> Chapter 9

Watching from the Steps
by Hyacinth Manning-Carner

How much pain have cost us the evils which have never happened.

—Thomas Jefferson, letter to Thomas Jefferson Smith (February 21, 1825)

Mood Disorders <

CHAPTER OVERVIEW

Extraordinary People

- **Kay Redfield Jamison: *An Unquiet Mind***

Unipolar Depression

People with unipolar depression experience sadness, loss of interest in their usual activities, changes in sleep and activity levels, and thoughts of worthlessness, hopelessness, and suicide.

Bipolar Mood Disorders

People with bipolar disorder experience both periods of depression and periods of mania, during which their mood is elevated or irritable, and they have great energy and self-esteem. Bipolar disorder is much less common than is unipolar depression.

Biological Theories of Mood Disorders

There clearly is a heritable component to bipolar disorder and for some forms of unipolar depression. Biochemical theories suggest that imbalances in certain neurotransmitters or the malfunctioning of receptors for these neurotransmitters contributes to mood disorders. People with depression show disturbances on neuroimaging scans. They also show chronic hyperactivity of the bodily system that regulates stress responses.

Psychological Theories of Mood Disorders

Behavioral theories suggest that a lack of positive reinforcements and the presence of many aversive circumstances lead to depression. Cognitive theories suggest that people with depression interpret stressful experiences in negative and distorted ways, contributing to their depression. Psychodynamic theories describe depression as anger turned inward on the self. Interpersonal theories of depression attribute it to maladaptive social roles and patterns of relationships.

Social Perspectives on Mood Disorders

Sociologists have examined the large age, gender, and cross-cultural differences in depression for clues to its origins.

Mood Disorders Treatments

Several drugs are effective in the treatment of depression. Electroconvulsive therapy is also used to treat serious depression. The newest treatments include repetitive transcranial magnetic stimulation (rTMS) and vagus nerve stimulation. Lithium, anticonvulsants, antipsychotics, and calcium channel blockers are used to treat mania. The psychological therapies aim to reverse the processes that specific theories say lead to depression. Prevention programs intervene with high-risk groups to prevent first onsets of depression.

Taking Psychology Personally

- **Primary Care Physicians Treating Depression**

Chapter Integration

New models of mood disorders describe how genetics may affect both the individual's biological sensitivity to stress and personality characteristics that heighten reactivity to stress, contributing to depression.

Extraordinary People

Kay Redfield Jamison: *An Unquiet Mind*

> I was a senior in high school when I had my first attack. At first, everything seemed so easy. I raced about like a crazed weasel, bubbling with plans and enthusiasms, immersed in sports, and staying up all night, night after night, out with friends, reading everything that wasn't nailed down, filling manuscript books with poems and fragments of plays, and making expansive, completely unrealistic plans for my future. The world was filled with pleasure and promise; I felt great. Not just great, I felt *really* great. I felt I could do anything, that no task was too difficult. My mind seemed clear, fabulously focused, and able to make intuitive mathematical leaps that had up to that point entirely eluded me. Indeed, they elude me still. At the time, however, not only did everything make perfect sense, but it all began to fit into a marvelous kind of cosmic relatedness. My sense of enchantment with the laws of the natural world caused me to fizz over, and I found myself buttonholing my friends to tell them how beautiful it all was. They were less than transfixed by my insights into the webbings and beauties of the universe although considerably impressed at how exhausting it was to be around my enthusiastic ramblings: You're talking too fast, Kay. Slow down, Kay. You're wearing me out, Kay. Slow down, Kay. And those times when they didn't actually come out and say it, I still could see it in their eyes: For God's sake, Kay, slow down.
>
> I did, finally, slow down. In fact, I came to a grinding halt. The bottom began to fall out of my life and my mind. My thinking, far from being clearer than a crystal, was tortuous. I would read the same passage over and over again only to realize that I had no memory at all for what I had just read. My mind had turned on me: It mocked me for my vapid enthusiasms; it laughed at all my foolish plans; it no longer found anything interesting or enjoyable or worthwhile. It was incapable of concentrated thought and turned time and again to the subject of death: I was going to die, what difference did anything make? Life's run was only a short and meaningless one; why live? I was totally exhausted and could scarcely pull myself out of bed in the mornings. It took me twice as long to walk anywhere as it ordinarily did, and I wore the same clothes over and over again, as it was otherwise too much of an effort to make a decision about what to put on. I dreaded having to talk with people, avoided my friends whenever possible, and sat in the school library in the early mornings and late afternoons, virtually inert, with a dead heart and a brain as cold as clay. (Jamison, 1995, pp. 35–38)

So writes author Kay Redfield Jamison in her autobiography, *An Unquiet Mind: A Memoir of Moods and Madness*. In *An Unquiet Mind*, Jamison describes her moods, her psychotic episodes, her suicide attempts, some outrageous things she did while manic, and her resistance to taking medication. It is an intimate look inside the life of a person with severe bipolar disorder, in all its mystery and tragedy.

