real life. A recent case series showed that following cognitive remediation therapy, individuals with anorexia nervosa improved on their cognitive flexibility, impulse control, and interoceptive awareness (the ability to accurately perceive internal bodily signals and states; Abbate-Daga, Buzzichelli, Marzola, Amianto, & Fassino, 2012). Another development is called integrative cognitive-affective therapy for bulimia nervosa (ICAT; Wonderlich et al., 2010). This treatment retains some elements of CBT but includes an emphasis on emotional responding and exposure to emotions (i.e., emotional regulation) and strategies to identify patterns of interpersonal and self-directed behavior, which may promote the avoidance of negative affect. A recent randomized controlled trial comparing ICAT with CBT-E for patients with bulimia nervosa showed that ICAT resulted in significant improvements and performed similarly to CBT-E, providing initial evidence of its efficacy treating bulimia nervosa (Wonderlich et al. 2014). Though promising, given the newness of both of these treatments, outcome research is in its early stages and further research is needed.

Treatment Research Issues Despite our poor understanding of the development of eating disorders, research into their treatment has made some progress. However, controlled trials comparing treatments may not reflect clinical reality, where therapists may sample from the available therapies and tailor treatments to specific clients. For instance, individual CBT might be paired with administration of drugs to help patients with bulimia nervosa, or interpersonal and family therapy might be bolstered with psychoeducation in the treatment of anorexia nervosa. These so-called multifaceted treatments (see Lansky & Levitt, 1992, for a review) may provide the best form of care, because the components of one therapy may enhance the effects of another or provide additional benefits beyond those available in the empirically established first-line treatment. Still, individually tailored treatments are difficult to evaluate systematically; although such tailoring makes some sense, it also makes it difficult for researchers to evaluate treatment efficacy.

Adding more complications to the study of treatment options for eating disorders is the high comorbidity rate mentioned earlier. As expressed by Harrop and Marlatt (2010), "though comorbidity rates are high, little research has been done concerning treatment" (p. 392). Additionally, individuals with comorbid conditions are often excluded from research studies examining the efficacy of eating disorder treatments, which makes it challenging to ascertain the effects of treatments in patients with comorbid psychopathology. Substance abuse, personality disorders, and mood disorders are often severe enough to warrant treatment even when no eating disorder is present; the fact that these and many other conditions occur at elevated levels amongst the eating disordered population only confounds the issue of treatment. In many cases, treating an underlying condition seemingly unrelated to the eating disorder (e.g., obsessive-compulsive disorder, depression) may be enough to help alleviate symptoms or at least prepare the individual to seek and attend eating disorder treatment. In other cases, patients with comorbidities in addition to the eating disorder may present challenges to treating either issue. As described earlier, the use of DBT for patients with multidiagnostic psychopathology and/or substance use is an emerging area that may provide treatment options for these types of patients (Courbasson et al., 2012; Federici & Wisniewski, 2013).

Prognosis for Eating Disorders

Eating disorders are serious problems. They have the highest mortality rate of all of the psychiatric disorders (e.g., Agras et al., 2004; Licht, Mortensen, Gouliaev, & Lund, 1993). Mortality-rate estimates range from approximately 5% (Casper & Jabine, 1996; Herzog et al., 2000; Sullivan, 1995) to 8% (Steinhausen et al., 2000). The most common causes of death among individuals with eating disorders are starvation and nutritional complications (e.g., electrolyte imbalance or dehydration), and suicide (Neumärker, 2000).

With respect to longer-term outcome, relapse rates following initially successful treatment are high with published rates ranging from approximately 20–60% for adults. Defining relapse and remission is difficult, however, and as a result, these rates vary considerably in the literature depending on methodological differences. Most reported rates are between 35% and 50% (e.g., Carter, Blackmore, Sutandar-Pinnock, & Woodside, 2004; Field et al., 1997; McFarlane et al., 2008; Walsh et al., 2006), comparable to relapse rates associated with alcohol and substance use among women (Walitzer & Dearing, 2006).

Theoretically, relapse signifies a return to disordered functioning following a period of symptom amelioration. Pike (1998) defined anorexia nervosa relapse relative to initial treatment response, remission, and recovery. An initial satisfactory response for anorexia nervosa (before which relapse cannot occur) is operationalized as increasing BMI to at least 20, consuming significantly more calories (although not necessarily normalized eating), reducing fears about weight gain, and medical stabilization. According to Pike, relapse involves weight dropping to a BMI below 18.5, restrictive eating or bingeing with compensatory behavior, a return to overvaluing weight and shape, and in some cases a return of associated medical problems.

Olmsted, Kaplan and Rockert (2005) also point out that relapse rates for bulimia nervosa are strongly influenced by definitions of remission and relapse. They recommend that researchers use consensus definitions; specifically, partial remission is defined as a maximum of two symptom episodes per month for 2 months, and relapse is defined as meeting full diagnostic criteria for 3 months.

Identification of factors that predict relapse may help to advance the understanding of the psychopathology of eating disorders, and also provide direction for the development of interventions aimed at relapse prevention. Previous research has identified younger age (Olmsted et al., 2005), higher frequency of vomiting (Olmsted, Kaplan, & Rockert, 1994) and restriction (McFarlane et al., 2008), longer duration of illness and greater psychiatric comorbidity (Deter & Herzog, 1994) as predictors of relapse. These variables may represent degree of impairment and severity of the eating disorder, indicating that those who are less well before treatment are more likely to relapse later. At the end of treatment, residual symptoms such as higher binge eating and/or vomiting frequency, weight-related self-esteem (McFarlane et al., 2008), higher dietary restraint (Halmi et al., 2002), exercise aimed at weight control (Carter et al., 2004; Strober et al., 1997), weight loss immediately following intensive treatment (Kaplan et al., 2008), and higher body dissatisfaction (Freeman et al., 1985) have also been shown to predict relapse. Finally, factors related to the process of treatment have been shown to predict continued wellness. These include a rapid response to intensive treatment (McFarlane et al., 2008) and abstinence from binge eating and vomiting during treatment (Halmi et al., 2002).

