# Depressive Disorders

CHAPTER 6

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## Chapter Outline



### *From the Case of Alina*

Alina had been a little scared about leaving home for college. She grew up in a small town, where she had always excelled at everything she attempted. Success was important to Alina. Her parents expected it, and she believed it was the key to her popularity.

At college, Alina made friends quickly and soon began enjoying campus life. She dated several men during her first year; then she met Jack early in her sophomore year. After 6 months of being a couple, though, their relationship began to sour. They quarreled often and felt increasingly tense when they were together. Jack suggested that they start dating other people, try to stay friends, and "see what happens." Alina was upset but also relieved.

Despite her relief, though, Alina has not bounced back emotionally. Now a 21-year-old college junior, she has been plagued with frequent headaches and stomach pains for about 3 months. She has no energy, has periods of feeling "down in the dumps," and sometimes feels hopeless and overwhelmed. A competent and motivated student, Alina's grades during the previous semester were lower than usual, and she has fallen behind in her current academic work. She started to skip classes, especially early in the morning, because she finds it difficult to get out of bed. Much of the time, she feels too tired to

**After reading this chapter, you will be able to answer the following key questions:**

- How are depressive disorders described and categorized in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*?
- What causes depressive disorders?
- What are the main treatments for helping people with depressive disorders?

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study. When she tries, her mind wanders, and she wastes time reading the same pages over and over. Even though she feels exhausted, on some nights she has difficulty falling asleep. Recently, she has begun to avoid her friends because she has to "fake it" to act like her former, happier self.

As she confided to her roommate, "It's as if I can't move; my body feels pulled down by some extra gravity in the room." She has also stopped calling her parents every week because she feels that they will just nag her to "snap out of it." In fact, she is increasingly annoyed with her parents for pushing her so hard all her life.

Alina finally went to the Student Health Center for help because of her physical problems, but a medical examination and initial lab tests failed to find any specific medical issue. Are Alina's symptoms a normal response to the end of a relationship, or do they reflect something more serious?

**mood disorders:** A group of mental disorders associated with serious and persistent difficulty maintaining an even, productive emotional state.

**mania:** An excited mood in which a person feels excessively and unrealistically positive and energetic.

**depression:** An extremely low, miserably unhappy mood along with other physical and cognitive symptoms.

**depressive disorders:** A group of emotional disturbances in which a person experiences a low, unhappy mood and has difficulty maintaining a more neutral or positive emotional state.

We saw in Chapter 5 how some **mood disorders** can include extremely high or agitated moods—known as **mania**—in which the person feels excessively and unrealistically positive. In *bipolar disorders*, individuals experience both episodes of depression and periods of mania. The most common mood disorder in Western cultures, however, involves only episodes of **depression**—a low, unhappy mood—without any manic (or hypomanic) periods. The term *unipolar depression* is sometimes used to refer to cases such as Alina's in which only depressive symptoms are present. **Depressive disorders** refer to a group of emotional disturbances associated with negative moods and persistent difficulty maintaining a comfortable and productive emotional state. However, these disorders usually involve more than just emotional symptoms; they also interfere with an individual's ability to work, to stay involved in relationships, to enjoy family life, and even to maintain good physical health. A depressive disorder can hinder almost all aspects of a person's functioning, and it can come and go many times in a person's life.

In this chapter, we consider the four main depressive disorders in detail—their physical, emotional, and cognitive symptoms; the main theories about their causes; the research data available to support these theories; and efforts at prevention and treatment. We then revisit the case of Alina to see how she fared and what her experience can tell us about depressive disorders.

## Depressive Disorders

Most people have bad moods and even entire days when they feel especially sad or unusually irritable. And most people feel dejected or demoralized when their grades are disap-

pointing or their friends let them down. Usually, these moods fade once individuals get past a difficult deadline, do something fun with a friend, or just catch up on their sleep (see Chapter 12). A depressive disorder is different from the common "bad mood" in the following ways:

1. The depressed mood seen in depressive disorders is, like Alina's, not temporary or easily shaken off. It typically persists for weeks, months, or even years.
2. A depressive disorder is severe enough to impair an individual's ability to work or interact with friends or family.
3. People suffering from depressive disorders show a cluster of other physical and behavioral symptoms, such as changes in appetite, sleep disturbance, or loss of interest in their usual pursuits. One way to think about this is that depression is a *full-body disorder*.

Major depressive disorder, the most common mood disorder and one of the most frequent mental disorders, is the number one cause of disability and a leading cause of suicide worldwide (World Health Organization [WHO], 2017a). An estimated 4.4% of the global population suffers from depression at any given time, with an 18% increase in the number of people living with depression between 2005 and 2015 (WHO, 2017a).

Depression affects approximately 17 million adults in the United States—over 7% of the population—every year (Kessler et al., 2005; National Institute of Mental Health, 2017b). In fact, researchers using prospective rather than retrospective data have found that *up to half of all individuals* in the general population may meet criteria for depression at some point in their life (Moffitt et al., 2010). Furthermore, almost half (45%) of American college students report difficulty functioning in the last year due to depression, according to the National College Health Assessment, which examined data from 68,000 students from 98 colleges and universities (American College Health Association, 2019).

Whereas depression appears relatively evenly across different ethnic groups in the United States, it is significantly lower in Asian American populations (Brody et al., 2018), whereas African Americans and Mexican Americans show significantly higher depression chronicity and significantly lower rates of treatment than Caucasians (González et al., 2010). The prevalence of depression decreases as family income levels increase (Brody et al., 2018). Women (10.4%) are almost twice as likely as were men (5.5%) to be diagnosed with depression (Brody et al., 2018), a difference to which we will return when we consider its causes in the "A Conversation With" feature later in this chapter. Men may experience depression in a different form—with higher rates of anger attacks/aggression, substance abuse, and risk taking compared with women (Martin et al., 2013), which may lead to underdiagnosing depression in this group.

As with other mental disorders, depression is often exacerbated by comorbid problems (Gadermann et al., 2012), ranging from hypertension (high blood pressure) and chronic lung diseases to diabetes; more than 25 medical diseases are disproportionally more likely in people with depression (Schoepf et al., 2014). Moreover, more than 90% of people with depression also meet criteria for another *Diagnostic and Statistical Manual (DSM)* disorder, with the most common being anxiety disorders, substance use disorders, and attention-deficit/hyperactivity disorder (ADHD; Gadermann et al., 2012; Kessler et al., 2005). Indeed, substance use disorders may reflect the efforts of people with

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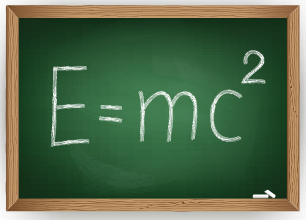
Many famous and creative people have suffered from depression, including Dwayne ("The Rock") Johnson. Whereas his roles in movies highlight a "tough guy" persona, Johnson has recently opened up about his lifelong struggles with depression, including frequent crying spells. In a recent interview with *People* magazine (Ellis, 2021), Johnson encouraged others to reach out for help: "I feel like the most important thing, obviously, is communicating and realizing that asking for help when you're down and you're feeling wobbly or when you're depressed is actually the most powerful thing you can do. Asking for help is not a weakness. As a matter of fact, asking for help is our superpower, and men, especially us, we fall into this trap of being really averse to vulnerability, because we always want to be strong and feel like we can take on the world."

Toby Allen, a prominent artist, completed a project in which he expertly illustrated a variety of mental disorders as anthropomorphized monsters, such as the depression monster shown here.


"Real Monsters" illustration by Toby Allen 2013. Reprinted with permission.




**MAPS - Attempted Answers**

depression to relieve their depressive symptoms (Grant et al., 2006). In other cases, people develop mood disorders as a result of their chronic struggles with substance abuse. In either case, depression makes the substance use disorder worse and more difficult to treat (McKowen et al., 2013).

Anxiety and depression often co-occur, and their comorbidity may be even more common in children and adolescents (Cummings et al., 2014). In such cases, individuals feel overwhelmed with negative emotions—despair and guilt over past problems, apprehension and fear about future threats—and they frequently have difficulty sleeping. In fact, the overlap between depression and anxiety is so extensive (over 50%; Hirschfeld, 2001) that some investigators believe they are both parts of a larger emotional state known as **negative affect**, made up of a mixture of anxious and depressive symptoms (Kendall & Watson, 1989). Low positive affect seems to distinguish depression from anxiety, whereas high negative affect is associated with both depression and anxiety (Riskind et al., 2013).

**negative affect:** An emotional state that is a mixture of anxious and depressive symptoms.

Depressive disorders tend to fall into two major categories: major depressive disorder and persistent depressive disorder (formerly called dysthymia). The core symptoms associated with these two categories overlap considerably, but they differ in severity and course. Major depressive disorder is typically more severe and episodic, whereas persistent depressive disorder is milder but more chronic.

## Major Depressive Disorder

**major depressive disorder:** One of the most severe forms of depression, characterized by constant sadness or despair, irritability, guilt, physical symptoms, insomnia, and lack of energy.

The diagnostic criteria for **major depressive disorder** are described in Table 6.1.

The predominant mood in major depressive disorder is typically a dull despair, a constant sadness that may leave the person thinking that nothing is worthwhile. A passage from Samuel Coleridge's "Dejection: An Ode," captures the oppressive sadness that often engulfs the person with depression:

*A grief without a pang, void, dark, and drear,*
*A stifled, drowsy, unimpassioned grief,*
*Which finds no natural outlet, no relief,*
*In word, sigh, or tear—*

**anhedonia:** Loss of the ability to enjoy activities central to a person's life.

Along with this profound sadness, the second most sensitive symptom of depression is **anhedonia**, which means that the person no longer gets joy or pleasure out of things or

**TABLE 6.1** The *DSM-5* in Simple Language: Diagnosing Depression

The person shows 5+ of the following 9 symptoms most days for 2+ weeks:

1. Sad mood
2. Lack of interest or pleasure in activities (anhedonia)

Physical changes, like:

3. Low energy
4. Sleeping more or less than usual
5. Eating more or less than usual
6. Moving faster or slower than usual

Changes in thinking, like:

7. Thinking negative thoughts about themselves
8. Trouble making decisions
9. Thoughts of suicide

*Source:* Adapted from the American Psychiatric Association (2013a).

activities that they used to like. Thus, the avid football fan loses interest in the game, the involved and active parent finds it difficult to attend to child care, or the once-productive employee becomes disengaged from their work.

In fact, these two features together—sadness (negative affect) and loss of interest or pleasure (lack of positive affect)—can accurately assess depression (Li et al., 2007). For instance, the Patient Health Questionnaire-2 (PHQ-2; Kroenke et al., 2003), which uses these symptoms in a two-item depression screening test in primary care settings, has good sensitivity (83%) and specificity (90%). The sensitivity of a clinical test refers to its ability to correctly identify those people with the disorder, whereas the specificity of a clinical test refers to its ability to correctly identify those people who do not have the disorder (Lalkhen & McCluskey, 2008). In other words, just using these two main symptoms of depression—depressed mood and anhedonia—can allow clinicians to correctly distinguish between those who have and do not have depression over 80% of the time.

As stated earlier, though, depression is a full-body disorder, rather than merely sad mood. Physical symptoms often accompanying major depressive disorder include change in appetite, persistent fatigue, and complaints about an upset stomach or a variety of aches and pains. As in the chapter-opening case about Alina, depression may cause the person to feel so drained of energy that body movements are slowed or reduced.

About three quarters of people with depression suffer from insomnia (see Chapter 12; Nutt et al., 2008). People with depression may have trouble falling asleep, but their most common sleep disturbances involve waking up during the night—or too early in the morning—and being unable to return to sleep, and they often have disordered REM (rapid eye movement) sleep (Hu et al., 2010). In fact, insomnia is a significant risk factor for the development of new or recurrent episodes of major depressive disorder in the first place (Perlis et al., 2006).

Although it is not an official criterion of major depressive disorder, people with depression also tend to suffer impairment in immune system functioning, which increases their vulnerability to infections and other illnesses (Tanaka & Kinney, 2011). Such impairment is particularly apparent among older people who are depressed and among people whose depression is severe (German et al., 2006).

Cognitive symptoms associated with depression include a sense of guilt and worthlessness and difficulty concentrating, even with simple daily activities such as reading or watching TV. People who are depressed often postpone decisions for fear of making mistakes and struggle to make even minor decisions such as what to cook for dinner. They may feel demoralized and hopeless. They are often self-critical, blaming themselves for their problems and feeling pessimistic about their lives and futures. Their recollection of


Skunky suspected he may be clinically depressed because spraying cocky teenagers with his vile odor did not give him the same sustained pleasure that it once did . . .

Cartoon written by Brian L. Burke and illustrated by Leslie B. Goldstein.

the past is also grim and unforgiving. This kind of thinking can lead to suicide attempts, as discussed in Chapter 5.

Because of their somatic (body) symptoms and preoccupation with physical well-being, many individuals with depression first seek relief from their primary care providers, such as Alina's visit to the Student Health Service of her college. In fact, major depressive disorder is one of the most common problems encountered in family practice and by primary care providers (Katon & Russo, 1989). However, these practitioners must be careful in their diagnoses because the physical symptoms associated with major depressive disorder can also stem from other conditions, including cancer, tumors, infections, hypothyroidism, nutrition deficiencies, and drugs such as steroids and narcotics (Tollefson, 1993).

## Course and Recurrence

Depressive disorders can occur across the entire lifespan (Hamilton, 1989). Although they usually first appear when people are in their late 20s—and are especially common between ages 20 and 45—depressive disorders can also occur in children and adolescents (Klerman, 1988; Ruderman et al., 2013), and they are a significant problem among older people (e.g., Solhaug et al., 2012).

Some episodes last only a couple of weeks, whereas others can last for years. The average duration of an untreated episode is between 8 to 10 months (Tollefson, 1993). However, perhaps in as many as two thirds of the cases, episodes of major depressive disorder are recurrent, meaning that a person recovers or improves for a period of time, only to suffer another episode at a later time (Yiend et al., 2009). According to Judd, the long-term course of major depression "typically consists of a dynamic and fluctuating continuum of different levels of depressive symptom severity, mostly below the diagnostic threshold for MDEs [major depressive episodes]" (2012, p. 169). Longitudinal research over periods as long as 20 years finds that people with depression suffer an average of 5 or 6 episodes in their lifetimes (Winokur, 1986; Yiend et al., 2009). As might be expected, recurrent depressive episodes take an ever-growing toll on a person's relationships and productivity.


Many professional athletes have opened up about their bouts with depression, including championship tennis player Naomi Osaka (shown here in 2018), gymnast Simone Biles, swimmer Michael Phelps, and NBA basketball players Kevin Love and DeMar DeRozan (ESPN, 2018; Futterman, 2021). Other celebrities who have suffered from depression include Meghan Markle, Bruce Springsteen, Kristen Bell, and Demi Lovato (USA Today, 2021).

Leonard Zhukovsky/Shutterstock

Results of a worldwide study showed that 15% of the population from high-income countries (compared to 11% from low/middle-income countries) were likely to get depression over their lifetime, with 5.5% having had depression in the last year (Bromet et al., 2011). The United States had one of the highest lifetime rates of depression (19.2%), along with France (21%) and the Netherlands (17.9%; Bromet et al., 2011). However, like for PTSD (see Chapter 9), wealthier countries have more depression but, within those countries, higher income is somewhat protective and lowers depression rates (Brody et al., 2018). Cross-national data also reveal that depression can lead to many other problems, including difficulties in role transitions (e.g., low education, high teen childbearing, marital disruption), reduced role functioning (e.g., low work performance, low earnings), elevated

(a)  (b) 

**MAPS - Superficial Syndromes**

These photos depict the superficial view of depression in saguaro cacti near Tucson, Arizona. The photo in *(a)* shows a single episode, whereas the photo in *(b)* illustrates the more common recurrent pattern, with more than one major depressive episode in the cactus's lifespan.

risk of a wide range of other mental disorders, and increased risk of early mortality due to physical disorders and suicide (Kessler & Bromet, 2013).

## Subgroups of Major Depressive Disorder

Differences in the pattern of depressive episodes and in their predominant symptoms have led mental health providers to propose subcategories of major depressive disorder. These subtypes, coded as **specifiers** in the *DSM*, may have different causes and prognoses and may respond best to different treatments. Of the nine different specifiers available in the *DSM-5*, clinicians often use one of the following six to describe the current or most recent episode of major depressive disorder:

**specifiers:** A descriptor used in the *DSM-5* to indicate the likely course, severity, and specific symptom characteristics of certain mental disorders.

1. **With atypical features.** This specifier applies to approximately 15–29% of individuals diagnosed with major depressive disorder or persistent depressive disorder (Thase, 2007). Rather than losing their appetite or having difficulty sleeping, people with atypical features sleep and eat more than usual, often gaining significant weight (Pae et al., 2009). They may even cheer up briefly following some positive event, such as a phone call from an old friend. In addition to the usual fatigue and psychomotor retardation that can accompany depression, people with atypical features may experience "leaden paralysis," heavy feelings in their arms or legs (like Alina's description of being weighed down by gravity). Many of these individuals also show such intense sensitivity to rejection that it disrupts their social relationships. This type of major depressive disorder is more likely to occur among younger people, and it frequently has a more chronic course than typical depression (Stewart et al., 2007). In addition, current findings suggest that atypical depression has preferential response to specific types of antidepressant medications (monoamine oxidase inhibitors over tricyclics, as described in more detail later in the chapter; Pae et al., 2009). However, the diagnostic reliability and validity of atypical depression remains elusive and open to further evolution (Łojko & Rybakowski, 2017).
2. **With melancholic features.** People with melancholic features tend to display severe anhedonia, along with a lack of reactivity to good events (i.e., no temporary mood

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Depression with melancholic features, a severe variety of major depressive disorder, occurs frequently in older people. People suffering this disorder are unable to feel pleasure from almost any activity, and their mood is usually worse in the morning.

brightening), sleep disturbances that involve being unable to get back to sleep after awakening as early as 4 or 5 a.m., changes in bodily activity characterized either by extreme agitation or slowness, and significant weight loss. The depressed mood has a distinct quality (sometimes called "empty mood") and is typically worse in the morning. Melancholic symptoms are more likely to be seen in older people with depression (Mallinckrodt et al., 2005). In one study, 36% of people with major depressive disorder met criteria for the melancholic subtype (Melartin et al., 2004). However, the validity of this subtype has been called into question, as there appear to be no major differences in comorbidity or course of depression between melancholic and nonmelancholic subtypes (Melartin et al., 2004).

