15

PSYCHOLOGICAL DISORDERS
when adaptation breaks down

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Below are descriptions of five actual patients (with names changed to safeguard their identities) drawn from the clinical experiences of two of your text’s authors. Read each description and ask yourself what these people have in common.

Ida, 43 years old, was strolling around a shopping mall by herself. Suddenly and out of the blue, she experienced a burst of incredibly intense anxiety that left her feeling terrified, faint, and nauseated. She thought she was having a heart attack and took a taxi to the nearest emergency room. The doctors found nothing wrong with her heart and told her that the problem was “all in her head.” Ida has since refused to leave her house or go anywhere without her husband. She’s scared to drive or take buses. Ida’s diagnosis: panic disorder (with agoraphobia).

Bill, 45 years old, hasn’t shaved or showered in over ten years. His beard is several feet long. Bill doesn’t want to shave or shower because he’s terrified that tiny “metal slivers” from the water will find their way into his skin. As much as possible, Bill avoids talking on the telephone or walking through doorways because he’s petrified of acquiring germs. Whenever he experiences a thought he feels he shouldn’t be having—such as a desire to kiss a married woman—he counts backwards from 100 by sevens. Bill recognizes these behaviours as irrational, but hasn’t been able to change them despite two decades of treatment. Bill’s diagnosis: obsessive-compulsive disorder.

A few days after having a baby at age 30, Ann became incredibly giddy. She felt on top of the world, barely needed any sleep, and for the first time began sleeping with men she’d just met. Ann also became convinced that she’d turned into a clown—literally. She was even persuaded that she had a bright red round nose, even though her nose was entirely normal. Ann’s diagnosis: bipolar disorder.

Terrell, 28 years old, has just been released from the intensive care unit of a city hospital. He had shot himself in the stomach after becoming convinced that fish were swimming there. He suspects that these fish are part of a government conspiracy to make him physically ill. Terrell’s diagnosis: schizophrenia.

Johnny is 13 years old. He’s charming, articulate, and fun loving. Yet he’s furious that his parents have helped commit him to the inpatient unit of a psychiatric hospital, and he blames them for his problems. Johnny is well aware that his actions, like cursing at teachers, holding live cats underwater until they drown, and attempting to blow up his junior high school with stolen dynamite, aren’t exactly popular among adults. Yet he sees nothing especially wrong with these behaviours and admits that he’s never felt guilty about anything. Johnny’s diagnosis: conduct disorder (with probable psychopathic personality).

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What Is Mental Illness? A Deceptively Complex Question

The answer to this question isn’t as simple as we might assume, because the concept of mental disorder doesn’t lend itself to a clear-cut dictionary definition. Instead, psychologists and psychiatrists have proposed a host of criteria for what mental disorder is. We’ll review five of them here. Each criterion captures something important about mental disorder, but each has its shortcomings (Gorenstein, 1984; Wakefield, 1992).

**Statistical Rarity.** Many mental disorders, like schizophrenia—Terrell’s condition—are uncommon in the population. Yet we can’t rely on statistical rarity to define mental disorder, because not all infrequent conditions—such as extraordinary creativity—are pathological, and many mental illnesses—such as mild depression—are quite common (Kendell, 1975).

**Subjective Distress.** Most mental disorders, including mood and anxiety disorders, produce emotional pain for individuals afflicted with them. But not all psychological disorders generate distress. For example, during the manic phases of bipolar disorder, which Ann experienced, people frequently feel better than normal and perceive nothing wrong with their behaviours. Similarly, many adolescents with conduct disorder, like Johnny, experience less distress than the typical adolescent.

**Impairment.** Most mental disorders interfere with people’s ability to function in everyday life. These disorders can destroy marriages, friendships, and jobs. Yet the presence of impairment by itself can’t define mental illness, because some conditions, like laziness, can produce impairment but aren’t mental disorders. Think about it: At exam time, students don’t eat or sleep especially well, but we don’t conclude that they’re mentally maladjusted—just that they’re coping!

**Societal Disapproval.** Nearly 50 years ago, psychiatrist Thomas Szasz (1960) argued that “mental illness is a myth” and that “mental disorders” are nothing more than conditions that society dislikes. He even proposed that psychologists and psychiatrists use diagnoses as weapons of control: By attaching negative labels to people whose behaviours they find objectionable, they’re putting these people “in their place.” Szasz was both right and wrong. He was right that our negative attitudes toward those with serious mental illnesses are often deep-seated and widespread. Szasz was also right that societal attitudes shape our views of abnormality.

Psychiatric diagnoses have often mirrored the views of the times. For centuries, some psychiatrists invoked the diagnosis of masturbational insanity to describe individuals whose compulsive masturbation supposedly drove them mad (Hare, 1962). Homosexuality was classified as a mental illness until members of the American Psychiatric Association (APA) voted to remove it from their list of disorders in 1973 (Bayer, 1981; see Chapter 11). As society became more accepting of homosexuality, mental health professionals came to reject the view that such behaviour is indicative of psychological disorder.

But Szasz was wrong in his assertion that society regards all disapproved-of conditions as mental disorders (Wakefield, 1992). To take just one example, racism is justifiably deplored by society but isn’t considered a mental disorder by either laypersons or mental health professionals (Yamey & Shaw, 2002). Neither is messiness or rudeness, even though they’re both considered undesirable by society.

**Biological Dysfunction.** Many mental disorders probably result from breakdowns or failures of physiological systems. For example, we’ll learn that schizophrenia is often marked by an underactivity in the brain’s frontal lobes. In contrast, some mental disorders, like specific phobias (intense and irrational fears; see Chapter 6), appear to be acquired largely through learning experiences and often require only a weak genetic predisposition to trigger them.
In fact, it's unlikely that any one criterion distinguishes mental disorders from normality, which explains why mental disorder is difficult or impossible to define. As a consequence, some authors have argued for a family resemblance view of mental disorder (Kirmayer & Young, 1999; Lilienfeld & Marino, 1995; Rosenhan & Seligman, 1989). According to this perspective, mental disorders don't all have one thing in common. Just as brothers and sisters within a family look similar but don't all possess exactly the same eyes, ears, or noses, mental disorders share a loose set of features. These features include those we've described—statistical rarity, subjective distress, impairment, societal disapproval, and biological dysfunction—as well as others, such as a need for treatment, irrationality, and loss of control over one's behaviour (Bergner, 1997). So Ida, Bill, Ann, Terrell, and Johnny aren't alike in precisely the same way. Yet they overlap enough in their features that we recognize each of them as having a mental disorder.

**Historical Conceptions of Mental Illness: From Demons to Asylums**

Throughout history, people have recognized certain behaviours as abnormal. Yet their explanations and treatments for these behaviours have shifted in tune with prevailing cultural conceptions. The history of society's evolving views of mental illness tells the fascinating story of a bumpy road from nonscience to science.

**The Demonic Model.** During the Middle Ages, many people in Europe and later in North America viewed mental illnesses through the lens of a demonic model. They attributed hearing voices, talking to oneself, and other odd behaviours to the actions of evil spirits infesting the body (Hunter & Macalpine, 1963). They also viewed at least some, but not all (Schoeneman, 1984), witches as having a mental illness. In 1486, two German priests released a detailed manual, the *Malleus Maleficarum* ("The Witches' Hammer"), to assist in identifying witches, whom many religious figures believed were possessed by the devil. For decades, this text was second only to the Bible as the world's best-selling book. According to the *Malleus Maleficarum*, one could detect witches by means of such foolproof indicators as the Devil's Mark, a spot on the skin that's insensitive to pain. The *Malleus Maleficarum* played a key role in the witch hunts of the sixteenth and seventeenth centuries, which resulted in the executions of tens of thousands of innocent individuals.

The often bizarre "treatments" of the day, including exorcisms, flowed directly from the demonic model. Yet the legacy of the demonic model lives on today in the thousands of exorcisms still performed in Italy, Mexico, and other countries (Harrington, 2005).

**The Medical Model.** As the Middle Ages faded and the Renaissance took hold, views of those with mental illness became more enlightened. Over time, more people came to perceive mental illness primarily as a physical disorder requiring medical treatment—a view that some scholars refer to as the medical model (Blaney, 1975). Beginning in the fifteenth century and especially in later centuries, European governments began to house these individuals in asylums—institutions for those with mental illness (Gottesman, 1991). Although the term asylum means a place of safety, it's acquired a considerably
more negative connotation because many institutions were massively overcrowded and understaffed. Indeed, the term bedlam, meaning “utter chaos,” derives from a shortened version of “Bethlehem,” the name of an insane asylum in London established in the Middle Ages (Scull, MacKenzie, & Hervey, 1996).

Moreover, the medical treatments of that era were scarcely more scientific than those of the demonic era, and several were equally barbaric. One gruesome treatment was “bloodletting,” which was premised on the mistaken notion that excessive blood causes mental illness. In some cases, physicians drained patients of nearly 1.8 kilograms (four pounds) of blood, about 40 percent of the body’s total. In still other cases, staff workers tried to frighten patients “out of their diseases” by tossing them into a pit of snakes, hence the term snake pit as a synonym for an insane asylum (Szasz, 2006).

Not surprisingly, most patients of this era deteriorated, and in the case of bloodletting, some died. Even those who improved in the short term may have merely been responding to the placebo effect—improvement resulting from the expectation of improvement (see Chapter 2). Yet few physicians of the day considered the placebo effect as a rival explanation for these treatments’ seeming effectiveness. Although most of these treatments seem preposterous to us today, it’s crucial to recognize that psychological and medical treatments are products of the times. Society’s beliefs about the causes of mental illness shape its interventions.

Fortunately, reform was on the way. Thanks to the heroic efforts of Phillippe Pinel (1748–1826) in France and Dorothea Dix (1802–1887) in the United States, an approach called moral treatment gained a foothold in Europe and North America. Advocates of moral treatment insisted that those with mental illness be treated with dignity, kindness, and respect. Prior to moral treatment, patients in asylums were often bound in chains; following moral treatment, they were free to roam the halls of hospitals, get fresh air, and interact freely with staff and other patients. Still, effective treatments for mental illnesses were virtually nonexistent, so many people continued to suffer for years with no hope of relief.

**THE MODERN ERA OF PSYCHIATRIC TREATMENT.** It wasn’t until the early 1950s that a dramatic change in society’s treatment of individuals with mental illness arrived on the scene. It was then that psychiatrists introduced a medication imported from France called chlorpromazine (its brand name is Thorazine) in mental hospitals (see Chapter 16). Chlorpromazine wasn’t a miracle cure, but it offered a modestly effective treatment for some symptoms of schizophrenia and other disorders marked by a loss of contact with reality. Many patients with these conditions became able to function independently, and some returned to their families. Others held jobs for the first time in years, even decades.

By the 1960s and 1970s, the advent of chlorpromazine and similar medications (see Chapter 16) became the primary impetus for a governmental policy called deinstitutionalization. Deinstitutionalization featured two major components: releasing hospitalized psychiatric patients into the community and closing mental hospitals (Torrey, 1997). Following deinstitutionalization, the number of hospitalized psychiatric patients plummeted through the beginning of the twenty-first century. But deinstitutionalization was a decidedly mixed blessing. Some patients returned to a semblance of a regular life, but tens of thousands of others spilled into cities and rural areas without adequate follow-up care. Many went off their medications and wandered the streets aimlessly. Some of the homeless people we can see today on the streets of major North American cities are a tragic legacy of deinstitutionalization (Leeper, 1988). Today, psychologists, social workers, and other mental health professionals are working to improve the quality and availability of community care for severely affected psychiatric patients. Among the consequences of these efforts are community mental health centres and halfway houses, which are free or low-cost care facilities in which people can obtain treatment.
Thankfully, our understanding of mental illness and its treatment today is considerably more sophisticated than it was centuries ago. Still, precious few of today’s treatments are genuine cures.

### Psychiatric Diagnoses across Cultures

Psychiatric diagnoses are shaped not only by history, but also by culture (Chentsova-Dutton & Tsai, 2006; Watters, 2010). Psychologists have increasingly recognized that certain conditions are culture-bound—specific to one or more societies—although most of these conditions remain poorly researched (see Table 15.1) (Kleinman, 1988; Simons & Hughes, 1986).

**CULTURE-BOUND SYNDROMES.** For example, some parts of Malaysia and several other Asian countries, including China and India, have witnessed periodic outbreaks of a strange condition known as koro. The victims of koro, most of whom are male, typically believe that their penis and testicles are disappearing and receding into their abdomen (female victims of koro sometimes believe that their breasts are disappearing). Koro is spread largely by social contagion. Once one man begins to experience its symptoms, others often follow suit, triggering widespread panics (see Chapter 13). In one region of India in 1982, the koro epidemic spun so out of control that the local government took to the streets with loudspeakers to reassure terrified civilians that their genitals weren’t vanishing. Government officials even measured male residents’ penises with rulers in an attempt to prove their fears unfounded (Bartholomew, 1994).

Another disorder specific to Malaysia, the Philippines, and some African countries is amok. This condition is marked by episodes of intense sadness and brooding followed by uncontrolled behaviour and unprovoked attacks on people or animals (APA, 2013). This condition gave rise to the popular phrase “running amok,” meaning “going wild.”

Other culture-bound syndromes seem to be variants of conditions in Western culture. In Japan, for example, social anxiety is typically expressed as a fear of offending others (called taijin kyofushu), such as by saying something offensive or giving off a terrible body odour (Kleinknecht et al. 1994). But in Canada and the United States, social anxiety is more commonly generated by fear of public embarrassment, such as what we might experience when giving a speech. Culture may influence how people express interpersonal anxiety. Because Japanese culture is more collectivistic (see Chapters 10 and 14) than Western culture, Japanese tend to be more concerned about their impact on others than are Westerners.

### Table 15.1 A Sampling of Culture-Bound Syndromes Not Discussed in the Text.

<table>
<thead>
<tr>
<th>SYNDROME</th>
<th>REGION OR PEOPLES AFFECTED</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arctic Hysteria</td>
<td>Inuit</td>
<td>Abrupt episode accompanied by extreme excitement and frequently followed by convulsive seizures and coma.</td>
</tr>
<tr>
<td>Couvade Syndrome</td>
<td>worldwide</td>
<td>An expectant father’s sympathetic labour pains, food cravings, nausea, even breast growth; in extreme cases, men may gain 25–30 pounds with a sympathetic belly lump (also called phantom pregnancy).</td>
</tr>
<tr>
<td>Gururumba</td>
<td>New Guinea</td>
<td>Theft and later deposit of neighbours’ possessions in the forest followed by amnesia of the entire episode.</td>
</tr>
<tr>
<td>Hwa-byung</td>
<td>Korea</td>
<td>Abdominal pain caused by emotional distress.</td>
</tr>
<tr>
<td>Mal de Ojo (Evil Eye)</td>
<td>Spain and Latin America</td>
<td>A common term to describe the cause of disease, misfortune, and social disruption.</td>
</tr>
<tr>
<td>Saora disorder</td>
<td>India</td>
<td>Inappropriate laughing or crying, fainting, memory loss, and the sensation you are being bitten by ants.</td>
</tr>
<tr>
<td>Windigo</td>
<td>First Nations in central and northeast Canada</td>
<td>Craving consumption of human flesh and fear of becoming a cannibal.</td>
</tr>
</tbody>
</table>

(Sources: Hall, 2001; Higgs, 2011; O’Neil, 2010; Klein, 2001; Park et al., 2001)
In contrast, Western culture is more individualistic, so people tend to worry more about what may happen to them as individuals. European-Canadian patients report more psychological symptoms than their Chinese counterparts, perhaps suggesting a tendency to “psychologize” symptoms of distress. Because Chinese patients frequently focus their thoughts “externally,” as opposed to “internally” on their emotional states, they may be more likely to notice somatic symptoms like aches and pains when distressed (Ryder et al., 2008). The focus on the self in individualistic societies may also contribute to certain culture-bound disorders in Western countries. Some eating disorders are largely specific to Canada, the United States, and Europe, where the media bombard viewers with images of thin models, probably making already self-conscious women even more self-conscious (see Chapter 11) (Keel & Klump, 2003; McCarthy, 1990).

CULTURAL UNIVERSALITY. Despite the cultural differences we’ve noted, we shouldn’t exaggerate the cultural relativity of mental disorders. Many mental disorders, especially those that are severe, appear to exist in most and perhaps all cultures. Jane Murphy (1976) conducted a classic study of two isolated societies—a group of Yorubas in Nigeria and a group of Inuit near the Bering Strait—that had experienced essentially no contact with Western culture. These societies possessed terms for disorders that are strikingly similar to schizophrenia, alcoholism, and psychopathic personality, a condition marked by dishonesty, manipulativeness, and an absence of guilt and empathy (see Chapters 11 and 14). For example, the Inuit use kunlangeta to describe a person who lies, cheats, steals, is unfaithful to women, and doesn’t obey elders—a description that fits almost perfectly the Western concept of psychopathic personality. When Murphy asked one of the Inuit how they dealt with such individuals, he replied that “somebody would have pushed him off the ice when no one was looking.” Apparently, Inuit aren’t much fonder of psychopaths than we are.

Special Considerations in Psychiatric Classification and Diagnosis

Because there are so many ways in which psychological adaptation can go awry, we’d be hopelessly lost without some system of diagnostic classification. Psychiatric diagnoses serve at least two crucial functions. First, they help us pinpoint the psychological problem a person is experiencing. Once we’ve identified this problem, it’s often easier to select a treatment. Second, psychiatric diagnoses make it easier for mental health professionals to communicate. When a psychologist diagnoses a patient with schizophrenia, she can be reasonably certain that other psychologists know the patient’s principal symptoms. Diagnoses operate as forms of mental shorthand, simplifying complex descriptions of problematic behaviours into convenient summary phrases.

Still, there are a host of misconceptions regarding psychiatric diagnosis. Before turning to our present system of psychiatric classification, we’ll examine four prevalent misconceptions.

Misconception 1: Psychiatric diagnosis is nothing more than pigeonholing—that is, sorting people into different “boxes.” According to this criticism, when we diagnose people with a mental disorder, we deprive them of their uniqueness: We imply that all people within the same diagnostic category are alike in all important respects. To the contrary, a diagnosis implies only that all people with that diagnosis are alike in at least one important respect (Lilienfeld & Landfield, 2008). Psychologists recognize that even within a diagnostic category, like schizophrenia or bipolar disorder, people differ dramatically in their other psychological difficulties, race and cultural background, personality traits, interests, and cognitive skills. People are far more than their disorders.

Misconception 2: Psychiatric diagnoses are unreliable. As we learned in Chapter 2, reliability refers to consistency of measurement. In the case of psychiatric diagnoses, the form of reliability that matters most is interrater reliability: the extent to which different raters (such as different psychologists) agree on patients’ diagnoses. The widespread perception that psychiatric diagnosis is unreliable is probably fuelled by high-profile media coverage of “duelling expert witnesses” in criminal trials, in which one expert witness diagnoses a defendant as experiencing schizophrenia and another diagnoses him as free of this disorder.