One study (Cockell, Zaitsoff, & Geller, 2004) obtained a retrospective account of self-reported factors that helped or hindered maintenance of treatment gains from 32 patients who had completed a 15-week inpatient treatment program at 6 months post-discharge. Factors identified as supporting maintenance were: a) Maintaining connections with professionals (for expertise and monitoring) and non-professionals (for support and understanding); b) application of the nutritional knowledge (what constitutes a normal meal, meal planning, etc.) and psychological skills (self-monitoring and emotional expression) learned in treatment; and c) focusing beyond the eating disorder (such as on fulfilling higher-order values). Factors that were believed to hinder maintenance included loss of structure and specialized support, self-defeating beliefs (e.g., need for control), and daily hassles that patients had been protected from while in treatment.

A recent study of individuals with anorexia nervosa found that over a 12-year follow-up period, 28% had a good outcome, 25% an intermediate outcome, 40% a poor outcome, and 8% were deceased. At the 12-year follow-up point, 19% met diagnostic criteria for anorexia nervosa, 10% for bulimia nervosa, 19% were classified as EDNOS, and 0% had binge eating disorder. A total of 52% did not appear to have a DSM-IV eating disorder (Fichter, Quadflieg, & Hedlund, 2006). It appears that despite the fact that a substantial number of individuals relapse and continue to have clinically significant eating disorders after receiving treatment, more patients recover after receiving treatment than exhibit spontaneous recovery without treatment. For example, in a study of the natural course of bulimia nervosa, at 5-year follow-up, approximately 50% of the women had an eating disorder of clinical severity, and only 35% did not show evidence of an eating disorder (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000). Another study examined relapse rates following day hospital treatment across all eating disorder diagnostic categories. Six months after completion of day hospital treatment the relapse rate was 38%, at 1 year it was 41%, and at 18 months it increased to 48% (McFarlane et al., 2008).

Current estimates of relapse rates in binge eating disorders are somewhat more optimistic than are those observed in anorexia nervosa and bulimia nervosa. One longitudinal study of binge eating disorders to assess relapse rates found that at 6 years post-treatment, most participants did not have a diagnosable eating disorder, although approximately 15% had shifted to another diagnostic category (7.4% to bulimia nervosa and 7.4% to EDNOS), and approximately 6% met the criteria for binge eating disorders once again (Fichter, Quadflieg, & Gnutzmann, 1998).

As is the case with both anorexia nervosa and bulimia nervosa, the severity of the disorder appears to be a prime determinant of negative outcome in binge eating disorders; the more frequent bingeing is at treatment intake, the more frequent it is following treatment (Peterson et al., 2000). Negative affect also predicts poorer treatment outcome for binge eating disorders (Eldredge & Agras, 1997). With respect to relapse, Safer, Lively, Telch and Agras (2002) found that the two strongest predictors of setbacks following a successful course of DBT were earlier onset of binge eating (i.e., at or before age 16), and greater (cognitive) dietary restraint.

Conclusion

We still have a long way to go toward a better understanding of why and how eating disorders develop, and the obstacles to making explanatory progress are significant. The most obvious obstacle is that the vast majority of studies examine correlates (or recollected precursors) of established eating disorders. This sort of research is inevitably inconclusive about causation. Although path-analytic strategies (e.g., Stice, 2001) attempt to extract causal patterns from correlational data, there are severe limits to the persuasiveness of such analyses. For ethical reasons, true experimentation is not likely to occur. For example, it is difficult to get permission to study bingeing in a patient with bulimia nervosa in the laboratory. Although such patients binge regularly, it is regarded as unethical for researchers to actively induce a binge. Further, although controlled experiments may provide insight into specific symptoms, the external validity of such measures would be questionable. Prospective research is more compelling than is retrospective research; but it too is correlational and therefore limited in what it can tell us.

Of course, although many of us would be most pleased with a tight, experimentally based analysis of eating disorders, there are other satisfactions to be had. Decades ago, Hilde Bruch (1975, 1978) provided us with an account of eating disorders, based on her vast clinical experience and insight. The empirical research that has been conducted since Bruch first presented her elegant formulations has done little to undermine her analysis. The reader who wants a clear understanding of eating disorders would be well-advised to refer to Bruch, or if not to her, to her patients. As one of them concluded, "The main thing I've learned is that the worry about dieting, the worry about being skinny or fat, is just a smokescreen. This is not the real illness. The real illness has to do with the way you feel about yourself" (Bruch, 1978, p. 127).

Note

1. References to EDNOS (rather than other specified feeding and eating disorder or unspecified feeding and eating disorder) in the current chapter are due to the fact that much of the referenced research occurred prior to the release of DSM-5, and therefore the term EDNOS was used to describe eating disorders not meeting criteria for anorexia nervosa, anorexia nervosa, or binge eating disorder.

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24

Gender Dysphoria

Jennifer T. Gosselin

Defining and Describing Gender Dysphoria

One's sex refers to anatomical indicators of being male or female, also termed *natal* sex, meaning the sex one has at birth (Byne et al., 2012). One's *gender* refers to the sense that one is male or female—in other words, the gender with which one identifies (Lips, 2006). *Gender dysphoria* is the distressing experience of an inconsistency between one's natal sex and one's gender (Edwards-Leeper & Spack, 2012). Individuals who take steps toward changing their gender or appearing as the opposite gender may be referred to as transsexuals, often specified by researchers as *male-to-female transsexuals* and *female-to-male transsexuals* (Stoller, 1985), although more recent terms that are gaining acceptance are *affirmed male* or *trans-man* and *affirmed female* or *trans-woman* (Edwards-Leeper & Spack, 2012). The term *transgender* is used as a broad term to include individuals (who may or may not have had sex reassignment surgery) who live as a different gender from their natal sex, and *gender variance* is used even more broadly to include any level of different gender identification that may or may not meet diagnostic criteria for gender dysphoria (see Carroll, Gilroy, & Ryan, 2002, and also Byne et al., 2012 for a comprehensive list of terms and acronyms).