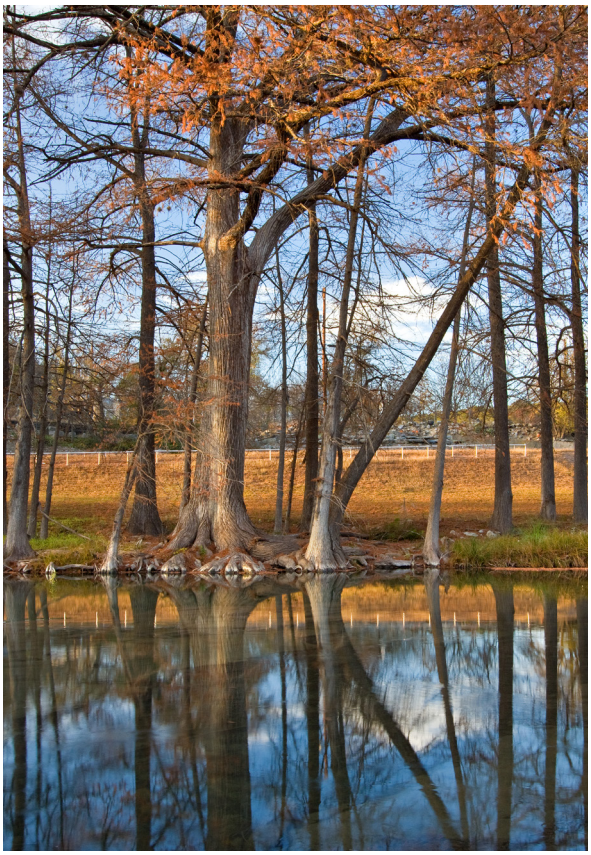
3. **With catatonia.** This specifier is used for depression marked by extreme psychomotor disturbances; similar problems are also observed in some cases of schizophrenia and bipolar disorders. Individuals may stay immobile for long periods or stay fixed in bizarre postures, sometimes showing a *waxy flexibility* that allows them to be manipulated like a toy action figure. Others may engage in agitated, purposeless behavior; may resist any attempt to move them; or may mimic every movement someone else has made, a condition called *echopraxia*. Some become mute or engage in *echolalia*, a parrot-like repetition of other people's speech (American Psychiatric Association, 2013a). Evidence suggests that catatonia may be best treated by benzodiazepine medication or electroconvulsive therapy (ECT; discussed in more detail later in the chapter; Carroll, 2001).
4. **With psychotic features.** Individuals with severe depression may experience psychotic symptoms, including delusions (e.g., false beliefs about being persecuted) or hallucinations (e.g., seeing or hearing things that are not actually there). Usually, these symptoms are *mood congruent*, meaning that they are consistent with the person's depressed thinking (Winokur et al., 1985). For example, people with depression who are preoccupied with death and dying might think that others are trying to kill them, or they might hear a voice telling them to kill themselves. *Mood incongruent* psychotic symptoms are less common in depression (more common in schizophrenia) and might involve the delusion that someone is trying to insert thoughts in their minds through electromagnetic airwaves. Individuals with depression and psychotic symptoms present a more severe course of illness, as indicated by longer hospitalizations and lower rates of remission (Buoli et al., 2013).

   Moreover, a proportion of individuals who initially appear to have major depressive disorder will prove, in time, to instead have a bipolar disorder, discussed in depth in Chapter 5. This change in diagnosis is more likely in those with a family history of bipolar disorders, early onset of depression, or psychotic features (American Psychiatric Association, 2013a). In a prospective longitudinal community study, a total of 3.6% of the initial unipolar major depression cases subsequently developed mania or hypomania, with particularly high rates (9%) in adolescent onset depression before age 17 (Beesdo et al., 2009). However, 13% of people who had major depressive disorder with psychotic features had a manic or hypomanic episode within a 2-year follow-up period, making it especially important to reassess and follow up with people who show this depression subtype (DelBello et al., 2003).
5. **With seasonal pattern.** This specifier, also known as **seasonal affective disorder**, refers to depressive episodes that have a clear seasonal pattern (Flaskerud, 2012). For example, some individuals experience depressive episodes only during the winter months, and then spontaneously recover in the spring. This type of depressive

**seasonal affective disorder:** Mood disorders that are linked to a particular season of the year; probably caused by shifts in overall exposure to light.

disorder is most commonly seen in locations where winter days are short and exposure to daylight is limited—that is, the rate in low-latitude countries is significantly lower than that in higher-latitude countries in the Northern Hemisphere (Whitehead, 2004). Similar to atypical depression, winter depression often presents with low energy, extreme fatigue, and greater than normal amounts of sleeping, along with increased appetite, often characterized by a craving for carbohydrates. Prevalence estimates of seasonal depression in community-based surveys have ranged from 1.4–9.7% in North America, 1.3–3.0% in Europe, and 0.0–0.9% in Asia (Levitt et al., 2000). However, in a study of over 6,500 participants, seasonal differences in severity or type of depressive symptoms were absent or small in effect size (Winthorst et al., 2011).

6. **With peripartum onset.** This specifier is used for cases of depressive disorder that begin during pregnancy or within 4 weeks after the birth of a child in 3–6% of women (American Psychiatric Association, 2013a). Young mothers and mothers with a low education level have a heightened risk of developing depression following delivery (Reck et al., 2008). The symptoms are similar to typical depressive disorder, but they tend to fluctuate more often and are frequently accompanied by attacks of severe anxiety and obsessive worries about harm befalling the baby. Women with peripartum episodes often feel guilty because their symptoms are at odds with the joy they were taught to expect following the birth of a baby. This shame often makes these women less willing to talk about their problems, thus making successful treatment less likely (Wisner et al., 2010). Many pediatrician offices screen for depression with new mothers to try to catch potential cases of depression before they worsen. However, existing empirical evidence casts doubt on whether peripartum onset depression actually constitutes a special category of the disorder (Cunningham et al., 2013). Other researchers question whether the peripartum period is even associated with any increased rate of depressive disorders (O'Hara et al., 1990). What does seem clear is that, if a mother suffers a depressive disorder shortly after delivering one child, she is at greater risk for depressive disorders following future deliveries (Depression Guideline Panel, 1993). In fact, this recurrence risk is especially high—30–50% with each subsequent delivery—once a woman has had a peripartum episode with psychotic features (American Psychiatric Association, 2013a).

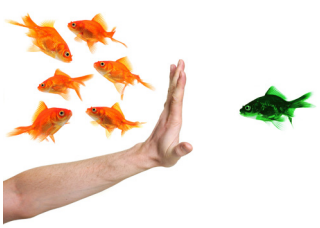


Sarah Fields Photography/Shutterstock

The Guadalupe River in Kerrville, Texas, near the mental hospital where Andrea Yates resides. Yates is a former Houston, Texas, resident who confessed to drowning her five children in their bathtub on June 20, 2001. In her 2006 retrial, a jury found that Yates was not guilty by reason of insanity because of her mental disorder (which, in *DSM-5*, would be diagnosed as major depressive disorder with peripartum onset and psychotic features, recurrent). Yates claimed that she killed her children to protect them from Satan. Yates was consequently committed by the court to the North Texas State Hospital and later to a mental hospital in Kerrville, Texas, where she remains today (Hlavaty, 2014).

## Cultural Factors and Limitations in Diagnosing Major Depressive Disorder

As noted in Chapter 1 and throughout this book, it is essential to consider cultural context when diagnosing any mental disorder. For instance, some countries describe depression using terms like "nerves" or "neurasthenia" rather than words that evoke suffering or sadness as per the Western construct of depression (Lehti et al., 2010). This may explain the high variation of lifetime depression rates worldwide, with the United States topping the list (Bromet et al., 2011). In addition, clinicians need to realize that, in most cultures, an individual with depression is more likely to present with somatic symptoms, most commonly low energy/fatigue or insomnia, rather than overt mood or anhedonia symptoms per se (American Psychiatric Association, 2013a). For instance, Alina's depression (from the chapter-opening case) began with frequent headaches and stomach pains, which is the most



MAPS - Prejudicial Pigeonholes

typical presentation of depression or anxiety in primary care (Kirmayer, 2001). However, the majority (80%; Kirmayer, 2001) of primary care patients will acknowledge a psychosocial dimension to their distress when asked ("What do you think might be causing your headaches and stomach pains, Alina?"). The key here is for the clinician to be culturally competent and able to communicate effectively with—and understand communication from—clients with a wide range of backgrounds and worldviews.

**bereavement:** Feelings of sadness that follow the death of a loved one and that are best characterized as normal grief reactions to loss.

Furthermore, the link between depression and **bereavement**—grieving the loss of a significant person in one's life—has been muddied by the current diagnostic manual. Its predecessor, *DSM-IV*, identified in the introduction that, to qualify as a mental disorder, "the syndrome or pattern must not be merely an expectable and culturally sanctioned response to a particular event, for example, the death of a loved one" (American Psychiatric Association, 1994, p. xxi). This bereavement exclusion prevented clinicians from diagnosing a major depressive episode in someone who had recently (within the past 2 months) lost a loved one. The approach of both the *DSM-5* and *ICD-11* has shifted, however, in that it is now possible to diagnose a major depressive disorder in individuals who are actively in mourning (Maj, 2012). The *DSM-5* provides a footnote outlining what it considers key differences between bereavement/grief, which typically involves feelings of emptiness and loss and waves of dysphoria (along with positive emotions) associated with thoughts of the deceased, and depression, which instead entails worthlessness and self-loathing as well as persistent sad mood (American Psychiatric Association, 2013a).

⚠ ***DSM-5-TR* Update:** The manual now outlines "disordered grief" in the recent addition as a new disorder, ***prolonged grief disorder***. Perhaps as many as 10 percent of people experience a pervasive inability to move past grief over the loss of a loved one, and these symptoms may affect their daily functioning (Moran, 2021). To qualify for this new diagnosis, the grieving person must display "intense yearning/longing" for the deceased, along with preoccupation with memories of their loved one that result in disruptions to their own relationships, identity, and/or sense of life meaning a year or more after the loss (APA, 2022). This diagnosis is already in ICD-11 and may become more prevalent following the global COVID-19 death toll.

This grief issue is closely linked to the more general question of what constitutes a mental disorder versus homeostatic reactions to major life events (Maj, 2012). A major depressive disorder is indeed an "expectable response" to the death of a loved one: In the United States, about 50% of all widows and widowers meet criteria for depression at some time during the first year of bereavement (Zisook et al., 1997). As pointed out in Chapter 1, some scholars now worry that "normal grief will become major depressive disorder, thus medicalizing and trivializing our expectable and necessary emotional reactions to the loss of a loved one and substituting pills and superficial medical rituals for the deep consolations of family, friends, religion, and the resiliency that comes with time and the acceptance of the limitations of life" (Frances, 2012). Parker (2007) further claims that depression was already overdiagnosed even before the slackening of the bereavement exclusion. However, others argue that the removal of the grief exclusion allows clinicians to treat major depressive disorder sooner and that it eliminates the arbitrary notion that grief should resolve in 2 months (the time frame included in the previous exclusion).

## Persistent Depressive Disorder

**persistent depressive disorder:** A depressive disorder in which depressed feelings and low self-esteem are present for at least 2 years but not as intensely as in major depressive disorder.

The *DSM-5* conceptualizes chronic forms of depression in a modified way. What was referred to as dysthymia in the *DSM-IV* now falls under the category of **persistent depressive disorder**, which includes both chronic major depressive disorder and the previous dysthymic disorder. An inability to find scientifically meaningful differences between these two conditions led to their combination (e.g., Blanco et al., 2010; Klein et al., 2006), with the same specifiers included as described previously for major depressive disorder in order to identify different presentations and courses.

In adults, the diagnosis of persistent depressive disorder is reserved for individuals who have had difficulties with chronically depressed mood and related symptoms for at least 2 years. In children, the prominent mood is often irritability rather than depression, and the minimum duration of symptoms is 1 year. Table 6.2 lists the key criteria for this diagnosis. Persistent depressive disorder tends to develop more gradually than major depression and typically does not involve an acute disruption of the person's life. It is analogous to a nagging cold that is never severe but, over time, can drag a person down (like the one you get during exam week). People with persistent depressive disorder often feel inadequate and brood about the past. They appear almost accustomed to their demoralized feelings, and, in some cases, will say such things as "I've always felt like this." Persistent depressive disorder is associated with increased risk for major depression (Keller et al., 1993), which suggests that the two disorders may share a common causal pathway.

**TABLE 6.2** The *DSM-5* in Simple Language: Diagnosing Persistent Depression

| |
|---|
| Consistently depressed (or irritable) mood for at least 1 year in children or 2 years in adults, which includes 2+ of the following 6 symptoms: |
| Changes in (1) appetite or (2) sleep; |
| Reduction in (3) energy levels, (4) self-esteem, (5) ability to concentrate, or (6) hope. |

*Source:* Adapted from American Psychiatric Association (2013a).

Around the world, there is little regional variation in prevalence estimates of persistent depressive disorder, which peaks at around 50 years old, with slightly higher frequency in women than men (Charlson et al., 2013). Lifetime prevalence of persistent depressive disorder in the United States is estimated at around 4% (Blanco et al., 2010), and it generally lasts far longer than the 2 years required for diagnosis; in one study, the median time to recovery was well over 4 years (52 months) and was associated with a high risk of relapse (Klein et al., 2006). However, due to its less acute (but more chronic) presentation than major depressive disorder, individuals with this disorder continue to face substantial unmet treatment needs (Blanco et al., 2010).

As many as one quarter of all people with major depressive disorder experience **double depression**, in which a major depressive episode is preceded or followed by persistent depressive disorder (Keller et al., 1992; Wells et al., 1992). Both persistent depressive disorder and double depression involve a similar course, which is worse than the course of major depression only, with longer duration and more relapses (Rhebergen et al., 2009).

**double depression:** A condition in which both major depression and dysthymia are experienced.

Despite findings indicating the utility of distinguishing between chronic and nonchronic forms of depression (Klein et al., 2006), persistent depressive disorder is a heterogeneous diagnosis that encompasses many different depressive conditions, as well as anxiety disorders (Chapter 7) and personality disorders (Chapter 16), and without clear evidence of its validity as a separate diagnostic entity (Rhebergen & Graham, 2014). It is feasible, therefore, that the disorder might eventually become a subtype or specifier of major depressive disorder in future *DSM*s but not as of *DSM-5-TR* (2022).

## Premenstrual Dysphoric Disorder

Based on scientific evidence, **premenstrual dysphoric disorder (PMDD)** has been moved from the *DSM-IV* Appendix B, "Criteria Sets and Axes Provided for Further Study," to the main body of *DSM-5* (American Psychiatric Association, 2013a). Premenstrual disorders, including PMDD and premenstrual syndrome (PMS, which is not a *DSM* disorder), involve significant mood, physical, and behavioral changes occurring during the premenstrual phase of the menstrual cycle (Sigmon et al., 2012). Typical symptoms mirror depression and include sad mood, anxiety, irritability, social withdrawal, sleep issues, food cravings, and physical pains like breast tenderness or headache (Sigmon et al., 2012). PMS occurs in 20–40% of menstruating women, whereas PMDD affects 2%–9% (Clayton, 2008). Note that, whereas PMS and PMDD have similar symptoms, those in PMDD are more severe/impairing and more strongly mood-related and motivation-sapping (Sigmon et al., 2012).

**premenstrual dysphoric disorder (PMDD):** A mental disorder that involves significant mood swings and irritability that occur during most menstrual cycles and then remit when menstruation occurs.

Even though the vast majority of women (95%) notice premenstrual changes, the symptoms of PMDD are stronger than these and enough to cause significant impairment (Craner et al., 2014). PMDD negatively impacts a plethora of social domains especially, ranging from dissatisfaction with relationships to more interpersonal difficulties in general (Di Guilio & Reissing, 2006); consequently, roughly 15% of women with PMDD report making a suicide attempt (Cunningham et al., 2009). PMDD also causes problems in health and occupational functioning (Craner et al., 2014), resulting in more healthcare service use and less productive (and more missing of) work (Borenstein et al., 2007).

Critics cite two concerns with PMDD becoming a full-fledged disorder, rather than an appendix listing in the *DSM-5*. First, one major concern is the potential stigmatization of women, who are already diagnosed with depression far more frequently than men

are. Second, some worry that the diagnostic criteria for PMDD are too easily met, which may result in unnecessary medical treatment of individuals whose symptoms are mild (Paris, 2013).

## Disruptive Mood Dysregulation Disorder

**disruptive mood dysregulation disorder:** A mental disorder in children between 6 and 18 years old that involves persistent irritability and temper outbursts that are grossly out of proportion to the situation or developmental level.

To address concerns about potential overdiagnosis and overtreatment of bipolar disorder in children, another new diagnosis that is classified within the depressive disorders is **disruptive mood dysregulation disorder**. This diagnosis was included in the *DSM-5* for children from age 6 up to age 18 who exhibit chronic, persistent irritability (American Psychiatric Association, 2013a). Alongside this irritability, the child has recurrent temper outbursts that are manifested verbally (e.g., screaming rages) and/or behaviorally (e.g., physical aggression) and that are grossly out of proportion to the situation or provocation or to the child's developmental level.

Irritability is a mood symptom present in the criteria for a large number of mental disorders and refers to easy annoyance and touchiness that can manifest in anger and temper outbursts (Stringaris, 2011). Prevalence rates of irritability range from 3% for severe, chronic irritability strictly (Brotman et al., 2006) to as high as 20% for milder versions (Pickles et al., 2010). A possible advantage of this new diagnosis is to cement what experienced clinicians already know—that depression frequently does not present with low mood (Miller, 2013). Irritability, anger, anhedonia, or disruptive behavior may be equally defining of a depressive disorder, and difficulty with regulating such emotion may be—at least for some—the primary deficit, more apparent than low mood per se (Miller, 2013). Most findings so far suggest that chronic, severe irritability is not a developmental presentation of mania or a precursor to bipolar disorder, thus justifying this new disorder for use instead of bipolar disorder in children (Mikita & Stringaris, 2013). In fact, children who suffer from disruptive mood dysregulation disorder typically develop major depressive disorders or anxiety disorders as adults, rather than bipolar disorders (Roy et al., 2013). However, offspring of parents with bipolar disorder are more likely to meet criteria for disruptive mood dysregulation disorder than are offspring of community control parents, suggesting that there may be at least some link between those two disorders (Sparks et al., 2014).

Critics contend that there is little research to support this new disorder (Paris, 2013), which is puzzlingly listed in the *DSM-5* under depressive disorders, rather than under disruptive behavioral disorders within the disorders of childhood and adolescence (see Chapter 3). In any case, because children often have temper tantrums and typically grow out of them, this diagnosis should be used only with extreme caution (Paris, 2013). In one community health clinic, 31% of youth met criteria for disruptive mood dysregulation disorder, and the disorder had extremely high (96%) overlap with oppositional defiant disorder (Freeman et al., 2016). Note that *ICD-11*, the World Health Organization's diagnostic manual, instead lists "6C90.0Z Oppositional defiant disorder with chronic irritability-anger," which many researchers recommended as a better fit for the available data and to avoid overdiagnosing children with mood disorders (Lochman et al., 2015; Runions et al., 2016).

## Section Review

Depressive disorders are among the most common mental disorders in the world and:

- are diagnosed in women about twice as often as in men;
- can occur at any age, but develop most frequently when people are in their 20s;
- are so often accompanied by anxiety that some experts believe that depression and anxiety are both components of one emotional state called (high) negative affect; and
- also involve a low positive affect, characterized by anhedonia, a loss of interest in previous activities.

The two most common depressive disorders are:

- major depressive disorder, characterized by at least one, but usually several, major depressive episodes throughout one's lifetime;

- persistent depressive disorder, characterized by chronically depressed mood lasting for at least 2 years in adults or 1 year in children;
- often experienced together or in sequence, a condition known as double depression; and
- sometimes separated into subtypes via specifiers based on their different symptom presentations and courses.