FACTOID

Another potential culture-bound syndrome is the unusual condition of body integrity identity disorder (also known as apotemnophilia; in Greek, apotemno “cut off” + philia “love of”), in which people experience persistent desires to amputate their limbs or body parts. Although responsible physicians won’t perform such operations, many patients with body integrity identity disorder have found doctors willing to amputate their limbs (First, 2004). This disorder has thus far been reported only in Canada, the United States, and Europe (Littlewood, 2004).
In fact, for major mental disorders, like schizophrenia, mood disorders, anxiety disorders, and alcoholism, interrater reliabilities are typically as high—correlations between raters of 0.8 or above out of a maximum of 1.0—as for most medical disorders (Matarazzo, 1983). Still, the picture isn’t entirely rosy. For many personality disorders, a class of disorders we’ll discuss later in this chapter, interrater reliabilities tend to be lower (Zimmerman, 1994).

**Misconception 3: Psychiatric diagnoses are invalid.** From the standpoint of Thomas Szasz (1960) and other critics, psychiatric diagnoses are largely useless because they don’t provide us with much, if any, new information. They’re merely descriptive labels for behaviours we don’t like.

When it comes to some popular psychology labels, Szasz probably has a point. Consider the explosion of diagnostic labels that are devoid of scientific support, such as codependency, sexual addiction, Internet addiction, road rage disorder, and compulsive shopping disorder (Cocarro & Danehy, 2006; Granello & Beamish, 1998; Koran et al., 2006; McCann, Shindler, & Hammond, 2003). Although frequently used in talk shows, television programs, movies, and self-help books, these labels aren’t recognized as formal psychiatric diagnoses.

Yet there’s now considerable evidence that many psychiatric diagnoses do tell us something new about the person. In a classic paper, psychiatrists Eli Robins and Samuel Guze (1970) outlined several criteria for determining whether a psychiatric diagnosis is valid. According to Robins and Guze, a valid diagnosis:

1. Distinguishes that diagnosis from other, similar diagnoses
2. Predicts diagnosed individuals’ performance on laboratory tests, including personality measures, neurotransmitter levels, and brain imaging findings (Andreasen, 1995)
3. Predicts diagnosed individuals’ family history of psychiatric disorders
4. Predicts diagnosed individuals’ natural history—that is, what tends to happen to them over time
5. Predicts diagnosed individuals’ response to treatment (Waldman, Lilienfeld, & Lahey, 1995)

There’s good evidence that unlike most popular psychology labels, many mental disorders fulfill Robins and Guze’s criteria for validity. **TABLE 15.2** illustrates these criteria using the example of attention-deficit/hyperactivity disorder (ADHD), a disorder we’ll discuss later in the chapter that’s characterized by inattention, impulsivity, and overactivity.

**TABLE 15.2** Criteria for Validity: The Case of ADHD. Although controversial in many respects, the diagnosis of attention-deficit/hyperactivity disorder (ADHD) largely satisfies the Robins and Guze criteria for validity.

<table>
<thead>
<tr>
<th>ROBINS &amp; GUZE CRITERIA</th>
<th>FINDINGS CONCERNING THE ADHD DIAGNOSIS</th>
</tr>
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<tbody>
<tr>
<td>1. Distinguishes a particular diagnosis from other similar diagnoses</td>
<td>The child’s symptoms can’t be accounted for by other diagnoses, such as substance abuse and anxiety disorders.</td>
</tr>
<tr>
<td>2. Predicts performance on laboratory tests (personality measures, neurotransmitter levels, brain imaging findings)</td>
<td>The child is likely to perform poorly on laboratory measures of concentration.</td>
</tr>
<tr>
<td>3. Predicts family history of psychiatric disorders</td>
<td>The child has a higher probability than the average child of having biological relatives with ADHD.</td>
</tr>
<tr>
<td>4. Predicts what happens to the individual over time</td>
<td>The child is likely to show continued difficulties with inattention in adulthood, but improvements in impulsivity and overactivity in adulthood.</td>
</tr>
<tr>
<td>5. Predicts response to treatment</td>
<td>The child has a good chance of responding positively to stimulant medications, like Ritalin (see Chapter 16).</td>
</tr>
</tbody>
</table>
Misconception 4: **Psychiatric diagnoses stigmatize people.** According to labelling theorists, psychiatric diagnoses exert powerful negative effects on people’s perceptions and behaviours (Scheff, 1984; Slater, 2004). Labelling theorists argue that once a mental health professional diagnoses us, others perceive us differently. Suddenly, we’re “weird,” “strange,” even “crazy.” This diagnosis leads others to treat us differently, in turn often leading us to behave in weird, strange, or crazy ways. The diagnosis thereby becomes a self-fulfilling prophecy.

In a sensational study, David Rosenhan (1973) asked eight individuals with no symptoms of mental illness (himself included) to pose as fake patients in 12 psychiatric hospitals. These “pseudopatients” (fake patients) presented themselves to admitting psychiatrists with a single complaint: They were hearing a voice saying “empty, hollow, and thud.” In all 12 cases, the psychiatrists admitted these pseudopatients to the hospital, almost always with diagnoses of schizophrenia (one received a diagnosis of manic depression, or what would today be called bipolar disorder). Remarkably, they remained there for an average of three weeks despite displaying no further symptoms of mental illness. The diagnosis of schizophrenia, Rosenhan concluded, became a self-fulfilling prophecy, leading doctors and nursing staff to view these individuals as disturbed. For example, the nursing staff interpreted one pseudopatient’s note taking as “abnormal writing behaviour.”

It’s true that there’s still stigma attached to some psychiatric diagnoses. If someone tells us that a person has schizophrenia, for instance, we may be wary of him at first or misinterpret his behaviours as consistent with this diagnosis. Yet the negative effects of labels last only so long. Even in Rosenhan’s study, all pseudopatients were released from hospitals with diagnoses of either schizophrenia or manic depression “in remission” (“in remission” means without any symptoms) (Spitzer, 1975). These discharge diagnoses tell us that psychiatrists eventually recognized that these individuals were behaving normally. Overall, there’s not much evidence that psychiatric diagnoses themselves generate long-term negative effects (Ruscio, 2003).

### Psychiatric Diagnosis Today: DSM-5

The official system for classifying individuals with mental disorders is the Diagnostic and Statistical Manual of Mental Disorders (DSM), which originated in 1952 and is now in its fifth edition, called DSM-5 (APA, 2013). There are 18 different classes of disorders in the DSM-5, several of which we’ll discuss in the pages to come.

**Diagnostic Criteria and Decision Rules.** Psychiatric classification has come a long way since the days of the *Malleus Malleficarum*. DSM-5 provides psychologists and psychiatrists with a list of diagnostic criteria for each condition, and a set of decision rules for deciding how many of these criteria need to be met. For example, to diagnose a person with major depressive disorder, DSM-5 requires the person to exhibit at least five of nine symptoms, including fatigue, insomnia, problems concentrating, and significant weight loss over a two-week period, with the requirement that the person experience either depressed mood or diminished interest or pleasure in everyday activities, or both.

“**THINKING ORGANIC.**” DSM-5 warns diagnosticians about physical, or “organic”—that is, medically induced—conditions that can simulate certain psychological disorders (Morrison, 1997). DSM-5 notes that certain substance use or medical disorders can mimic the clinical picture of depression. For example, it informs readers that *hypothyroidism*, a disorder marked by underactivity of the thyroid gland (in our lower necks), can produce depressive symptoms. If a patient’s depression appears due to hypothyroidism, the psychologist shouldn’t diagnose major depression. It’s essential to “think organic,” or to first rule out medical causes of a disorder, when diagnosing psychological conditions.

**The DSM-5: Other Features.** DSM-5 is more than a tool for diagnosing mental disorders; it’s a valuable source of information concerning the characteristics, such as the prevalence, of many mental disorders. **Prevalence** refers to the percentage of people in the population with a disorder. In the case of major depression, the lifetime prevalence is at least 10 percent among women and at least 5 percent among men (some estimates are even higher). That means that...
for a woman, the odds are at least 1 in 10 she’ll experience an episode of major depression at some point in her life; for a man, the odds are at least 1 in 20 (APA, 2013).

DSM-5 also recognizes that there’s more to people than their disorders. The manual adopts a biopsychosocial approach, which acknowledges the interplay of biological (like hormonal abnormalities), psychological (like irrational thoughts), and social (interpersonal interactions) influences. Specifically, it reminds diagnosticians to attend carefully to patients’ ongoing life stressors, past and present medical conditions, and overall level of functioning when evaluating their psychological status.

Finally, DSM-5 acknowledges that we live in a diverse world filled with people from different ethnic, socioeconomic, and cultural backgrounds. Some of them embrace unconventional beliefs, sexual identities, and behaviours that are “abnormal” from the vantage point of our contemporary society. DSM-5 provides information about how differing cultural backgrounds can affect the content and expression of symptoms. This information is vital to ensuring that diagnosticians do not incorrectly label someone from a different culture with a mental disorder merely because that person is exhibiting behaviours that those of us in Western culture might find unusual or unfamiliar.

THE DSM-5: CRITICISMS. There’s little dispute that DSM-5 is a helpful system for slicing up the enormous pie of psychopathology into more meaningful and manageable pieces. Yet DSM-5 has received more than its share of criticism, and sometimes for good reason (Frances & Widiger, 2012; Widiger & Clark, 2000). There are well over 300 diagnoses in DSM-5, not all of which meet the Robins and Guze criteria for validity. To take only one example, the DSM-5 diagnosis of “Mathematics Disorder” describes little more than difficulties with performing arithmetic or math reasoning problems. It seems to be more of a label for learning problems than a diagnosis that tells us something new about the person. In addition, although the diagnostic criteria and decision rules for many DSM-5 disorders are based primarily on scientific findings, others are based largely on subjective committee decisions. Another problem with DSM-5 is the high level of comorbidity among many of its diagnoses (Angold, 1999; Cramer et al., 2010; Lilienfeld, Waldman, & Israel, 1994), meaning that individuals with one diagnosis frequently have one or more additional diagnoses. For example, it’s extremely common for people with a major depression diagnosis to meet criteria for one or more anxiety disorders. This extensive comorbidity raises the troubling question of whether DSM-5 is diagnosing genuinely independent conditions as opposed to slightly different variations of one underlying condition.

Another problem with DSM-5 is its reliance on a categorical model of psychopathology (Trull & Durett, 2005). In a categorical model, a mental disorder—such as major depression—is either present or absent, with no in-between. Categories differ from each other in kind, not degree. Pregnancy fits a categorical model, because a woman is either pregnant or she’s not. Yet scientific evidence suggests that some disorders in DSM-5 better fit a dimensional model, meaning that they differ from normal functioning in degree, not kind (Krueger & Piasecki, 2002). Height fits a dimensional model, because although people differ in height, these differences aren’t all or none. The same may be true of many forms of depression and anxiety, which most research suggests lie on a continuum with normality (Kollman et al., 2006; Slade & Andrews, 2005). These findings square with our everyday experience, because we all feel at least a bit depressed and anxious from time to time.

Some authors have proposed that the Big Five, a system of personality dimensions we encountered in Chapter 14, may better capture the true “state of nature” than many of the categories in DSM-5 (Widiger & Clark, 2000; Wright et al., in press). For example, depression is typically characterized by high levels of neuroticism and introversion. Indeed, DSM-5 initially planned to include a system of personality dimensions similar to the Big Five in the main text of the manual (Krueger et al., 2007), but this dimensional system was eventually placed in a secondary section of the manual dedicated to future research. Yet many psychologists and psychiatrists have resisted a dimensional model, perhaps because they, like the rest of us, are cognitive misers (see Chapter 2); they strive to simplify the world. Most of us find it easier to think of the world in terms of simple black or white categories than complex shades of grey (Lilienfeld & Waldman, 2004; Macrae & Bodenhausen, 2000).
A particular concern voiced regarding DSM-5 is its tendency to “medicalize normality,” that is, to classify relatively mild psychological disturbances as pathological (Frances & Widiger, 2012). For example, in a sharp break from previous versions of the DSM, DSM-5 now allows individuals to be diagnosed with major depressive disorder following the loss of a loved one (assuming they meet the pertinent DSM-5 criteria), including the death of a spouse. Although this change may be justified by research (Pies, 2012), critics worry that it will open the floodgates to diagnosing many people with relatively normal grief reactions as disordered (Wakefield & First, 2012).

Like virtually all documents crafted by human beings, DSM-5 is vulnerable to political influences (Kirk & Kutchins, 1992). For example, some researchers have lobbied successfully for the inclusion of their “favourite” disorder or area of specialty. Several critics have viewed the business of psychopathology as driven by the pharmaceutical industry (rather than by scientific theory and evidence). Arguably, those in control of drug treatments will have a considerable stake in both the cataloguing and description (even prescription) of psychological disorders. But like all scientific endeavours, the system of psychiatric classification tends to be self-correcting. Just as homosexuality was stricken from the DSM in the 1970s, science will continue to weed out invalid disorders, ensuring that future editions of the DSM will be based on better evidence.

NORMALITY AND ABNORMALITY: A SPECTRUM OF SEVERITY. As you read case histories or descriptions in this chapter, you may wonder, “Is my behaviour abnormal?” or “Maybe my problems are more serious than I thought.” At times like this, it’s useful to be aware of medical students’ syndrome (Howes & Salkovskis, 1998). As medical students first become familiar with the symptoms of specific diseases, they often begin to focus on their bodily processes. Soon they find it hard to stop wondering whether a slight twinge in their chest might be an early warning of heart trouble or a mild headache the first sign of a brain tumour. Similarly, as we learn about psychological disorders, it’s only natural to “see ourselves” in some patterns of behaviour, largely because in meeting the complex demands of daily life we all experience disturbing impulses, thoughts, and fears from time to time. So don’t become alarmed as you learn about these conditions, as many are probably extremes of psychological difficulties we all experience on occasion.

Answers are located at the end of the text.

ONLINE TESTS FOR MENTAL DISORDERS

We’re all familiar with medical websites that allow us to type in our symptoms (“itchy throat, headache, no fever”) and receive a free, instant diagnosis of our physical ailments. But did you know that similar sites exist for the diagnosis of mental illnesses, including attention-deficit/ hyperactivity disorder? Let’s evaluate some claims and statements, which are modelled after information on actual sites devoted to the self-diagnosis of adult ADHD.

“This 20-question self-test is the most valid and reliable screening measure for adult ADHD available on the Internet!”

Be suspicious of claims like “most valid and reliable” put forward with no supportive evidence. Because information on the Internet is vast and constantly subject to change, it’s difficult to evaluate this extraordinary claim. Moreover, most online diagnostic tests have never been evaluated in peer reviewed studies.

“Keep in mind that this is a screening test. Remember, it’s only the first step in arriving at an accurate diagnosis of ADHD.”

This statement rightly cautions against arriving at an ADHD diagnosis based on information contained in a brief questionnaire. It’s often necessary to consider historical information and current behaviours and performance in different settings (such as school, workplace, and home), as well as tests of attention and input from different professionals (such as physicians, teachers, and family members).

“High scores on the self-test may result from depression, anxiety, and bipolar disorder, so it’s important to rule out these conditions before a diagnosis of ADHD can be made with confidence.”

The site states accurately that ADHD symptoms often overlap with those of other disorders, and that it may be challenging to distinguish the symptoms of ADHD, anxiety, depression, and bipolar disorder.
But at some point in your life, you may experience a psychological problem that’s so disturbing and persistent that you’ll want to talk about it. If so, you’ll probably find it worthwhile to consult with a family member, friend, physician, dormitory counsellor, clergy person, or mental health professional, such as a social worker, psychologist, or psychiatrist. In Chapter 16, we’ll present some tips for what to look for and avoid in a psychotherapist.

MENTAL ILLNESS AND THE LAW: A CONTROVERSIAL INTERFACE. Psychological problems not only affect our mental functioning; they can place us at risk for legal problems. There are few topics about which the general public is certain it knows more, yet actually knows less, than the interface between mental illness and the law.

Mental Illness and Violence. One of the most pervasive myths in psychology is that people with mental illness are at greatly heightened risk for violence (Link et al., 1999). In fact, the overwhelming majority of people with schizophrenia and other psychotic disorders aren’t physically aggressive toward others (Friedman, 2006; Steadman et al., 1998; Teplin, 1985). One might have hoped that the seemingly endless parade of “true crime” shows on television would have helped to combat this misconception, but it’s probably done the opposite. Although only a few percent of people with mental disorders commit aggressive acts, about 75 percent of televised characters with mental illness are violent (Wahl, 1997).

THE INSANITY DEFENCE: FREE WILL VERSUS DETERMINISM

In courts of law, mental illnesses and the law occasionally collide head-on, often with unpredictable consequences. The best-known example of this clash is the insanity defence, which is premised on the idea that we shouldn’t hold people legally responsible for their crimes if they weren’t of “sound mind” when they committed them. The insanity defence comes in many forms, which differ somewhat between Canada and the United States. In fact, four U.S. states—Utah, Montana, Idaho, and Kansas—have opted out of it. The two nations also differ on the outcome should the plaintiff be found incompetent to stand trial due to mental incapacity: Most of the U.S. states offer the plaintiff a full acquittal, whereas in Canada a court will withhold the acquittal but hold the individual criminally responsible.

Most contemporary forms of this defence are based loosely on the M’Naghten rule, formulated during an 1843 British trial. This rule requires that to be declared insane, persons must either have (1) not known what they were doing at the time of the crime or (2) not known that what they were doing was wrong (Melton et al., 1997). A defendant (accused person) who was so disoriented during an epileptic seizure that he didn’t realize he was attacking a police officer might fulfill the first prong of M’Naghten; a defendant who believed he was actually murdering Adolf Hitler when he shot his next-door neighbour might fulfill the second. Several other versions of the insanity defence strive to determine whether defendants were incapable of controlling their impulses at the moment of the crime. In April 2012, Peter Lefebvre from Stittsville, Ontario, severely beat his wife before taking his own life. Although the couple was undergoing a separation, Lefebvre’s mental state (particularly anxiety and depression) was suggested as a possible cause for his erratic behaviour. Because this judgment is exceedingly difficult (how can we know whether a man who murdered his wife in the heat of overwhelming anger could have controlled his temper had he really tried?), some courts ignore it.

The insanity defence is controversial, to put it mildly. To its proponents, this defence is necessary for defendants whose mental state is so deranged that it impairs their freedom to decide whether to commit a crime (Sadoff, 1992; Stone, 1982). To its critics, this defence is nothing more than a legal cop-out that excuses criminals of responsibility (Lykken, 1982; Szasz, 1991). These divergent perspectives reflect a more deep-seated disagreement about free will versus determinism (see Chapter 01). The legal system assumes that our actions are freely chosen, whereas scientific psychology assumes that our actions are completely determined by prior variables, including our genetic makeup and learning history. So lawyers and judges tend to view the insanity defence as a needed exception for the small minority of defendants...
who lack free will. In contrast, many psychologists view this defence as illogical, because they see all crimes, including those committed by people with severe mental disorders, as equally “determined.”