According to the fifth edition of the *Diagnostic and statistical manual of mental disorders* (DSM-5; American Psychiatric Association, 2013), gender dysphoria is defined as: "A marked incongruence between one's experienced/expressed gender and assigned gender, of at least 6 months' duration" (p. 452). In other words, the person asserts that he/she has been born in the wrong body. *Gender identity* (one's subjective sense of being male or female) is not to be confused with *sexual orientation* (to whom one is attracted) or with *gender noncomformity*, which is presenting or identifying oneself in a manner that is different from the conventional gender roles of one's culture (Coleman et al., 2011). In other words, a girl who self-identifies as a "tomboy" may be opposing stereotypical gender roles, while remaining happy to be a girl. Being sexually attracted to a particular gender or both genders is also separate from either adopting or opposing certain gender roles, as individuals who are gay, straight, or bisexual can demonstrate varying degrees of masculinity or femininity, regardless of their gender or sexual orientation. Similarly, sexual orientation is separate from being satisfied or dissatisfied with one's natal sex, as transgender individuals may be attracted to men, women, both, or neither. Any combination of sexual orientation, gender-role nonconformity, and gender identity can therefore occur.

Although more diagnostic criteria must be met to diagnose gender dysphoria in children (six criteria needed) compared with adolescents/adults (two criteria needed), the criteria for both age groups are similar in that the individuals strongly dislike and would like to change their assigned gender, often including their genitalia and/or secondary sex characteristics (if present), and they would prefer to live their lives as a different gender and to be treated as such by others, with clinically significant distress or impairment resulting from this gender inconsistency (American Psychiatric Association, 2013). In children, gender dysphoria typically involves preference for opposite-sex dress, play behaviors, playmates, and urinary position. Among adolescents and adults with gender dysphoria (the second diagnostic category based on age), behaviors may include cross-dressing, as well as breast-binding in women to reduce the appearance of their breasts. Unlike transvestic fetishism, however, the aim of cross-dressing in gender dysphoria is to appear to be the opposite gender

in order to feel consistent with one's sense of self, rather than for sexual arousal, although a person could have both aims. Researchers have highly conflicting views about the extent to which *autogynephilia* (sexual arousal due to the concept of oneself as a woman) is related to gender dysphoria (Blanchard, 2010; Moser, 2010). As noted in DSM-5, when gender dysphoria occurs in an individual with a history of transvestic disorder, there is often little-to-no sexual arousal remaining that is associated with cross-dressing (American Psychiatric Association, 2013). Additionally, natal women may feel sexy in lingerie, just as women who are male-to-female transgender may feel sexy in lingerie (Moser, 2010).

When making a differential diagnosis, the clinician should rule out gender nonconformity, variant gender expression, or dissatisfaction with society's gender roles that reflect a separate issue from the distressing disconnect between one's sex and gender (American Psychiatric Association, 2013). Disgust with one's genitals could also be a symptom of *body dysmorphic disorder* (BDD); however, BDD includes an obsessive preoccupation with a perceived imperfection in the body part, while gender dysphoria would typically involve viewing one's genitals as an unwanted reminder of one's natal sex (American Psychiatric Association, 2013). Similarly, *skoptic syndrome* is an obsessive dislike of one's primary and secondary sex characteristics, which may lead to genital self-mutilation, but it may or may not co-occur with gender dysphoria (Coleman & Cesnik, 1990). A standard clinical assessment may include items pertaining to psychotic and delusional symptoms, and an individual may experience both gender dysphoria and psychotic symptoms as well (American Psychiatric Association, 2013). However, gender dysphoria must not be deemed a fleeting psychotic symptom for a diagnosis to be made. By the same token, the insistence that one was born in the wrong body in terms of one's sex is a symptom of gender dysphoria, rather than a delusion (American Psychiatric Association, 2013).

Specific Modifications From the DSM-IV-TR to the DSM-5 for Gender Dysphoria

The American Psychiatric Association has made a number of changes to gender dysphoria in DSM-5, with the most notable changes being the modification of the name of the disorder, the placement of the disorder in a separate chapter from the sexual dysfunctions and paraphilias, clearer primary diagnostic criteria, inclusion of more diagnostic criteria, and the separation of the two diagnostic categories in terms of age (child vs. adolescent/adult). A generally supported alteration is that *gender identity disorder* is now *gender dysphoria*, with the rationale that the updated term "focuses on dysphoria as the clinical problem, not identity per se" (American Psychiatric Association, 2013, p. 451). This modification also explicitly drops the word *disorder*, which addresses the criticism that the disorder label is stigmatizing and that the symptoms of gender dysphoria are not pathological (Zucker et al., 2013). In keeping with the conceptualization of gender identity and sexual orientation as separate characteristics, the previous diagnostic specifiers of *sexual attraction* (to males, females, both, or neither) have been eliminated in DSM-5, and new specifiers have been added for the clinician to indicate whether the individual also has a *disorder of physical sex development* (such as androgen insensitivity syndrome or having intersex genitalia due to chromosomal anomalies) and if the person is in *posttransition* (such as living as the desired gender and planning to make or having already made physical changes to become a different gender).

A point of contention that still applies to some extent to the current DSM is that gender nonconforming behaviors are included as diagnostic criteria, which may lead to the overdiagnosis of children who simply do not wish to follow conventional gender norms (Haldeman, 2000). For example, the diagnostic criteria "A strong preference for the toys, games, or activities stereotypically used or engaged in by the other gender" and "A strong preference for playmates of the other gender" (American Psychiatric Association, 2013, p. 452) are generally considered to be normal variations among children and are not pathological (Hein & Berger, 2012; Meyer-Bahlburg, 2010). This potential for overdiagnosis has been addressed, however, in DSM-5 in that Criterion A1 is now required for a diagnosis: "A strong desire to be of the other gender or an insistence that one is the other gender (or some alternative gender different from one's assigned gender)" (American Psychiatric Association, 2013, p. 452; see also Zucker et al., 2013).