The other depressive disorders in the *DSM-5* are both new and include:

- premenstrual dysphoric disorder—disruptive mood swings and irritability that occur during most menstrual cycles; and
- disruptive mood regulation disorder, which can be diagnosed in children who show profound irritability and temper tantrums beyond what is developmentally expected.

Both of these diagnoses have been subject to criticism about stigmatizing women and children and labelling typical hormonal or developmental trajectories as disordered.

## Causes of Depressive Disorders

Now that you understand how to detect depressive disorders, let's move on to a discussion about causality. How do these disorders develop in the first place? Like most other mental disorders, depressive disorders result from the complex interplay of biological, psychological, and social factors. And, as with most other disorders, the relative contribution of each of these factors is probably different in each person's case.

People from Western countries strongly endorse social stressors as being the chief causes of mental disorders (Link et al., 1999; Nakane et al., 2005). Social factors covered in these surveys included stressful life events, traumatic experiences, family problems, and social/economic disadvantages. One study compared public perceptions of depression in Japan and Australia, again illustrating that the public had a predominant belief in social causes and risk factors. However, the belief in weakness of character as a cause of depression was more prominent in Japan (Nakane at al., 2005). In the United States, life stress (a social cause) was endorsed by 95% of participants as being the most likely cause of a person's depression, whereas biological causes of a "chemical imbalance in the brain" (73%) and a genetic or inherited problem (53%) were seen as second and third most likely causes, respectively (Link et al., 1999). Public notion of the cause of depression is of particular interest and concern because it may reduce the likelihood of seeking professional help and support from others (e.g., if "weakness of character" is seen as the cause) or result in improper frontline treatment (e.g., if "chemical imbalance" is seen as the cause).

We discuss the biopsychosocial approach to understanding and sorting through the potential causes of depression next.

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Most researchers believe that depression results from an interaction between biological, psychological, and social factors, consistent with a biopsychosocial model of abnormal behavior as discussed in Chapter 2.

### Biological Causes of Depressive Disorders

Most researchers—like the public—view depressive disorders as stemming at least partly from biological causes. Biological systems calibrate our response to stress based on early life experiences, which may then enhance the risk of depression following exposure to stressful events later in life (Patten, 2013). Biological findings regarding depression and bipolar disorders (see Chapter 5) show some interesting similarities, but also key differences, suggesting that these are distinct entities (Hickie, 2014).

### Genetic Influences on Depressive Disorders

It is estimated that 30–40% of the risk for major depression is heritable (Heim & Binder, 2012), defined as the proportion of observable differences in the disorder between individuals that is due to genetic differences. Recall from Chapter 2 that one way to explore the contribution of genetic factors to disorders is to contrast the appearance of a disorder in identical (*monozygotic*) twins who have exactly the same genes versus nonidentical (*dizygotic*) twins who share only about 50% of their genetic endowment. If a depressive disorder appears in both members of monozygotic twin pairs more frequently than in dizygotic pairs, there is evidence of a genetic contribution to the disorder. Indeed, major depressive disorder is about four times more likely to occur in both members of identical twins, compared with nonidentical twins (Bowman & Nurnberger, 1993).

Evidence for a genetic component in depressive disorders has also been found in family studies comparing the risk for various relatives of people with such disorders (see Figure 6.1). These studies have consistently shown that close relatives of adults with major depressive disorder are at higher risk for such disorders than are more distant relatives. One study suggested that loss of appetite or weight in parents with a history of recurrent depression may be the strongest marker of risk for depression in their offspring (Mars et al., 2013).

Of course, greater environmental similarities in the lives of close relatives might help account for the results of family studies, so researchers have also used adoption studies to determine the relative contributions of genetic versus environmental factors. If depressive disorders are determined to any significant degree by genetic factors, then depression should be more frequent among the biological relatives of an individual with depression, even if that individual was raised in an adoptive family. However, several studies have shown that children living with parents (especially mothers) who have depression are at higher risk to later develop depression themselves, regardless of whether or not these parents were adoptive or biological, indicating that the risk may be primarily environmental (Marmorstein et al., 2012; Tully et al., 2008).

Taken together, twin, adoption, and family studies suggest that genetic factors play a weaker role in unipolar depression versus bipolar disorders (see Chapter 5) and that there is a more clearly defined genetic risk for major depressive disorder than for persistent depressive disorder (Newman & Bland, 2009). Furthermore, genetic models do not tell us *how* genetic endowment leads to depression. To understand that link, we must look at how alterations in biological functioning might affect mood.

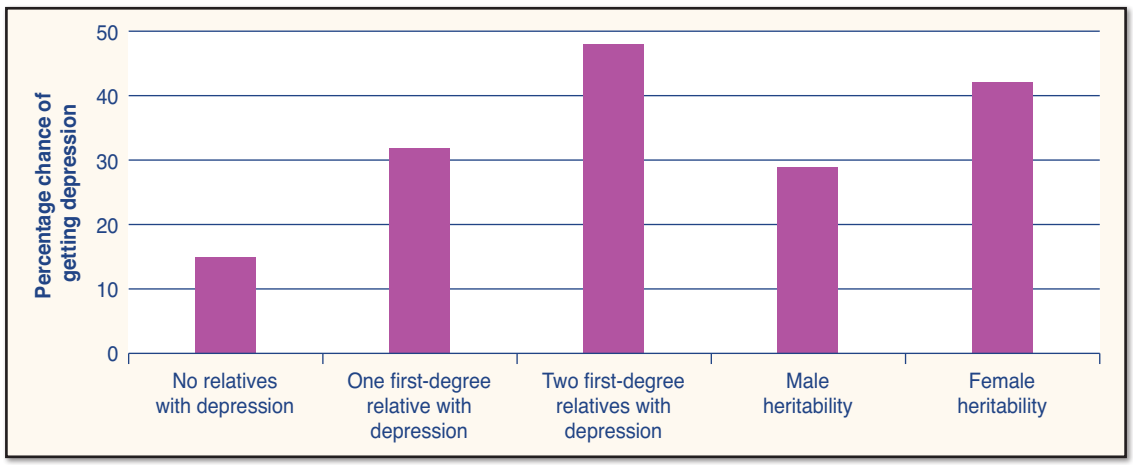



**FIGURE 6.1** Depressive Disorders Run in Families

First-degree relatives of people with major depressive disorder have about two or three times the lifetime risk of developing major depression compared to those whose first-degree relatives do not have any major depressive disorders. If that relative has chronic depression, then the person has a 2.5-to-1 chance of also having a chronic form of the disorder. In addition, the heritability of depression is higher in women (42%) than in men (29%).

*Source:* Based on data from Kendler et al. (2006); Mondimore et al. (2006); and Wilde et al. (2014).

## Neurobiological Influences on Depressive Disorders

Depressive disorders are accompanied by a number of abnormalities in the central nervous system (Pandey & Dwivedi, 2009). These may include abnormalities in the body's regulatory functions—especially in the production and utilization of the chemical messengers in the brain known as *neurotransmitters* and in the production and impact of stress hormones. Much research on the relationship between neurotransmitters and depression has focused on dopamine, serotonin, and the *catecholamines*—norepinephrine and epinephrine. These four neurotransmitters are thought to regulate several important behavioral systems relevant to depressive disorders, including motivation, concentration, and interest in others (Rogeness et al., 1992).

***Neurotransmitters and Depression*** Theories about the biological processes underlying depression first appeared in the 1950s, when physicians noted symptoms of depression in patients being treated for high blood pressure with reserpine—a drug that lowers catecholamine levels (France et al., 2007). Further evidence for this "chemical imbalance" theory came from studies showing that medications that *increased* levels of norepinephrine in the brain could diminish depressive symptoms (France et al., 2007).

However, later research discovered that not all people with depression have low levels of norepinephrine, nor do they all improve after taking drugs that increase norepinephrine levels (France et al., 2007). Gold and Wong (2021) found that norepinephrine levels are *elevated* in people with melancholic depression (see specifiers described previously). Since the **catecholamine theory** was first proposed, scientists have discovered that many other neurotransmitters and related chemicals may be involved in depressive disorders. Furthermore, mood-related neural activity may be affected not only by the amount of a neurotransmitter at a synapse but also by a neurotransmitter's effects on other neurotransmitters and on the number and receptivity of receptor sites. Finally, the long-term effects of a change in the amount of a neurotransmitter may differ from its short-term effects. These discoveries help explain why there is no simple, direct correspondence between moods and the amount of any one neurotransmitter in the brain.

**catecholamine theory:** The idea that low levels of norepinephrine lead to depression and high levels of norepinephrine lead to mania.

The most prominent (though likely incorrect) current theory—the serotonin monoamine theory—holds that low serotonin levels may allow other neurotransmitters such as dopamine and norepinephrine to swing increasingly out of control, leading to extreme moods (Leventhal & Antonuccio, 2009). Some studies, including a meta-analysis, reveal that one or two different polymorphisms (a specific nucleotide sequence in one's genetic code) in the single gene that makes a serotonin precursor may play a role in depression (Gao et al., 2012). This line of research provides a vital integration of genetics and neurotransmitters in the possible causes of depressive disorders.

**Connections**

What other mental disorders are treated with medications that affect neurotransmitters? See Chapters 4, 5, 7, and 8.

Further circumstantial evidence for the role of neurotransmitters in the etiology of depression comes from the fact that medications such as fluoxetine (sold as Prozac), which inhibits the brain's reuptake of serotonin, and bupropion (sold as Wellbutrin), which selectively blocks reuptake of dopamine, have both proved to be somewhat effective antidepressants, although with significant caveats to be discussed later.

Despite the mixed evidence, the lay public in the United States—including 54% of college students—has shown a strong adoption of the chemical imbalance theory of depression as depicted in many drug company advertisements (France et al., 2007). Overall, though, studies have failed to show a consistent and predictable causal link between depression and serotonin or norepinephrine levels (Healy, 2004). A meta-analysis showed that monoamine neurotransmitter (e.g., serotonin, norepinephrine) depletion did not directly or reliably decrease mood (Ruhé et al., 2007). In addition, neuroscientists have pointed out that, even when chemical abnormalities have been found in people with depression, they are as likely to be effects of depression as causes. Thus, there is no good answer to the question: Are the behaviors commonly found in people with depression, such as inactivity, low appetite, sleep disturbance, excessive alcohol consumption, and so forth, caused by *or the cause of* chemical changes in the brain (Horwitz & Wakefield, 2007)?

**MAPS - Medical Myths**

Thus, after many decades of research, and contrary to popular belief (and drug company pamphlets), neurotransmitter theories of depression have failed to achieve consistent empirical support (Leventhal & Antonuccio, 2009) and are no longer tenable (Cowen & Browning, 2015). Because it is unlikely that depression is related to only one neurotransmitter or another, even in a particular person, interactions among various biochemical, as well as life events, may ultimately prove to be the key causal factors in depressive disorders (e.g., Karg et al., 2011; Sharpley et al., 2013).

***Depression and the Endocrine System*** With these caveats in mind, depression has also been related to the functioning of the endocrine system. A key part of this system (described in Chapter 2) is the hypothalamic-pituitary-adrenal (HPA) axis, which plays a critical role in the body's response to stress. In times of stress, the adrenal glands (above the kidneys) respond to messages from the hypothalamus/pituitary gland (in the brain) by increasing their output of cortisol and adrenaline/epinephrine, hormones that help the body cope with stressors via "fight-or-flight-or-freeze" mechanisms (see Chapter 9).

Certain groups of people with depression show elevated levels of cortisol and abnormal daily variations in cortisol secretion (Nabeta et al., 2014), which supports the theory that disruptions in the regulation of the HPA axis contribute to (or are caused by) depression. This theory has also been explored through *biological challenge tests*, which involve giving people **dexamethasone**, a substance that temporarily suppresses the production of cortisol. Initial dexamethasone challenges showed that some people with depression may have an overactive HPA axis, as they failed to show normal cortisol suppression after being given the drug (e.g., Gibbons, 1969). However, further research revealed that similar "nonsuppression" effects occur in people without depression, as well as in individuals who had suffered broken bones or other physical trauma (Carroll, 1986; Knorr et al., 2010). Therefore, despite its early promise, the *dexamethasone suppression test* is no longer considered a specific or reliable tool for exploring the neuroendocrinological aspects of depression (Knorr et al., 2010).

**dexamethasone:** A substance that temporarily suppresses the production of cortisol in healthy adults.

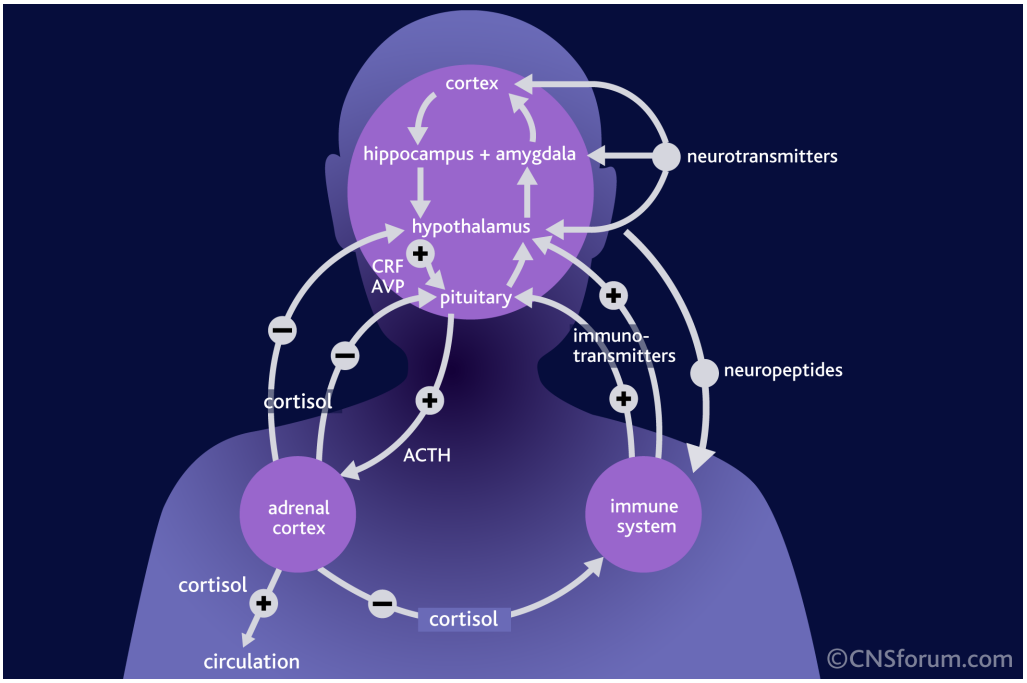


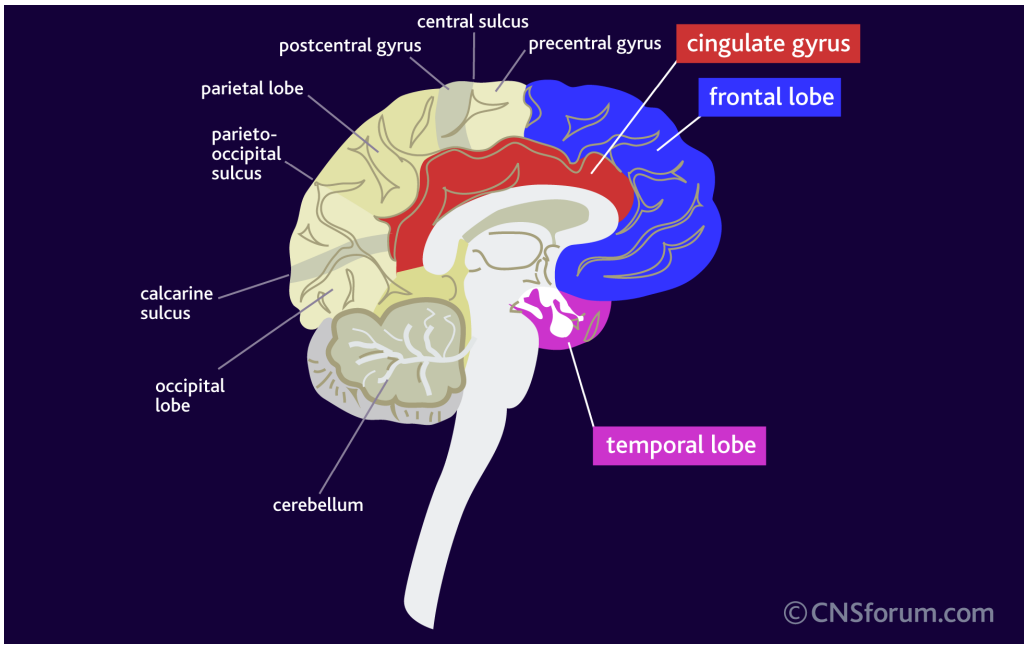

Image supplied by The Lundbeck Institute, www.cnsforum.com

The hypothalamic-pituitary-adrenal (HPA) axis is a feedback loop that includes the hypothalamus, the pituitary, and the adrenal glands. The release of cortisol into the circulation has a number of effects, including elevation of blood glucose. Cortisol also negatively affects the immune system and prevents the release of certain immune cells. Interference from other brain regions (e.g., the hippocampus and amygdala) can modify the HPA axis, as can neurotransmitters (Goodman & Gilman, 2001).

Nevertheless, researchers have continued to investigate a possible connection between depression and the HPA axis, which also fits with the catecholamine theory of depression discussed earlier, because the hypothalamus is strongly influenced by catecholamines. One study showed a higher cortisol awakening response among both participants with current and remitted major depressive disorder (Vreeburg et al., 2009). People with premenstrual dysphoric disorder showed a flattened daily cortisol slope (Beddig et al., 2019), which has been associated with stress-related dysregulation (Adam et al., 2017). Other research indicates that dysregulation of the HPA axis may be related to poor verbal memory functioning (Hansson et al., 2013) and poor coping strategies in people with depression (Hori et al., 2014). Depressed people with psychosis had higher evening (but not morning) cortisol than depressed people without psychosis or nondepressed controls, who did not differ from each other (Keller et al., 2017). Finally, higher cortisol levels were significantly associated with persistent depressive symptoms, especially physical (sleep, appetite, fatigue) rather than cognitive-affective symptoms (Iob et al., 2020).

***Other Biological Factors in Depression*** Researchers are also using sophisticated technology—particularly functional magnetic resonance imaging (fMRI) and positron emission tomography (PET)—to explore differences in the brain activity of people with and without depressive symptoms. PET studies have found, for example, that people with major depressive disorder have reduced blood flow and metabolism in the prefrontal cortex, particularly when they exhibit psychomotor retardation (Videbech, 2000), and increased activity in the limbic system, which processes emotions (Mayberg et al., 1999). Accordingly, fMRI studies reveal that people with depression have left amygdala hyperarousal (a part of the limbic system), especially for fearful faces, which normalizes with antidepressant treatment (Sheline et al., 2001). The frontal lobe and limbic system contain brain circuits that are involved in attention, alertness, reward, and emotion and are influenced by the neurotransmitters described earlier. Functional and structural anomalies in these brain regions may be stable vulnerability markers for major depressive disorder that precede the onset of symptoms (Gotlib et al., 2014).