There are numerous misconceptions regarding the insanity verdict (see **TABLE 15.3**). For example, although most people believe that a sizeable proportion, perhaps 15 to 20 percent, of criminals are acquitted (found innocent) on the basis of the insanity verdict, the actual percentage is less than 1 percent (Silver, Cirincione, & Steadman, 1994). This erroneous belief probably stems from the availability heuristic (see Chapter 2): Because we hear a great deal about a few widely publicized cases of defendants acquitted on the grounds of insanity, we overestimate this verdict’s prevalence (Butler, 2006).

**TABLE 15.3** Popular Misconceptions Regarding the Insanity Defence.

<table>
<thead>
<tr>
<th>MYTH</th>
<th>REALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insanity is a psychological or psychiatric term.</td>
<td>Insanity is a purely legal term that refers only to whether the person was responsible for the crime, not to the nature of his or her psychiatric disorder.</td>
</tr>
<tr>
<td>The determination of insanity rests on a careful evaluation of the person’s current mental state.</td>
<td>The determination of insanity rests on a determination of the person’s mental state at the time of the crime.</td>
</tr>
<tr>
<td>The insanity defence requires a judgment of the defendant’s incompetence to stand trial.</td>
<td>Competence to stand trial bears on the defendant’s ability to assist in his or her own defence.</td>
</tr>
<tr>
<td>A large proportion of criminals escape criminal responsibility by using the insanity defence.</td>
<td>The insanity defence is raised in only about 1 percent of criminal trials and is successful only about one-fourth of the time.</td>
</tr>
<tr>
<td>Most people acquitted on the basis of an insanity defence quickly go free.</td>
<td>The average insanity acquittee spends close to three years in a psychiatric hospital, often longer than the length of a criminal sentence for the same crime.</td>
</tr>
<tr>
<td>Insanity defences are complicated and frequently fool juries as a result.</td>
<td>Most successful insanity verdicts are delivered by judges, not juries.</td>
</tr>
<tr>
<td>Most people who use the insanity defence are faking mental illness.</td>
<td>The rate of faking mental illness among insanity defendants appears to be low.</td>
</tr>
</tbody>
</table>

(Source: Butler, 2006; Grisso, 2003; McCutcheon & McCutcheon, 1994; Pasewark & Pantele, 1979; Phillips, Wolf, & Coons, 1988; Silver et al., 1994)

Still, like many misconceptions, this one contains a kernel of truth. Although most people with mental illness aren’t at increased risk for violence, a subset—especially those who are convinced that they’re being persecuted (by the government, for example) and those with substance abuse problems—are (Douglas, Guy, & Hart, 2009; Monahan, 1992; Steadman et al., 1998).

**Involuntary Commitment**. We’re all familiar with criminal commitment, which is just a fancy term for putting someone in jail or prison. Yet society possesses another mechanism for committing individuals against their will. Known as involuntary commitment or civil commitment, it’s a procedure for protecting us from certain people with mental disorders, and protecting them from themselves. Jurisdictions in both Canada and the United States specify that individuals with mental illness can be committed against their will only if they (1) pose a clear and present threat to themselves or others or (2) are so psychologically impaired that they can’t care for themselves (Appelbaum, 1997; Werth, 2001). Although psychiatrists (but not psychologists) can recommend involuntary commitment to a hospital, only a judge can formally approve it following a hearing. Nevertheless, in most states, two psychiatrists or other physicians can place an emergency

In March 2009, Vince Li pleaded not guilty by reason of insanity for the beheading of a fellow passenger on a Winnipeg-bound Greyhound bus. His lawyers argued that Li was not criminally responsible for his behaviour due to mental illness.

Many anxiety disorders, including phobias, frequently have an initial onset in childhood.

**involuntary commitment** procedure of placing some people with mental illnesses in a psychiatric hospital or other facility based on their potential danger to themselves or others, or their inability to care for themselves.
“hold” on patients to hospitalize them involuntarily for a brief period of time, typically three days. When that period expires, the patient is legally entitled to a judicial hearing.

Involuntary commitment raises difficult ethical questions. Advocates of this procedure contend that the government has the right to assume the role of “parent” over individuals with mental illness who are dangerous and don’t possess sufficient insight to appreciate the impact of their actions (Chodoff, 1976; Satel, 1999). In contrast, critics argue that by involuntarily institutionalizing people who haven’t committed crimes, the government is depriving them of their civil liberties (Schaler, 2004; Szasz, 1978). Critics of involuntary commitment also point to research demonstrating that mental health professionals typically do a poor job of forecasting violence (Monahan, 1992), often predicting that patients will commit violence when they won’t. Research conducted in the United States suggests that African-American psychiatric patients are especially likely to be misclassified as potentially violent (Garb, 1998). Still, mental health professionals can predict violence at better-than-chance levels, especially when patients have very recently engaged in, or are immediately threatening, violence (Kramer, Wolbransky, & Heilbrun, 2007; Lidz, Mulvey, & Gardner, 1993; Monahan et al., 2000).

### Assess Your Knowledge

**FACT OR FICTION?**

1. According to a family resemblance view, no one criterion distinguishes mental disorder from normality.  
   **True / False**

2. Once the medical model began to take hold in the Renaissance, treatments for mental disorders came to be based on strong scientific evidence.  
   **True / False**

3. Almost all deinstitutionalized patients returned successfully to their families and communities.  
   **True / False**

4. Some mental disorders appear to be present in most, if not all, cultures.  
   **True / False**

5. Virtually all psychiatric diagnoses are unreliable.  
   **True / False**

6. Most individuals with severe mental illness are not prone to violence.  
   **True / False**

**Answers:**

1. T (p. 600)  
2. F (p. 600)  
3. F (p. 601)  
4. T (p. 603)  
5. F (p. 603)  
6. T (p. 608)

### Anxiety-Related Disorders: The Many Faces of Worry and Fear

**LO 15.4** Describe the many ways people experience anxiety.

We’ll begin our tour of psychological disorders with problems stemming from anxiety. Fortunately, most everyday anxieties generally don’t last long or feel especially uncomfortable. Anxiety in small doses can even be adaptive. It can permit a lightning-quick response to danger, steer us away from harmful behaviours, and inspire us to solve festering problems. Yet sometimes anxiety spirals out of control, becoming excessive and inappropriate. It may even feel life-threatening (Mendelowicz & Stein, 2000).

Anxiety disorders are among the most prevalent of all mental disorders; 31 percent of us will meet the diagnostic criteria for one or more such disorders at some point in our lives (Kessler et al., 2007). The average age of onset for anxiety disorders (11 years) is earlier than for most other disorders, including substance use disorders (20 years) and mood disorders (30 years; Kessler et al., 2005). **TABLE 15.4** displays the lifetime prevalence of anxiety disorders, along with many other disorders we’ll consider in this chapter.

Yet anxiety isn’t limited to anxiety disorders. Anxiety can seep into numerous aspects of our functioning, including our perceived physical health. In a controversial condition
called **somatic symptom disorder**, new to DSM-5, anxieties about physical symptoms—that are either medically verified or purely psychological in origin—can become so intense and "over the top" that they interfere with daily living. In cases of **illness anxiety disorder** (another new diagnosis that is similar to what was previously called hypochondriasis), people become so preoccupied with the idea that they're suffering from a serious undiagnosed illness, that no amount of reassurance can relieve their anxiety. Much like radar operators who stay on their toes for signs of incoming enemy planes, people with illness anxiety disorder seem continually on the alert for signs of physical illness, constantly checking the Internet, for example, for information about symptoms and signs of diseases. Despite repeated medical reassurance and physical examinations, they may insist that their mild aches, pains, and twinges are signs of serious diseases, like cancer, AIDS, or heart disease.

### Generalized Anxiety Disorder: Perpetual Worry

We all get caught up with worry from time to time. Yet for the 3 percent of us who have **generalized anxiety disorder (GAD)**, worry is a way of life. People with GAD spend an average of 60 percent of each day worrying, compared with 18 percent for the rest of the general population (Craske et al., 1989). Many describe themselves as “worrywarts.” They tend to think anxious thoughts, feel irritable and on edge, have trouble sleeping, and experience considerable bodily tension and fatigue (Barlow, Chorpita, & Turovsky, 1996; Wittchen, 2002). Often they worry too much about the small things in life, like an upcoming meeting at work or social event. One-third of those with GAD develop it following a major stressful event—like a wedding, illness, physical abuse, or death of a relative—or as the result of lifestyle changes, such as completing school and embarking on a career (Hazlett-Stevens, Pruitt, & Collins, 2008; Mellingier & Lynn, 2003). People with GAD are more likely to be female than male—as is the case with most anxiety disorders—as well as middle aged, widowed or divorced, poor, and prone to “self-medication” with alcohol and drugs to relieve symptoms (Grant et al., 2005; Noyes, 2001). U.S. data also show that Asians, Hispanics, and African Americans are at relatively low risk for GAD (Grant et al., 2005).

GAD may be the core anxiety disorder out of which all others develop (Barlow, 2002). Indeed, people with GAD often experience other anxiety disorders, including phobias and panic disorder, which we’ll consider next.

### Panic Disorder: Terror That Comes out of the Blue

The Greek god Pan was a mischievous spirit who popped out of the bushes to scare the living daylight out of travellers. Pan lent his name to **panic attacks**, which occur when nervous feelings gather momentum and escalate into intense bouts of fear, even terror. Panic attacks can occur only rarely, or on a daily basis, for weeks, months, or even years at a time. People are diagnosed with **panic disorder** when they experience panic attacks that are repeated and unexpected, and when they either experience persistent concerns about panicking or change their behaviour (for example, change jobs) as a result of the attacks (APA, 2013).

Panic attacks peak in less than ten minutes and can include sweating, dizziness, light-headedness, a racing or pounding heart, shortness of breath, feelings of unreality, and fears of going crazy or dying. Because many patients experiencing their initial panic attacks believe that they’re having heart attacks, many first go to emergency rooms, only to be sent home and—like Ida, whom we met at the outset of the chapter—told “it’s all in your head.” Some panic attacks are associated with specific situations, such as riding in elevators or shopping in supermarkets, but others come entirely out of the blue—that is, without warning, often generating fears of the situations in which they occur.

Panic attacks can occur in every anxiety disorder, as well as in mood and eating disorders. Even high-functioning people can experience panic attacks in anticipation of stressful events (Cox & Taylor, 1998): About 20 to 25 percent of college and university students report at least one panic attack in a one-year period, with about half that number reporting unexpected attacks (Lilienfeld, 1997). Panic disorder often develops in early adulthood (Kessler et al., 2005), and is associated with a history of fears of separation from a parent during childhood (Lewinsohn et al., 2008).
FICTOID

MYTH: Most people with agoraphobia are housebound.
REALITY: Being unable or afraid to leave the house occurs only in severe cases of agoraphobia.

Some of the most common fears involve insects and animals, such as spiders and snakes.

First Person Account:
SOCIAL ANXIETY DISORDER.

“When I would walk into a room full of people, I’d turn red and it would feel like everybody’s eyes were on me. I was embarrassed to stand off in a corner by myself, but I couldn’t think of anything to say to anybody. It was humiliating. I felt so clumsy, I couldn’t wait to get out.”

(Dickey, 1994).

Phobias: Irrational Fears

A phobia is an intense fear of an object or situation that’s greatly out of proportion to its actual threat. Many of us have mild fears—of things like spiders and snakes—that aren’t severe enough to be phobias. For a fear to be diagnosed as a phobia, it must restrict our lives, create considerable distress, or both.

Phobias are the most common of all anxiety disorders. One in nine of us has a phobia of an animal, blood or injury, or a situation like a thunderstorm. Social fears are just as common (Kessler et al., 1994). Agoraphobia, which we’ll examine next, is the most debilitating of the phobias, and occurs in about 1 in 20 of us (Keller & Craske, 2008; Kessler et al., 2006).

AGORAPHOBIA. Some 2700 years ago in the city-states of ancient Greece, agoraphobia acquired its name as a condition in which certain fearful citizens couldn’t pass through the central city’s open-air markets (agoras). A common misconception is that agoraphobia is a fear of crowds or public places. But agoraphobia actually refers to a fear of being in a place or situation in which escape is difficult or embarrassing, or in which help is unavailable in the event of a panic attack (APA, 2013).

Agoraphobia typically emerges in the mid-teens and is usually a direct outgrowth of panic disorder. In fact, most people with panic disorder develop agoraphobia (Cox & Taylor, 1998) and become apprehensive in a host of settings, such as malls, crowded movie theatres, tunnels, bridges, or wide-open spaces. The manifestation of agoraphobia seems to differ across cultures. For example, some First Nations people in Greenland suffer from a condition called “kayak angst,” marked by a pronounced fear of going out to sea by oneself in a kayak (Barlow, 2000; Gusow, 1963).

In some cases, agoraphobia reaches extreme proportions. Two clinicians saw a 62-year-old woman with agoraphobia who hadn’t left her house—even once—for 25 years (Jensvold & Turner, 1988). Having experienced severe panic attacks and terrified by the prospect of still more, she spent almost all of her waking hours locked away in her bedroom, with curtains drawn. The therapists attempted to treat her agoraphobia by encouraging her to take short trips out of her house, but she repeatedly refused to walk even a few steps past her front door.

SPECIFIC PHOBIA AND SOCIAL ANXIETY DISORDER. Phobias of objects, places, or situations—called specific phobias—commonly arise in response to animals, insects, thunderstorms, water, elevators, and darkness. Many of these fears, especially of animals, are widespread in childhood but disappear with age (APA, 2013).

Surveys show that most people rank public speaking as a greater fear than dying (Wallechinsky, Wallace, & Wallace, 1977). Given that statistic, imagine how people with social anxiety disorder, formerly called social phobia in DSM-IV, must feel. They experience a marked fear of public appearances in which embarrassment or humiliation seems likely, such as speaking or performing in public or, more rarely, swimming, swallowing, or signing their cheques in public (Mellinger & Lynn, 2003). Their anxiety goes well beyond the stage fright that most of us feel occasionally (Heimberg & Juster, 1995).

Posttraumatic Stress Disorder: The Enduring Effects of Experiencing Horror

One of the most significant changes in DSM-5 is that posttraumatic stress disorder and obsessive-compulsive disorders, which were formerly included in the category of anxiety disorders, now are positioned in their own separate diagnostic categories. Yet because both disorders are associated with significant anxiety, we consider them under the heading of anxiety-related disorders. When people experience or witness a traumatic event, such as front-line combat, an earthquake, or sexual assault, they may develop posttraumatic stress disorder (PTSD) (see Chapter 12). In the DSM-5, PTSD is in a new class of “trauma and stressor-related disorders” in which the definition of a traumatic event is broad. It includes direct exposure to a traumatic event, such as a rape, wartime combat, or a natural disaster. Nevertheless, it also includes situations in which people learn about an event from a friend or
relative who experienced threatened or actual death or in which people are exposed repeatedly to distressing details of a traumatic event, such as the sexual abuse of an elderly person.

We learned in Chapter 12 that flashbacks are among the hallmarks of PTSD. The terror of war can return decades after the original trauma and be reactivated by everyday stressful experiences (Foa & Kozak, 1986). Canadian Lieutenant-General Romeo Dallaire was a United Nations peacekeeper who between 1993 and 1994 was responsible for curbing Rwanda’s genocide. He commented:

Rwanda will never ever leave me. It’s in the pores of my body. My soul is in those hills, my spirit is with the spirits of all those people who were slaughtered and killed that I know of, and many that I didn’t know…. Fifty to sixty thousand people walking in the rain and the mud to escape being killed, and seeing a person there beside the road dying. We saw lots of them dying. And lots of those eyes still haunt me, angry eyes or innocent eyes, no laughing eyes. But the worst eyes that haunt me are the eyes of those people who were totally bewildered.

Other symptoms of PTSD include efforts to avoid thoughts, feelings, places, and conversations associated with the trauma; recurrent dreams of the trauma; and increased arousal, such as sleep difficulties and startling easily (APA, 2013). Reminders of the incident can trigger full-blown panic attacks, as in the case of a Vietnam veteran who hid under his bed whenever he heard a helicopter in the distance—over 20 years after the war ended (Baum, Cohen, & Hall, 1993; Foa & Rothbaum, 1998; Jones & Barlow, 1990). PTSD isn’t easy to diagnose. Some of its symptoms, such as anxiety and difficulty sleeping, may have been present before the stressful event. Moreover, some people malingering (fake) PTSD to obtain government benefits, so diagnosticians must rule out this possibility (Rosen, 2006).

■ Obsessive-Compulsive and Related Disorders: Trapped in One’s Thoughts and Behaviours

Just about all of us have had a thought or even a silly song jingle that we just couldn’t get out of our heads. Patients with obsessive-compulsive disorder (OCD) know all too well what this experience is like, except that their symptoms are much more severe. Like Bill, whom we’ll recall from the opening of the chapter, they typically suffer from obsessions: persistent ideas, thoughts, or impulses that are unwanted and inappropriate and cause marked distress. Unlike typical worries, obsessions aren’t extreme responses to everyday stressors. They usually centre on “unacceptable” thoughts about such topics as contamination, sex, aggression, or religion (Franklin & Foa, 2008). For example, individuals with OCD may be consumed with fears of being dirty or thoughts of killing others. Unlike ordinary worriers, or people who experience compulsions that aren’t especially disturbing to them—such as checking repeatedly to see whether a door is locked—people with OCD typically are disturbed by their thoughts and usually see them as irrational or nonsensical (Fullana et al., 2009). They often label themselves “crazy” or dangerous. Despite their best efforts, people with OCD can’t find a way to make these thoughts stop.

Most OCD patients also experience symptoms linked closely to obsessions—namely, compulsions: repetitive behaviours or mental acts that they undertake to reduce or prevent distress, or relieve shame and guilt (Abramowitz, Taylor, & McKay, 2009). In most cases, patients feel driven to perform the action that accompanies an obsession, prevent some dreaded event, or “make things right.” A patient treated by one of your text’s authors awoke early each morning to wash the hood of his car until it was spotless, and felt compelled to repeat this ritual as soon as he arrived home at the end of the workday. Common OCD rituals include

- Repeatedly checking door locks, windows, electronic controls, and ovens
- Performing tasks in set ways, like putting on one’s shoes in a fixed pattern

First Person Account:

**OBSESSIVE-COMPULSIVE DISORDER.**

“I couldn’t do anything without rituals. They transcended every aspect of my life. Counting was big for me. When I set my alarm at night, I had to set it to a number that wouldn’t add up to a ‘bad’ number. I would wash my hair three times as opposed to once because three was a good-luck number and one wasn’t. It took me longer to read because I’d count the lines in a paragraph. If I was writing a term paper, I couldn’t have a certain number of words on a line if it added up to a bad number. I was always worried that if I didn’t do something, my parents were going to die.”

(Dickey, 1994).

ruling out rival hypotheses

HAVE IMPORTANT ALTERNATIVE EXPLANATIONS FOR THE FINDINGS BEEN EXCLUDED?