To further reduce the risk of false positives, the diagnostic criteria for children have been expanded to the requirement of six of eight criteria, and a duration of 6 months is now required for children and adolescents/adults (American Psychiatric Association, 2013). Not all individuals who strongly wish to change their gender actually want to become the opposite gender, and the revised DSM criteria reflect that. For example, Criterion A4 for adolescents/adults is "A strong desire to be the other gender (or some alternative gender different from one's assigned gender)" (p. 452), and the term *cross-gender identification* has been removed. In order to be inclusive toward individuals with disorders of sex development and those in posttransition who are dissatisfied with their new gender, the term *assigned gender* is used in place of *sex*. Unlike disorders such as schizophrenia, for which there are specifiers that indicate the client is currently in remission, the client who has made a successful transition to the desired gender without significant regret would no longer be diagnosable with gender dysphoria (Zucker et al., 2013).

Epidemiology of Gender Dysphoria

Gender dysphoria is relatively rare, and estimates of prevalence depend on the definition of the disorder, the population studied, and the method of data collection. Research

conducted in the Netherlands estimates that transsexualism occurs in one in 11,900 males and one in 30,400 females (Bakker, van Kesteren, Gooren, & Bezemer, 1993), with similar, slightly higher rates found more recently in Belgium (De Cuypere et al., 2007). While completing transsexual surgery is rare, gender dysphoria is more common (although still rare), with rates of the experience of gender dysphoria at approximately 1% for both genders and a desire for transsexual treatment at 0.6% for men and 0.2% for women in the Netherlands (Kuyper & Wijsen, 2013). Rates of very often wishing one were the opposite sex were higher in Taiwanese undergraduate women (2.2%) compared with men (0.7%), although it is unclear whether this result reflected actual discomfort with natal sex among women or gender-role dissatisfaction based on a cultural bias of valuing men (Lai, Chiu, Gadow, Gau, & Hwu, 2010; Tu & Liao, 2005). Among middle-school students in San Francisco, 1.3% self-identified as transgender (Shields et al., 2013). Treatment providers have noted a substantial increase over the past several decades in the number of children and adolescents who are seeking treatment for gender dysphoria, although these changes likely reflect greater identification of and acceptance of treatment for gender variance, rather than an actual increase in gender dysphoria, *per se* (Edwards-Leeper & Spack, 2012; Zucker, Bradley, Owen-Anderson, Kibblewhite, & Cantor, 2008).

Developmentally, all children progress through different stages of gender constancy, or full comprehension of gender, including an initial ability to label boys and girls in toddlerhood, followed by an understanding that (without intervention) natal sex is permanent, lifelong, and cannot be changed with clothing, for example (Berk, 2009). Gender dysphoria could therefore have an onset that coincides with this understanding, which solidifies around 4–6 years of age, although discontent with one's gender could occur before gender constancy is fully grasped, as in a young boy who asserts that he will grow up to be a mommy. The majority of adults with gender dysphoria recall an onset in childhood, and among those who seek treatment, male-to-female transsexuals are more likely to have a later onset compared to female-to-male transsexuals (Johansson, Sundbom, Hojerback, & Bodlund, 2010; Nieder et al., 2011).

Etiology of Gender Dysphoria

Biological/Neurological Factors in Gender Dysphoria
Research on biological factors in the development of gender dysphoria have primarily focused on prenatal sex hormones, genetic explanations, and structural and functional differences in the brain (see Gender Identity Research and Education Society, 2006, for a review). Neuroimaging research supports that male-to-female transsexuals seem to have different brain activation compared with heterosexual, non-gender-dysphoric men during mental rotation tasks (Schoning et al., 2010) and in response to sexual stimuli (Gizewski et al., 2009). Postmortem brain dissection has revealed similarities between natal females and male-to-female transsexuals. More specifically, there is a greater similarity in particular limbic system neuronal clusters of male-to-female transsexuals and heterosexual, non-transsexual females, as compared with homosexual and heterosexual non-transsexual men (who also showed greater similarity to each other), even after ruling out the potential effects of hormone treatment, testicle removal, sexual orientation, and cause of death (Kruijver et al., 2000; Zhou, Hofman, Gooren, & Swaab, 1995). Additional research is needed, however, owing to the small sample sizes typically used in brain research.

One biological hypothesis of the origin of gender dysphoria is that prenatal exposure to varying levels of sex hormones influences subsequent gender identity development (Gender Identity Research and Education Society, 2006). Androgen exposure in the womb has been linked to comparative finger length on one's hand, particularly between the "index" and "ring" fingers, called the second to fourth digit ratio (Lutchmaya, Baron-Cohen, Raggatt, Knickmeyer, & Manning, 2004; Manning, Scutt, Wilson, & Lewis-Jones, 1998). Among male-to-female transsexuals, the second to fourth digit ratio was more similar to natal females' than to non-transsexual males' digit ratio (Schneider, Pickel, & Stalla, 2006). Similarly, the right-hand digit ratios of female-to-male transsexuals were more similar to natal males' than natal female controls' ratios (Hisasue, Sasaki, Tsukamoto, & Horie, 2012). Handedness has also been studied, with results showing that both types of transsexuals (male-to-female and female-to-male), as well as boys who were diagnosed with gender identity disorder, were significantly more likely to be left-handed than people in the general population (Orlebeke, Boomsma, Gooren, Verschoor, & van den Bree, 1992; Zucker, Beaulieu, Bradley, Grimshaw, & Wilcox, 2001).