Further, a new theory on biological causes of depression has emerged that contends that inflammation may play a significant role (Dantzer et al., 2008). This inflammatory response has been postulated to lead to depression via several mechanisms, including

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Many areas of the brain appear to be involved in depression, including the frontal and temporal lobes and parts of the limbic system, such as the cingulate gyrus. However, it is not clear if the changes in these areas are the causes of or the result of depression (Gotlib et al., 2014).

by influencing serotonin (a neurotransmitter) and/or cortisol (a hormone). This "cytokine theory" of depression is certainly attractive for a field (biological causes of depression) that has been short of real innovations, but it remains far from proven at this point (Dantzer et al., 2008). In fact, the findings of a recent systematic review highlight the lack of prospective evidence for biomarkers as predictors of onset of major depressive disorder and relapse/recurrence (Kennis et al., 2020). Whereas these results do not indicate that there are no causal biomarkers, they nevertheless highlight the dearth of prospective evidence that *any* biomarkers explain the onset of depression.

Finally, there may be a link between depression and the microbiota in our gut (Evrensel & Ceylan, 2015). A meta-analysis showed that gut microorganisms are decreased in major depressive disorder compared to nondepressed controls in observational studies, and depressive symptoms improve in interventional studies with probiotics (Sanada et al., 2020). The gut-brain axis will likely be the focus of expanded research in the coming years and could explain why a healthy diet is often a lifestyle intervention that can reduce symptoms of depression. To date, much of the research in this area has focused on animals, and expanded research is needed to understand this link in humans and to explore if specific microorganisms might be implicated the most in depression (Cheung et al., 2019).

## Section Review

Among the biological factors believed to contribute to depressive disorders are:

- genetic risks, which are not as strong for major depressive disorder as for bipolar disorder, but stronger than for persistent depressive disorder;
- disturbances in the level, functioning, or regulation of one or more neurotransmitters such as norepinephrine, serotonin, and dopamine;
- abnormalities in the endocrine (hormone) system, particularly the HPA axis, which is a critical system in the body's response to stress;
- changes in blood flow or inflammation that could interact with the neurotransmitters and/or endocrine system; and
- possible influences related to links between our brain and the microbiota in our gut that may impact symptoms of depression.

However, not one of these biomarkers has been clearly established as a *cause* of depression.

## Psychological Causes of Depressive Disorders

Most current psychological theories view biological factors as one of many risks (diatheses) that predispose some people to develop depressive disorders, as per the diathesis-stress model. Ultimately, though, psychological theories assert that depression results from a person's sense of lost control and diminished power (Gilbert, 1992). When people believe they have lost the ability to direct their own lives, they feel hopeless and become demoralized. Eventually, their actual power does diminish, and they give up many of their former productive or enjoyable activities.

### Intimate Relationships and Depression

Some psychological theories suggest that problems with intimate relationships can create a predisposition (diathesis) or act as a trigger (stress) for depression.

***Psychoanalytic Theories*** Psychoanalytic theories of depression are based on a classic paper by Sigmund Freud called *Mourning and Melancholia* (1917/1957). In Freud's model, individuals prone to depression suffered the loss of caregivers or were disappointed by them in some way during their childhood. The diathesis for later depression was their reaction to this early disappointment, which may have led them to become abnormally dependent on others to make them feel adequate or prone to anger when their dependency needs were not met. Freud said that depression results when this anger is eventually turned inward against the self.

It is vital to understand that "Freud's theory was embedded in a particular historical and scientific context and was constrained by the metaphors of his day" (Dozois, 2000, p. 188). In fact, Freud himself postulated that this theory would not necessarily generalize to other places and times, but researchers (e.g., Bradbury, 2001) have continued to be overattached to Freud's work on depression's causes (Dozois, 2000). One kernel of wisdom that emerged from Freud's theory, though, is the distinction between mourning and depression—notably, that the self-loathing and loss of self-esteem that is more typical of depression than mourning, part of what the *DSM-5* describes in an effort to help users distinguish these two states.

***Attachment Theories*** Other psychoanalytic theorists have downplayed the importance of Freud's "anger turned inward" view of depression and emphasized other factors instead (Arieti & Bemporad, 1978). For example, John Bowlby (1980, 1988a,b) proposed a model of psychopathology that draws on biological and social research on animals and humans. Like Freud, Bowlby stressed the importance of early mother-infant attachment. He noted that the nature of this attachment serves as the child's working model of the world and helps the child learn to regulate emotions. As discussed in Chapter 3, disturbance of this attachment can lead to impaired emotional adaptation (Cassidy, 1988; Kobak & Sceery, 1988). Relatedly, Stephen Suomi (1991) has demonstrated that separating baby rhesus monkeys from their mothers can produce symptoms of depression and anxiety in the infants that mimic the signs of insecure attachment in children. Bowlby suggested that insecure attachments provide a basis for depression because the individual fails to develop successful methods for dealing with the stressors of life and negative emotions such as anxiety.

**Connections**

What kinds of family relationships might place children at risk for depression? See Chapter 3 for some answers.

***Interpersonal Theories*** Bowlby's ideas, along with earlier contributions by Adolf Meyer and Harry Stack Sullivan, laid the groundwork for current interpersonal theories about the origins of depression. These theories suggest that unsatisfactory relationships place people at increased risk for depression. Intimate interpersonal relationships can protect against depression; however, divorce, loss of friendships, and other deterioration in social support are potential triggers for depression (Karasu, 1990; Monroe & Depue, 1991).

But does loss of social support precede or follow depression? James Coyne's (1976) classic interactional model of depression has emerged as one of the most influential frameworks for studying interpersonal aspects of depression (Starr & Davila, 2008). In this model, people with mild depression attempt to assuage feelings of guilt and low self-worth by seeking reassurance from others. At first, others provide support, but people with depression doubt the authenticity of the support and continue to seek reassurance until others grow annoyed and reject them. The rejection exacerbates the symptoms of people with depression as the cycle continues (Starr & Davila, 2008). In short, people with depression may provoke frustrating interpersonal encounters, thus assuring that their relationships become increasingly unstable and contentious (Coyne & Downey, 1991).

Several other studies have found that the behavior of people with depression adversely affects the mood and behavior of their family members and friends, thereby decreasing the social support otherwise available to individuals with depression (e.g., Coyne et al., 1987). Further, a meta-analysis supported the significant link between excessive reassurance seeking and depression (Starr & Davila, 2008). Finally, romantic partners' criticism may play a significant role in the maintenance of depressive symptoms (Meuwly et al., 2012).

In all likelihood, decreased social support is both a contributor to and a consequence of depression. As people with depression

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Because baby rhesus monkeys form close attachments to their mothers, they provide an excellent opportunity for researchers to study the consequences of disrupted attachment. Stephen Suomi (1991) has found, for example, that some baby monkeys suffer an emotional reaction similar to depression after being briefly separated from their mothers.

"I'll only give you the paper if you promise not to let the news upset you."

Cartoon Resource/Shutterstock

withdraw and shut off contact with existing friends and loved ones (as shown in the chapter-opening case about Alina), fewer people are available to provide the support and understanding that could lessen their depressed feelings.

### Learning, Cognition, and Depression

Cognitive and behavioral theories seek to explain depression by focusing on current patterns of thinking and reinforcement, rather than on early childhood events per se. These theories fit with interpersonal models of depression described earlier: People with depression lose their social reinforcement or begin to think about themselves differently as a result of their loss of important relationships, which may have been caused, in part, by their own excessive reassurance seeking in the first place. Most prominent among these theories are the hopelessness model (Abramson et al., 1989), Beck's (1987) seminal cognitive theory, and Nolen-Hoeksema's (1991) response style theory. We discuss these in the context of other important ideas about the psychological causes of depression.

***The Role of Reinforcement*** Peter Lewinsohn and his colleagues (1974, 1979, 1984) proposed that depression develops when people stop receiving adequate positive reinforcement from their environments, while also having many "punishing" experiences. Lewinsohn suggests three general reasons for the development of such reinforcement patterns:

1. An individual's environment may actually contain few positive elements and many negative ones; for example, living in an isolated area (or during a pandemic lockdown) would be a deprivation for someone who craves many friendships.
2. Even more important, the individual may lack the skills necessary to obtain positive results or cope with negative consequences; a person who desires friendships may be too shy or fearful of criticism to talk to strangers.
3. The individual may interpret events in a way that minimizes the positive and accentuates the negative, as when a person who desires friendships avoids new acquaintances because they all seem to be "snobs."

If some combination of these environmental and personality characteristics triggers a decrease in people's efforts to obtain life's rewards, a downward spiral of depressed behavior and reduced reward may appear. As the depressed behavior interferes with reward-seeking efforts, the likelihood of finding positive reinforcement is further reduced, causing depression to deepen and eventually to eliminate efforts to find rewards (Lewinsohn et al., 1979). See Figure 6.2.

**hopelessness:** A chronic tendency to view negative events as inevitable and positive events as unlikely, with no prospect for changing this pattern.

***Learned Helplessness, Hopelessness, and Depression*** The twice-revised **hopelessness** model of depression postulates that if people feel they are unable to control life events—especially stressful events—they learn a sense of hopelessness that will eventually lead to depressive symptoms. This theory grew out of research on the response of animals to uncontrollable stressors, an environment that Lewinsohn (discussed previously) would describe as having many more "punishers" than positive reinforcers. In this classic (though no longer ethical) experiment, dogs were exposed to episodes of electric shock from which they could not escape. When these animals later experienced shocks from which they *could* escape, many did not even try to do so; they just tolerated the shock, looking helpless and miserable (Seligman & Maier, 1967).

Similar results were observed in humans who had been exposed to sessions of inescapable aversive noise (Hiroto & Seligman, 1975). These and other results led Seligman

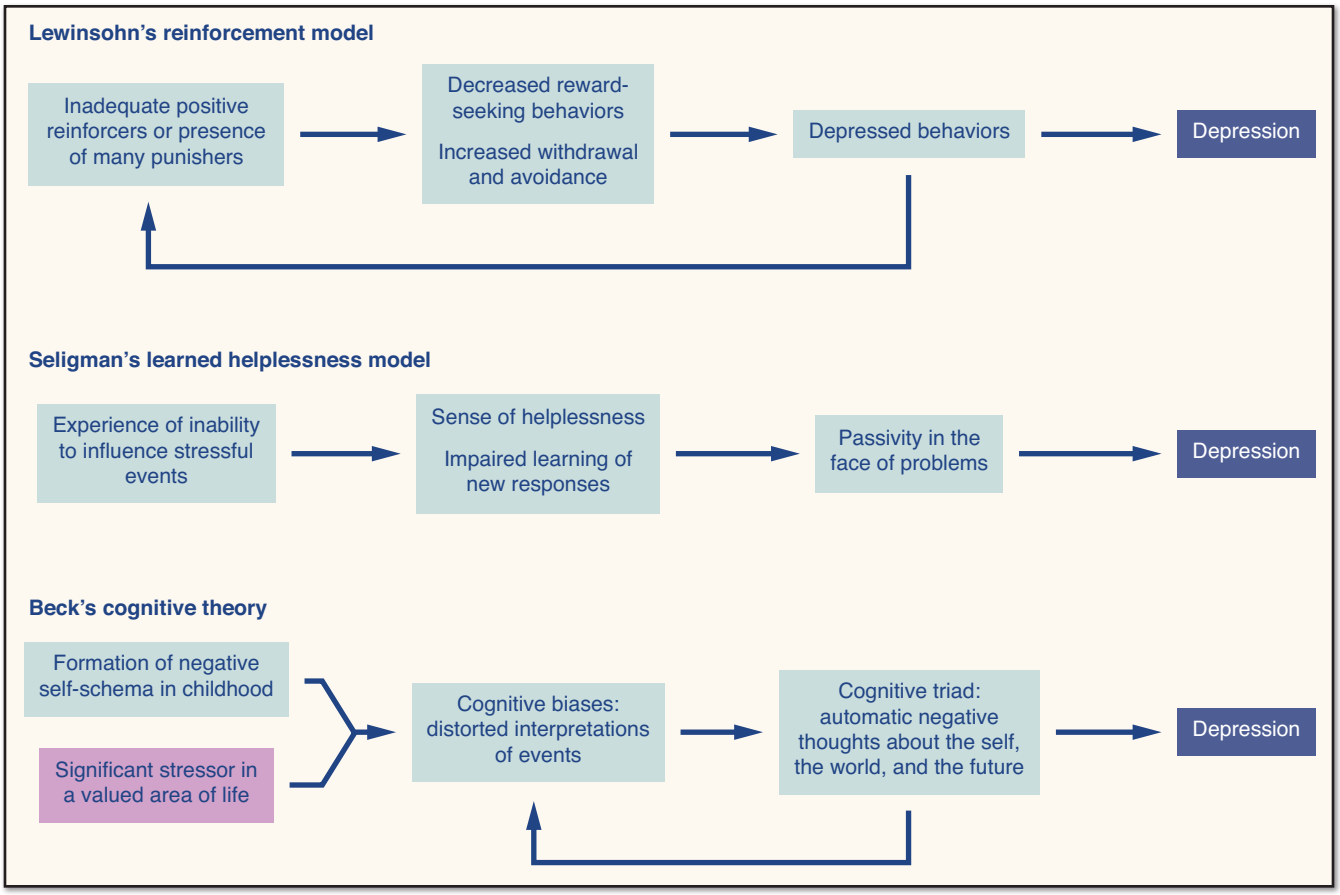



**FIGURE 6.2** Psychological Mechanisms Involved in Depression

Several important psychological theories explain how depression might develop. Each of the theories above has garnered empirical support and has spawned key psychotherapeutic approaches to treating the disorder.

(1975) to hypothesize that this **learned helplessness** (a precursor to hopelessness) in humans (1) interferes with the ability to learn responses that could solve or help them cope with life's problems, (2) causes them to give up even trying to solve such problems, and (3) eventually so impairs motivation, mood, and self-efficacy as to leave them in a state of depression.

Updated versions of this model of depression—known as the **hopelessness theory**—have stressed the importance of the individual's interpretation of aversive events in determining whether a sense of helplessness occurs (Abramson et al., 1989). According to this theory, certain people have a cognitive vulnerability that interacts with stressful life events to enhance their susceptibility to depression. The cognitive vulnerability is a **negative inferential style**, "the tendency of an individual to make particular kinds of inferences about the cause, consequences, and self-worth implications of stressful life events. . . . Specifically, when faced with a stressful life event, an individual who has a cognitive vulnerability is likely to: (a) attribute the event to stable and global causes; (b) view the event as likely to lead to other negative consequences; and (c) construe the event as implying that he or she is unworthy or deficient" (Haeffel et al., 2017, p. 543). Stable explanations view negative outcomes as enduring and unchangeable ("I will *never* be able to improve my grade"), whereas global attributions involve negative inferences about the generalizability and scope of negative events ("I will not do well in *any* college class"). Conversely, people whose thinking is characterized by optimism tend to explain bad events in terms of unstable and specific causes, thereby making hopelessness and depression less likely (e.g., "I will make sure my bad grade won't happen again, plus I'm still doing well in all my other classes"). Hopelessness theory provides yet another application of a *diathesis-stress model* in which a negative attributional or inferential style is the diathesis or predisposition

**learned helplessness:** An explanation of depression suggesting that, if people feel chronically unable to control life events, they learn a sense of helplessness that leads to depressive symptoms.

**hopelessness theory:** A theory that posits that some individuals have a cognitive vulnerability (a negative inferential style) that interacts with stressful life events to increase the likelihood of depression.

**negative inferential style:** A tendency to interpret failures in life as the result of stable and global factors within a person, to assume negative consequences of these failures, and to infer negative self-characteristics as a result (e.g., "I am a bad person.")

that leads to depression in the face of stressful life events (Metalsky et al., 1993). A systematic review determined that 23 of the 24 studies conducted to date have supported the vulnerability-stress hypothesis featured in hopelessness theory (Liu et al., 2015). Furthermore, individuals from two diverse samples (56% to 70% minority) with high levels of cognitive vulnerability were harder to treat for their depression (Haeffel et al., 2017).

***Beck's Cognitive Triad*** One of the most influential theories of depression has been and remains Aaron Beck's (1987) *cognitive theory*. According to this theory, vulnerability to depression develops during childhood when basic beliefs about the self are formulated. Beck says that these beliefs—a person's **self-schemas**, such as "I'm likable" or "I'm unlikable"—are determined by the quality of the developing child's interactions with the environment.

**self-schemas:** Core assumptions and beliefs about the self.

Negative self-schemas may have little influence until they are activated by the threat that accompanies significant life stressors. For example, in the chapter-opening case, Alina placed high value on her popularity and her academic achievement; after the breakup of her relationship, she began to falter in both of these areas. When a person suffers a loss in the arena that they value most, negative self-schemas become activated as the person's thinking and interpretation of events become distorted, In fact, Beck and his colleagues (1979) identified several cognitive distortions or "thinking errors" that characterize how people with depression process information (see Table 6.3). These irrational thoughts pop up involuntarily ("automatic thoughts") to bias our view of events. Soon, the person begins to see neutral or even pleasant events in a negative light. For example, Alina might interpret her professor's compliments as insincere or her friend's cancellation of dinner plans as a sign of rejection and her waning popularity. As a consequence of these cognitive processes, the person begins to experience sadness and other symptoms of depression, including loss of motivation and interest in activities, which could make their cognitions even more negative.

**cognitive triad:** Automatic, repetitive, and negative thoughts about the self, the world, and the future that are characteristic of people with depression.

Ultimately, the thinking of people with depression is characterized by a **cognitive triad** of automatic, repetitive, and negative thoughts about the self, the world, and the future (Beck

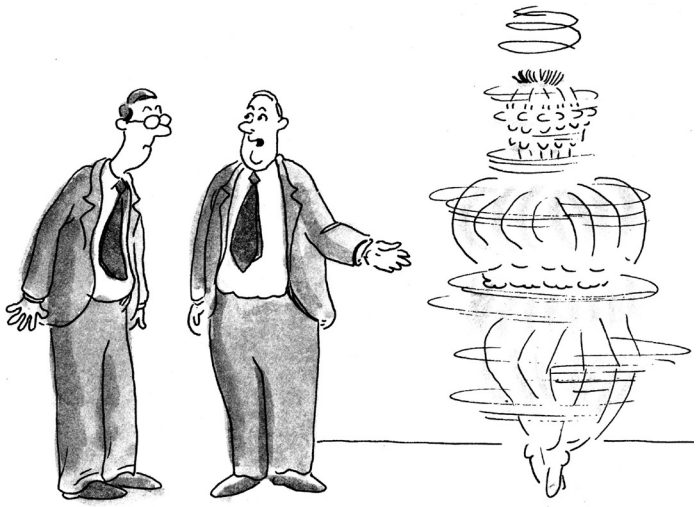
**TABLE 6.3 Typical Systematic Errors (also known as Cognitive Distortions) in the Thinking of People with Depression**

| Type of Error | Description |
|---|---|
| Arbitrary inference | Drawing a specific conclusion without solid evidence (e.g., assuming someone does not like you without any proof) |
| Selective abstraction | Taking a piece of data out of context (e.g., if your coach gives you nine compliments and one constructive criticism, you would come away thinking you are a bad team member) |
| Overgeneralization | Drawing a general rule or conclusion on the basis of isolated incidents (e.g., thinking the universe is against you because you spilled your coffee and got a flat tire in the same day) |
| Magnification and minimization | Overfocusing on a perceived failure and ignoring or discounting a perceived success (e.g., thinking that the test you did poorly on will cause you to fail out of school and that the test you aced was easy; focusing only on a critical comment from a professor, even though your overall grade was an A) |
| Personalization | Assuming that other people's behavior is about you (e.g., thinking that people are in a bad mood today because of you) |
| Dichotomous (all-or-none) thinking | Placing all experiences in one of two opposite categories, positive and negative (e.g., thinking that if you do not win "employee of the month" this time, then you are a bad employee) |

*Source:* Adapted from Beck et al. (1979).

et al., 1979). Individuals with depression see themselves as inadequate and, therefore, worthless; they perceive the world's demands as overwhelming; and they dread that the future will bring nothing but more of the same (similar to the hopelessness model discussed earlier).