Some dogs suffer from a condition called canine lick acral dermatitis, in which they lick themselves compulsively, resulting in severe skin damage (Derr, 2010). Some scientists believe that this condition may be an animal variant of obsessive-compulsive disorder; interestingly, this condition sometimes responds to the same medications used to treat human obsessive-compulsive disorder (see Chapter 16).

| Watch | Margo: Obsessive-Compulsive Disorder on mypsychlab.com |
| Simulate | The Obsessive-Compulsive Test on mypsychlab.com |

obsessive-compulsive disorder (OCD)

condition marked by repeated and lengthy (at least one hour per day) immersion in obsessions, compulsions, or both

obsession

persistent idea, thought, or impulse that is unwanted and inappropriate, causing marked distress

compulsion

repetitive behaviour or mental act performed to reduce or prevent stress
chapter 15  PSYCHOLOGICAL DISORDERS

- Repeatedly arranging and rearranging objects
- Washing and cleaning repeatedly and unnecessarily
- Counting the number of dots on a wall or touching or tapping objects

Many people without OCD occasionally engage in one or more of these activities (Mataix-Cols, Rosario-Campos, & Lackman, 2005). Nevertheless, by definition, individuals diagnosed with OCD spend at least an hour a day immersed in obsessions, compulsions, or both; one patient spent 15 to 18 hours per day washing his hands, showering, getting dressed, and cleaning money. Still, many individuals with OCD lead remarkably successful lives. Howard Hughes, the billionaire industrialist, struggled for years with severe, untreated symptoms of OCD. As his disorder progressed, he became so obsessed with possible contamination by germs that he refused to leave the expensive hotel rooms in which he lived. He refused to shake hands with anyone and instructed his servants to engage in elaborate rituals concerning food and utensils. For example, he insisted that they wrap the handle of a spoon in tissue paper and seal it with tape. Yet Hughes rarely bathed or brushed his teeth, and he cut his hair and fingernails only once a year (Bartlett & Steele, 2004; Brownstein & Solyom, 1986). More recently, celebrities like Cameron Diaz, Billy Bob Thornton, and David Beckham have spoken publicly about their struggles with the disorder.

Explanations for Anxiety-Related Disorders: The Roots of Pathological Anxiety, Fear, and Repetitive Thoughts and Behaviours

How do anxiety disorders arise? Differing theories propose explanations focusing on the environment, catastrophic thinking, and biological influences.

LEARNING MODELS OF ANXIETY: ANXIOUS RESPONSES AS ACQUIRED HABITS. According to learning theories, fears are—you guessed it—learned. John B. Watson and Rosalie Rayner’s (1920) famous demonstration of classical conditioning of the fear of a small furry animal (remember poor little Albert from Chapter 6?) powerfully conveys how people learn fears.

Operant conditioning, which relies on reinforcements and punishments (see Chapter 6), offers another account of how fears are maintained. If a socially awkward girl repeatedly experiences rejection when she asks boys to go to movies, she may become shy around them. If this pattern of rejection continues, she could develop a full-blown social anxiety disorder. Paradoxically, her avoidance of boys provides negative reinforcement, because it allows her to escape the unpleasant consequences of social interaction. This sense of relief perpetuates her avoidance, and ultimately her anxiety.

Learning theorists (Rachman, 1977) believe that fears can arise in two additional ways. First, we can acquire fears by observing others engage in fearful behaviours (Mineka & Cook, 1993). A father’s fear of dogs might instill the same in his child. Second, fears can stem from information or misinformation from others. If a mother tells her children that riding in elevators is dangerous, they may end up taking the stairs.

CATASTROPHIZING AND ANXIETY SENSITIVITY. People with social phobias predict that many social encounters will be interpersonal disasters, and some people with fears of lightning are so fearful that they seek the shelter of a basement when mild thunderstorms are detected on radar 80 kilometres (50 miles) away (Voncken, Bogels, & de Vries, 2003). As these examples illustrate, catastrophizing is a core feature of anxious thinking (Beck, 1976; Ellis, 1962; Ellis & Dryden, 1997). People catastrophize when they predict terrible events—such as contracting a life-threatening illness from turning a doorknob—despite their low probability (A. T. Beck, 1964; J. Beck, 1995).

People with anxiety disorders tend to interpret ambiguous situations in a negative light (Matthews & MacLeod, 2005; see TABLE 15.5). Many people with anxiety disorders harbour high levels of anxiety sensitivity, a fear of anxiety-related sensations (Reiss & McNally, 1985; Stein, Jang, & Livesley, 1999). Think of the times you felt a bit dizzy when you stood up quickly or your heart raced after climbing a flight of stairs. You probably dismissed these physical symptoms as harmless. Yet people with high anxiety sensitivity...
Describe how life events can interact with characteristics of the individual to produce mood disorders and suicide.

**LO 15.5** Identify the characteristics of different mood disorders.

**LO 15.6** Describe how life events can interact with characteristics of the individual to produce depression symptoms.

**LO 15.7** Identify common myths and misconceptions about suicide.

**ANXIETY: BIOLOGICAL INFLUENCES.** Twin studies show that many anxiety disorders, including panic disorder, phobias, PTSD, and OCD, are genetically influenced (Andrews et al., 1990; Roy et al., 1995; Van Grootheest et al., 2007). In particular, genes influence people's levels of neuroticism—a tendency to be high strung and irritable (see Chapter 14)—which can set the stage for excessive worry (Anderson, Taylor, & McLean, 1996; Zinbarg & Barlow, 1996). On a genetic basis, people who experience GAD are virtually indistinguishable from those who experience major depression, which is also associated with elevated neuroticism (Kendler & Karkowski-Shuman, 1997). This finding suggests a shared genetic pathway for these disorders.

Studies that have attempted to identify a single gene associated with OCD have provided mixed results at best, although evidence is accumulating that genes that transport serotonin and glutamate probably play some role in the development of OCD (Samuels et al., 2011; see Chapter 3). Much like a car that's stuck in gear, people with OCD experience problems with shifting thoughts and behaviours (Schwartz & Bayette, 1996). Brain scans reveal abnormalities in white matter and increased activity in portions of the frontal lobes where information is filtered, prioritized, and organized (Zohar et al., 2012). Under these circumstances, people can't seem to get troubling thoughts out of their minds or inhibit repeated rituals.

Some children with OCD and related anxiety disorders also meet criteria for Tourette's disorder, a condition marked by motor tics, like twitching and facial grimacing, and vocal tics, like grunting or throat clearing (although Tourette's disorder sometimes occurs without OCD). This finding has led some researchers to suggest that OCD and Tourette's disorder share biological roots (Mell, Davis, & Owens, 2005). A number of children develop OCD or Tourette's disorder after experiencing strep throat or scarlet fever infections caused by streptococcal (strep) bacteria. Scientists are seeking to determine whether strep triggers an immune system response that affects the brain and brings about OCD symptoms, or whether the relationship between strep and OCD symptoms is coincidental (Gause et al., 2009; Kurlan & Kaplan, 2004). Another possibility is that children with strep feel irritable and uncomfortable, which worsens OCD symptoms.

**TABLE 15.5 Anxiety and Interpretation of Ambiguity.** Anxiety leads us to interpret ambiguous stimuli negatively. Researchers have asked anxious and nonanxious subjects to listen to homophones—words that sound the same but have two different meanings and spellings—and to write down the word they heard. In these studies, they've used homophone pairs in which one meaning (and spelling) is threatening and the other is nonthreatening. Compared with nonanxious subjects, anxious subjects are more likely to write down the version of the homophone that is threatening, such as “bury” as opposed to “berry” (Blanchette & Richards, 2003; Mathews, Richards, & Eysenck, 1989).

<table>
<thead>
<tr>
<th>SELECTED HOMOPHONES</th>
<th>THREATENING MEANING/SPELLING</th>
<th>NONTHREATENING MEANING/SPELLING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bury</td>
<td>Berry</td>
<td>Dye</td>
</tr>
<tr>
<td>Die</td>
<td>Berry</td>
<td>Patience</td>
</tr>
<tr>
<td>Patients</td>
<td>Brews</td>
<td>Flu</td>
</tr>
<tr>
<td>Bruise</td>
<td>Brews</td>
<td>Sword</td>
</tr>
<tr>
<td>Flu</td>
<td>Soared</td>
<td>Boar</td>
</tr>
</tbody>
</table>

**MYTH:** Most individuals with Tourette's disorder have coprolalia.

**REALITY:** Seventy percent or more of patients with Tourette's disorder do not curse (Goldenberg, Brown, & Weiner, 1994).

**MOOD DISORDERS AND SUICIDE**

Campers participate in a team-building exercise during a summer camp for children with Tourette’s disorder, a syndrome marked by motor and vocal tics.
Imagine that we’re therapists interviewing someone who’s come to us for help. As the client begins to talk about his life, it becomes clear that even the simplest activities, like dressing or driving to work, have become enormous acts of will. He reports difficulty sleeping and unaccountably wakes up before dawn each day. He refuses to answer the telephone. He lies listlessly for hours staring at the television set. His mood is downcast, and occasionally tears well up in his eyes. He’s recently lost a fair amount of weight. His world is grey, a void. Toward the end of the interview, he tells us that he’s begun to contemplate suicide.

We’ve just interviewed a person with a mood disorder, so called because his difficulties centre on his bleak mood, which colours all aspects of his existence. When we check his symptoms we can see that he meets the criteria for a major depressive episode. We’ll soon encounter another mood disorder, bipolar disorder, in which people’s mood is often the mirror image of depression. TABLE 15.6 outlines the range of mood disorders in DSM-5. Consider these famous Canadians: Prime Minister Sir John A. Macdonald, actress Margot Kidder, Olympic silver medallist in figure skating Elizabeth Manley, comedian Jim Carrey, poet Leonard Cohen, ballerina Karen Kain, singer Alys Robi, musician Glenn Gould, and Prime Minister Pierre Elliot Trudeau. They have all at one point suffered from mood disorders.

**TABLE 15.6 Mood Disorders and Conditions.**

<table>
<thead>
<tr>
<th>DISORDER/CONDITION</th>
<th>SYMPTOMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depressive disorder</td>
<td>Chronic or recurrent state in which a person experiences a lingering depressed mood or diminished interest in pleasurable activities, along with symptoms that include weight loss and sleep difficulties</td>
</tr>
<tr>
<td>Manic episode</td>
<td>Markedly inflated self-esteem or grandiosity, greatly decreased need for sleep, much more talkative than usual, racing thoughts, distractibility, increased activity level or agitation, and excessive involvement in pleasurable activities that can cause problems (like unprotected sex, excessive spending, reckless driving)</td>
</tr>
<tr>
<td>Bipolar disorder I</td>
<td>Presence of one or more manic episodes</td>
</tr>
<tr>
<td>Persistent depressive disorder (dysthymia)</td>
<td>Low-level depression of at least two years’ duration; feelings of inadequacy, sadness, low energy, poor appetite, decreased pleasure and productivity, and hopelessness</td>
</tr>
<tr>
<td>Hypomanic episode</td>
<td>A less intense and disruptive version of a manic episode; feelings of elation, grouchiness or irritability, distractibility, and talkativeness</td>
</tr>
<tr>
<td>Bipolar disorder II</td>
<td>Patients must experience at least one episode of major depression and one hypomanic episode</td>
</tr>
<tr>
<td>Cyclothymic disorder</td>
<td>Moods alternate between numerous periods of hypomanic symptoms and numerous periods of depressive symptoms. Cyclothymia increases the risk of developing bipolar disorder.</td>
</tr>
<tr>
<td>Postpartum depression</td>
<td>A depressive episode that occurs within a month after childbirth in up to 15 percent of women. A much more serious condition, postpartum psychosis, occurs in about one or two per 1000 childbirths, with psychotic symptoms, including command hallucinations to kill the infant or delusions that the infant is possessed by an evil spirit (Beck &amp; Gable, 2001).</td>
</tr>
<tr>
<td>Seasonal affective disorder</td>
<td>Depressive episodes that display a seasonal pattern, most commonly beginning in fall or winter and improving in spring</td>
</tr>
<tr>
<td>Disruptive mood dysregulation disorder</td>
<td>For children under 18 years; they experience persistent irritability and frequent episodes of extreme, out-of-control behaviour</td>
</tr>
<tr>
<td>Premenstrual dysphoric disorder</td>
<td>Occurs in women during the final week before the onset of menses, with marked mood swings, irritability, anger, and anxiety</td>
</tr>
</tbody>
</table>

(Source: Data from DSM-5, APA, 2013; www.dsm5.org)
Major Depressive Disorder: Common, But Not the Common Cold

Over the course of a lifetime, more than 20 percent of us will experience a mood disorder. Major depression alone darkens the lives of more than 16 percent of North Americans (Kessler et al., 2005). Due to its frequency, some have dubbed depression the “common cold” of psychological disorders (Seligman, 1975). Yet we’ll soon see that this description doesn’t begin to capture the profound depths of suffering that people with this condition experience. Depressive disorders can begin at any age, but are most likely to strike people in their thirties. Contrary to popular misconception, they’re less common in elderly adults than in younger people (Kessler et al., 2010; Klerman, 1986).

As we mentioned earlier, women are about twice as likely to experience depression as men. In Chapter 12, we learned that this gender difference may be associated with women’s tendency to ruminate more than men (Nolen-Hoeksema, 2002, 2003). Yet it may also be associated with differences between men and women in economic power, sex hormones, social support, and history of physical or sexual abuse (Howland & Thase, 1998). The sex difference in depression is widespread but not universal. In some cultures, such as certain Mediterranean populations, Orthodox Jews, and the Amish, this sex difference is largely absent (Piccinnelli & Wilkinson, 2000). But researchers don’t know why. One possibility is that the differences in the rates of depression across genders in Western cultures reflect an underdiagnosis of depression in men. In Canada and the United States, women are more willing to admit to depression and seek psychological services than men, who are socialized to “act tough” (Kilmartin, 2006).

Over the past century, the rate of diagnosed major depression has gradually crept up, and psychologists have diagnosed depression at earlier ages with succeeding generations (Compton et al., 2006; Klerman et al., 1985). Nevertheless, the earlier onset of depression, which today peaks at 15 to 24 years of age, may be more illusory than real. Older people may have difficulty remembering past depressive episodes, leading scientists to underestimate the prevalence of depression among the elderly (Giuffra & Risch, 1994).

The symptoms of depression may develop gradually over days or weeks; in other cases, they may surface rather suddenly. Depression, like the common cold, is recurrent. The average person with major depression experiences five or six episodes over the course of a lifetime. Most episodes last from six months to a year. But in as many as a fifth of cases, depression is chronic—that is, present for decades with no relief (Ingram, Scott, & Siegle, 1998). Generally, the earlier depression strikes the first time, the more likely it will persist or recur (Coryell et al., 2009). In sharp contrast to the common cold, depression can produce severe impairment. In extreme cases, people may fail to feed or clothe themselves or take care of basic health needs, like brushing their teeth or showering.

Explanations for Major Depressive Disorder: A Tangled Web

The multifaceted phenomenon of depression illustrates the biopsychosocial approach (see Chapter 12), underscoring how multiple factors can combine to produce psychological symptoms. Let’s reconsider the depressed man we imagined interviewing at the beginning of this section. From his severely depressed father and perpetually anxious mother, he may have inherited a tendency to respond to stressful situations with negative emotions (neuroticism). He felt that a competitive colleague tried continually to undermine his authority as a television producer. In response, he felt insecure, and began to second-guess his every decision. Each day he wasted hours ruminating about losing his job. The quality of his work nose-dived. He withdrew socially and began to refuse invitations to go golfing with his buddies. His friends tried to cheer him up, but the black cloud that hung over his head wouldn’t budge. Feeling rebuffed, his friends stopped inviting him to do anything. His once-bright social world became a black void, and he moped around doing virtually nothing. He felt helpless. Eventually, his dark thoughts turned to suicide.

This example highlights a key point. To fully understand depression, we must appreciate the complex interplay of inborn tendencies, stressful events, interpersonal relationships, the loss of reinforcers in everyday life, negative thoughts, and feelings of helplessness (Akiskal & McKinney, 1973; Ilardi & Feldman, 2001).
DEPRESSION AND LIFE EVENTS. Sigmund Freud (1917/1953) suggested that early loss can render us vulnerable to depression later in life. He may have been on to something, because the loss of beloved people or even the threat of loss can trigger depression in adulthood. Stressful life events that represent loss or threat of separation are especially tied to depression (Brugha, 1995; Mazure, 1998; Paykel, 2003). But the loss created by a blow to our sense of self-worth can sting every bit as much as the loss of a close relationship (Finlay-Jones & Brown, 1981). As McGill University's David Zuroff explains, a crucial determinant of whether we'll become depressed is whether we've lost or are about to lose something we value dearly, like someone we love, financial support, or self-esteem (Beck, 1983; Blatt, 1974; Prince et al., 1997; Zuroff, Mongrain, & Santor, 2004). Recall that in DSM-5, people who become depressed following the loss of a loved one can be formally diagnosed with depression, whereas this was not the case in previous editions of the manual.

As we learned in Chapter 12, pessimism and other symptoms of depression can set the stage for negative life circumstances, like getting fired from a job or losing a close relationship (Hammen, 1991; Harkness & Luther, 2001). The causal arrow of this association thus points in both directions. Negative life events set us up to bring us down, but depression can create problems in living.

INTERPERSONAL MODEL: DEPRESSION AS A SOCIAL DISORDER. James Coyne hypothesized that depression creates interpersonal problems (Coyne, 1976; Joiner & Coyne, 1999; Rudolph et al., 2006). When people become depressed, he argued, they seek excessive reassurance, which in turn leads others to dislike and reject them. Coyne (1976) asked undergraduates to talk on the telephone for 20 minutes with patients with depression, patients without depression, or nonpatient women drawn from the community. He didn’t inform students that they’d be interacting with patients with depression. Yet following the interaction, students who spoke with these patients became more depressed, anxious, and hostile than those who interacted with individuals without depression. Moreover, subjects were more rejecting of patients with depression and expressed much less interest in interacting with them in the future. For Coyne, depression is a vicious cycle. People with depression often elicit hostility and rejection from others, which in turn maintains or worsens their depression.

Many, but not all, studies have replicated Coyne’s findings that people with depression seek excessive reassurance and tend to stir up negative feelings in others (Burns et al., 2006; Joiner & Coyne, 1999; Starr & Davila, 2008). Constant worrying, mistrust, fears of rejection and abandonment, and socially inappropriate behaviours can also be a social turnoff to many people (Wei et al., 2005; Zborowski & Garske, 1993).

BEHAVIOURAL MODEL: DEPRESSION AS A LOSS OF REINFORCEMENT. Peter Lewinsohn’s (1974) behavioural model proposes that depression results from a low rate of response-contingent positive reinforcement. Put in simpler terms, when people with depression try different things and receive no payoff for them, they eventually give up. They stop participating in many pleasant activities, affording them little opportunity to obtain reinforcement from others. In time, their personal and social worlds shrink, as depression seeps into virtually every nook and cranny of their lives. Lewinsohn later observed that some people with depression lack social skills (Segrin, 2000; Youngrid & Lewinsohn, 1980), making it harder for them to obtain reinforcement from people they value. To make matters worse, if others respond to individuals with depression with sympathy and concern, they may reinforce and maintain their withdrawal. This view implies a straightforward recipe for breaking the grip of depression: pushing ourselves to engage in pleasant activities. Sometimes merely getting out of bed can be the first step toward conquering depression (Dimidjian et al., 2006).