Genetic research has emphasized twin studies; more specifically, monozygotic (identical) twins have much higher rates of concordance for gender dysphoria than dizygotic (fraternal) twins of the same or different genders, although the majority of identical twins are also discordant for gender dysphoria (Heylens et al., 2012). Parents' ratings of their twins' gender dysphoric symptoms also showed substantial concordance (Coolidge, Thede, & Young, 2002). Twin studies can be difficult to interpret, however, since twins' family and social environments may be similar, and in that twins may choose similar environments and evoke similar responses from others based on their shared appearance and personality factors. Other family studies indicate that individuals with a transsexual sibling (particularly brothers with a transsexual sibling and/or if the sibling is a male-to-female transsexual) are more likely to become transsexual compared to the general population, although the rates of concordance for siblings was still less than 1% (Gomez-Gil et al., 2010).

On a micro level, researchers have also investigated genes that may contribute to transsexualism, particularly in male-to-female transsexuals, using DNA analysis. Genetic variations that affect androgen and estrogen receptors in the brain have received increasing attention, given that these hormone receptors seem to be involved in sex differentiation during brain development (Bao & Swaab, 2011). Although some findings have indicated a significant difference in particular genes or gene combinations influencing sex hormone receptors between male-to-female transsexuals and non-transsexual men (Hare et al., 2009; Henningsson et al., 2005), other research has failed to replicate these significant differences (Ujike et al., 2009). There have also been mixed findings with respect to genes for enzymes that are involved in sex hormone synthesis (Bentz et al., 2008; Hare et al., 2009; Ujike et al., 2009). Continued research in this area will likely reveal polygenetic influences on gender identity development, rather than a single genetic factor.

Other research has examined chromosomal and gonadal or hormonal anomalies that blur the line between the male and female sexes (see Nabhan & Lee, 2007, for a review of disorders of sex development). For example, infants born with congenital adrenal hyperplasia (CAH) may have female chromosomes, but their bodies produce excessive amounts of androgens (masculinizing hormones) owing to adrenal gland dysfunction. At birth, these infants may have large clitorises or may even appear male. Regardless of whether they are assigned to the female or male gender, most grow up without developing gender dysphoria, although they do have higher rates of gender dysphoria when compared with the very low rates in the general population. Individuals with CAH who are assigned to the male gender, and particularly those whose gender is reassigned later in childhood, tend to have higher rates of gender identity issues (Dessens, Slijper, & Drop, 2005).

In addition to CAH, a number of other physiological and chromosomal anomalies can occur that result in ambiguous genitalia. One abnormality (5 alpha-reductase-2 deficiency), which has been documented among individuals in villages in the Dominican Republic, occurred in genetically male (XY) children who had ambiguous genitalia, but appeared female, and most were reared as girls (Imperato-McGinley, Guerrero, Gautier, & Peterson, 1974). These children then developed masculine secondary sex characteristics at puberty, their testes descended, and their ambiguous genitalia developed into penises. They were known in the community as "*machihembra*," meaning "half man, half woman" or "*guevedoces*," meaning "penis at age 12." Most of these individuals adopted male gender roles as adults, although follow-up research shows that one of them in particular expressed the desire for sex reassignment surgery in adulthood to become a woman (Sobel & Imperato-McGinley, 2004). This rare condition and a similar genetic condition, 17 beta-hydroxysteroid dehydrogenase-3 deficiency, have also been reported in a variety of other regions, including Papua New Guinea, Mexico, Brazil, and in the Gaza strip, with similar findings that even when reared as girls, approximately half of these individuals (or more, depending on the sample) developed a male gender identity in adulthood (Cohen-Kettenis, 2005). Moreover, when reared as boys, they typically kept their male identity (Cohen-Kettenis, 2005). Finally, in separate research, the famous case of the boy (John/Joan) with the botched circumcision who was unsuccessfully reared as a girl also points to the role that biology plays in gender identity (Diamond & Sigmundson, 1997).

Psychosocial and Environmental Factors in Gender Dysphoria The concept of gender is socially constructed. For example, in most societies, there are two gender categories: male and female. In some societies, however, such as in India and Thailand, there is a third category for individuals who wear women's clothing, but have male (or sometimes intersex or ambiguous) genitalia and do not try to pass as female (Marecek, Crawford, & Popp, 2004). From a social cognitive perspective, gender identity development involves learning gender roles through observational learning (learning by mimicking a model), experiencing others' evaluative reactions to one's behavior, and being rewarded and punished for gender-typical or gender-atypical behavior (Bussey & Bandura, 2004). These environmental forces typically exert pressure on children to conform to gender norms, which is readily apparent by observing the pink, frilly, sparkly toys marketed to girls and the combat-oriented, explosive, mechanical toys marketed to boys. Despite this pressure to conform, individuals with gender dysphoria may persist in their sex-atypical behavior and their assertion that they are the opposite or a different sex. Many explanations have been provided for this unexpected result. For example, researchers have proposed that children with gender dysphoria have identified with and imitated a person of the opposite gender, or that they have been reinforced for gender-atypical behaviors (Halle, Schmidt, & Meyer, 1980; King, 1996). Given that these explanations seem to fall short, a biopsychosocial model may better capture the phenomenon of gender identity development.

Age, Cultural, and Gender-Related Factors in Gender Dysphoria In children, more boys than girls are typically diagnosed with gender dysphoria, although this may be partly due to greater parental disapproval of these symptoms in boys than in girls (Devor, 1996; Zucker & Bradley, 1995). The majority of children with gender dysphoria do not continue to have this disorder in adulthood (Wallien & Cohen-Kettenis, 2008). Instead of a persistence of gender dysphoria, most children with this disorder indicate at follow-up that they have come to accept their gender and that they also have bisexual or homosexual feelings of attraction as they mature (Drummond, Bradley, Peterson-Badali, & Zucker, 2008; Zucker & Bradley, 1995). Adolescents who had been diagnosed with gender identity disorder

as children noted that their gender dysphoric symptoms began around ages 6–7, and that these symptoms heightened with the social and physiological changes occurring between 10 and 13 years of age (Steensma, Biemond, de Boer, & Cohen-Kettenis, 2011).