In general, research supports Beck's ideas that people with depression engage in an excess of negative thinking, are prone to the negative triad of beliefs, and tend to distort even positive feedback as negative (Beck & Alford, 2009). It is less clear whether these dysfunctional cognitions precede or follow the onset of depression (Haaga et al., 1991; Gotlib et al., 2014). In fact, studies have shown that such dysfunctions may be both the cognitive consequences of being depressed *and* the long-term styles of thinking that predispose people to either first or subsequent depressive episodes (Otto et al., 2007). However, negative self-schemas predict depressive episodes and severity years later (Halvorsen et al., 2010), and children at familial risk for depression who were not (yet) depressed themselves show depression-prone cognitive styles prior to their depressive symptoms (Gotlib et al., 2014).




Cartoon Resource/Shutterstock

The way people interpret their life events and whether they develop learned helplessness (or hopelessness) has to do with their attributional/inferential style and cognitive schemas.

Other studies provide support for combined models/theories of depression. For instance, perceived trauma and insecure adult attachment (discussed earlier) predicted cognitive schemas such as irrational beliefs, which in turn predicted depression (Riggs & Han, 2009). Moreover, interpersonal schemas—irrational beliefs about other people—may be particularly important in cognitive vulnerability to depression (Dozois, 2007). Findings from the Cognitive Vulnerability to Depression (CVD) Project, a prospective and collaborative two-site study, provided strong support for both the hopelessness and cognitive models of depression (Alloy et al., 2009), and these two theories also show promise for explaining depression in children and adolescents (Lakdawalla et al., 2007). In another study offering support for both of these theories, hopelessness interacted with stress to predict adolescent depression 1 year later, whereas positive cognitive styles and low stress levels were protective factors for females specifically (Morris et al., 2008).

### Response Style, Personality, and Depression

Major life stressors, especially in the form of loss or trauma, appear to be social factors that can trigger depressive reactions. However, they do not always do so. Why do some people become depressed after a trauma but others do not? According to response styles theory, the way people define and cope with stressors is essential in determining the severity and length of a depressive episode (Nolen-Hoeksema et al., 1993).

For example, using *distraction* as a way of coping with stressful events appears to soften the events' impact and help ameliorate depression. Distracting responses, such as doing something fun with a friend, may be beneficial because they allow people to temporarily get their minds off their depression, while also providing an opportunity for positive feedback from others. However, *ruminative* responses tend to amplify and prolong periods of depression, as the individual obsesses about the causes, symptoms, or consequences of depression (Nolen-Hoeksema et al., 1993). Ruminative responses include endlessly thinking, writing, or talking about the depression in an effort to understand it. As suggested by Pyzsczynski and Greenberg's (1987) self-awareness model of depression, excessive ruminative responses may keep the individual so obsessed with depression as to alienate those who might provide social support and, at the same time, may preclude more rewarding social interactions. For instance, Alina (from the chapter-opening case) made her symptoms worse by withdrawing from her parents, her friends, and her classmates in the wake of her relationship breakup.

Nolen-Hoeksema (1987) proposed that men and women differ in their characteristic responses to stressors and that these differences may explain why women are more prone to depression. She points out that, in many cultures, socialization throughout childhood teaches boys to emphasize action rather than feelings and teaches girls to be introspective and passive. Consequently, when faced with stressors, men are more likely than women to employ beneficial distracting strategies, whereas women fall into ruminative patterns that accentuate personal responsibility for the problems at hand and therefore facilitate depression. Indeed, research indicates that learning to decrease ruminative responses and increase distracting strategies leads to improvement in depressed mood (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema et al., 1993).

Several studies have investigated the neural mechanisms—what is happening in the brain—for these three prominent psychological theories of depression: hopelessness, Beck's cognitive model, and response styles theory (Auerbach et al., 2013). For instance, negative thoughts about the future (Beck's model) have been linked to reactivity of the amygdala (part of the limbic system), and that same brain structure may be critically implicated in the negative self-referential processing that leads to rumination (Auerbach et al., 2013). In addition, high rumination is associated with delayed post-stressor cortisol recovery among adolescents with depression, indicating another possible cognitive-biological connection (Stewart et al., 2013).

### Creativity and Depression

An ongoing line of research may help illuminate the depression-rumination connection. Remember the link between bipolar disorders and creativity discussed in Chapter 5? Studies of creative individuals and historical analyses reveal an even stronger link between creative behavior and depression (Verhaeghen et al., 2005). For instance, in reviewing the biographies of over 1,000 eminent individuals living in the 20th century, Ludwig (1995) found a lifetime prevalence of depression of 50% for people working in the creative arts, compared with 20% of those in the field of enterprise and 27% of important social figures. Verhaeghen et al. propose that "a common underlying psychological characteristic, namely, a tendency for self-reflective rumination, may be the source of this correlation. . . . Specifically, . . . self-reflection independently (a) increases the risk for depression and (b) spurs interest in and ability for creative behavior" (2005, p. 229).

Krikkiat/Shutterstock

Actor Ewan McGregor (famous for his role as Obi-Wan Kenobi in *Star Wars*—see photo) has acknowledged bouts of depression, as have a number of other creative people, including talk-show guru Oprah Winfrey, late-night TV host Conan O'Brien, singers Beyoncé Knowles and Gwen Stefani (who claimed depression was the reason she dyed her hair pink in 2000), actors Jim Carrey and Angelina Jolie, and baseball players such as hall-of-famer Ken Griffey, Jr. and future hall-of-famer Zack Greinke.

Other research has highlighted two distinct types of rumination: *brooding*, characterized by a neurotic tendency to dwell passively on undesirable aspects of the self and the sense that one's feelings are threatening, confusing, and inescapable; and *reflectiveness*, characterized by an openness to explore negative feelings, a sense that one's feelings are clear and controllable, and a willingness to contemplate strategies for improving one's mood (Trapnell & Campbell, 1999). How individuals ruminate determines whether they will be more prone to creativity or depression: Reflectiveness has a positive effect on creative behaviors but no effect on low mood, whereas brooding is linked positively to low mood but has no effect on creativity (Verhaeghen et al., 2014).

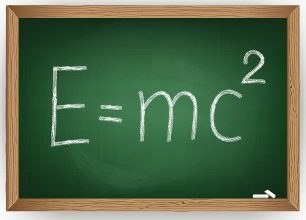
### The Value of Depression

The psychological models just reviewed suggest that depression is associated with problems in cognitive processing, particularly with distorted, negatively-

biased thinking. Other research suggests, however, that distortion or bias at certain junctures may be more characteristic of nondepressed people and that depression may actually have several positive aspects to it, in addition to creativity.

Taylor and her colleagues (1994) have found that most nondepressed people have views of themselves, their accomplishments, and their futures that are slightly unrealistic. They also tend to selectively remember the positive aspects of their lives, a style similar to what Seligman terms "optimism." This tendency to see themselves and the future in a positive, if slightly illusory, light appears to promote feelings of happiness (Gibbons, 1986; Taylor & Brown, 1988), adaptive social functioning (Diener, 1984), the capacity for productive work (Isen et al., 1987), and a measure of protection from the stress of life (Taylor & Brown, 1988). Overall, research studies (such as Robins & Beer, 2001) "converge on the adaptive value of positive illusions in the short term, and these may be particularly constructive in instances that would typically incur depression and lack of motivation" (Noble et al., 2011, p. 652). One study, for example, showed that adolescents who were below average in their math achievement overestimated their performance, and these positive illusions were inversely related to depressive symptoms (Noble et al., 2011).

Taylor (1994) believes that depression may result from perceiving life without the protective benefits of rose-colored glasses; as a result, people with depression are often more deliberate, careful, and skeptical—and at times more accurate—in how they process information from their environment (Rottenberg, 2014). Thus, negative cognitive processes of people with depression may simply reflect an unforgivingly accurate pattern of self-judgment. For this reason, some investigators refer to people with depression as "sadder but wiser" (Alloy & Abramson, 1979, 1988).



MAPS - Attempted Answers

Other aspects of depression—in addition to creativity and accuracy—may also be valuable in certain situations. For instance, the dark pull of depressive disorders may arise from adaptations that evolved to help our ancestors ensure their survival (Rottenberg, 2014). Low mood may operate as a kind of "stop mechanism," activated in situations in which persisting in a goal is likely to be futile or even dangerous (Rottenberg, 2014). Several studies reveal that a negative or sad mood can actually enhance memory performance, lower errors, improve motivation, and lead to more effective social actions in the short-term (Forgas, 2013). Further, some researchers suggest that depression—with its links to the immune system and inflammatory response described earlier in the chapter—may even help individuals fight existing infections (Anders et al., 2013). However, whereas low mood may have worked well for our ancestors, our modern environment—in which daily survival is no longer a sole focus and life stressors take on a more chronic form—makes it all too easy for a sad mood to slide into a severe, long-lasting depression that does not enhance our survival (Rottenberg, 2014).

## Section Review

Depressive disorders are influenced by several psychological factors, including:

- early experiences with loss or disappointment that may make a person unusually sensitive to later adversities;
- insecure attachments that leave a child less able to feel worthwhile, regulate emotions, and be satisfied in intimate relationships;
- a relative lack of positive reinforcement and an excess of punishment;
- a perceived lack of control over important events, leading to hopelessness;
- distorted thinking in which a person exaggerates negative aspects about the self, the world, and the future and ignores or minimizes positive information;
- a tendency to perseverate or ruminate about the self, particularly brooding about inescapable faults or failures; and
- the possibility that some aspects of depression (such as low mood) may have been advantageous to our ancestors for a variety of reasons.

## Social Causes of Depressive Disorders

One of the key social causes of depression is likely the nature of our modern world. Two well-done, large-scale studies revealed that the lifetime prevalence of depression in young people growing up in the 1990s exceeded by roughly a factor of 10 the prevalence in young people growing up in the 1950s (Seligman, 1990). As Figure 6.3 shows, there is also evidence that the rise in depression over the second half of the last century has continued into this one: In Denmark, rates of depression more than doubled in the first 6 years of the new millennium (Andersen et al., 2011), whereas chronicity and symptoms in the United States have continued to increase in women (Eaton et al., 2007) and in adolescent populations (Bertha & Balázs, 2013). Worldwide, there was an 18% increase in the number of people living with depression between 2005 and 2015 (WHO, 2017a). Martin Seligman (1998), then president of the American Psychological Association, spoke about an American depression epidemic:

> We discovered two astonishing things about the rate of depression across the century. The first was there is now between 10 and 20 times as much of it as there was 50 years ago. And the second is that it has become a young person's problem.

These changes have occurred over too brief a time span to be explained by genetic factors. Psychologist Stephen Ilardi (2009) began examining the social causes of depressive disorder after being inspired by the resilience and strength of certain aboriginal groups who almost never experience depression, such as the Kaluli of Papua New Guinea. The Kaluli practice a similar lifestyle to our ancestors, as they hunt, forage, and garden for their food, and live what we might consider challenging lives, with none of the material comforts or medical advances that we often take for granted. So what is it about their lifestyle that makes depression so rare (about 0.05% or 1 in 2,000 people, compared to 20–25% in our culture)?

There may be something about modern industrialized life that creates fertile soil for depression (Seligman, 1990). Rising depression rates are thought to reflect the growing influence of social stressors, such as disintegration of the family, unemployment, increased mobility, violence, and the resulting disillusionment found particularly in urban populations (Gershon et al., 1987). As Ilardi (2013) put it in his excellent TEDx talk at Emory University: "We were never designed for the sedentary, indoor, socially isolated, fast-food-laden, sleep-deprived, frenzied pace of modern life." Seligman (1990) further

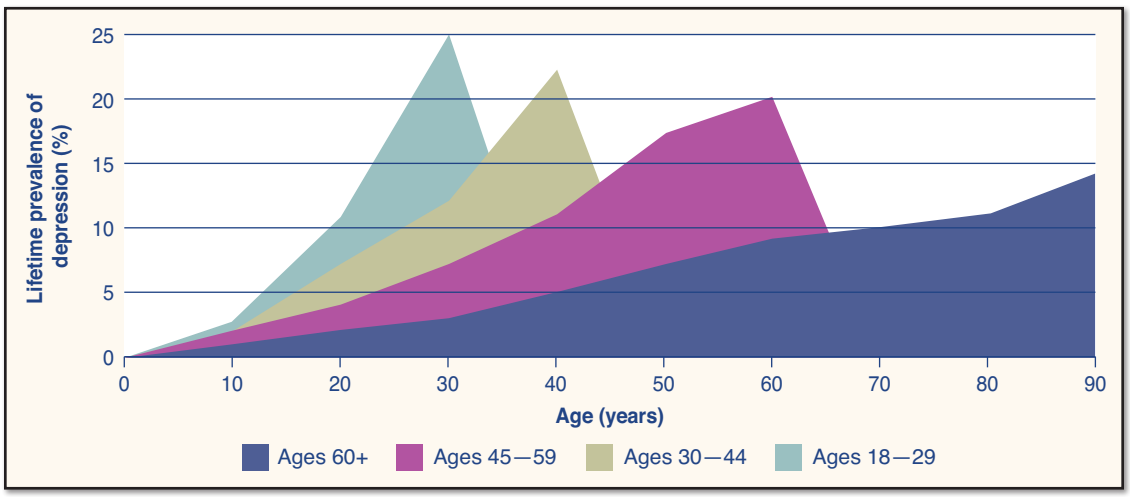



**FIGURE 6.3** Age Changes in Depression

According to one large-scale study of over 9,000 Americans, more people are becoming depressed and are experiencing their first major depressive episodes at increasingly earlier ages. The graph shows how the youngest cohort in the study (18–29-year-olds) has already had a higher lifetime prevalence (almost 25% depressed) than any other cohort. Depression expert Constance Hammen discusses the reasons behind these alarming trends in the feature "A Conversation With" that closes this chapter.

*Source:* Based on data from Kessler et al. (2003).

suggests that the modern Western exaltation of the self, along with the weakening of common institutions (such as religious or cultural organizations), has made it so that when people do not succeed at something, they have few larger beliefs to fall back on for consolation. In his view, hope lies in striking a healthier balance between commitment to the self and dedication to the common good.

### Stressors as Triggers of Depression

As noted, most theorists view depression from a diathesis-stress perspective (see Figure 6.4) in which depressive symptoms do not emerge until a diathesis such as genetic risk and/or disturbances in early parent-child relationships and/or cognitive vulnerability combines with stressful events and/or harmful environments. In a classic study of social environment and depression, Brown and Harris (1978) found that depression was more likely in British women if certain vulnerability factors were coupled with specific stressors or "provoking agents." Vulnerability factors (diatheses) included lack of a close confidant or friend; death of their mother in childhood, having four or more children under the age of 14 living at home, and lack of employment outside of the home. The most common provoking factors (stressors) were the recent death of a significant person or other events that posed a serious threat of loss. The greater the number of stressors a woman experienced in the previous several months, the greater her chances of being depressed. Certain aspects of the women's current social situations—especially a lack of social support—increased the risk of depression following a loss.

Subsequent research has consistently documented that onset or relapse of depression is more likely following a major loss, either of a significant relationship through death or divorce or loss of self-worth related to unemployment or the like (Brown et al., 1988; Monroe & Depue, 1991). Both acute and chronic stressors have been implicated in

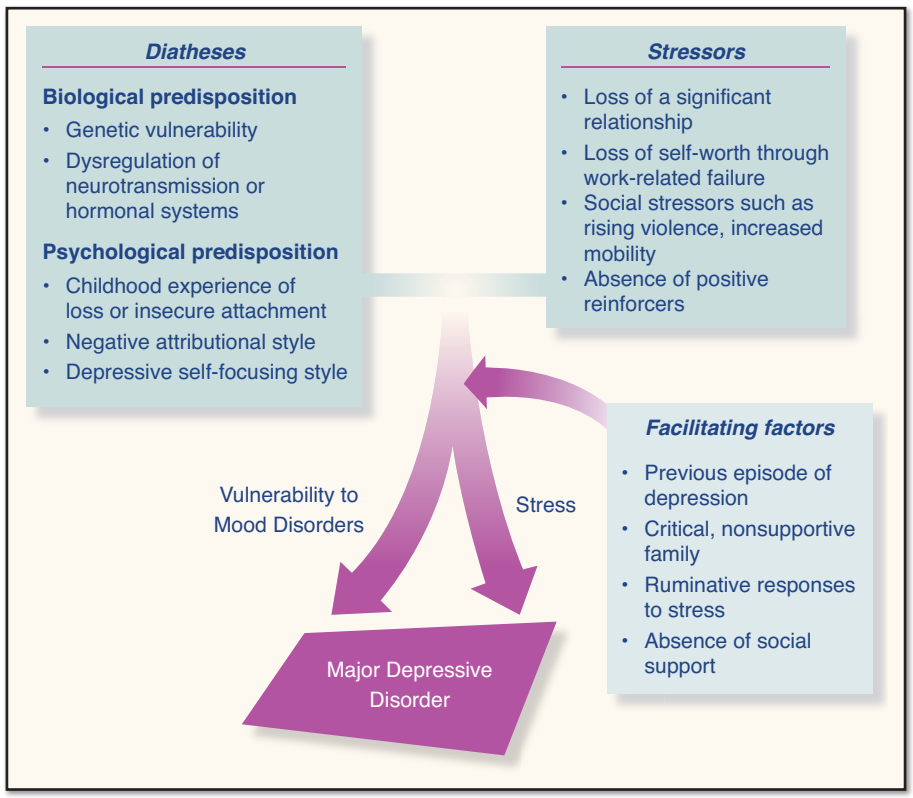



**FIGURE 6.4** A Diathesis-Stress Perspective on Major Depressive Disorder

There are a multitude of theories about major depressive disorder and a multitude of biopsychosocial factors that might be involved in its etiology. Perhaps there are also many ways in which the disorder develops. Shown here are the major factors we have discussed that seem to play a role.

depression (Harkness et al., 2014). For instance, after the 2010 BP Deepwater Horizon oil spill spewed more than 200 million gallons of oil into the Gulf of Mexico, more than 80% of those who suffered loss of income became depressed in the following year (Morris et al., 2013). Moreover, COVID-19 has increased depression rates worldwide (Castaldelli-Maia et al., 2021). In the United States, the prevalence of depressive symptoms was threefold higher during COVID-19 compared with before the pandemic: Those with fewer socioeconomic resources and higher exposure to stressors such as unemployment reported more symptoms of depression (Ettman et al., 2020).

Peer stressors through adolescence also increase the incidence of depressive disorders, especially in the context of poor relationships with parents during that span (Hazel et al., 2014). Academic stressors, such as Alina's falling college grades in the chapter-opening case, can contribute to or maintain symptoms of depression (O'Reilly et al., 2014). Interestingly, adult women are significantly more likely to report a severe life event prior to major depressive disorder onset than adult men, although this gender difference does not apply to adolescent depression (Harkness et al., 2010).