COGNITIVE MODEL: DEPRESSION AS A DISORDER OF THINKING. In contrast, Aaron Beck’s influential cognitive model of depression holds that depression is caused by negative beliefs and expectations (Beck, 1976, 1987). Beck focused on the cognitive triad, three components of depressed thinking: negative views of oneself, the world, and the future. These habitual thought patterns, called negative schemas, presumably originate in early experiences of loss, failure, and rejection. Activated by stressful events in later life, these schemas reinforce people with depression’s negative experiences (Scher, Ingram, & Segal, 2005).
Depressed persons’ view of the world is bleak because they put a decidedly negative mental spin on their experiences. They also suffer from cognitive distortions, which are skewed ways of thinking. One example is selective abstraction, in which people come to a negative conclusion based on only an isolated aspect of a situation. A man might consistently single out a trivial error he committed in a softball game and blame himself completely for the loss. It’s as though people with depression are wearing glasses that filter out all of life’s positive experiences and bring all of life’s negative experiences into sharper focus.

There’s considerable support for Beck’s idea that people with depression hold negative views of themselves, the future, and the world (Haaga, Dyck, & Ernst, 1991; Ingram, 2003). But the evidence for the role of cognitive distortions in nonhospitalized, or not seriously depressed, individuals isn’t as strong (Haack et al., 1996). In fact, some research suggests that compared with people without depression, individuals with mild depression actually have a more accurate view of circumstances, a phenomenon called depressive realism. In contrast, people who aren’t depressed experience illusionary control over their environments. This surprising conclusion comes from research in which people without depression were more likely than people with depression to believe that they controlled a light bulb when it came on, even though experimenters had rigged when it turned on and off (Alloy & Abramson, 1979, 1988). Thus, people with depression were more realistic in their estimates of personal control, because they had no control over the light bulb. Researchers (Msetfi et al., 2005) replicated this finding, but suggested that depressed individuals experience difficulties attending to and processing information about the light. So rather than being more realistic, people with depression may be less attentive to “reality,” a finding consistent with cognitive models.

Not tuning in to reality may exact serious costs. Janet Kistner and her colleagues (Kistner et al., 2006) studied 9- and 10-year-old children’s perceptions of how their classmates felt about them. The researchers discovered that children with accurate perceptions of their level of social acceptance were less likely to develop depression than those with inaccurate perceptions. Beck’s theory implies that negative schemas shape emotional tone rather than the other way around. Nevertheless, Kistner’s research suggests that depression itself can create a negative bias. As time went on, children who were depressed at the onset of the study became more negatively biased about how much others liked and perceived them. These findings point to a cycle in which inaccurate perceptions lead to depression, and depression leads to inaccurate perceptions.

**LEARNED HELPLESSNESS: DEPRESSION AS A CONSEQUENCE OF UNCONTROLLABLE EVENTS.** Martin Seligman (1975; Seligman & Maier, 1967) accidentally stumbled across an unusual finding related to depression in his work with dogs. He was testing dogs in a shuttle box, depicted in Figure 15.1; one side of the box was electrified and the other side, separated by a barrier, wasn’t. Ordinarily, dogs avoid painful shocks by jumping over the barrier to the nonelectrified side of the box. Yet Seligman found something surprising. Dogs first restrained in a hammock and exposed to shocks they couldn’t escape later often made no attempt to escape shocks in the shuttle box, even when they could easily get away from them. Some of the dogs just sat there, whimpering and crying, passively accepting the shocks as though they were inescapable. They’d learned to become helpless.

Bruce Overmier and Seligman (1967) described learned helplessness as the tendency to feel helpless in the face of events we can’t control and argued that it offers an animal model of depression. Seligman noted striking parallels between the effects of learned helplessness and depressive symptoms: passivity, appetite and weight loss, and difficulty learning that one can change circumstances for the better. But we must be cautious in drawing conclusions from animal studies because many psychological conditions, including depression, may differ in animals and humans (Raulin & Lilienfeld, 2008).
Provocative as it is, Seligman’s model can’t account for all aspects of depression. It doesn’t explain why people with depression make internal attributions (explanations) for failure. In fact, the tendency to assume personal responsibility for failure contradicts the notion that people with depression regard negative events as beyond their control. The original model also doesn’t acknowledge that the mere expectation of uncontrollability isn’t sufficient to induce depression. After all, people don’t become sad when they receive large amounts of money in a lottery, even though they have no control over this event (Abramson, Seligman, & Teasdale, 1978).

When data don’t fit a model, good scientists revise it. Seligman and his colleagues (Abramson et al., 1978) altered the learned helplessness model to account for the attributions people make to explain their worlds. They argued that persons prone to depression attribute failure to internal as opposed to external factors, and success to external as opposed to internal factors. A person with depression might blame a poor test grade on a lack of ability, an internal factor, and a good score on the ease of the exam, an external factor. The researchers also observed that depression-prone persons make attributions that are global and stable: They tend to see their failures as general and fixed aspects of their personalities. Still, internal, global, and stable attributions may be more a consequence than a cause of depression (Harvey & Weary, 1984). The depression brought on by undesirable life events may skew our thinking, leading us to make negative attributions (Beidel & Turner, 1986; Gibb & Alloy, 2006; Ilardi & Craighead, 1994).

Whether we develop depression depends not only on our attributions of outcomes, but also on the difference between how we feel—our actual affect—and how we want to feel—our ideal affect (Tsai, 2007). Jeanne Tsai and her colleagues found that cultural factors influence people’s ideal affect (Tsai, Knutson, & Fung, 2006). Compared with Hong Kong Chinese, both Caucasians and Asians value excitement, whereas compared with Caucasians, Hong Kong Chinese and Asians value calm. Yet across all three cultural groups, the size of the gap between ideal and actual affect predicts depression.

**DEPRESSION: THE ROLE OF BIOLOGY.** Twin studies indicate that genes exert a moderate effect on the risk of major depression (Kendler et al., 1993). Some researchers have suggested that specific variations in the serotonin transporter gene (which affects the rate of reuptake of serotonin; see Chapter 3) play a role in depression, especially in conjunction with life experiences. Scientists first reported that people who inherit two copies of this stress-sensitive gene are two and a half times more likely to develop depression following four stressful events than people with another version of the gene that isn’t sensitive to stress (Caspi et al., 2003). The stress-sensitive gene appears to affect people’s ability to dampen negative emotions in the face of stress (Kendler, Gardner, & Prescott, 2003). Nevertheless, researchers who recently reviewed all of the available evidence concluded that there was no basis for a link between the gene and stressful life events on the one hand and depression on the other hand (Risch et al., 2009). In response, other scientists challenged how these authors analyzed previous findings (Rutter, 2009). To resolve questions regarding the role of gene–life events interactions in depression, researchers will need to conduct well-designed studies to determine whether the initially promising findings are replicable. Hopefully, these studies will clarify whether any genetic irregularities that surface are specific to depression; they may be associated with anxiety, too (Hariri et al., 2002).

Depression also appears to be linked to low levels of the neurotransmitter norepinephrine (Leonard, 1997; Robinson, 2007) and diminished neurogenesis (growth of new neurons), which brings about reduced hippocampal volume (see Chapter 3)(Pittenger & Duman, 2008; Videbech & Ravnkilde, 2004). Many patients with depression have problems in the brain’s reward and stress-response systems (Depue & Iacono, 1989; Forbes, Shaw, & Dahl, 2007) and decreased levels of dopamine, the neurotransmitter most closely tied to reward (Martinot et al., 2001). This finding may help to explain why depression is often associated with an inability to experience pleasure.
Bipolar Disorder: When Mood Goes to Extremes

In April 2012, the Manitoba Theatre Centre staged the rock opera Next to Normal, depicting the Goodman family’s struggle with bipolar disorder. When we say “bipolar,” what two poles are we talking about? We have already discovered one pole or extreme of mood, namely depression; so let’s consider the other extreme. Recall that Ann, the giddy patient with bipolar disorder whom we met at the beginning of the chapter, experienced many classic symptoms of a manic episode. These episodes are typically marked by dramatically elevated mood (feeling “on top of the world”), decreased need for sleep, greatly heightened energy, inflated self-esteem, increased talkativeness, and irresponsible behavior. People in a manic episode often display “pressured speech,” as though they can’t get their words out quickly enough, and are difficult to interrupt (Goodwin & Jamison, 1990). Their ideas often race through their heads quickly, which may account for the heightened rate of creative accomplishments in some individuals with bipolar disorder (see Chapter 9). These and other symptoms, which we can find in Table 15.6 (refer to page 616), typically begin with a rapid increase over only a few days. People usually experience their first manic episode after their early twenties (Kessler et al., 2005).

Bipolar disorder, formerly called manic-depressive disorder, is diagnosed when there’s a history of at least one manic episode (APA, 2013). In contrast to major depression, bipolar disorder is equally common in men and women. In the great majority of cases—upward of 90 percent (Alda, 1997)—people who’ve had one manic episode experience at least one more. Some have episodes separated by many years and then have a series of episodes, one rapidly following the other. More than half the time, a major depressive episode precedes or follows a manic episode. Manic episodes often produce serious problems in social and occupational functioning, such as substance abuse and unrestrained sexual behavior. Because their judgment is so impaired, people in the midst of manic episodes may go on wild spending sprees, or drive while intoxicated. One of your text’s authors treated a manic patient who passed himself off to a financial company as his own father, gained access to his father’s savings for retirement, and gambled away his entire family fortune. Another frittered away most of his life’s savings by purchasing more than 100 bowling balls, none of which he needed. The negative effects of a manic episode, including loss of employment, family conflicts, and divorce, can persist for many years (Coryell et al., 1993).

Bipolar disorder is among the most genetically influenced of all mental disorders (Miklowitz & Johnson, 2006). Twin studies suggest that its heritability may be as high as 85 percent (Alda, 1997; Lichtenstein et al., 2009; McGuffin et al., 2003). Scientists believe that genes that increase the sensitivity of the dopamine receptors (Willner, 1995) and decrease the sensitivity of serotonin receptors may boost the risk of bipolar disorder (Ogden et al., 2004). Many genes appear to be culprits in increasing the risk of bipolar disorder, and there is at least some genetic overlap between psychotic symptoms in bipolar disorder and schizophrenia (Craddock, O’Donovan, & Owen, 2005; Lichtenstein et al., 2009; Purcell et al., 2009).

Brain imaging studies suggest that people with bipolar disorder experience increased activity in structures related to emotion, including the amygdala (Chang et al., 2004; Yergelun-Todd et al., 2000), and decreased activity in structures associated with planning, such as the prefrontal cortex (Kruger et al., 2003). Still, the cause-and-effect relationship between physiological findings and mood disorders isn’t clear. For example, the high levels of norepinephrine and differences in brain activity observed in people with bipolar disorder may be an effect rather than a cause of the disorder (Thase, Jindal, & Howland, 2002).

Bipolar disorder is influenced by more than biological factors. Stressful life events are associated with an increased risk of manic episodes, more frequent relapse, and a longer recovery from manic episodes (Johnson & Miller, 1997). Interestingly, some manic episodes appear to be triggered by positive life events associated with striving for and achieving goals, such as job promotions or winning poetry contests (Johnson et al., 2000, 2008). Once again, we can see that psychological disorders arise from the intersection of biological, psychological, and sociocultural forces.

Can We Be Sure That A Causes B?

manic episode experience marked by dramatically elevated mood, decreased need for sleep, increased energy, inflated self-esteem, increased talkativeness, and irresponsible behavior

bipolar disorder condition marked by a history of at least one manic episode
Suicide: Facts and Fictions

Major depression and bipolar disorder are associated with a higher risk of suicide than most other disorders (Miklowitz & Johnson, 2006; Wolsdorf et al., 2003). This is an especially escalating problem among Canada’s Aboriginal peoples. Estimates suggest that the suicide rate of people with bipolar disorder is about 15 times higher than that of the general population (Harris & Barracough, 1997; Novick, Swartz, & Frank, 2010). Some anxiety disorders, like panic disorder, social anxiety disorder, and substance abuse are also associated with heightened suicide risk (Spirito & Esposito-Smythers, 2006). In both Canada and the United States, vital statistics data show that suicide is ranked as the eleventh leading cause of death (National Institute of Mental Health [NIMH], 2004; Statistics Canada, 2012) and the third leading cause of death for children, adolescents, and young adults (Kochanek et al., 2004).

Typically, almost 4000 Canadians commit suicide every year, three times as many males as females (a number that surely underestimates the problem because relatives report many suicides as accidents). While the national rate is 11.6 suicides per 100 000 people in the population, that rate spikes to 18.0 per 100 000 for people aged 35 to 55 years. For each completed suicide, there are an estimated 8 to 25 attempts. Contrary to what many believe, most people are more of a threat to themselves than to others. For every two people who are victims of homicide, three take their own lives (NIMH, 2004). In Table 15.7 we present a number of other common myths and misconceptions about suicide.

It’s essential to try to predict suicide attempts because most people are acutely suicidal for only a short period (Schneidman, Farberow, & Litman, 1970; Simon, 2006), and intervention during that time can be critical. Unfortunately, the prediction of suicide poses serious practical problems. First, we can’t easily conduct longitudinal studies (see Chapter 10) to determine which people will attempt suicide. It would be unethical to allow people believed to be at high risk for suicide to go through with attempts to allow us to pinpoint predictors of suicide. Second, it’s difficult to study the psychological states associated with suicide because the period of high risk for a suicide attempt is often brief. Third, the low prevalence of suicide makes predicting it difficult (Finn & Kamphuis, 1995; Meehl & Rosen, 1955). Nevertheless, the social costs of failing to predict a suicide are so great that efforts to accurately predict suicide attempts continue.

Fortunately, research has taught us a great deal about risk factors for suicide. The single best predictor of suicide is a previous attempt, because 30 to 40 percent of all people who kill themselves have made at least one prior attempt (Maris, 1992;

### Table 15.7 Common Myths and Misconceptions about Suicide.

<table>
<thead>
<tr>
<th>MYTH</th>
<th>REALITY</th>
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<tr>
<td>Talking to persons with depression about suicide often makes them more likely to commit the act.</td>
<td>Talking to persons with depression about suicide makes them more likely to obtain help.</td>
</tr>
<tr>
<td>Suicide is almost always completed with no warning.</td>
<td>Many or most individuals who commit suicide communicate their intent to others, which gives us an opportunity to seek help for a suicidal person.</td>
</tr>
<tr>
<td>As a severe depression lifts, people’s suicide risk decreases.</td>
<td>As a severe depression lifts, the risk of suicide may actually increase, in part because individuals possess more energy to attempt the act.</td>
</tr>
<tr>
<td>Most people who threaten suicide are seeking attention.</td>
<td>Although attention seeking motivates some suicidal behaviours, most suicidal acts stem from severe depression and hopelessness.</td>
</tr>
<tr>
<td>People who talk a lot about suicide almost never commit it.</td>
<td>Talking about suicide is associated with a considerably greater risk of suicide.</td>
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Pelkonnen & Marttunen, 2003). About three times as many men as women commit suicide, but nearly three times as many women attempt it (NIMH, 2004). Hopelessness may be an even better predictor of suicide than depressed mood (Beck et al., 1990; Goldston et al., 2001), because people are most likely to try to kill themselves when they see no escape from their pain. Intense agitation is also a powerful predictor of suicide risk (Fawcett, 1997). A list of risk factors for suicide appears in TABLE 15.8.

**TABLE 15.8 Major Suicide Risk Factors.**

<p>| | |</p>
<table>
<thead>
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<tbody>
<tr>
<td>1.</td>
<td>Depression</td>
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<tr>
<td>2.</td>
<td>Hopelessness</td>
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<tr>
<td>3.</td>
<td>Substance abuse</td>
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<tr>
<td>4.</td>
<td>Schizophrenia</td>
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<tr>
<td>5.</td>
<td>Homosexuality, probably because of social stigma</td>
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<tr>
<td>6.</td>
<td>Unemployment</td>
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<tr>
<td>7.</td>
<td>Chronic, painful, or disfiguring physical illness</td>
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<tr>
<td>8.</td>
<td>Recent loss of a loved one; being divorced, separated, or widowed</td>
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<td>9.</td>
<td>Family history of suicide</td>
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<tr>
<td>10.</td>
<td>Personality disorders, such as borderline personality disorder (see later discussion)</td>
</tr>
<tr>
<td>11.</td>
<td>Anxiety disorders, such as panic disorder and social anxiety disorder</td>
</tr>
<tr>
<td>12.</td>
<td>Old age, especially in men</td>
</tr>
<tr>
<td>13.</td>
<td>Recent discharge from a hospital</td>
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</tbody>
</table>

**FACT OR FICTION?**

1. Men and women are equally likely to suffer from major depression.  True / False
2. Depression is associated with stressful life events.  True / False
3. According to Lewinsohn, depression is caused by a low rate of response-contingent positive reinforcement.  True / False
4. According to Seligman, depression-prone people make specific and unstable attributions for negative life events.  True / False
5. Depressed mood is a better predictor of suicide than hopelessness.  True / False

Answers:

1. F  (p. 617)
2. T  (p. 617)
3. T  (p. 618)
4. F  (p. 620)
5. F  (pp. 622–623)

**PERSONALITY AND DISSOCIATIVE DISORDERS: THE DISRUPTED AND DIVIDED SELF**

**LO 15.8** Identify the characteristics of borderline and psychopathic personality disorders.

**LO 15.9** Explain the controversies surrounding dissociative disorders, especially dissociative identity disorder.

Most of us are accustomed to thinking of ourselves as one coherent unified identity. But some individuals—especially those with personality and dissociative disorders—experience a serious disruption in their thoughts or behaviours that prevents them from experiencing a healthy, consistent identity. Distinguishing normal variations in personality and sense of self from personality and identity disorders isn’t easy because we all have our personal quirks.
Fatal Attraction

The homicidal and unstable character in the movie Fatal Attraction would probably qualify for a diagnosis of borderline personality disorder, although most people with this diagnosis aren’t violent.

First Person Account:
**PSYCHOPATHIC PERSONALITY.**

“[Whatever] I’ve done in the past, you know—the emotions of omissions or commissions—doesn’t bother me…. Guilt? It’s this mechanism we use to control people. It’s an illusion. It does terrible things to our bodies. And there are much better ways to control our behaviour than that rather extraordinary use of guilt” (excerpt from interview with serial killer Ted Bundy shortly before his execution) (Hare, 1993).