Evidence-Based Interventions for Gender Dysphoria

Unlike treatments for most disorders, which involve helping individuals to change their thoughts, feelings, and behaviors, successful treatment for gender dysphoria may involve helping individuals explore and change their gender and/or sex, if indicated (Byne et al., 2012; Coleman et al., 2011). This transition may include a variety of treatments, including medications, such as hormone treatments, voice and communication therapy, hair transplants, electrolysis for men to remove unwanted facial and body hair, and varying degrees of surgery to masculinize or feminize the face, neck, chest, genitals, and other areas as needed. It is important for clients/patients to be advised of all of the treatment possibilities and their potential consequences and degree of permanence to the body, including fertility issues and potential health risks (Byne et al., 2012; Coleman et al., 2011).

Not all gender dysphoric individuals will decide to change their gender or will choose the opposite gender identity, or any constant gender for that matter, however. Individuals may desire varying degrees of treatment outside of the typical male-to-female or female-to-male sex reassignment surgery (Lev, 2005). One group of natal men, termed male-to-eunuch transgender, have been or wish to be castrated (have their testicles removed or rendered useless) in order to become a gender that is neither male nor female (Vale et al., 2010). These individuals typically dislike being a man, including the effects of testosterone and their genitalia, but they may have difficulty finding treatment, since they do not fit the male-to-female transsexual treatment guidelines (Vale et al., 2010). Overall, the predominant goal for treatment should be assisting individuals through the process of determining what will allow them to feel content with their chosen gender identity (Byne et al., 2012; Coleman et al., 2011).

Biological/Pharmacological and Surgical Interventions for Gender Dysphoria Although different medical centers around the world have different requirements for medical treatments for gender dysphoria, many centers have protocols that are consistent with the standards of care provided by the World Professional Association for Transgender Health (WPATH; Coleman et al., 2011) and/or the Endocrine Society (Hembree et al., 2009). These guidelines specify that certain conditions must be met before sex reassignment surgery can be performed, including a thorough clinical assessment, recommendation from one or more mental health professionals and/or physicians or other qualified treatment providers, and living as the opposite gender for 1 year while receiving continuous hormone injections (Coleman et al., 2011; Hembree et al., 2009). Requirements prior to genital surgery typically involve stages of intervention that proceed from the least invasive and most reversible treatments to the most permanent treatments, with careful assessment at every benchmark to help to ensure a positive outcome (Byne et al., 2012). Whereas requiring psychotherapy is standard in many cases, its necessity has been questioned. For example, the current standards of care by WPATH state that although an initial assessment is necessary, the hurdle of a particular number of psychotherapy sessions should not be mandated prior to medical interventions (Coleman et al., 2011). Some researchers have also expressed concern that individuals who want sex reassignment surgery have learned to report certain expected case histories, including that they are homosexual, that they have been gender dysphoric since childhood, and that they do not have transvestic fetishism because they believe it will increase their chances of being accepted for surgery (see Lev, 2005, for a review).

Some argue that gender dysphoria is not a disorder and that the requirements for sex reassignment surgery are expensive, demeaning, and invalid (Denny, 2004). It could further be argued that this type of surgery should not be treated differently than other elective surgeries, which do not have such extensive preoperative requirements. Patients with disorders of sex development, for example, are typically readily accepted for genital surgery, given that they have a clear medical diagnosis (Meyer-Bahlburg, 2010). Moreover, research suggests that transgender patients who do not comply with the 1-year opposite-gender experience guideline prior to surgery have postoperative outcomes that are no different than patients who do comply (Lawrence, 2003). Regardless of these preoperative requirements, a major obstacle to treatment—including hormone administration and surgery—is that these costs are typically out-of-pocket expenses, which can be cost-prohibitive for many patients (Edwards-Leeper & Spack, 2012). When patients can afford treatment, sex reassignment surgery typically results in positive mental and physical health outcomes (De Cuypere et al., 2005; Smith, Van Goozen, Kuiper, & Cohen-Kettenis, 2005), as well as satisfaction with sexual functioning (see Klein & Gorzalka, 2009, for a review). For example, 76% of patients reported an improvement in sexual functioning (De Cuypere et al., 2005), 92% reported high levels of satisfaction with their overall physical appearance, and 98% reported having no regrets after sex reassignment surgery (Smith et al., 2005). When negative outcomes do occur, they often involve dissatisfaction or problems with the surgical procedure, which usually cannot be predicted (Lawrence, 2003).

Hormone therapies and sex reassignment surgery are not without serious risks. Laboratory testing should be performed prior to the start of cross-sex hormone treatment to ensure that the patient does not have significant

and/or untreated artery, heart, blood clotting, or red blood cell (polycythemia) conditions, cancers (such as breast cancer), liver disease, tumors, cysts on the ovaries (polycystic ovarian syndrome), or any other unique health risks that may be exacerbated by hormone administration, and tests should also ensure that the patient is not pregnant (Coleman et al., 2011; Hembree et al., 2009). An evaluation prior to medical treatment should also include a comprehensive family medical history and personal health history, including health-related behaviors. For example, smoking poses a significantly increased health risk when combined with hormone use. Upon the start of treatment, continued assessment should occur to detect any signs of adverse effects. Treatment may need to be discontinued if clinically indicated (Coleman et al., 2011; Hembree et al., 2009).

Psychosocial Interventions for Gender Dysphoria For clients who do decide to change their gender, the primary focus of psychotherapy may be preparing them for changes in their relationships with others and with the broader society through the "coming-out" process (Byne et al., 2012). It is not uncommon for gender dysphoric individuals to also struggle with psychiatric symptoms (particularly during adolescence and prior to their transition), such as depression, social anxiety, other types of anxiety, substance use, and suicidal thoughts and actions, which also should be addressed in therapy (Gomez-Gil et al., 2012; Hepp, Kraemer, Schnyder, Miller, & Delsignore, 2005; Hoshiai et al., 2010; Nuttbrock et al., 2010; Spack et al., 2012). These symptoms are not surprising, given that gender is such a salient part of social identity, and given the increased risk for suffering caused by a number of possible stressors, such as social and familial rejection, a history of being bullied and victimized, and experiences of prejudice and discrimination (Carroll et al., 2002; Nuttbrock et al., 2010; Spack et al., 2012). The concept of *minority stress* applied to this population entails the stress of living in a world that stigmatizes transgender individuals, along with the internalization of this deprecating view of oneself (Hendricks & Testa, 2012). Further, there is an added burden of trying to present oneself in a manner that seems acceptable, while anticipating and preparing oneself for negative encounters with others (Hendricks & Testa, 2012). Coping strategies may therefore become the focus of treatment. In addition to individual therapy, couples counseling, family therapy, and group therapy may also be beneficial, particularly in affirming that despite the rarity of the disorder, the individual is not alone in his/her experience (Coleman et al., 2011).