Stressful family environments also influence the course of a depressive disorder (Davila et al., 1995). For example, individuals with depression who live with highly critical, unsupportive families with poor problem-solving skills are more likely to suffer recurrent episodes of depression and be less able to get back to a productive life than those whose families have a more positive emotional climate (Hooley, 1987; Miller et al., 1992). Higher expressed emotion (EE, see Chapter 4) is associated with a greater likelihood of having a future onset of a depressive episode in children and adolescents with depression (Silk et al., 2009). Further, continued exposure to maternal criticism appears to be an important risk factor for initial depression in children (Burkhouse et al., 2012).

**Connections**

Does a negative emotional climate in families ("expressed emotion") contribute to relapses of other types of mental disorders? For more on this subject, see Chapter 4.

## Section Review

Our modern social environment may be a poor fit for our current bodies and brains, with a lack of outdoor activity, improper nutrition and sleep, and an inappropriate pace of life leading to a rise in depression compared to previous generations.

Depressive disorders are also influenced by other social factors, such as exposure to major stressful events, especially if these events:

- occur to someone lacking in social support or with a critical family, and
- involve some kind of chronic or acute loss.

# Treatment of Depressive Disorders

During the last seven-plus decades, clinicians have made significant progress in developing and testing both biological and psychological treatments for depressive disorders. Here, we consider the results of this work.

## Drug Treatments for Depressive Disorders

**antidepressants:** A broad class of medications commonly used in the treatment of depressive and sometimes bipolar disorders; these drugs include monoamine oxidase inhibitors, tricyclics, and selective serotonin reuptake inhibitors (SSRIs), such as Prozac.

Drugs have been used to treat depressive disorders for many years, with varying effectiveness. Researchers discovered the first **antidepressants** purely by chance in the 1950s. Seeking a treatment for schizophrenia, scientists at the Münsterlingen asylum in Switzerland found that a drug that tweaked the balance of the brain's neurotransmitters sent patients into bouts of euphoria. On first trying it in 1955, some patients became newly sociable and energetic and called the drug a "miracle cure." The drug, called imipramine, was quickly followed by dozens of rivals—known as tricyclics for their three-ring chemical structure—as drug companies rushed to take advantage of a burgeoning market (Fitzpatrick, 2010). Tricyclics have now largely been replaced as the first-line drug treatment of depression by selective serotonin reuptake inhibitors (SSRIs, discussed shortly), and there is also a third (less widely used) class of antidepressant medications called monoamine oxidase inhibitors (Nemeroff & Schatzberg, 2007).

The net effect of antidepressant drug action is to alter activity in the brain cells that use norepinephrine, serotonin, and other mood-related neurotransmitters discussed earlier in the chapter. The best-known antidepressants fall into the three categories already mentioned:

- **Monoamine oxidase (MAO) inhibitors**, such as phenelzine (sold as Nardil) and tranylcypromine (sold as Parnate), block monoamine oxidase, an enzyme that breaks down neurotransmitters such as serotonin and norepinephrine, resulting in greater availability of these neurotransmitters at neural synapses.
- **Tricyclics**, such as imipramine (sold as Tofranil), amitriptyline (sold as Elavil), and desipramine (sold as Norpramin), also increase levels of neurotransmitters such as norepinephrine and serotonin, but they do so by blocking the reuptake of these neurotransmitters.
- **Selective serotonin reuptake inhibitors (SSRIs)** include fluoxetine (sold as Prozac), sertraline (sold as Zoloft), citalopram (sold as Celexa), and paroxetine (sold as Paxil). These drugs slow the reabsorption of serotonin by the neurons that secrete it, thus keeping more serotonin in the synapse longer. There are also drugs that act as serotonin-norepinephrine reuptake inhibitors (SNRIs), such as venlafaxine (sold as Effexor) and duloxetine (sold as Cymbalta). Another antidepressant—bupropion (sold as Wellbutrin)—acts by blocking reuptake of dopamine.

**monoamine oxidase (MAO) inhibitor:** A drug that blocks monoamine oxidase, an enzyme that breaks down neurotransmitters such as serotonin and norepinephrine, resulting in greater availability of these neurotransmitters at neural synapses.

**tricyclics:** Drugs used primarily to treat depression; they increase levels of neurotransmitters such as norepinephrine and serotonin by blocking their reuptake.

**selective serotonin reuptake inhibitors (SSRIs):** A type of drug used to treat mental disorders such as depression that works by slowing the reuptake of serotonin in the brain.

Studies suggest that 50–60% of adults with depression are helped by these antidepressant medications, with various therapeutic effects, including brightened mood, improved sleep, and increased energy (Ruhé et al., 2006). Antidepressants may work better for persistent depressive disorder than for major depression (von Wolff et al., 2013).

Although the chemical action of many antidepressants is known, it is still unclear how they actually alter depressive symptoms. For example, antidepressants have an immediate impact on neurotransmitter levels, but depressive symptoms usually are not alleviated until after at least 2 weeks (and sometimes up to 8 weeks) of treatment. However, some studies suggest that antidepressants can work as early as 3 days after treatment and that early response may predict eventual outcome (Katz et al., 2006; Posternak & Zimmerman, 2005). In any case, scientists speculate that this delay—ranging from days to weeks—may reflect the time it takes for neuronal receptors to adapt to changes in neurotransmitter levels (Frazer & Benmansour, 2002). In other words, it may be a change in how brain cells use neurotransmitters—and not simply the levels of available neurotransmitters—that is critical to symptom reduction. Still others have postulated that antidepressants do not act as direct mood enhancers but rather change the relative balance of positive to negative emotional processing and actually work in a manner consistent with cognitive theories of depression (Harmer et al., 2009).

In clinical studies of antidepressants, approximately one third of people with depression achieve a full remission, one third experience a response, and one third are nonresponders (Tranter et al., 2002). Several meta-analyses have suggested that the differences between drug and placebo are not clinically significant for most people with depression (Kirsch, 2009); in other words, much of the response to antidepressant medications may be due to the placebo effect (see Chapter 2), which results from the person's expectancy rather than any specific chemical action of the drug per se. One meta-analysis (Fournier et al., 2010, p. 47) rocked the media and the pharmaceutical industry with the following conclusion:

> The magnitude of benefit of antidepressant medication compared with placebo increases with severity of depression symptoms and may be minimal or nonexistent, on average, in patients with mild or moderate symptoms. For patients with very severe depression, the benefit of medications over placebo is substantial.

MAPS - Medical Myths

Interestingly, herbal antidepressants such as hypericum (a medicinal plant known as St. John's wort) have produced similar effects to prescribed medications in treating depression (Walach & Kirsch, 2003). Whereas hypericum has only minor effects over placebo (Linde et al., 2005), it works as well as SSRIs with significantly fewer withdrawals (i.e., people

CONTROVERSY

## Prozac: Wonder Drug or Perilous Pill?

Since it was introduced in the late 1980s, Prozac (fluoxetine, an SSRI) has been at the center of a storm of controversy. Heralded by some as a wonder drug and condemned by others as a dangerous substance, Prozac has been prescribed to many millions of people worldwide. Headlined in major news publications and the topic of countless TV talk shows, the debate over Prozac rages on.

Advocates for Prozac, including Peter Kramer, the psychiatrist whose book *Listening to Prozac* (1993) popularized the drug for millions of readers, claim that it relieves depression and is also safer than other antidepressants. According to case reports, Prozac can even improve basic personality features, helping shy introverts become assertive extroverts or converting pessimists into optimists. No wonder Prozac is known in some circles as the drug that can help people become "better than well." In the book and later feature film *Prozac Nation*, the author laments that the drug did not really work for her but describes it as "a pill that doesn't make you happy but does make you feel not sad" (Wurtzel, 1995, p. 340).

Critics of Prozac blame it for a number of problems, including what they claim is a heightened risk for suicide and violent behavior among people taking the drug. They point to incidents such as on September 14, 1989, when Joseph Wesbecker killed 8 of his coworkers (and later himself) in a printing plant in Kentucky with an AK-47 assault rifle. When it was subsequently discovered that Wesbecker had begun taking Prozac about a month before the shootings, families of the victims filed a lawsuit against Eli Lilly, the manufacturer of Prozac. This lawsuit, the first of hundreds like it across the nation, claimed that Prozac causes previously nondangerous people to turn violent.

Whereas the Westbecker case (resolved in 1994) and many others like it ended with judges or juries deciding that Prozac was not responsible for the violence, a precedent-setting case in Canada in 2011 had a different result. In that case, Judge Robert Heinrichs listened to expert psychiatric testimony for the defense by Dr. Peter Breggin and agreed that Prozac caused a stimulant-like syndrome leading to manic-like behavior, suicidality, and violence in a teenage high school student with no prior history of violence who, while chatting in his home with two friends, abruptly stabbed one of them to death.

Accordingly, the risk of possible dangerous side effects, plus the ethical concerns about a drug that offers the false promise of making over personalities, is the topic of *Talking Back to Prozac* (Breggin & Breggin, 1994), a book intended to refute Kramer's book. The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Breggins pick through the studies used to justify Prozac's safety, often uncovering flaws and shoddy science, and detail the FDA approval process, including who on the panel was paid by whom. Other books in the new millennium have attacked the pharmaceutical industry even more directly. *Let Them Eat Prozac: The Unhealthy Relationship Between the Pharmaceutical Industry and Depression* (Healy, 2004) draws on the author's own research and expertise to demonstrate the potential hazards associated with these drugs and Big Pharma's abuses of power. *Medicines Out of Control? Antidepressants and the Conspiracy of Goodwill* (Medawar & Hardon, 2004) is another broad-scale critique of drug companies and their regulation in England and the United States. Relatedly, *Antidepressants and Advertising: Marketing Happiness* (Hunter, 2007) examines the volatile issue of marketing antidepressants through direct-to-consumer appeals, which is only legal in the United States (since 1997) and New Zealand (since 1981) among Western nations.

What evidence is there for the pro- and anti-Prozac positions? First, thoughts about suicide and violence do increase in a small number of people who are treated with antidepressant medication. After individuals take their medicine and begin to feel less depressed, their energy level rises, thereby increasing, at least temporarily, their risk for harming themselves or others. Moreover, the United States Food and Drug Administration (FDA; 2004) issued warnings that use of antidepressant medications poses a small but significantly increased risk of suicidal ideation/suicide attempt for children and adolescents. However, subsequent research has shown that antidepressants reduce more suicides in children than they produce (Bridge et al., 2007), and

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### Prozac: Wonder Drug or Perilous Pill? *(Continued)*

FDA warnings since 2007 point out that scientific data did not show this increased risk in adults older than 24 and that adults ages 65 and older taking antidepressants actually have a decreased risk of suicidality (U.S. Food and Drug Administration, 2007). Whittington and colleagues (2004) reported *no increased risk of suicidal behavior with fluoxetine* in children compared to placebo (3.6% versus 3.8%), but an increased risk of suicidal ideation or attempting suicide was observed for other SSRIs. Currently, Prozac is the only SSRI approved by the FDA to treat moderate to severe depression in children 8 years of age and older (Dwyer & Bloch, 2019).

Both sides in this controversy have often relied on dramatic anecdotes and uncontrolled case histories rather than well-designed scientific studies to support their arguments. As a result, both proponents and opponents of Prozac have exaggerated the powers of the drug, and the public has been too influenced by the latest incident reported in the media. The likely truth is that Prozac works for some people in some situations and not others, but that psychotherapy is a better first-line treatment for depression and related issues, as described later in this chapter.

#### Thinking Critically

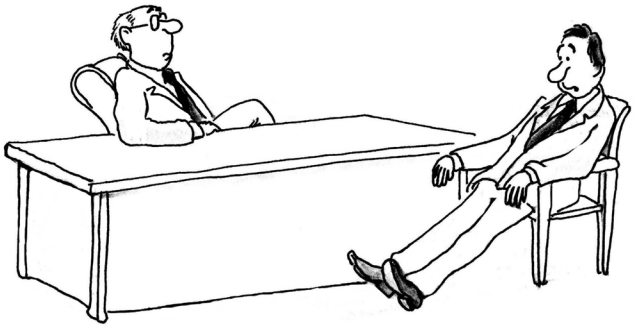
1. What other factors besides medication might account for suicides by people with depression?
2. How often does any adverse effect of a medication need to occur before it is considered a serious side effect? Is once enough, or should several examples be required before we decide that the risks of the drug outweigh its benefits?
3. Has the development and widespread marketing of Prozac and other drugs helped or hurt our society?

who stopped taking the pills) due to side effects (Rahimi et al., 2009). Finally, there is little evidence that either of the common medical practices of augmentation (prescribing more than one antidepressant at a time) or switching (prescribing a different antidepressant if no response is observed to the first medication) is an effective treatment option, despite their widespread usage in clinical practice (Bschor & Baethge, 2010; Yury et al., 2009).

The STAR*D (Sequenced Treatment Alternatives to Relieve Depression) project has been the largest antidepressant clinical trial in the United States to date conducted without pharmaceutical company support and continues to stimulate debate (Insel & Wang, 2009). Of the 4,041 people enrolled in STAR*D, all of whom were initially started on citalopram (Celexa, an SSRI), only 1,518 of them (37.6%) achieved remission after up to four medication trials over a year, and only 108 (7.1%) survived continuing care without relapsing and/or dropping out of the study (Pigott, 2011). One researcher (Ghaemi, 2008, p. 957), reflecting on STAR*D and other outcome studies, concluded: "The widely held clinical view of 'antidepressants' as highly effective and specific for the treatment of all types of depressive disorders is exaggerated." Similarly, a more recent meta-analysis of 131 randomized placebo-controlled trials enrolling a total of 27,422 participants concluded that "the potential small beneficial effects seem to be outweighed by harmful effects" (Jakobsen et al., 2017, p. 2).

The major types of antidepressants are all about equally effective, so the choice of which one to prescribe depends on a variety of other considerations (Del Re et al., 2013). Common side effects include sluggishness, weight gain, sexual dysfunction (especially with SSRIs), suicidal ideation (Smith, 2009), and with tricyclics, possible death from overdose (Palmer et al., 1998). FDA (2007) warning labels on these drugs further state that suicidality, anxiety, agitation, panic attacks, insomnia, irritability, hostility, aggressiveness, impulsivity, akathisia (psychomotor restlessness), hypomania, and mania have been reported in people being treated with antidepressants. The tricyclics also have cardiac side effects and can trigger manic episodes in people prone to bipolar disorder (Geller et al., 1993; Prien & Potter, 1993), and some MAO inhibitors cause high blood pressure if combined with certain foods.

Despite these significant limitations, with direct-to-consumer advertising from the pharmaceutical companies legally permitted since 1997, antidepressant use in the United States doubled over the following decade (Olfson & Marcus, 2009), making antidepressants the



*"What is the point of filling my prescription? My primary care doctor told me that the medication would only work if I took it while drinking an organic fruit smoothie lying on a tropical beach with waves lapping at the shore and a majestic orange sunset on the horizon after I finished my surfing lesson. . . ."*

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most frequently used class of medications by Americans ages 18–44 (Levine, 2013). From 2015 to 2018, 18% of American women and 8% of American men took the medication over the last 30 days (Brody & Gu, 2020). Worldwide, use has continued to rise, where Iceland, Portugal, Canada, Australia, and Sweden all had more than 10% of their population consuming these pills daily in 2019 (OECD, 2021). As a result, the world's 11 largest drug companies (6 of which are headquartered in the United States) made a net profit of over $700 billion in a decade, and "Big Pharma" remains the largest political lobbying group worldwide (Drugwatch, n.d.), spending almost $230 million in the United States alone in 2019 (double that of the next industry; Evers-Hillstrom, 2019; Wouters, 2020). After a predicted decline in 2021 following a post-COVID surge, the drug market is expected to exceed $21 billion in 2025 at a growth rate of 7.6% (Business Wire, 2021). See the "Controversy" feature for more on this topic.

Ideally, drug treatment of depressive disorders begins with the gradual introduction of medication, followed by a period of active treatment lasting for 4 to 6 months after depressive symptoms have been alleviated (Andreasen & Black, 1991). Further to this, antidepressant *prophylaxis*—sustained treatment for a few years or indefinitely—may be beneficial in any person with a history of three or more major depressive episodes, or two or more episodes in the last 5 years (Montgomery, 2006). Some clinical guidelines suggest switching to a different antidepressant at 3 months if remission does not occur (Olgiati et al., 2013). Moreover, these medications need to be gradually reduced if the person wants to discontinue drug treatment, as sudden withdrawal can cause several unpleasant side effects, ranging from headaches, dizziness, fatigue, insomnia and flu-like symptoms to irritability, aggression, anxiety, panic attacks, and "brain zaps," a feeling people describe as a jolt of electricity to their brain (Weir, 2020).

Scientists have searched for gene expression (via mRNA biomarkers) that could predict antidepressant response, with some success, although this has yet to be translated into clinical practice (Mariani et al., 2021). Psychologist Robert DeRubeis and colleagues (2014) developed a computer model to predict who is likely to respond to drugs versus psychotherapy based on five elements of their history/case: marital status, employment status, life events, comorbid personality disorder, and prior medication trials. However, decisions on which treatment to use (or which drug to try) largely remain guesswork at this point.

## Other Biological Treatments for Depressive Disorders

**electroconvulsive therapy (ECT):** A treatment for severe depression that induces brief seizures through the use of an electric current.

In the 1930s, clinicians noted the remission of psychotic and depressive symptoms in some people who experienced spontaneous seizures (Endler, 1988). On the assumption that the convulsions were responsible for the improvement, **electroconvulsive therapy (ECT)** was developed at a clinic at the University of Rome in Italy—and imported to the United States in the 1940s—to purposely induce brief seizures in individuals with severe mental disorders by passing an electric current through their brains (Endler, 1988).

The early ECT treatments were frightening procedures associated with many negative side effects (Winokur, 1986), including broken bones due to muscle stiffening during the ECT-induced seizures, marked memory loss, periods of disorientation, and even some deaths. Consequently, throughout the 1960s and 1970s, clinicians and hospitals began to limit or abandon the use of ECT.

To minimize problematic side effects, ECT is now administered only on one side of the head (a procedure known as *unilateral ECT*) and is accompanied by medication to control heart rate and relax muscles. Oxygen is also administered because most people stop breathing on their own during the seizure activity (Avery, 1993; Winokur, 1986). These

precautions have eliminated ECT's most serious long-term side effects, although temporary memory loss and disorientation are still common, and one third of people treated with ECT still report persistent memory loss (Rose et al., 2003).

The use of ECT persists in spite of these difficulties because it is effective for people whose depression is severe or who do not respond to antidepressant medication (UK ECT Review Group, 2003). Remission rates of just over 50% have been reported for people with depression following treatment with ECT (Berlim et al., 2013; Dierckx et al., 2012). A meta-analysis showed that the chance of response with ECT was about 4 times greater than with the antidepressant drugs (Pagnin et al, 2004). In addition, ECT is more effective in people without previous pharmacotherapy failure, as compared with people for whom antidepressants did not work (Heijnen et al., 2010).