Borderline Personality: A Volatile Blend of Traits. Persons with borderline personality’s impulsivity and rapidly fluctuating emotions often have a self-destructive quality: Many engage in drug abuse, sexual promiscuity, overeating, and even self-mutilation, like cutting themselves when upset. They may threaten suicide to manipulate others, reflecting the chaotic nature of their relationships. Because many experience intense feelings of abandonment when alone, they may jump frantically from one unhealthy relationship to another.

Explanations of Borderline Personality Disorder. Psychoanalyst Otto Kernberg (1967, 1975) traced the roots of borderline personality to childhood problems with developing a sense of self and bonding emotionally to others. According to Kernberg, individuals with borderline personality disorder can’t integrate differing perceptions of people, themselves included. This defect supposedly arises from an inborn tendency to experience intense anger and frustration from living with a cold, unempathetic mother. Kernberg argued that borderline individuals experience the world and themselves as unstable because they tend to “split” people and experiences into either all good or all bad. Although influential, Kernberg’s model of borderline personality remains inadequately researched.

According to Marsha Linehan’s (1993) sociobiological model, individuals with borderline personality disorder inherit a tendency to overreact to stress and experience...
lifelong difficulties with regulating their emotions (Crowell, Beauchaine, & Linehan, 2009). Indeed, twin studies suggest that borderline personality traits are substantially heritable (Torgerson et al., 2000). Difficulties in controlling emotions may be responsible for the rejection that many individuals with borderline personality disorder encounter, as well as their concerns about being validated, loved, and accepted. Edward Selby and Thomas Joiner's emotional cascade model holds that intense rumination about negative events or emotional experiences may result in uncontrolled “emotional cascades,” which prompt self-injurious actions, like cutting. Although these impulsive and desperate actions succeed in providing brief distraction from rumination, they often fuel further bouts of rumination, creating a vicious cycle of problems with regulating emotions (Selby et al., 2009; Selby & Joiner, 2009).

**PSYCHOPATHIC PERSONALITY: DON'T JUDGE A BOOK BY ITS COVER.** We don’t intend to alarm you. Yet the odds are high that in your life you’ve met—perhaps even dated—at least one person whom psychologists describe as a psychopathic personality, which used to be known informally as a psychopath or sociopath. Psychopathic personality is not formally a psychological disorder and is not listed in DSM-5. Nevertheless, it overlaps moderately to highly with the DSM-5 diagnosis of antisocial personality disorder (ASPD). In contrast to ASPD, which is marked by a lengthy history of illegal and irresponsible actions, psychopathic personality is marked by a distinctive set of personality traits (Lilienfeld, 1994). Because much more psychological research has concentrated on psychopathic personality than on ASPD (Hare, 2003; Patrick, 2006), we focus on psychopathy here.

Psychopathic Personality: A Dangerous Mixture of Traits. Those with psychopathic personality—most of them male—are guiltless, dishonest, manipulative, callous, and self-centred (Cleckley, 1941/1988; Lykken, 1995). Because of these distinctly unpleasant personality traits, one might assume we’d all go out of our way to avoid individuals with this disorder—and we’d probably be better off if we did. However, many of us seek out people with psychopathic personality as friends and even romantic partners because they tend to be charming, personable, and engaging (Hare, 1993). This was certainly the case with Johnny, whom we’ll recall from the beginning of the chapter. Like Johnny, many people with this condition have a history of conduct disorder, marked by lying, cheating, and stealing in childhood and adolescence.

If the traits we’ve described fit someone you know to a T, there’s no need to panic. Despite popular conception, most people with psychopathic personality aren’t physically aggressive. Nevertheless, they are at heightened risk for crime compared with the average person, and a handful—probably a few percent—are habitually violent. Serial killers Paul Bernardo and Ted Bundy almost certainly met the criteria for psychopathic personality disorder, as do about 25 percent of prison inmates (Hare, 2003). Also, despite scores of movie portrayals of crazed serial killers, people with this disorder typically aren’t psychotic. To the contrary, most are entirely rational. They know full well that their irresponsible actions are morally wrong; they just don’t care.

There’s reason to suspect that people with this condition populate not only much of the criminal justice system, but also positions of leadership in corporations and politics (Babiak & Hare, 2006). Many psychopathic traits, such as interpersonal skills, superficial likeability, ruthlessness, and risk taking, may give people with this disorder a leg up for getting ahead of the rest of the pack. Still, there’s surprisingly little research on “successful psychopaths”—people with high levels of psychopathic traits who function well in society (Hall & Benning, 2006; Widom, 1977).

**Causes of Psychopathic Personality.** Despite over five decades of research, the causes of psychopathic personality remain mysterious. Classic research shows that individuals with this disorder don’t show much classical conditioning to unpleasant unconditioned

**Figure 1:** In rare cases, violent individuals with psychopathic personality are women (Arrigo & Griffin, 2004). Aileen Wuornos (pictured above), a serial killer called the Damsel of Death, was executed for the murders of six men she lured to their death by posing as a stranded driver. Charlize Theron won an Academy Award for her portrayal of Wuornos in the movie Monster.
Dissociative Disorders

When speaking about ourselves, we use the words *me* and *I* without giving it a second thought. That’s not the case in most dissociative disorders, which involve disruptions in consciousness, memory, identity, or perception (APA, 2013). The idea that one person can have more than one identity—let alone more than a hundred, as in the case of Nadean Cool, whom we met in Chapter 7, is an extraordinary claim. So it’s no wonder that dissociative identity disorder (DID) is one of the most controversial of all diagnoses. Before we consider the debate that swirls around this condition, we’ll consider several other dissociative disorders.

DEPERSONALIZATION/DEREALIZATION DISORDER. If you’ve ever felt detached from yourself, as though you’re living in a movie or a dream or observing your body from the perspective of an outsider, you’ve experienced depersonalization. More than half of adults have experienced one brief episode of depersonalization, and such experiences are especially common among adolescents and college and university students (APA, 2013; Simeon et al., 1997). Derealization, the sense that the external world is strange or unreal, often accompanies both depersonalization and panic attacks. Only if people experience multiple episodes of depersonalization, derealization, or both, do they qualify for a diagnosis of depersonalization/derealization disorder.

DISSOCIATIVE AMNESIA. In dissociative amnesia, people can’t recall important personal information—most often following a stressful experience—that isn’t due to ordinary forgetting. Their memory loss is extensive, and can include suicide attempts or violent outbursts (Sar et al., 2007). More commonly, psychologists diagnose dissociative amnesia when adults report gaps in their memories for child abuse.

This diagnosis has proven controversial for several reasons. First, memory gaps regarding nontraumatic events are common in healthy individuals and aren’t necessarily stress-related or indicative of dissociation (Belli et al., 1998). Second, most people may not be especially motivated to recall child abuse or other upsetting events. As Richard McNally (2003) pointed out, not thinking about something isn’t the same as being unable to remember it, which is amnesia. Third, careful studies have turned up no convincing cases of amnesia that can’t be explained by other factors, like disease, brain injury, normal forgetting, or an unwillingness to think about disturbing events (Kihlstrom, 2005; Pope et al., 2007).

At times, we’ve all felt like running away from our troubles. In dissociative fugue, a type of dissociative amnesia, people not only forget significant events in their lives, but also flee their stressful circumstances (*fugue* is Latin for “flight”). In some cases, they...
move to another city or another country, assuming a new identity. Fugues can last for hours or, in unusual cases, years. Dissociative fugue is rare, occurring in about 2 of every 1000 people (APA, 2013), with more prolonged fugue states even rarer (Karlin & Orne, 1996).

In 2006, a 57-year-old husband, father, and Boy Scout leader from New York was found living under a new name in a homeless shelter in Chicago after he left his garage near his office and disappeared. When a tip to America’s Most Wanted uncovered his true identity six months later, his family contacted him, but he claimed to have no memory of who they were (Brody, 2007).

In this and other fugue cases, it’s essential to find out whether the fugue resulted from a head injury, stroke, or other neurological cause. Moreover, some people merely claim amnesia to avoid responsibilities or stressful circumstances, relocate to a different area, and get a fresh start in life. Even when fugues occur shortly after a traumatic event, it’s difficult to know whether the trauma caused the amnesia. Scientists don’t fully understand the roles that trauma, psychological factors, and neurological conditions play in fugue states (Kihlstrom, 2005).

**Dissociative Identity Disorder:**

**Multiple Personalities, Multiple Controversies.** Dissociative identity disorder (DID) is characterized by the presence of two or more distinct personality states (the term “identities” was deleted in DSM-5) that markedly disrupt the person’s usual sense of identity and may be observed by others or reported by the individual. These personality states, or “alters,” as they’re sometimes called, are often very different from the primary or “host” personality and may be of different names, ages, genders, and even races. In some cases, these features are the opposite of those exhibited by the host personality. For example, if the host personality is shy and retiring, one or more alters may be outgoing or flamboyant. Psychologists have reported the number of alters to range from one (the so-called split personality) to hundreds or even thousands, with one reported case of 4500 personalities (Acocella, 1999).

In general, women are more likely to receive a DID diagnosis and report more alters than men (APA, 2013). Researchers have identified intriguing differences among alters in their respiration rates (Bahnson & Smith, 1975), brain wave activity (EEG; Ludwig et al., 1972), eyeglass prescriptions (Miller, 1989), handedness (Savitz et al., 2004), skin conductance responses (Brende, 1984), voice patterns, and handwriting (Lilienfeld & Lynn, 2003). As fascinating as these findings are, they don’t provide conclusive evidence for the existence of alters. These differences could stem from changes in mood or thoughts over time or from bodily changes, such as muscle tension, that people can produce on a voluntary basis (Allen & Movius, 2000; Merckelbach, Devilly, & Rassin, 2002). Moreover, scientists have falsified claims that alters are truly distinct. When psychologists have used objective measures of memory, they’ve typically found that information presented to one alter is available to the other, providing no evidence for amnesia across alters (Allen & Movius, 2000; Huntjens et al., 2012).

The primary controversy surrounding DID revolves around one question: Is DID a response to early trauma, or is it a consequence of social and cultural factors (Merskey, 1992)? According to the posttraumatic model (Gleaves, May, & Cardena, 2001; Ross, 1997), DID arises from a history of severe abuse—physical, sexual, or both—during childhood. This abuse leads individuals to “compartmentalize” their identity into distinct alters as a means of coping with intense emotional pain. In this way, the person can feel as though the abuse happened to someone else.
Advocates of the posttraumatic model claim that 90 percent or more of individuals with DID were severely abused in childhood (Gleaves, 1996). Nevertheless, many studies that reported this association didn’t check the accuracy of abuse claims against objective information, such as court records of abuse (Coons, Bowman, & Milstein, 1988). Moreover, researchers haven’t shown that early abuse is specific to DID, as it is present in many other disorders (Pope & Hudson, 1992). These considerations don’t exclude a role for early trauma in DID, but they suggest that researchers must conduct further controlled studies before drawing strong conclusions (Gleaves, 1996; Gleaves et al., 2001).

According to advocates of the competing sociocognitive model (see Chapter 5), the claim that some people have hundreds of personalities is extraordinary, but the evidence for it is unconvincing (Giesbrecht et al., 2008; Lilienfeld et al., 1999; McHugh, 1993; Merskey, 1992; Spanos, 1994, 1996). According to this model, people’s expectancies and beliefs—shaped by certain psychotherapeutic procedures and cultural influences, rather than early traumas—account for the origin and maintenance of DID. Advocates of this model claim that some therapists—like those of Nadean Cool (see Chapter 7)—use procedures, like hypnosis and repeated prompting of alters, that suggest to patients that their puzzling symptoms are the products of indwelling identities (Lilienfeld & Lynn, 2003; Lilienfeld et al., 1999). The following observations and findings support this hypothesis:

- Many or most DID patients show few or no clear-cut signs of this condition, such as alters, prior to psychotherapy (Kluft, 1984).
- Mainstream treatment techniques for DID reinforce the idea that the person possesses multiple identities. These techniques include using hypnosis to “bring forth” hidden alters, communicating with alters and giving them different names, and encouraging patients to recover repressed memories supposedly housed in dissociated selves (Spanos, 1994, 1996).
- The number of alters per DID individual tends to increase substantially when therapists use these techniques (Piper, 1997).
- Researchers have reported a link between dissociation and the tendency to fantasize in everyday life (Giesbrecht et al., 2008), which may be related to the production of false memories, although the interpretation of these findings has proven controversial (Dalenberg et al., 2012).

As of 1970, there were 79 documented cases of DID in the world literature. As of 1986, the number of DID cases had mushroomed to approximately 6000 (Lilienfeld et al., 1999), and some estimates in the early twenty-first century are in the hundreds of thousands. The sociocognitive model holds that the popular media have played a pivotal role in the DID epidemic (Elzinga et al., 1998). Indeed, much of the dramatic increase in DID’s prevalence followed closely on the release of the bestselling book Sybil (Schreiber, 1973) in the mid-1970s, later made into an Emmy Award-winning television movie starring Sally Field. The book and film told the heartbreaking story of a young woman with 16 personalities who reported a history of sadistic child abuse. Subsequently released audiotapes of Sybil’s therapy sessions suggested that she had no alters or memories of child abuse prior to treatment, and that her therapist urged her to behave differently on different occasions (Rieber, 1999).

Over the past two decades, media coverage of DID has skyrocketed (Showalter, 1997; Spanos, 1996; Wilson, 2003), with some celebrities, like comedian Roseanne Barr and football star Herschel Walker, claiming to suffer from the disorder. Although DID is virtually nonexistent in Japan and India, it is now diagnosed with considerable frequency in some countries, such as Holland, in which it has recently received more publicity (Lilienfeld et al., 1999). In summary, there’s considerable support for the sociocognitive model and the claim that therapists, along with the media, are creating alters rather than discovering them. The dissociative disorders provide a powerful, although troubling, example of how social and cultural forces can shape psychological disorders.
THE ENIGMA OF SCHIZOPHRENIA

LO 15.10 Recognize the characteristic symptoms of schizophrenia.
LO 15.11 Explain how psychosocial, neural, biochemical, and genetic influences create the vulnerability to schizophrenia.

Psychiatrist Daniel Weinberger has called schizophrenia the “cancer” of mental illness: It is perhaps the most severe of all disorders, and the most mysterious (Levy-Reiner, 1996). As we’ll discover, it is a devastating disorder of thought and emotion associated with a loss of contact with reality.

■ Symptoms of Schizophrenia: The Shattered Mind

Even today, many people confuse schizophrenia with DID (Wahl, 1997). Swiss psychiatrist Eugen Bleuler gave us the modern term schizophrenia in 1911. The term literally means “split mind,” which no doubt contributed to the popular myth that the symptoms of schizophrenia stem from a split personality. You may have even heard people refer to a “schizophrenic attitude” when explaining that they’re “of two minds” regarding an issue. Don’t be misled. As Bleuler recognized, the difficulties of individuals with schizophrenia arise from disturbances in attention, thinking, language, emotion, and relationships with others. In contrast to DID, which is supposedly characterized by multiple intact personalities, schizophrenia is characterized by one personality that’s shattered.

Schizophrenia causes most of its sufferers’ levels of functioning to plunge. More than half suffer from serious disabilities, such as an inability to hold a job and maintain close relationships (Harvey, Reichenberg, & Bowie, 2006). Indeed, a large proportion of homeless people would receive diagnoses of schizophrenia (Cornblatt, Green, & Walker, 1999). Individuals who experience schizophrenia comprise less than 1 percent of the population, with most estimates ranging from 0.4 to 0.7 percent (Saha et al., 2005). Yet they make up half of more than 100,000 patients in mental institutions in North America (Grob, 1997). But there’s some good news. Today, more than ever, people with schizophrenia can function in society, even though they may need to return periodically to hospitals for treatment (Harding, Zubin, & Strauss, 1992; Lamb & Bachrach, 2001; Mueser & McGurk, 2004).

Researchers have struggled with the problem of describing schizophrenia since the eighteenth century, when Emil Kraepelin first outlined the features of patients with dementia praecox, meaning psychological deterioration in youth. But Kraepelin didn’t get it quite right. Even though the typical onset of schizophrenia is in the mid-twenties for men and the late twenties for women, schizophrenia can also strike after age 45 (APA, 2013). Moreover, as many as one-half to two-thirds of people with schizophrenia improve significantly, although not completely, and a small percentage recover completely after a single episode (Harrow et al., 2005; Robinson et al., 2004). DSM-5 states that to be diagnosed with schizophrenia, the individual must exhibit at least one of the following three symptoms: delusions, hallucinations, and/or disorganized speech (discussed below).
Among the hallmark symptoms of schizophrenia are **delusions**—strongly held, fixed beliefs that have no basis in reality. Delusions are called **psychotic symptoms** because they represent a serious distortion of reality. Terrell, whom we met at the beginning of the chapter, experienced delusions that led to a suicide attempt.

Delusions commonly involve themes of persecution. One of your text’s authors treated a man who believed that coworkers had tapped his phone and conspired to get him fired. Another was convinced that a helicopter in the distance beamed the Beatles song “All You Need Is Love” into his head to make him feel jealous and inadequate. The authors of your text have also treated patients who reported delusions of grandeur (greatness), including one who believed that she’d discovered the cure for cancer even though she had no medical training. Other delusions centre on the body and may include a firm belief that one is infested with brain parasites or even that one is dead (so-called Cotard’s syndrome). Still others involve elaborate themes of sexuality or romance. John Hinckley, Jr., the man who attempted to assassinate U.S. President Ronald Reagan in 1981, was convinced that murdering the president would gain him the affection of actress Jodie Foster.

**Hallucinations: False Perceptions.** Among the other serious symptoms of schizophrenia are **hallucinations**: sensory perceptions that occur in the absence of an external stimulus. They can be auditory (invoking hearing), olfactory (invoking smell), gustatory (invoking taste), tactile (invoking the sense of feeling), or visual. Most hallucinations in schizophrenia are auditory, usually consisting of voices. In some patients, hallucinated voices express disapproval or carry on a running commentary about the person’s thoughts or actions. **Command hallucinations**, which tell patients what to do (“Go over to that man and tell him to shut up!”), may be associated with a heightened risk of violence toward others (McNiel, Eisner, & Binder, 2000).Incidentally, extremely vivid or detailed visual hallucinations—especially in the absence of auditory hallucinations—are usually signs of an organic (medical) disorder or substance abuse rather than schizophrenia (Shea, 1998).

Do your thoughts sound like voices in your head? Many people experience their thoughts as inner speech, which is entirely normal. Some researchers suggest that auditory hallucinations occur when people with schizophrenia believe mistakenly that their inner speech arises from a source outside themselves (Bentall, 2000; Frith, 1992; Thomas, 1997). Brain scans reveal that when people experience auditory hallucinations, brain areas associated with speech perception and production become activated (McGuire, Shah, & Murray, 1993).

**Disorganized Speech.** Consider the reply of a 39-year-old patient with schizophrenia to the question of whether he felt that people imitated him. “Yes… I don’t quite gather. I know one right and one left use both hands but I can’t follow the system that’s working. The idea is meant in a kind way, but it’s not the way I understand life. It seems to be people taking sides. If certain people agree with me they speak, and if not, they don’t. Everybody seems to be the doctor and Mr. H. [his own name] in turn” (Mayer-Gross, Slater, & Roth, 1969, p. 177).