Treatment of Gender Dysphoria in Children and Adolescents A particularly contentious area of research is treating minors who are suffering from gender dysphoria. Owing to a lack of compelling data, no psychotherapies are considered empirically validated at this time for this population, providing even more impetus for a variety of helpful and potentially harmful approaches to be used (see Moller, Schreier, Li, & Romer, 2009, for a review). Psychotherapeutic approaches range from teaching the child or adolescent to develop more socially accepted, natal sex-consistent behaviors, to taking an impartial, accepting stance that does not endorse a particular gender identity outcome, to assisting the child to transition to the opposite gender (Byne et al., 2012). Many approaches include a family therapy or psychoeducation component, support groups, and/or play dates with same-sex peers, with a variety of treatment goals that may or may not support gender transformation, often consistent with parental goals (Moller et al., 2009).

While some clinicians argue that retraining children to act in a more gender-role consistent manner will reduce peer and other interpersonal problems, Lev notes that

> in other areas where children are routinely bullied, for example racial or ethnic discrimination and physical or mental disabilities, the focus of intervention has been policy directed towards changing the social conditions that maintain abuse, not on changing children to better fit in to oppressive circumstances.
>
> (Lev, 2005, p. 49).

Some researchers therefore assert that this treatment approach is unethical and that it involves imposing narrowly defined social expectations for gender roles onto children and adolescents (Isay, 1999; Lev, 2005). Researchers further note that children or adolescents with gender-atypical behavior may be lesbian, gay, bisexual, or questioning, and thus psychotherapy should follow the American Psychological Association's (2012) Guidelines for Psychological Practice with Lesbian, Gay, and Bisexual Clients, which involves support and acceptance of feelings and behaviors that vary from socially prescribed gender roles and sexual orientation norms (Moller et al., 2009).

Others argue that detection of gender dysphoria in children can be useful in assisting them to make a transition to the opposite sex during adolescence, if clearly indicated (Cohen-Kettenis, Delemarre-van de Waal, & Gooren, 2008; Edwards-Leeper & Spack, 2012). This early transition allows the individual to avoid the continued stress of living life as a gender that is inconsistent with the sense of self. Additionally, secondary sex characteristics during puberty can be particularly problematic for adolescents with gender dysphoria. In cases involving unwavering gender dysphoria since childhood, intensifying gender dysphoria in adolescence, psychological stability, a clear desire for sex reassignment surgery, a positive response to partial and then full hormone treatment, and a successful experience living as the other gender, surgery for both male-to-female and female-to-male adolescent transsexuals have resulted in a positive outcome (Cohen-Kettenis & van Goozen, 1997). Other recommended, preoperative conditions include parental support for treatment and ongoing psychotherapy (Edwards-Leeper & Spack, 2012; see Shumer & Spack, 2013, for a review of treatment

guidelines). Not all researchers agree, however, that adolescents are developmentally ready to make such a consequential decision as a transsexual transformation, and irreversible genital procedures should not be performed on individuals under the age of legal consent in the respective country, which is 18 in the United States (Coleman et al., 2011). Further, the Endocrine Society guidelines state that treatment of preadolescent children should specifically exclude having the child live as the opposite gender and that cross-sex hormones should not be administered before the age of approximately 16 (Hembree et al., 2009).

Another treatment option for minors is to administer medications that postpone puberty by suppressing sex hormones or their effects (Coleman et al., 2011; Edwards-Leeper & Spack, 2012; Hembree et al., 2009). These medications (such as gonadotropin-releasing hormone agonists) have reversible effects on the body and therefore do not have the same magnitude of commitment as cross-sex hormone treatments or performing surgery. This treatment strategy has the benefits of giving the patient more time to work through gender identity issues, avoiding the stress caused by secondary sex characteristics, potentially minimizing the invasiveness of future surgeries (such as breast reduction), and allowing the individual to more convincingly pass as the other gender (Cohen-Kettenis et al., 2008; Edwards-Leeper & Spack, 2012). Some concerns have been raised, however, about the potential effects of these medications on bone development, and bone density testing is therefore recommended as part of the medical monitoring of these adolescents (Hembree et al., 2009).

Since gender dysphoria among children may or may not translate into gender dysphoria in adulthood, one strategy clinicians may use to serve their clients (and the families involved) is to avoid specific gender identity goals and to instead address coping strategies, psychoeducation, and support for the gender variant child as the process of gender identity development unfolds over time (Byne et al., 2012). One such approach that emphasizes identity exploration is termed *true gender self child therapy* (Ehrensaft, 2012). This approach emphasizes supportive, attentive listening and encouragement of creativity in order for the child to safely explore and express his or her self-harmonious gender identity, called the *true gender self*, while liberating the child from the stifling gender expectations imposed by others that involve playing a role, termed the *false gender self*. As explained by Ehrensaft (2012), "The job of the clinician is not to ward off a transgender outcome, but to facilitate the child's authentic gender journey" (p. 339).