However, relapse remains a problem. Even with continuation treatment of some sort (e.g., medication), over half of the people who initially respond well to ECT suffer a recurrence of their depressive disorder in the next year (Jelovac et al., 2013). Furthermore, ECT was once thought to be the treatment of choice for people with melancholic depression (discussed earlier in the chapter) but that turns out not to be the case (Rasmussen, 2011). Finally, it is still not clear why ECT works, although it appears that the seizure, not the shock itself, somehow alters the activity of neurotransmitters (Fink, 2013). One research team hypothesizes that ECT affects the brain in a similar manner as severe stress or brain trauma in that it activates the HPA axis (discussed earlier) and the dopamine system at the same time as it reduces activity in the frontal and temporal lobes (Fosse & Read, 2013). Another theory stipulates that ECT changes the wiring in the hippocampus and amygdala, key components of the brain's limbic system (Tendolkar et al., 2013). Overall, though, the uncertainty surrounding its mechanism of action, its inherently (though misleading) frightening nature, and its negative side effects make ECT one of the most underutilized treatments on the menu of options for depressive disorders (Kraus et al., 2019).

One proposed alternative to ECT has been repetitive transcranial magnetic stimulation (rTMS), a noninvasive technique for modulating cortical and subcortical function through the use of rapidly changing electromagnetic fields generated by a coil placed over the scalp (George & Post, 2011). TMS of the (left) dorsolateral prefrontal cortex has been FDA-approved since 2008 to treat depression in patients who failed to respond to one standard antidepressant treatment (Kraus et al., 2019). However, the remission rates with rTMS have been significantly lower (34%) than those with ECT (52%; Berlim et al., 2013).

Another biological treatment—one with far fewer risks—being employed for depressive disorders is **light therapy**, especially with depression that has a seasonal pattern. Individuals with these disorders appear to develop a phase delay in their circadian rhythms due to reductions in the amount of early morning light they experience (Avery et al., 1990; Lewy et al., 1987). Light therapy consists of exposure to bright lights during the day as well as "dawn simulation," a bright light source that comes on gradually during the early morning beginning about 2 hours before normal waking (Avery et al., 1994). It has been associated with remission of the symptoms of seasonal pattern depression and corrects phase delays in functions such as body temperature and hormone output (Lewy, 1993; Sack et al., 1990). Meta-analyses suggest that bright light treatment and dawn simulation for seasonal affective disorder, as well as bright light alone for nonseasonal depression, yield a reduction in depression equivalent to those in most antidepressant pharmacotherapy trials (Golden et al., 2005; Pjrek et al., 2020).

**light therapy:** Exposing patients to a bright light source during the early morning hours to reduce symptoms of seasonal depression and to correct problems in body temperature or hormone output.

Hailed as one of the most exciting innovations in psychiatry in the last several decades has been the accidental discovery that one low-dose infusion of ketamine could yield quick antidepressant effects (Kraus et al., 2019). Ketamine was initially used as a horse and dog tranquilizer and was approved by the FDA for use as a human anesthetic in 1970. Meta-analytic results now provide support for the controlled use of ketamine in the rapid management of depressive symptoms (Marcantoni et al., 2020). Nevertheless, whereas ketamine appears promising in the short-term treatment of depression, more clinical and experimental data are needed with regards to its efficacy, tolerance, and security of long-term administration (Marcantoni et al., 2020). Similarly, researchers are beginning

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This photo shows one of your textbook authors with his teenage son after climbing Table Mountain in Cape Town, South Africa, in 2021. Exercise is a biological and behavioral treatment for depression that works as well as antidepressant medication when used as prescribed, especially when combined with nature (e.g., sunlight).

to explore the potential therapeutic benefit of psilocybin in treating depression that has not responded to other trials of medicine. Early evidence suggests that psilocybin-assisted therapy reduces symptoms of treatment-resistant depression and that it should be a focus of future research (Davis et al., 2021).

However, one of the most promising biological treatments for depression may actually not be medications, electricity, or light, but rather lifestyle in general. For instance, several meta-analyses show that physical exercise—whether aerobic or strength training—is as effective in treating depression as medication (Josefsson et al., 2014; Silveira et al., 2013). Another meta-analysis found moderate evidence for the short-term beneficial effects of yoga, compared to usual care in the treatment of depression (Cramer et al., 2013). Unfortunately, two other meta-analyses suggest that the benefits of exercise may fade over time (Krogh et al., 2011) and that it has only a small positive effect on depression in children and adolescents (Brown et al., 2013).

In addition to physical exercise and yoga, other lifestyle factors that have been implicated in improving depression include nutrition and sleep. For example, weight loss in obese individuals reduces symptoms of depression (Fabricatore et al., 2011), omega-3 fatty acids have antidepressant properties (Lin et al., 2010), and sleep abnormalities are early markers for later depression (Augustinavicius et al., 2014). Combining all these lifestyle data, psychologist Stephen Ilardi created a treatment for depression called Therapeutic Lifestyle Change (TLC). TLC encourages people to integrate the following six elements into their lives: (1) an omega-3 rich diet, (2) exercise, (3) plenty of natural sunlight, (4) ample sleep, (5) social connections, and (6) participation in meaningful tasks that leave little time for negative thoughts—all things that our ancestors had in abundance (Ilardi, 2009). Preliminary studies of TLC revealed that, whereas 25–33% of people with depression in community-based treatment (typically medication) improved, the response rate among people receiving TLC was over three times higher (Ilardi, 2009; Karwoski, 2008). However, no further research on TLC has been published to date.

## Psychotherapy for Depressive Disorders

Although drugs and other biological elements may be an important aspect of treatment for depressive disorders, especially for severe depression, these do not address the social, emotional, or personality factors that may also underlie people's problems. Therefore, psychotherapy that emphasizes psychodynamic, interpersonal, behavioral, cognitive, or motivational aspects are also commonly used to treat people with depressive disorders. In fact, the psychotherapy approaches described here are as effective as antidepressant medications in the short-term treatment of depression and are likely more effective than medication in the longer-term management of depressive symptoms (Spielmans et al., 2011). Network meta-analysis (NMA) is a relatively recent development, which extends principles of meta-analysis to the evaluation of multiple treatments in a single analysis (Rouse et al., 2017). An NMA revealed that all psychotherapies for depression outperformed waitlist control and all—except nondirective supportive counseling and psychodynamic therapy—were more efficacious than pill placebo (Cuijpers et al., 2021).

### Psychodynamic Approaches

Traditionally, psychodynamic therapies have attempted to alter the individual's personality structure—usually by exposing and working through various unconscious conflicts—rather than to treat a specific problem such as depression. Contemporary versions of psychodynamic treatment seek to address depression specifically and more directly. Examples of these therapies are *time-limited dynamic psychotherapy* (Strupp & Binder, 1984), *short-term dynamic psychotherapy* (Davanloo, 1994), and *supportive-expressive therapy* (Luborsky, 1984). Short-term psychodynamic psychotherapies (defined as 40 or fewer sessions) may also be effective in children and adolescents (Abbass et al., 2013). Overall, though, psychodynamic therapies for depression are the least-examined treatment methods for depression, compared to cognitive-behavioral therapy and interpersonal therapy (discussed in the next sections; Dekker et al., 2014). Accordingly, it is difficult at present to draw firm conclusions about the value of psychodynamic therapies in the treatment of depressive disorders (Jakobsen et al., 2011).

### Interpersonal Therapy

Emerging from psychodynamic therapy, the interpersonal approach to treating depressive disorders focuses on the client's current social support system (Klerman et al., 1984). In the case of a depressive disorder, the therapist begins by asking the client to view depression as an illness so as to minimize any sense of guilt over being depressed. Then, attention is directed to one of four interpersonal problems presumed to be central to depression: (1) severe or prolonged grief reactions, (2) role conflicts in interpersonal relationships (as when someone tries to excel in occupational, marital, and parental roles, all at the same time), (3) role transitions (such as becoming a widow or widower, a parent, or a college student), and (4) deficits in interpersonal skills (such as extreme shyness or social awkwardness).

The specific treatment strategies depend on which interpersonal problem is most important. For instance, if the core problem is a role transition, attention might center on exploring the losses associated with the change and on preparing for the new role. Overall, the goal is to reduce dependency and increase self-esteem by helping people improve their relationships in family and work environments (Karasu, 1990).

One of the largest comparison studies of different treatments for depression was the National Institute of Mental Health's Treatment of Depression Collaborative Research Study, which compared the effects of the antidepressant imipramine, placebo medication, cognitive therapy, and interpersonal psychotherapy (Elkin et al., 1989). In this study, conducted at three clinical sites in the United States, 250 adults who had been diagnosed with depression were randomly assigned to one of the four treatments. Among the most important findings was that most people tended to improve, regardless of whether they received the antidepressant drug, cognitive therapy, or interpersonal therapy. In addition, antidepressants were superior to cognitive therapy for patients with severe depression (Elkin et al., 1995). The long-term effectiveness of the treatments was less impressive, but there were tendencies for the psychotherapies to outperform imipramine (a tricyclic antidepressant, the first one on the market). At 18-month follow-ups, only 30% of the original cognitive therapy clients, 26% of the interpersonal therapy clients, 19% of the antidepressant drug clients, and 20% of the placebo clients had recovered and not suffered any relapses. Overall, the value of interpersonal therapy in the treatment of depression is clear but probably small (Jakobsen et al., 2011), yet it deserves a place in treatment guidelines as one of the evidence-based treatments for depression (Cuijpers et al., 2011).

### Behavioral Approaches

As might be expected from the discussion of theories of depression, behavioral therapists seek to reduce clients' depressive symptoms by helping them increase or gain access to positive events in their lives. Sometimes, this means teaching or enhancing the skills (e.g., assertiveness) that clients need in order to experience support and other rewards in

## MULTICULTURAL CONSIDERATIONS: Managing Depression

Gabriela ("Gabby") is a 28-year-old medical resident in a highly competitive general surgery residency in Seattle, Washington. She was recently referred for mental health treatment by her program director after multiple co-residents shared their concern about a recent change in her mood, her withdrawal from others, and her lack of follow-through on critical tasks. Although initially reluctant, Gabby agreed to try psychotherapy. During early sessions with her therapist, Gabby was extremely tearful and disclosed feelings of sadness, inadequacy, and worthlessness. She described a lack of motivation or interest in her job, which she used to love. She continued to meet basic requirements of her work but admitted she was not giving her full effort due to her fatigue and difficulty concentrating. Whereas her relationship with her husband was strong, she noted she has not been interested in sex at all recently, which has led to some stress in their relationship. Her husband has expressed concern about her but perceives that she is pushing him away when he offers help. Gabby also described frequent thoughts of wishing she was dead, which were highly distressing to her as these thoughts were inconsistent with her religious values. Gabby's symptoms met criteria for major depressive disorder, and she began outpatient therapy in an in-house program designed to increase access to mental health services for physicians (and other healthcare providers) in training.

Gabby was born and raised in New Mexico. Her parents moved there from Mexico when her brothers were 4 and 8. Gabby was born shortly after they emigrated, and her younger sister was born around 3 years later. Both Gabby and her younger sister are American citizens, but her brothers and parents still have an undocumented status. She describes her family as very "tight-knit." Her family is Catholic, and she still considers herself a devout Catholic, even though her work schedule prevents her from attending mass regularly. She married her husband, a Caucasian man from her hometown, in a large Catholic ceremony around 3 years ago.

In Gabby's family, she was always known as the high achiever. She maintained a 4.0 GPA through both high school and college and was accepted to all five of the medical schools to which she applied. She opted to attend medical school in New Mexico to stay close to her family. She graduated with top honors from medical school and was widely regarded as one of the most successful students in her class. Although she knew relocating to Seattle would be a significant change for her, she believed the quality of the residency training was worthwhile. Gabby has always set high standards for herself, especially because she knows how important education is to her family. Neither of Gabby's parents attended college, and Gabby was the first in her extended family to attend medical school. Gabby's parents frequently talked about the fact that providing their children with a high-quality education was one of their motivators for moving to the United States.

As the psychologist began working with Gabby, it was evident that a number of cultural considerations were necessary in understanding what might be contributing to and exacerbating Gabby's symptoms of depression.

- **How did Gabby identify?** No matter what therapeutic approach is selected for treatment, a person's own identities and culture should be a part of planning and implementing treatment. Gabby and her psychologist explored her varied identities as a woman of color, a wife, a physician, a Catholic, and more. Though she identified broadly as Mexican-American, she noted that she considered herself more American than Mexican due to being raised in the United States. Whereas her family did hold strong communal values, she placed a lot of individual responsibility (and pressure) on herself to succeed.
- **How was she impacted by her multiple identities?** Despite Gabby's clear success and expertise, she described that it was not uncommon for her to enter a room and have a patient or a patient's family member ask to see "the doctor" and then appear surprised or flustered when Gabby identified herself as such. Although this situation occurred with some of her other female residents, Gabby felt like it happened more with her, and she wondered if she got this question more because she was female *AND* Mexican-American. She was also worried she was overreacting and might be labeled as "over-sensitive" or "not tough enough" for surgery. She was familiar with the term *microaggression* and expressed frustration that her training program did not address these issues more directly.
- **How did her family's status as undocumented immigrants impact her upbringing?** Given Gabby's citizenship, she had more opportunities available to her than her brothers and parents. Although her brothers worked odd jobs here and there to help support her parents, she had been able to start working consistently in high school, including a paid internship experience with a physician in her hometown. Gabby loved school, so college and medical school made sense for her, but she still experienced feelings of guilt that her brothers had not had the same options due to their undocumented status. She did not want to let any of her

**MULTICULTURAL CONSIDERATIONS: Managing Depression *(Continued)***

family members down as she viewed her opportunities as wider than others in her family.

- **How did familial expectations influence her?** As noted, Gabby was extremely close with her family, and many of her personal goals for herself were linked to her desire to help them. She noted that her current lack of motivation was extremely uncomfortable for her. She admitted that moving so far from family, even with her husband's support, had been more difficult than she imagined. She had anticipated being able to remain in regular contact with phone and video calls, but her intense work schedule had made that contact more difficult, which contributed to her feeling less supported than in the past.
- **What role does her faith play in her life?** Another key identity for Gabby was her Catholicism. Being raised in a strong Catholic family, where church was central to many of their family and community events, Gabby considered her faith a big piece of who she was. In residency, she had struggled with feeling more disconnected from her church due to her work hours. She also perceived that there was little room for religion in the scientific-focused world of surgery, so she sometimes felt as though she had to hide or minimize her spiritual views in the context of her work setting. This conflict had taken a toll on her mental health, and reconnecting with her church and spirituality became a key focus of treatment.

social situations. The most prominent current behavioral therapies are social skills training, problem-solving therapy, and behavioral activation. Behavioral activation, which involves increasing people's overt behavior to bring them in contact with reinforcing environmental activities or events (as opposed to the social withdrawal and avoidance that is typical of depression), is a well-established and advantageous alternative to other treatments of depression (Mazzucchelli et al., 2009).

Furthermore, a series of meta-analyses has shown that behavioral therapies perform as well as—and in some cases better than—other psychotherapies in the treatment of depression (Cuijpers et al., 2011, 2021). Evidence also suggests that starting with a behavioral component in psychotherapy may actually increase client retention over the span of treatment (Ahmed & Lawn, 2012). However, whereas behavioral activation may outperform cognitive therapy in the short term for people with severe depression (Dimidjian et al., 2006), long-term relapse is reduced when therapists also include a cognitive component to the treatment (Dobson et al., 2008).

### Cognitive-Behavioral Therapy

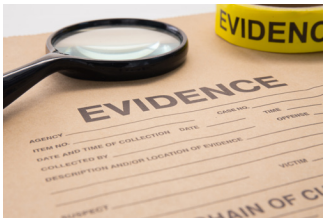
Cognitive-behavioral treatments for depression usually include educational, behavioral, and cognitive techniques and are known overall as CBT. One of the most prominent of these therapies is a short-term (8–16 sessions) intervention based on Beck's (1987) cognitive model of depression discussed earlier in the chapter (and in Chapter 2). Research is beginning to show that CBT works via processes specified by Beck's seminal theory to generate changes in thinking that then produce a reduction in the person's depressive symptoms (Driessen & Hollon, 2010).

In cognitive therapy, the client and therapist work together as collaborators to identify and change the client's maladaptive thinking patterns (negative self-schemas). The therapist first introduces the cognitive-behavioral model of depression, explaining that how people interpret events influences how they feel. Next, the therapist helps the client monitor their daily activities and mood to clarify which thoughts and activities are associated with changes in mood. Clients learn, for example, that, even when they are depressed, their moods fluctuate during the course of the day. They may also begin to see links between brighter moods and certain activities, companions, or thoughts. Finally, clients are helped to identify and test the validity of their "automatic thoughts" and often distorted underlying assumptions about the world. Since these assumptions guide how clients interpret their experiences, this phase of treatment usually challenges clients' personal belief systems, as illustrated by the ongoing case of Alina:

### *From the Case of Alina*, Continued

Alina, the 21-year-old woman whose case opens this chapter, has suffered from symptoms of depression since she was a sophomore in college. The youngest of four children in her family, Alina has always felt as if she has lived in the shadows of her older brothers, each of whom has his own family and has already been successful in the business world. Alina has always felt pressure to do well academically to impress her parents, who had hoped she would attend medical school after college. Alina's breakup with Jack, as well as her subsequent academic decline, has left her with many negative self-thoughts. In cognitive therapy, Alina has begun to learn that her depressive symptoms are worse after events in which she feels like a failure. As an example, the therapist asked Alina to concentrate on a recent incident in which a friend from her residence hall wordlessly walked by Alina in the hallway. Because of Alina's underlying sense of inferiority and current mood, she automatically assumed that her friend was ignoring her, thus adding to her feelings of inferiority and insecurity. However, the therapist asked Alina to think of alternative explanations for her friend's behavior and asked her to test these explanations by talking to the friend about the event and by watching the interactions of other people in the residence halls. The goal was to help Alina recognize that the friend could have been preoccupied, might not have seen her, or was perhaps too ill to be sociable. The therapist also helped Alina realize that, had she automatically assumed one of these other explanations, she would have likely experienced different, nondepressed feelings afterwards.

Mindfulness-based cognitive therapies (MBCT) emerged from the cognitive-behavioral tradition as *the third wave* of these therapy models (Williams et al., 2014). One type of MBCT is a manualized group skills training program (Segal et al., 2002) that integrates psychological educational aspects of CBT for depression with meditation components of mindfulness-based stress reduction developed by Kabat-Zinn (1990). The program teaches skills that enable participants to disengage from their habitual dysfunctional cognitive routines (negative self-schemas) and thus reduce the risk of relapse into depression (Lau et al., 2004). MBCT has garnered growing empirical support (McCarney et al., 2012). However, whereas MBCT prevented relapse for depressed people with a history of childhood trauma, it did not yield any significant advantage compared to an active control treatment and the usual care overall for people with recurrent depression (Williams et al., 2014). Another third-wave model is acceptance and commitment therapy (ACT; Hayes et al., 2013), which employs mindfulness, philosophy, and metaphor to teach psychological flexibility. A meta-analysis found that ACT significantly reduces depressive symptoms compared with controls, especially at 3 months of follow-up, for adults, and for people with mild depression (Bai et al., 2020). A recent network meta-analysis (NMA) found MCBT and ACT to be equivalent to classic CBT and significantly better than usual care or control treatment through 1 year of follow-up (Cuijpers et al., 2021).