We can see that his language skips from topic to topic in a disjointed way. Most researchers believe that this peculiar language results from thought disorder (Meehl, 1962; Stirk et al., 2008). The usual associations that we forge between two words, such as mother–child, are considerably weakened or highly unusual for individuals with schizophrenia (for example, mother–rug) (Kuperberg et al., 2006). In severe forms, the resulting speech is so jumbled that it’s almost impossible to understand, leading psychologists to describe it as **word salad**. Language problems, like thought disorder, point to fundamental impairments in schizophrenia in the ability to shift and

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**Factoid**

One of the more unusual delusions is **folie à deux** (French for “folly of two”), known technically as “shared psychotic disorder” in DSM-5. In folie à deux, one person in a close relationship, often a marriage, induces the same delusion in his or her partner. For example, both partners may end up convinced that the government is poisoning their food (Silveira & Seeman, 1985). Rare cases of folie à deux in identical twins, folie à trois (involving three people), and folie à famille (involving an entire family) have also been reported.

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**delusion**

strongly held, fixed belief that has no basis in reality

**psychotic symptom**

psychological problem reflecting serious distortions in reality

**hallucination**

sensory perception that occurs in the absence of an external stimulus
maintain attention, which influence virtually every aspect of affected individuals’ daily lives (Cornblatt & Keilp, 1994; Fuller et al., 2006).

**GROSSLY DISORGANIZED BEHAVIOUR AND CATATONIA.** When people develop schizophrenia, self-care, personal hygiene, and motivation often deteriorate. They may avoid conversation; laugh, cry, or swear inappropriately; or wear a warm coat on a sweltering summer day.

Catatonic symptoms involve motor (movement) problems, such as extreme resistance to complying with even simple suggestions, holding the body in bizarre or rigid postures, or curling up in a fetal position. Catatonic individuals’ withdrawal can be so severe that they refuse to speak and move, or they may pace aimlessly. They may also repeat a phrase in conversation in a parrotlike manner, a symptom called *echolalia*. At the opposite extreme, they may occasionally engage in bouts of frenzied, purposeless motor activity.

- **Explanations for Schizophrenia: The Roots of a Shattered Mind**

Today, virtually all scientists believe that psychosocial factors play some role in schizophrenia. Nevertheless, they also agree that these factors probably trigger the disorder only in people with a genetic vulnerability.

**THE FAMILY AND EXPRESSED EMOTION.** Early theories of schizophrenia mistakenly laid the blame for the condition on mothers, with so-called *schizophrenogenic* (schizophrenia-producing) mothers being the prime culprits. Based on informal observations of families of children with schizophrenia, some authors described such mothers as overprotective, smothering, rejecting, and controlling (Arieti, 1959; Lidz, 1973). Other theorists pointed the finger of blame at the interactions among all family members (Dolnick, 1998).

But as important as clinical experience can be in generating hypotheses, it doesn’t provide an adequate arena for testing them (see Chapter 2). Indeed, these early studies were severely flawed, largely because they lacked control groups of people without schizophrenia. A now widely accepted rival hypothesis is that family members’ responses aren’t the cause of schizophrenia, but instead are typically a response to the stressful experience of living with a severely disturbed person.

It’s widely acknowledged that parents and family members don’t “cause” schizophrenia (Gottesman, 1991; Walker et al., 2004). Still, families may influence whether patients with the disorder relapse. After leaving hospitals, patients experience more than twice the likelihood of relapse (50 to 60 percent) when their relatives display high expressed emotion (EE)—that is, criticism, hostility, and overinvolvement (Brown et al., 1962; Butzlaff & Hooley, 1998). Criticism is especially predictive of relapse (Halweg et al., 1989; McCarty et al., 2004), and may result in part from relatives’ frustrations in living with a person with schizophrenia who displays disruptive behaviours. Indeed, EE may reflect family members’ reactions to their loved one’s schizophrenia as much as contribute to their loved one’s relapse (King, 2000).

The apparent effects of EE vary across ethnic groups. Critical comments from family members may undermine recovering patients’ confidence and sense of independence, which are valued in Caucasian culture (Chentsova-Dutton & Tsai, 2007). In contrast, in Latin American culture, independence isn’t as highly valued, so criticism doesn’t predict relapse. Nevertheless, a lack of family warmth, which is prized in Latin American families, does predict relapse (Lopez et al., 2004). Moreover, in African-American families, high levels of EE predict better outcomes among individuals with schizophrenia, perhaps because family members perceive EE as an expression of openness, honesty, and caring (Rosenfarb, Bellack, & Aziz, 2006). Although EE often predicts relapse, well-controlled studies don’t support the hypothesis that child rearing directly causes schizophrenia, any more than does extreme poverty, childhood trauma, or parental conflict, all of which are correlated with schizophrenia (Cornblatt, Green, & Walker, 1999; Schofield & Balian, 1959).
SCHIZOPHRENIA: BRAIN, BIOCHEMICAL, AND GENETIC FINDINGS. Research using a variety of technologies has uncovered intriguing biological clues to the causes of schizophrenia. We’ll focus on three such clues: brain abnormalities, neurotransmitter differences, and genetic findings.

Brain Abnormalities. Research indicates that one or more of four fluid-filled structures called ventricles (see Chapter 3), which cushion and nourish the brain, are typically enlarged in individuals with schizophrenia. This finding is important for two reasons. First, these brain areas frequently expand when others shrink (Barta et al., 1990; Raz & Raz, 1990), suggesting that schizophrenia is a disorder of brain deterioration. Second, deterioration in these areas is associated with thought disorder (Vita et al., 1995).

Other brain abnormalities in schizophrenia include increases in the size of the sulci, or spaces between the ridges of the brain (Cannon, Mednick, & Parnas, 1989), and decreases in (a) the size of the temporal lobes (Boos et al., 2007; Job et al., 2005), (b) activation of the amygdala and hippocampus (Hempel et al., 2003), and (c) the symmetry of the brain’s hemispheres (Luchins, Weinberger, & Wyatt, 1982; Zivotofsky et al., 2007). Functional brain imaging studies show that the frontal lobes of people with schizophrenia are less active than those of nonpatients when engaged in demanding mental tasks (Andreasen et al., 1992; Knyazeva et al., 2008), a phenomenon called hypofrontality. Still, it’s not clear whether these findings are causes or consequences of the disorder. For example, hypofrontality could be due to the tendency of patients with schizophrenia to concentrate less on tasks compared with other individuals.

Researchers also need to rule out alternative explanations for brain underactivity that could arise from patients’ diet, drinking and smoking habits, and medication use (Hanson & Gottesman, 2005).

Some studies have suggested that marijuana use in adolescence can bring about schizophrenia and other psychotic disorders in genetically vulnerable individuals (Compton et al., 2009; Degenhardt et al., 2009; Degenhardt & Hall, 2006). Nevertheless, it’s difficult to pin down a causal relationship between marijuana use and schizophrenia for three reasons: (1) People who use marijuana are likely to use a variety of other drugs, which may be reluctant to report on surveys; (2) individuals with schizophrenia may be more likely to use marijuana, so the causal arrow may be reversed, and (3) the rates of schizophrenia remained stable between 1970 and 2005 in the United Kingdom, although marijuana use increased over this period (Frisher et al., 2009). Still, people with a personal or family history of psychotic disorders, including schizophrenia, would be particularly ill advised to use marijuana.

Neurotransmitter Differences. The biochemical of the brain is one of the keys to unlocking the mystery of schizophrenia. One early explanation was the dopamine hypothesis (Carlsson, 1995; Keith et al., 1976; Nicol & Gottesman, 1983). The evidence for the role of dopamine in schizophrenia is mostly indirect. First, most antischizophrenic drugs block dopamine receptor sites. To put it crudely, they “slow down” nerve impulses by partially blocking the action of dopamine (see Chapters 3 and 16). Second, amphetamine, a stimulant (see Chapter 5) that blocks the reuptake of dopamine, tends to worsen the symptoms of schizophrenia (Lieberman & Koreen, 1993; Snyder, 1975).

Nevertheless, the hypothesis that a simple excess of dopamine creates the symptoms of schizophrenia doesn’t seem to fit the data. A better-supported rival hypothesis is that abnormalities in dopamine receptors produce these symptoms. Receptor sites in the brain appear to be highly specific for dopamine transmission. These sites respond uniquely to drugs designed to reduce psychotic symptoms and are associated with difficulties in attention, memory, and motivation (Busatto et al., 1995; Keefe & Henry, 1994; Reis et al., 2004).

These findings provide evidence for a direct tie between dopamine pathways and symptoms of schizophrenia, such as paranoia. As we’ve seen, some symptoms of schizophrenia represent excesses of normal functions and include hallucinations, delusions, and disorganized speech and behaviour. We can contrast these positive symptoms with

**FICTOID**

**MYTH:** Many non-Western cultures regard people with schizophrenia and other severe mental illnesses as “shamans,” or medicine men, and often worship them as possessing divine powers.

**REALITY:** Jane Murphy’s (1976) work suggests that the Yoruba of Nigeria and other cultures distinguish people with mental illness from shamans. The Yoruba, for example, referred to a man with mental illness living on an abandoned anthill as “out of mind and crazy.” In contrast, they referred to a shaman during a healing session as “out of mind but not crazy,” meaning that he was in a temporary trance state.
negative symptoms, which reflect decreases or losses of normal functions. These symptoms include social withdrawal and diminished motivations, decreased expression of emotions, and brief and limited speech (Andreasen et al., 1997). People with schizophrenia are less impaired when their symptoms are predominantly positive rather than negative (Harvey, Reichenberg, & Bowie, 2006). There’s evidence that positive symptoms result from dopamine excesses in some brain regions, and that negative symptoms result from dopamine deficits in other brain regions (Davis et al., 1991). However, the causes of negative symptoms are difficult to pinpoint, because they may arise from prolonged institutionalization and medication side effects (see Chapter 16). Dopamine is probably only one of several neurotransmitters that play a role in schizophrenia; other likely candidates are norepinephrine, glutamate, and serotonin (Cornblatt, Green, & Walker, 1999; Grace, 1991).

Genetic Influences. Still unresolved is the question of which biological deficits are present prior to schizophrenia and which appear after the disorder begins (Seidman et al., 2003). The seeds of schizophrenia are often sown well before birth and lie partly in individuals’ genetic endowment. As we can see in Figure 15.2, being the offspring of someone diagnosed with schizophrenia greatly increases one’s odds of developing the disorder. If we have a sibling with schizophrenia, we have about a one in ten chance of developing the disorder; these odds are about ten times higher than those of the average person. As genetic similarity increases, so does the risk of schizophrenia.

Still, it’s possible that the environment accounts for these findings because siblings not only share genes, but also grow up in the same family. To eliminate this ambiguity, researchers have conducted twin studies, which provide convincing support for a genetic influence on schizophrenia. If we have an identical twin with schizophrenia, our risk rises to about 50 percent. An identical twin of a person with schizophrenia is about three times as likely as a fraternal twin of a person with schizophrenia to develop the disorder, and about 50 times as likely as an average person (Gottesman & Shields, 1972; Kendler & Diehl, 1993; Meehl, 1962). Adoption data also point to a genetic influence. Even when children who have a biological parent with schizophrenia are adopted by parents with no hint of the disorder, their risk of schizophrenia is greater than that of a person with no biological relative with schizophrenia (Gottesman, 1991). Interestingly, scientists have identified structural brain abnormalities, like ventricular enlargement and decreases in brain volume, in the healthy close relatives of patients with schizophrenia, further suggesting that genetic influences produce a vulnerability to schizophrenia (Staal et al., 2000).

VULNERABILITY TO SCHIZOPHRENIA: DIATHESIS-STRESS MODELS. Diathesis-stress models incorporate much of what we know about schizophrenia. Such models propose that schizophrenia, along with many other mental disorders, is a joint product of a genetic vulnerability, called a diathesis, and stressors that trigger this vulnerability (Meehl, 1962; Walker & DiFiorio, 1997; Zubin & Spring, 1977).

Paul Meehl (1990) suggested that approximately 10 percent of the population has a genetic predisposition to schizophrenia. What are people with this predisposition like? During adolescence and adulthood, they may strike us as “odd ducks.” They may seem socially uncomfortable, and their speech, thought processes, and perceptions may impress us as unusual. They’re likely to endorse items on psychological tests such as “Occasionally, I have felt as though my body did not exist” (Chapman, Chapman, & Raulin, 1978). Such individuals display symptoms of psychosis-proneness or schizotypal personality disorder. Most people with schizotypal personality disorder don’t develop full-blown schizophrenia, perhaps because they have a weaker genetic vulnerability or because they’ve experienced fewer stressors.

Well before people experience symptoms of schizophrenia, we can identify “early warning signs” or markers of vulnerability to this condition. People with schizotypal personality disorder display some of these markers, which include social
CHILDHOOD DISORDERS: RECENT CONTROVERSIES

LO 15.12 Describe the symptoms and debate surrounding disorders diagnosed in childhood.

Although in this chapter we’ve focused primarily on disorders of adulthood, we’ll now close with a few words about childhood disorders, especially those that have been front and centre in the public eye. Each of the disorders we’ll consider—autism spectrum disorders, attention-deficit/hyperactivity disorder, and early-onset bipolar disorder—has garnered its share of controversy in the popular media and the scientific community.

Autism Spectrum Disorders

So, 1 in 150 is the widely proclaimed proportion of individuals with autism spectrum disorder (ASD), a category in DSM-5 that includes autistic disorder (better known as autism) and Asperger’s disorder, a less severe form of autism. DSM-5 contends that the symptoms of autism can best be described as on a continuum of severity, rather than in categorical terms, with many children with Asperger’s disorder being able to function effectively in a school or occupational setting. Although this proportion may not seem all that high,
it’s remarkably high compared with the figure of 1 in 2000 to 2500, which researchers had until recently accepted for many years (Wing & Potter, 2002). These dramatic upsurges in the prevalence of autism have led many researchers and educators, and even some politicians, to speak of an autism “epidemic” (Kippes & Garrison, 2006). But is the epidemic real?

As we learned in Chapter 2, individuals with autism are marked by severe deficits in language, social bonding, and imagination, usually accompanied by intellectual impairment (APA, 2013). The DSM-5 breaks down the symptoms of autism spectrum disorders into social impairments and repetitive or restrictive behaviours, which can include repetitive speech or movements, resistance to change, and highly specialized and limited interests and preoccupation with certain foods or unusual objects such as lightbulbs. The causes of autism remain mysterious, although twin studies suggest that genetic influences play a prominent role (Rutter, 2000). Still, genetic influences alone can’t easily account for an astronomical rise in a disorder’s prevalence over the span of a decade. It’s therefore not surprising that researchers have looked to environmental variables to explain this bewildering increase. In particular, some investigators have pointed their fingers squarely at one potential culprit: vaccines (Rimland, 2004).

Much of the hype surrounding the vaccine–autism link was fuelled by a study of only 12 children in the late 1990s (Wakefield et al., 1998) demonstrating an apparent linkage between autistic symptoms and the MMR vaccine—the vaccine for mumps, measles, and rubella, also known as German measles. (The journal Lancet, which published the study, officially retracted it in 2010, saying that Wakefield never received ethical clearance for the investigation and that the article contained false claims about subject recruitment.) The symptoms of autism usually become most apparent shortly after the age of 2, not long after infants have received MMR and other vaccinations for a host of diseases. Indeed, tens of thousands of parents insist that their children developed autism following the MMR vaccine, or following vaccines containing a preservative known as thimerosol, which is present in many mercury-bearing vaccines. Nevertheless, studies in the United States, Europe, and Japan failed to replicate the association between the MMR vaccine and autism, strongly suggesting that the seeming correlation between vaccinations and autism was a mirage (Offit, 2008). The results of several large U.S., European, and Japanese studies show that even as the rate of MMR vaccinations remained constant or declined, the rate of autism diagnoses continued to soar (Herbert, Sharp, & Gaudiano, 2002; Honda, Shimizu, & Rutter, 2005). Moreover, even after the Danish government stopped administering thimerosol-containing vaccines, the prevalence of autism still skyrocketed (Madsen et al., 2002).

Many parents of children with autism probably fell prey to illusory correlation (see Chapter 2); they’d “seen” a statistical association that didn’t exist. Their error was entirely understandable. Given that their children had received vaccines and developed autistic symptoms at around the same time, it was only natural to perceive an association between the two events.

Making matters more complex, recent research calls into question the very existence of the autism epidemic (Grinker, 2007; Wilson, 2005). Most previous investigators had neglected to take into account an alternative explanation—changes in diagnostic practices over time, which have expanded the autism diagnosis to include more mildly affected children, including some with a condition called Asperger’s disorder (which appears to be a mild form of autism). Evidence suggests that more liberal diagnostic criteria rather than vaccines can account for most, if not all, of the reported autism epidemic (Gernsbacher, Dawson, & Goldsmith, 2005; Lilenfeld & Arkowitz, 2007). In addition, both the Canadians Disability Act and the Americans with Disabilities Act indirectly encouraged school districts to classify more children as having autism and other developmental disabilities, as these children could now receive more extensive educational accommodations.

Of course, at least a small part of the epidemic might be genuine, and some still unidentified environmental cause could account for the increase. But in evaluating the evidence, we should ask ourselves a critical question. Which is more parsimonious as an
explanation of a 657 percent increase within one decade: a vaccine that’s yet to be shown to produce any increase in the symptoms of autism, or a simple change in diagnostic practices?

■ Attention-Deficit/Hyperactivity Disorder and Early-Onset Bipolar Disorder

Even the best-adjusted children often appear overactive, energetic, and restless. But children with attention-deficit/hyperactivity disorder (ADHD) often behave like caricatures of the exuberant child. You probably know or have known someone with ADHD: 3 to 7 percent of school-aged children satisfy the diagnostic criteria for the disorder (Barkley, 2006; Bird, 2002). The male-to-female ratio of ADHD ranges from 3.4 to 1 across studies, and between 30 and 80 percent of children with ADHD continue to display ADHD symptoms into adolescence and adulthood (Barkley, 2006; Monastra, 2006). The ADHD diagnosis subsumes two subtypes—one with hyperactivity and one without hyperactivity—in which inattention is predominant (APA, 2013).

SYMPTOMS OF ADHD. The first signs of ADHD may be evident as early as infancy. Parents often report that children with ADHD are fussy, cry incessantly, and frequently move and shift their position in the crib (Wolke, Rizzo, & Woods, 2002). By 3 years of age, they’re constantly walking or climbing, restless, and prone to emotional outbursts. But it’s not until elementary school that their behaviour patterns are likely to be labelled “hyperactive” and a treatment referral made. Teachers complain that such children won’t remain in their seats, follow directions, or pay attention, and that they display temper tantrums with little provocation. Such children often struggle with learning disabilities, difficulties with processing verbal information, and poor balance and coordination (Jerome, 2000; Mangeot et al., 2001). By middle childhood, academic problems and disruptive behaviour are frequently evident.