Interventions That Are Counterindicated by Research for Gender Dysphoria

Psychotherapeutic treatment for gender dysphoria has been highly controversial and is similar to treatment for homosexuality in that there is a history of treatment strategies designed to change the client to fit with societal norms, rather than to accept the client and support the client's wishes and view of him/herself (Dickinson, Cook, Playle, & Hallett, 2012). With the rise of behaviorism, different strategies were used with the goal of making individuals—especially men who were gay, transvestites, or transgender—become heterosexual men without any fetishes, if present (Jordan & Deluty, 1995). Primarily in the 1960s and 1970s, aversion treatments were used on lesbian, gay, bisexual or transgender, or who are questioning their sexual orientation (LGBTQ) individuals and cross-dressers, involving pain by electric shock and/or nausea and vomiting through medications. The goal was for the client to pair the aversive condition (such as induced vomiting or an electric shock, administered to the feet in some cases) with the client's cross-dressing, nude photos of men, or with homosexual fantasies or the reading of passages of homosexual encounters, sometimes with shocks corresponding to any detected erections (Dickinson et al., 2012; McConaghy, 1970). Some clients volunteered for treatment, while others faced an ultimatum of treatment or imprisonment, based on archaic laws (Dickinson et al., 2012). These treatments are likely to be harmful, not only in that they can be dehumanizing, painful, and generate suffering and subsequent psychological symptoms, but inducing vomiting via medication also carries a risk of death due to choking or severe side effects (Smith, Bartlett, & King, 2004).

Although less physically painful, talk therapies have also been used with the goal of sexual orientation change efforts and gender conformity, in some cases with religious clients who desire this change (APA Task Force on Appropriate Therapeutic Responses to Sexual Orientation, 2009). These therapies (called reparative or conversion therapies) have demonstrated the potential for harm to the individual's personal and social well-being, from anxiety and relationship difficulties to depression and suicidality, with minimal or questionable efficacy findings (APA Task Force on Appropriate Therapeutic Responses to Sexual Orientation, 2009; Blackwell, 2008; Hein & Matthews, 2010). Complicit with the treatment of homosexuality and gender dysphoria to encourage the person to become heterosexual or to refrain from a successful gender transition is the value that heterosexuality or a non-LGBTQ identity is preferable. Clinicians who engage in sexual orientation change efforts and gender conformity treatment are therefore imposing this value judgment on their clients, which is considered unethical and inconsistent with the standards of care for working with LGBTQ individuals (American Psychological Association, 2012; Coleman et al., 2011). Further, a conflict between the individual and society is specifically not considered pathological according to the DSM; thus, gender variance should be accepted and supported.

Summary and Diagnostic Issues

Gender dysphoria involves a conviction that one is or should have been born a different sex. This condition

reflects a wider range of beliefs about one's gender and desired physical changes than was originally thought, including individuals who want to live as the opposite gender with or without hormone therapy and with or without sex reassignment surgery, or individuals who do not wish to choose a gender or who view gender as nonstatic (Lev, 2005). The examination of the etiology of gender dysphoria is dominated by biological and genetic research (Gender Identity Research and Education Society, 2006). Treatment may include a variety of hormone therapies, surgeries, voice/behavioral coaching, and supportive counseling (Byne et al., 2012; Coleman et al., 2011). For those individuals who opt for sex reassignment surgery, there are commonly used guidelines that typically must be met before the surgery can be performed, but most transsexuals have a successful surgery and are relieved of their gender dysphoria (Smith et al., 2005).

The DSM as an Evolving Document Gender dysphoria has been at the center of much debate. Given that the distress of gender dysphoria can be removed through physical changes and/or surgery and because the diagnosis may be stigmatizing, some argue that gender dysphoria be eliminated from the DSM and that it instead be considered a treatable medical condition (Ault & Brzuzy, 2009; Coleman et al., 2011). A basic comparison is that an individual who strongly dislikes his/her nose and is pleased with the results of a rhinoplasty would not be diagnosed with BDD. There are many implications for removing gender dysphoria from the DSM, however, ranging from: potential effects on court cases, treatment coverage, the sense of validation that this is a genuine cluster of symptoms, the accuracy of diagnosis if other diagnoses are used instead, the awareness and identification of these symptoms and related training among treatment providers, and the sustainability of relevant research and treatment centers (Meyer-Bahlburg, 2010). For example, some have argued that removal of gender dysphoria could make discrimination lawsuits on behalf of transgender individuals more difficult (Romeo, 2008,[1] as cited in Meyer-Bahlburg, 2010). Aside from gender dysphoria in adults, other researchers argue that this disorder should not be diagnosed in children because it may stigmatize them during their identity development and may be used as the basis for gender conformity treatment (Hein & Berger, 2012; Lev, 2005).

Among researchers who agree that gender dysphoria should continue to be a diagnostic category, there are still criticisms of the diagnostic criteria (see Dragowski, Scharron-del Rio, & Sandigorsky, 2011, for a review). Debate continues around the inclusion of the clinically significant distress criterion for gender dysphoria, given that "Socially deviant behavior (e.g., political, religious, or sexual) and conflicts that are primarily between the individual and society are not mental disorders unless the deviance or conflict results from a dysfunction in the individual" (American Psychiatric Association, 2013, p. 20). In other words, at least a portion of the distress or difficulty caused by gender dysphoria may involve stigma and rejection by society, which is explicitly not a symptom of a disorder (Ault & Brzuzy, 2009; Dragowski et al., 2011). Others argue, however, that gender dysphoria is distressing to the individual in that he/she feels inconsistent with his/her body, which is separate from a conflict with society or social norms (Zucker et al., 2013).

Regardless of whether gender dysphoria is retained as a disorder in the future, clinical, medical, and legal practices should promote acceptance, empathy, and empowerment to gender variant individuals. Our gender is our first social identity of interest to others when we are born and continues to be an important social identity throughout our lives. Individuals should therefore be able to self-identify and self-define this central aspect of themselves in a manner that feels genuine.

Note

1. F. Romeo, "Overview of transgender legal strategies and implications for DSM-V." Lecture presented at the Work Group on Gender, Department of Psychiatry, Columbia University, New York, NY, September 19, 2008.

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Note: The following abbreviations have been used – *f* = figure; *n* = note; *t* = table