***Evidence-Based Treatment***

Overall, research evaluating all types of CBT has found that it is an effective treatment for depression that may also reduce the risk of relapse (Cuijpers et al., 2021; Dobson et al., 2008; Hollon, 1993). In addition, CBT may have an advantage over medication in terms of long-term benefits (Antonuccio et al., 1995). For instance, CBT is equal to SSRIs in initial response and symptom reduction, but superior in terms of relapse prevention (Driessen & Hollon, 2010). Similar to medication and to other psychological treatments, some of the effect of CBT may be due to placebo effects, but it also has specific and active effects beyond mere placebo (Cuijpers et al., 2021; Honyashiki et al., 2014).

CBT is highly amenable to different delivery formats, ranging from groups to books and electronic media. One meta-analysis showed that group CBT had a moderate effect on the level of people's depression and a small effect on the relapse rate of depression (Feng et al., 2012). CBT can even be implemented in a self-guided manner, which typically

involves using a book, the Internet, or a phone app with or without the support of a therapist, such as Lewinsohn's *Coping with Depression* course (Lewinsohn & Clarke, 1984), Burns's (1999) *The Feeling Good Handbook*, or apps such as MoodKit (Thriveport, 2011) or the Depression CBT Self-Help Guide (Excel at Life, 2014). CBT self-help treatments for depression have a small but significant effect on depressive symptoms (Cuijpers et al., 2011).

### Motivational Interviewing

Motivational interviewing (MI; Miller & Rollnick, 2013) is a therapeutic approach that integrates the relationship-building principles of humanistic therapy (Rogers, 1951) with more active cognitive-behavioral strategies targeted to the client's stage of change (Prochaska et al., 1992). In the past few decades, MI has become a well-recognized brand that has been used in psychotherapy, medicine, addictions, public health, and beyond (Lundahl & Burke, 2009). Whereas MI has primarily been implemented in the treatment of substance use disorders, as discussed further in Chapter 14, it may also be valuable in treating depression (Arkowitz & Burke, 2008). The specific focus of MI on increasing motivation may be a good fit—either as an adjunct to another type of psychotherapy or as a stand-alone treatment—to the motivational deficit that people with depression show. First, loss of interest and pleasure in most or all usual activities—that is, low motivation—is a core symptom of depression, as described earlier in the chapter (American Psychiatric Association, 2013a). Second, motivation—as measured by willingness to participate actively in treatment, to explore one's problems, and to make changes and sacrifices to improve—significantly predicted improvement in depression treatment (Burns & Nolen-Hoeksema, 1991).

Two studies showed early promise for training primary care providers to use MI with their medical patients who suffer from depression (Keeley et al., 2014, 2016). First, providers were able to effectively learn MI in 8–16 hours of training (Keeley et al., 2014). Further, a single brief session (15 minutes) of MI tripled depressive remission for a group of patients with comorbid diabetes and depression, from 7% (control group) to 23% (MI group) at 8-month follow-up (Keeley et al., 2016).

### Comparing and Combining Treatments

Overall, psychotherapy of any type yields over a 60% remission rate of depression at follow-up, generally with about 12–18 sessions (Cuijpers et al., 2014, 2021). Psychotherapy is not only effective in adults with depression in general, but also in older adults, women with postpartum depression, people with general medical disorders, inpatients, primary care patients, individuals with chronic depression, and individuals with sub-threshold depression (Cuijpers et al., 2011). Note, however, that psychotherapy is not as effective for people with persistent depressive disorder as it is for people with other depressive disorders; it still has a small but significant positive effect, although SSRIs are significantly more effective for these chronic depressive symptoms (Cuijpers et al., 2010). Further, several meta-analyses have shown that all bona fide psychotherapies for depression are about equally effective, with the exception of supportive psychotherapy (Rogers, 1951), which has a slightly lower effect size than the other treatment models (Braun et al., 2013; Cuijpers et al., 2008, 2021). Finally, problem-solving therapy (a type of behavioral therapy/CBT) may be significantly more effective than standard CBT and third-wave therapies at 1-year follow-up (Cuijpers et al., 2021).

Some researchers suggest that the optimal treatment program for depression may be a combination of antidepressants and either CBT or IPT (Hollon, 1993). The logic of this advice is that drugs can quickly relieve physical symptoms, such as sleep disturbance, whereas psychotherapies address the cognitive and behavioral patterns that perpetuate depressive symptoms and increase the risk of recurrence. One way to think about it is via a car metaphor: Imagine having a car that is out in the cold all winter. You might need a boost (e.g., medication) to start your car, but then you still need to figure out where and how to drive (i.e., psychotherapy). In one meta-analysis, medications and cognitive

therapy led to different (and counterintuitive) patterns of response to specific symptoms of depression; medications worked faster than cognitive therapy or placebo on suicidal ideation and other cognitive symptoms, whereas cognitive therapy was superior at treating vegetative (i.e., low energy) symptoms of depression (Fournier et al., 2013). Overall, a network meta-analysis determined that combined treatment was more effective than psychotherapy alone or pharmacotherapy alone in achieving response or remission at the end of treatment, and this pattern held for chronic and treatment-resistant depression as well (Cuijpers et al., 2020).

Psychotherapies for children and adolescents with depression are in a more formative stage than the adult counterpart; most of them focus on group interventions. School-based CBT interventions for depression in youth hold considerable promise, although investigation is still needed to identify features that optimize service delivery and outcome (Mychailyszyn et al., 2012). Beneficial treatments have common elements, "including activities designed to promote competence, enhance relationship and communication skills, teach systematic problem solving, change unrealistic negative cognitions, and use behavioral activation strategies to increase activity and show its relationship to feelings" (McCarty & Weisz, 2007, p. 886).

Relatedly, mental health content has exploded on the immensely popular social media platform TikTok. The hashtag #mentalhealth has 15+ billion views and #therapistsoftiktok has hundreds of million views (Dastagir, 2021). The pandemic has probably accelerated the space's evolution, but therapists credit its existence to the broader destigmatization of mental health issues as well as the app's younger users who are more comfortable publicly processing everything from childhood trauma to relationship abuse and openly sharing their depressive symptoms. For better or for worse (you decide), TikTok is exposing people to mental health education. In a video captioned *Why Mental Health TikTok Is Powerful*, therapist Jaime Mahler shared a user's intriguing comment: "10 years of therapy and what I needed to hear I heard on TikTok. And it has changed the entire way I process my past and view myself now" (Dastagir, 2021, para. 1).

Overall, the prognosis for people suffering from depressive disorders is mixed. The good news is that most people recover, even without any treatment, from an episode of depression, in a matter of months. And treatment with psychotherapy and/or medication is relatively effective for the majority of adults (60%+). The bad news is that the risk of relapse is high no matter which treatment or combination of treatments is employed. Therefore, many individuals face a life of repeated episodes that result in heavy financial and personal burdens. Even with the plethora of treatment options, depression is, as stated previously, the number one cause of disability and a leading cause of suicide worldwide (World Health Organization [WHO], 2017a). Whether psychotherapy or medication is the preferred treatment for adult depression remains a hotly debated topic among mental health professionals and often comes down to advertising and accessibility. Drug companies market their medications very heavily, and it is easier to get people access to pills than a trained psychotherapist or counselor, although this may be changing with the current push for more integrated (and online) health care in the United States. Despite these obstacles, though, the fact that psychotherapy carries fewer side effects and medical risks and achieves slightly superior long-term and relapse prevention effects than medication suggests that it should be the treatment of first choice for depressive disorders—with the possible exception of persistent depressive disorder or very severe depression—whenever feasible.

### Culturally Sensitive Treatments

In the last two decades, several studies explored the development and implementation of culturally sensitive treatments (CSTs; also called culturally responsive treatments or culturally informed treatments) for depression for different ethnic groups, with the goal of reducing mental health disparities among historically-disadvantaged groups (Kalibatseva & Leong, 2014). CSTs entail "the tailoring of psychotherapy to specific cultural contexts" (Hall, 2001, p. 502). At the same time, a prominent debate in the field of CSTs has

been whether evidence-based treatments should be adapted or not (Atkinson et al., 2001; La Roche & Christopher, 2008). Whereas some researchers argue that such treatments should be employed as initially designed to maintain fidelity to the model, a growing body of clinicians have sought to make modifications or cultural adaptions to existing evidence-based treatments with the aim of expanding their generalizability to diverse populations (La Roche & Christopher, 2008).

A systematic review reveals that the majority of CSTs for depression employ an evidence-based bottom-up approach, which involves general and practical adaptations, such as translating materials or infusing specific cultural values into the treatment. For instance, Kalibatseva and Leong found that "all studies that included Spanish-speaking populations emphasized the importance of values such as *respeto* and *simpatia*, and instructed the staff to be warmer and more personalized in their interactions with their clients" (Kanter et al., 2010; Miranda et al., 2003; 2014, p. 436). Most CST studies used CBT strategies and included Latinos and African Americans; overall, culturally-adapted CBT showed strong effects in the treatment of depression, was more effective in individual (vs. group) format, and outperformed culturally-adapted IPT (Kalibatseva & Leong, 2014). In addition to adapting evidenced-based interventions, some researchers advocate using alternative research methods, such as participatory action research (PAR; discussed in Chapter 2) and integration of community perspectives to build interventions from within communities to address that group's needs (Wendt & Gone, 2011).

## Section Review

The three main categories of antidepressant medications are:

- monoamine oxidase (MAO) inhibitors,
- the tricyclics, and
- selective serotonin reuptake inhibitors (SSRIs).

These medications:

- are about equally effective, producing improvements in 50–60% of adults;
- are heavily marketed by pharmaceutical companies; and
- have significant side effects that could be distressing to people taking them.

Other biological treatments for depression include electroconvulsive therapy (ECT), light therapy, and lifestyle factors, such as nutrition, sleep, and exercise.

Depression can also be treated effectively with psychotherapy—in particular, interpersonal therapy, behavioral therapy, and cognitive therapy. These therapies

- achieve improvement rates (60%+) that do not usually differ from those of medication and lead to relapse rates that may actually be lower than with medication and may have fewer adverse side effects.
- can be modified to be more culturally sensitive, though much work remains to be done in this regard.

Even with all of these treatment options, however, depression remains a serious and potentially debilitating disorder that often recurs throughout a person's lifespan.

### *Revisiting the Case of Alina*

The primary care doctor at the Student Health Center referred Alina to the psychologist, Dr. Jane, who interviewed Alina and asked her to complete an MMPI-3 personality test (see Chapter 1). Alina's MMPI-3 restructured clinical (RC) scales showed an elevated RCd (demoralization) and RC2 (low positive emotions), in the context of a low RC9 (hypomanic activation). This is a typical profile for someone suffering from depression, whereas a person with bipolar disorder is more likely to have an elevated RC9 (Watson et al., 2011). Based on the clinical interview and MMPI-3 profile, the psychologist

## A CONVERSATION WITH

### Constance Hammen

Constance Hammen

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***Q*** *What do you think underlies the growing rates of depression in our population? Why do we see so much depression, especially in younger people?*

***A*** Rates of depression seem to be increasing worldwide, and in the United States, most studies have shown that younger people were reporting more major depression by their early 20s than did previous generations. Depression rates rise in the late teens and early 20s and throughout young adulthood and then are relatively lower in older age populations. Probably the simplest explanation is that higher rates tend to occur in relation to stressful events and circumstances. Early adulthood is a period of considerable change and challenge for many people, and stressors that overwhelm one's coping skills and resources may trigger depression. It has also been argued that younger people have more exposure to stress now than did previous generations. Their lives are full of family disruption, pressure to succeed and be financially independent, and social mobility. I think they also have many more negative cognitions about themselves and their ability to control circumstances. Certainly, families and social support are very important in helping people deal with stressful circumstances. Young people today may have fewer of those ties, and many do not have resources such as a good education or a good job.

***Q*** *Depression is still more common among women. Any thoughts about why?*

***A*** In my view, four separate factors contribute to the gender differences. One factor has to do with the expression of the symptomatology itself. We know that women are more willing to admit to emotional difficulties, to express emotions, and to play the role of the weak and needy. So in that sense, they are more likely to admit and feel the symptoms of depression than men are. But there is more to it than that.

A second factor has to do with causal factors that may be more prevalent with women. Women have greater exposure to stress because women experience not only their own stressors but also those of people they are close to. In a sense, women are lightning rods for the stressors of other people, as well as feeling their own greater exposure to stress. Women are raised to value close relationships, whereas men are raised to value achievement. Because women tend to value social relationships, they feel stressed when those relationships are threatened.

A third factor would be differences in how people cope with stress. Men and women cope somewhat differently with provoking situations. Men's coping methods may reduce the likelihood of depression. Men, generally speaking, are more active copers. They are more likely to go out and do things to solve the problem, avoid the problem, or distract themselves. Men also may turn to pathological forms of coping, such as drugs and alcohol; nonetheless, they try not to let themselves experience depressive symptoms.

Women, on the other hand, are somewhat less active copers, more likely to use what we call emotion-focused coping; that is, they will think about their problems and symptoms, cry, talk about their problems with other people, and focus on their feelings. Women tend to ruminate about their problems and feelings, and this kind of focus and repetition of negative experiences may consequently exacerbate the symptoms of depression.

Fourth, it has long been suspected that biological differences affecting hormonal balance and interactions among brain circuits, neurotransmitters, and gonadal hormones play a role. However important those factors may be, they are subtle and highly complex to study, and need to be thought of in terms not of levels of female hormones but of interactions among very complicated levels of biological and environmental factors.

***Q*** *What new directions should clinicians and researchers take to better understand and treat depression?*

***A*** One fruitful approach to the origins of depression might be to study how previous stressful experiences may alter the brain or neurochemistry to make people susceptible to depression in response to later stress-

## A CONVERSATION WITH

### Constance Hammen *(Continued)*

ors. One strong finding in our field is that past depression predicts future depression. Part of the explanation might be that past depression alters the organism in ways that make the person more susceptible to depression. Depression may also alter the brain's processing of information so that it is difficult to inhibit negative cognitions or to shift the focus away from negative content. More knowledge of the specific mechanisms behind becoming depressed and remaining depressed will help to focus on both cognitive-behavioral and pharmacotherapy treatments that are more specific to the underlying deficits.

I think we are also going to see more research on the heterogeneity of depression. It really is a group of different disorders, both mixtures of depression with other disorders and different forms or aspects of depression. The better we can describe the specific clinical and underlying features, the more we might be able to tailor treatment for those specific disorders.

Finally, our treatments just do not cover the need. We must conduct more research on the problem of lower-grade chronic depression. It may be subclinical depression, but if it is enduring, if it persists or recurs, it can still cause a lot of disruption in people's lives and in the lives of those around them. Also, we need better methods of disseminating treatments and making them more accessible to people. Thus, treatments must be better developed to apply to people who might not typically seek out help and should include Internet-based applications (or mobile apps), coordination within the medical primary care system, making interventions appealing and acceptable to all ages and ethnicities, and greatly expanded services for children and adolescents.

diagnosed Alina with depression. Depression can take many forms, and Alina had many of the common symptoms. She felt hopeless and overwhelmed; she could not sleep or eat well. She withdrew from her friends and from school. The decades of research reflected in the findings discussed in the chapter proved to be helpful in Alina's treatment.

The psychologist, Dr. Jane, also assessed Alina for suicidal ideation by asking her directly if she thought about killing herself, how or when she might do it, and whether she had access to lethal means. Alina reported having passive thoughts of "not wanting to be here anymore" but denied any active ideation and said she would never take her own life and had never tried to do so in the past. She was therefore considered low risk for a suicide attempt. Dr. Jane was aware of research suggesting that cognitive or interpersonal psychotherapy is at least as effective as medication in treating moderate depression such as Alina's. Based on that knowledge, she discussed those treatment options with Alina, who chose to return to the Student Counseling Center for 8–10 sessions of CBT focused on Alina's excessively high standards and expectations for herself.

Gradually, through therapy and related homework ("daily thought records"), Alina learned to think more flexibly and realize that she did not have to be perfect to be happy. After eight sessions of therapy, Alina reported feeling much better. She began going out with her friends again and, by the end of the semester, she had caught up on her academic work. Although her grades were not as good as in the past, Alina was satisfied with them. Her parents were disappointed, but, for once in her life, Alina decided that she would not apologize to her parents because of their disappointment. Two years after her treatment concluded, Alina has not suffered a relapse of depression, and she continues to use her "daily thought record" (from her CBT) sporadically to combat her distorted thoughts when she is in a bad mood.

## Summary

### Depressive Disorders

The most serious depressive disorder—major depressive disorder—involves prolonged periods of sad moods and demoralized, hopeless feelings; a loss of interest in almost all activities; and disturbances in appetite, sleep, and energy levels. Persistent depressive disorder is a depressive disorder in which depressed feelings and low self-esteem are present for at least 2 years but not as intensely as (though more chronically than) in major depressive disorder. Premenstrual dysphoric disorder involves mood swings and irritability that occur during most menstrual cycles and that remit when menstruation occurs. Disruptive mood dysregulation disorder can be diagnosed (with caution) in children between 6 and 18 years old and involves persistent irritability and temper outbursts that are grossly out of proportion to the situation or developmental level.

### Causes of Depressive Disorders

Depressive disorders are caused by a complex interplay of biological, psychological, and social factors. Genetic factors play an important role in depressive disorders, perhaps because they are linked to disturbances in chemical neurotransmitters, such as norepinephrine, serotonin, and dopamine. Depressive disorders have also been related to problems in the endocrine system (hypothalamic-pituitary-adrenal [HPA] axis), as well as possible inflammatory responses of the immune system.

Several psychological variables have been proposed as causes of depressive disorders, including heightened sensitivity to loss or failure, problems in early attachment relationships, interpersonal conflicts, learned helplessness/hopelessness, deficient reinforcement, cognitive distortions, negative self-schemas, and specific types of rumination. Environmental and sociocultural factors involving our modern world, stressful life events, and a lack of adequate social support have also been implicated.

### Treatment of Depressive Disorders

Both medication and various kinds of psychotherapy are somewhat effective treatments for depressive disorders. Antidepressant medications, such as monoamine oxidase (MAO) inhibitors, tricyclics, and selective serotonin reuptake inhibitors (SSRIs), are effective for many adults with major depressive disorder and for persistent depressive disorder, but they are less useful for children and adolescents and may have serious side effects. Some people with severe depression, especially those who do not respond well to medication, may be helped by electroconvulsive therapy (ECT). Other biological treatments include ketamine, psilocybin, light therapy, and lifestyle factors, including exercise, sleep, and nutrition.

Because of its effectiveness in preventing relapse and lack of side effects, psychotherapy is the best treatment in most cases of depression, with the exception of persistent depressive disorder or very severe depression. In many instances, psychotherapy and medications are used in combination to treat depressive disorders. Cognitive-behavioral therapy and interpersonal therapy have been particularly successful in cases of depression, but well-controlled comparison studies have not clearly established any one psychotherapy to be consistently superior to another in the treatment of depression. Even with adequate treatment of any type, the chances of a relapse of depression remain high for many people.

## Key Terms

anhedonia, p. 212
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bereavement, p. 218
catecholamine theory, p. 223
cognitive triad, p. 230
depression, p. 210
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