A high level of physical activity often diminishes as children with ADHD mature and approach adolescence. Nevertheless, by adolescence, impulsiveness, restlessness, inattention, problems with peers, delinquency, and academic difficulties comprise a patchwork of adjustment problems (Barkley, 2006; Hoza, 2007; Kelly, 2009). Alcohol and substance abuse are frequent (Molina & Pelham, 2003), and many adolescents with ADHD appear in juvenile courts as a result of running away from home, school truancy, and theft (Foley, Carlton, & Howell, 1996). Adults with ADHD are at increased risk for accidents and injuries (Woodward, Ferguson, & Horwood, 2000), divorce (Wymbus et al., 2008), unemployment, and contact with the legal system (Hinshaw, 2002).

ADHD appears to be genetically influenced in many cases, with estimates of its heritability as high as 0.80 (Swanson & Castellanos, 2002). What may be inherited are abnormalities in genes that influence (a) serotonin, dopamine, and norepinephrine; (b) a smaller brain volume; and (c) decreased activation in the frontal areas of the brain (Monastra, 2008).

As we’ll see in Chapter 16, people with ADHD can be treated successfully with stimulant medications. Nevertheless, these medications occasionally have serious side effects, making accurate diagnosis a serious public health issue. Yet an accurate diagnosis of ADHD can be dicey. A host of conditions that can cause problems in attention and behavioural control, including traumatic brain injuries, diabetes, thyroid problems, vitamin deficiencies, anxiety, and depression, must first be ruled out (Monastra, 2008). Additionally, there’s sometimes a fine line between highly energetic children and those with mild ADHD. As a consequence, some scholars have expressed concerns that ADHD is overdiagnosed in some settings (LeFever, Arcona, & Antonuccio, 2003), although
Many children have problems concentrating. There can be a fine line between children who have trouble paying attention in class and children diagnosed with ADHD. Others point to evidence that some children with ADHD are actually overlooked by many diagnosticians (Scuotto & Eisenberg, 2007).

**THE CONTROVERSY OVER EARLY-ONSET BIPOLAR DISORDER.** Perhaps the most controversial diagnostic challenge is distinguishing children with ADHD from children with bipolar disorder (Meyer & Carlson, 2008). The diagnosis of early-onset bipolar disorder was once rare, but ballooned from only 0.42 percent of cases of outpatient mental health visits in the early 1990s to 6.67 percent of such visits in 2003 (Moreno et al., 2007), raising concerns about its overdiagnosis. Children are particularly likely to receive a diagnosis of early-onset bipolar disorder when they show rapid mood changes, reckless behaviour, irritability, and aggression (McClellan, Kowatch, & Findling, 2007). Popular books like *The Bipolar Child* (Papolos & Papolos, 2007), which list these and other symptoms, catch the eye of many parents with troubled children and raise concerns about bipolar disorder. Yet a moment’s reflection suggests that many children fit this description, and surely many children with ADHD can be so characterized. Because 60 to 90 percent of children with bipolar disorder share an ADHD diagnosis, a hypothesis to consider is that many children diagnosed with bipolar disorder are merely those with severe symptoms of ADHD, such as extreme temper outbursts and mood swings (Geller et al., 2002; Kim & Miklowitz, 2004).

To address concerns about the overdiagnosis of bipolar disorder in children, DSM-5 developed a new category of *disruptive mood dysregulation disorder* to diagnose children with persistent irritability and frequent behaviour outbursts (APA, 2013). Nevertheless, the validity of this condition remains controversial, and some experts have expressed concerns that it may result in labelling children with repeated temper tantrums as pathological (Frances, 2012). Meanwhile, a thorough evaluation involving parents, teachers, and mental health professionals is essential to an accurate diagnosis of early-onset bipolar disorder, as well as ADHD.

### assess your knowledge

**FACT OR FICTION?**

1. The rates of diagnosed autism have sharply declined in recent years. **True / False**
2. Most cases of autism are caused by vaccines. **True / False**
3. More boys than girls are diagnosed with ADHD. **True / False**
4. Parents first notice their children’s attention problems in elementary school. **True / False**
5. Children with bipolar disorder are likely to also be diagnosed with ADHD. **True / False**
CONCEPTIONS OF MENTAL ILLNESS: YESTERDAY AND TODAY 598–610

**LO 15.1 IDENTIFY CRITERIA FOR DEFINING MENTAL DISORDERS.**
The concept of mental disorder is difficult to define. Nevertheless, criteria for mental disorders include statistical rarity, subjective distress, impairment, societal disapproval, and biological dysfunction. Some scholars argue that mental illness is best captured by a family resemblance view.

1. Describe the family resemblance view of mental disorder. (p. 600)

**LO 15.2 DESCRIBE CONCEPTIONS OF DiAGNOSES ACROSS HISTORY AND CULTURES.**
The demonic model of mental illness was followed by the medical model of the Renaissance. In the early 1950s, medications to treat schizophrenia led to deinstitutionalization. Some psychological conditions are culture-specific. Still, many mental disorders, such as schizophrenia, can be found in most or all cultures.

2. During the Middle Ages, ________ was often used to treat mental illness. (p. 600)
3. Institutions for the mentally ill created in the fifteenth century were known as ________. (p. 600)
4. In the United States in the nineteenth century, Dorothea Dix advocated for ________, ________, an approach calling for dignity, kindness, and respect for those with mental illness. (p. 601)
5. In the early 1950s, medications that treated schizophrenia, like chlorpromazine, led to a government policy called ________. (p. 601)
6. Some eating disorders are examples of a ________ disorder specific to Western cultures. (p. 603)

**LO 15.3 IDENTIFY COMMON MISCONCEPTIONS ABOUT PSYCHIATRIC DiAGNOSES, AND THE STRENGTHS AND LIMITATIONS OF THE CURRENT DIAGNOSTIC SYSTEM.**
Misconceptions include the ideas that a diagnosis is nothing more than pigeonholing, and that diagnoses are unreliable, invalid, and stigmatizing. The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) is a valuable tool. However, its limitations include high levels of comorbidity and an assumption of a categorical model in the absence of compelling evidence.

7. What misconception regarding psychiatric diagnosis is fuelled by high-profile media coverage of “duelling expert witnesses” in criminal trials? (p. 603)

8. Critics have suggested that DSM-5 is driven more by ________ than by science. (p. 607)
9. The diagnostic criteria used to classify individuals with mental disorders in DSM-5 (are/are not) based completely on scientific evidence. (p. 606)
10. One of the problems with DSM-5 is the high level of ________ among many of its diagnoses. (p. 606)

ANXIETY-RELATED DISORDERS: THE MANY FACES OF WORRY AND FEAR 610–615

**LO 15.4 DESCRIBE THE MANY WAYS PEOPLE EXPERIENCE ANXIETY.**
Panic attacks involve intense yet brief rushes of fear that are greatly out of proportion to the actual threat. People with generalized anxiety disorder spend much of their day worrying. In phobias, fears are intense and highly focused. In posttraumatic stress disorder, extremely stressful events produce enduring anxiety. Obsessive-compulsive disorder is marked by intensely disturbing thoughts, senseless or irrational rituals, or both. Learning theory proposes that fears can be learned via classical and operant conditioning and observation. Anxious people tend to catastrophize or exaggerate the likelihood of negative events. Many anxiety disorders are genetically influenced.

11. People with ________ ________ ________ spend an average of 60 percent of each day worrying. (p. 611)
12. People suffer from ________ ________ when they experience panic attacks that are repeated and unexpected and when they change their behaviour in an attempt to avoid panic attacks. (p. 611)
13. A ________ is an intense fear of an object or a situation that’s great out of proportion to its actual threat. (p. 612)
14. What are the symptoms of PTSD? Who is at high risk for developing this disorder? (p. 613)
15. Persistent ideas, thoughts, or impulses that are unwanted and inappropriate and cause distress are called ________. (p. 613)
16. Repetitive behaviours or mental acts initiated to reduce or prevent stress are called ________. (p. 613)
17. What anxiety disorder did the billionaire industrialist Howard Hughes suffer from? Can you name some other well-known people who suffer from this disorder? (p. 614)
18. People ________ when they predict terrible events, despite the low probability of their actual occurrence. (p. 614)
19. Anxious people tend to interpret ambiguous situations in a (negative/positive) light. (p. 614)
20. Many people with anxiety disorders harbour high levels of ________, ________, a fear of anxiety-related sensations. (p. 614)
MOOD DISORDERS AND SUICIDE 615–623

LO 15.5 IDENTIFY THE CHARACTERISTICS OF DIFFERENT MOOD DISORDERS.
The sad mood of major depression is the mirror image of the expansive mood associated with a manic episode, seen in bipolar disorder. Depression can be recurrent or, more rarely, chronic. Manic episodes are often preceded or followed by bouts of depression. Bipolar disorder is among the most genetically influenced of all mental disorders.

21. The state in which a person experiences a lingering depressed mood or diminished interest in pleasurable activities is called a(n) __________ __________ __________. (p. 616)

22. Over the course of a lifetime, more than ______ percent of us will experience a mood disorder. (p. 617)

23. Bipolar disorder is (equally common/more common) in women compared with men. (p. 621)

24. Twin studies suggest that the ________ of bipolar disorder may be as high as 85 percent. (p. 621)

LO 15.6 DESCRIBE HOW LIFE EVENTS CAN INTERACT WITH CHARACTERISTICS OF THE INDIVIDUAL TO PRODUCE DEPRESSION SYMPTOMS.
Stressful life events are linked to depression. Depressed people may face social rejection, which can amplify depression. According to Lewinsohn’s behavioural model, depression results from a low rate of response-contingent positive reinforcement. Aaron Beck’s cognitive model holds that negative schemas play an important role in depression, whereas Martin Seligman’s model emphasizes learned helplessness. Genes exert a moderate effect on the risk of developing depression.

25. Describe James Coyne’s interpersonal model of depression. (p. 618)

26. Lewinsohn’s behavioural model assumes that depression results from a (low/high) rate of response-contingent positive reinforcement. (p. 618)

27. Aaron Beck’s cognitive model focused on three components of depressed thinking: negative views of __________, the __________, and the __________. (p. 618)

LO 15.7 IDENTIFY COMMON MYTHS AND MISCONCEPTIONS ABOUT SUICIDE.
Myths about suicide include the misconception that talking to depressed people about suicide makes them more likely to commit the act, suicide is almost always completed with no warning, suicide risk decreases as severe depression lifts, most people who threaten suicide are seeking attention, and people who talk a lot about suicide almost never commit it.

29. __________ __________ and __________ __________ are associated with a higher risk of suicide than most other disorders. (p. 622)

30. Many or most individuals who commit suicide (communicate/do not communicate) their intent to others. (p. 622)

PERSONALITY AND DISSOCIATIVE DISORDERS: THE DISRUPTED AND DIVIDED SELF 623–629

LO 15.8 IDENTIFY THE CHARACTERISTICS OF BORDERLINE AND PSYCHOPATHIC PERSONALITY DISORDERS.
Borderline personality disorder is marked by instability in mood, identity, and impulse control. People with psychopathic personality are guiltless, dishonest, callous, and self-centred.

31. Of all psychiatric conditions, personality disorders are historically the (least/most) reliably diagnosed. (p. 624)

32. Persons with borderline personality disorder’s impulsivity and rapidly fluctuating emotions often have a __________ quality. (p. 624)

33. A diagnosis of __________ personality disorder is characterized by a lengthy history of illegal and irresponsible actions. (p. 625)

34. Most people with psychopathic personalities (are/are not) physically aggressive. (p. 625)

Answers are located at the end of the text.
35. From a psychological perspective, what's rare about the case of serial killer Aileen Wuornos? (p. 625)

**LO 15.9 EXPLAIN THE CONTROVERSIES SURROUNDING DISSOCIATIVE DISORDERS, ESPECIALLY DISSOCIATIVE IDENTITY DISORDER.**

Dissociative disorders involve disruptions in consciousness, memory, identity, or perception. The role of severe child abuse in DID is controversial. The sociocognitive model holds that social influences, including the media and suggestive procedures in psychotherapy, shape symptoms of DID.

36. In DID, the alternate identities are often very (similar to/different from) the primary personality. (p. 627)

37. The intriguing differences that researchers have identified among alters in their respiration rates, brain wave activity, eyeglass prescription, handedness, skin conductance responses, voice patterns, and handwriting (do/don’t) provide conclusive evidence for the existence of different alters. (p. 627)

38. According to the __________ model, DID arises from a history of severe abuse during childhood. (p. 627)

39. According to the sociocognitive model, how might a therapist contribute to the origin and maintenance of DID? (p. 628)

40. Some psychologists hold that the __________ have played a pivotal role in the DID epidemic. (p. 628)

**THE ENIGMA OF SCHIZOPHRENIA 629–634**

**LO 15.10 RECOGNIZE THE CHARACTERISTIC SYMPTOMS OF SCHIZOPHRENIA.**

The symptoms of schizophrenia include delusions, hallucinations, disorganized speech, and grossly disorganized behaviour or catatonia.

41. Despite the origin of the term schizophrenia, it shouldn’t be confused with __________ __________ __________. (p. 629)

42. Strongly held, fixed beliefs that have no basis in reality are called __________. (p. 630)

43. __________ symptoms represent serious reality distortions. (p. 630)

44. Most __________ in schizophrenia are auditory, usually consisting of voices. (p. 630)

45. People with schizophrenia can exhibit __________, in which their language becomes severely jumbled and skips from topic to topic in a disjointed way. (p. 630)

46. __________ symptoms involve motor problems, extreme resistance to complying with simple suggestions, holding the body in rigid postures, and refusing to speak or move. (p. 631)

47. It is widely acknowledged today that parents and family members (cause/don’t cause) schizophrenia. (p. 631)

48. Research indicates that one or more of four fluid-filled structures called ventricles, which cushion and nourish the brain, are typically (enlarged/diminished) in individuals with schizophrenia. (p. 632)

49. As illustrated in this figure, the lifetime risk of developing schizophrenia is largely a function of what? (p. 633)

50. A __________ model proposes that schizophrenia, along with many other mental disorders, is a product of genetic vulnerability and stressors that trigger this vulnerability. (p. 634)

**CHILDHOOD DISORDERS: RECENT CONTROVERSIES 634–637**

**LO 15.12 DESCRIBE THE SYMPTOMS AND DEBATE SURROUNDING DISORDERS DIAGNOSED IN CHILDHOOD.**

Individuals with autistic disorder are marked by severe deficits in language, social bonding, and imagination, usually accompanied by intellectual disability. Asperger’s disorder is a less severe form of autism on the autism spectrum. Children with ADHD experience problems with inattention, impulsivity, and hyperactivity, and often struggle with learning disabilities, difficulties processing verbal information, and poor balance and coordination. Some scholars have expressed concerns that ADHD is overdiagnosed in some settings, although others point to evidence that some children with ADHD are actually overlooked by many diagnosticians.

One of the most controversial diagnostic challenges is distinguishing children with ADHD from children with bipolar disorder.
51. Genetic influences alone (can/can't) easily account for an astronomical rise in autism's prevalence over the span of a decade. (p. 635)

52. Research studies have (failed/succeeded) in replicating the association between the MMR vaccine and autism. (p. 635)

53. Many parents of children with autism probably fall prey to _______ _______; they “see” a statistical association that didn’t exist. (p. 635)

54. What really accounts for most of the reported autism epidemic, despite many parents’ conviction that vaccines trigger the disorder? (p. 635)

55. The diagnosis of ADHD subsumes two subtypes, one with _______ and one without, in which _______ is predominant. (p. 636)

56. The first signs of hyperactivity may be evident in _______. (p. 636)

57. Although a high level of physical activity often diminishes as children with ADHD mature and approach adolescence, adjustment problems such as _______ and _______ _______ are frequent. (p. 636)

58. ADHD is associated with (increased/decreased) activation in the frontal areas of the brain. (p. 636)

59. List at least three conditions that must be first ruled out before an accurate diagnosis of ADHD can be made. (p. 637)

60. Because _______ to _______ percent of children with bipolar disorder share an ADHD diagnosis, a hypothesis to consider is that many children diagnosed with bipolar disorder are merely those with more severe symptoms of ADHD. (p. 637)

**DO YOU KNOW THESE TERMS?**

- demonic model (p. 600)
- medical model (p. 600)
- asylum (p. 600)
- moral treatment (p. 601)
- deinstitutionalization (p. 601)
- labelling theorists (p. 605)
- Diagnostic and Statistical Manual of Mental Disorders (DSM) (p. 605)
- prevalence (p. 605)
- comorbidity (p. 606)
- categorical model (p. 606)
- dimensional model (p. 606)
- insanity defence (p. 608)
- involuntary commitment (p. 609)
- somatic symptom disorder (p. 611)
- illness anxiety disorder (p. 611)
- generalized anxiety disorder (GAD) (p. 611)
- panic attack (p. 611)
- panic disorder (p. 611)
- phobia (p. 612)
- agoraphobia (p. 612)
- specific phobia (p. 612)
- social anxiety disorder (p. 612)
- posttraumatic stress disorder (PTSD) (p. 612)
- obsessive-compulsive disorder (OCD) (p. 613)
- obsession (p. 613)
- compulsion (p. 613)
- anxiety sensitivity (p. 614)
- major depressive episode (p. 616)
- cognitive model of depression (p. 618)
- learned helplessness (p. 619)
- manic episode (p. 621)
- bipolar disorder (p. 621)
- personality disorder (p. 624)
- borderline personality disorder (p. 624)
- psychotic personality (p. 625)
- antisocial personality disorder (ASPD) (p. 625)
- dissociative disorder (p. 626)
- depersonalization/derealization disorder (p. 626)
- dissociative amnesia (p. 626)
- dissociative fugue (p. 626)
- dissociative identity disorder (DID) (p. 627)
- schizophrenia (p. 629)
- delusion (p. 630)
- psychotic symptom (p. 630)
- hallucination (p. 630)
- catatonic symptom (p. 631)
- autism spectrum disorder (ASD) (p. 634)
- attention-deficit/hyperactivity disorder (ADHD) (p. 636)

**APPLY YOUR SCIENTIFIC THINKING SKILLS**

Use your scientific thinking skills to answer the following questions, referencing specific scientific thinking principles and common errors in reasoning whenever possible.

1. The popular media use many diagnostic labels, such as codependency or sexual or Internet addiction, that have minimal scientific support. Go online or to your local bookstore and locate at least three examples of these labels. How is each disorder or addiction diagnosed? Do the disorders fulfill the criteria for validity outlined by Robins and Guze (refer to Table 15.2)? Why or why not?

2. As the text notes, the insanity defence is the subject of many popular misconceptions. Locate two popular articles that discuss the use of this defence in a recent legal case. To what extent was the coverage of the insanity defence factually accurate? Did it fall prey to any of the myths described in the chapter? Explain.

3. Locate two to three articles or websites that argue for a connection between childhood vaccinations and the increase in autism rates. What evidence do they provide to support a link between vaccines and autism? Have the sources considered alternative explanations for the increase in autism rates?