When Jamison published this book in 1995, it garnered a great deal of attention in both the professional psychology and psychiatry literatures as well as in the general public media. This was because it was one of the most eloquent accounts of the experience of bipolar disorder published in years. It was also because Dr. Jamison is one of the most prolific and respected researchers of mood disorders in the field. Seldom does a person in Jamison's position—a

(*continued*)

professor of psychiatry at Johns Hopkins Medical School, a leading researcher and author in the field of mood disorders, an active clinician who specializes in treating people with mood disorders, and a winner of a MacArthur Foundation "genius" award—reveal that she suffers from the very disorder she researches and treats. Throughout this chapter, we will hear more of Jamison's powerful descriptions of what it is like to have a serious mood disorder.

The emotional roller-coaster ride Kay Jamison describes is known as **bipolar disorder,** or *manic-depression*. First, Jamison had **mania,** with great energy and enthusiasm for everything, fizzing over with ideas, talking and thinking so fast that her friends could not keep up with her. Eventually, though, she crashed into a **depression.** Her energy and enthusiasm were gone, and she was slow to think, to talk, and to move. The joy was drained from her life. Bipolar disorder is one of the two major types of mood disorders. The other type is **unipolar depression.** People with unipolar depression experience only depression, no mania.

The symptoms of unipolar depression and bipolar disorder may, at first glance, seem very familiar. We often talk of feeling depressed when something bad happens. And some people get a "fizzing over" feeling of exuberance and invincibility when things are going really well in their world. People who develop mood disorders, however, experience highs and lows that most of us can only imagine.

UNIPOLAR DEPRESSION

VOICES

> From the time I woke up in the morning until the time I went to bed at night, I was unbearably miserable and seemingly incapable of any kind of joy or enthusiasm. Everything—every thought, word, movement—was an effort. Everything that once was sparkling now was flat. I seemed to myself to be dull, boring, inadequate, thick brained, unlit, unresponsive, chill skinned, bloodless, and sparrow drab. I doubted, completely, my ability to do anything well. It seemed as though my mind had slowed down and burned out to the point of being virtually useless. The wretched, convoluted, and pathetically confused mass of gray worked only well enough to torment me with a dreary litany of my inadequacies and shortcomings in character and to taunt me with the total, the desperate hopelessness of it all. (Jamison, 1995, p. 110)

Symptoms of Depression

Depression takes over the whole person—emotions, bodily functions, behaviors, and thoughts (see DSM-IV-TR criteria in Table 9.1 on page 304).

Emotional Symptoms

The most common emotion in depression is *sadness*. This sadness is not the garden variety type, which we all feel sometimes, but is a deep, unrelenting pain. As Kay Jamison wrote, she was "unbearably miserable and seemingly incapable of any kind of joy or enthusiasm." In addition, many people diagnosed with depression report that they have lost interest in everything in life—a symptom referred to as *anhedonia*. Even when they try to do something enjoyable, they may feel no emotional reaction.

Physiological and Behavioral Symptoms

In depression, many bodily functions are disrupted. These *changes in appetite, sleep, and activity levels* can take many forms. Some people with depression lose their appetite, but others find themselves eating more, perhaps even binge eating. Some people with depression want to sleep all day. Others find it difficult to sleep and may experience a form of insomnia known as *early morning waking*, in which they awaken at 3 or 4 A.M. every day and cannot go back to sleep.

Behaviorally, many people with depression are slowed down, a condition known as *psychomotor retardation*. They walk more slowly, gesture more slowly, and talk more slowly and quietly. They

TABLE 9.1 DSM-IV-TR

Symptoms of Depression

Depression includes a variety of emotional, physiological, behavioral, and cognitive symptoms.

Emotional Symptoms

Sadness
Depressed mood
Anhedonia (loss of interest or pleasure in usual activities)
Irritability (particularly in children and adolescents)

Physiological and Behavioral Symptoms

Sleep disturbances (hypersomnia or insomnia)
Appetite disturbances
Pyschomotor retardation or agitation
Catatonia (unusual behaviors ranging from complete lack of movement to excited agitation)
Fatigue and loss of energy

Cognitive Symptoms

Poor concentration and attention
Indecisiveness
Sense of worthlessness or guilt
Poor self-esteem
Hopelessness
Suicidal thoughts
Delusions and hallucinations with depressing themes

have more accidents, because they cannot react to crises as quickly as necessary to avoid them. Many people with depression *lack energy* and report feeling chronically *fatigued.* A subset of people with depression have *psychomotor agitation* instead of retardation. They feel physically agitated, cannot sit still, and may move around or fidget aimlessly.

Cognitive Symptoms

The thoughts of people with depression may be filled with themes of *worthlessness, guilt, hopelessness,* and even *suicide.* They often have trouble concentrating and making decisions. Again, as Kay Jamison described, "It seemed as though my mind had slowed down and burned out to the point of being virtually useless."

In some severe cases, the cognitions of people with depression lose complete touch with reality, and they experience delusions and hallucinations. **Delusions** are beliefs with no basis in reality, and **hallucinations** involve seeing, hearing, or feeling things that are not real. The delusions and hallucinations that people with depression experience usually are depressing and negative in content. For example, people have delusions that they have committed a terrible sin, that they are being punished, or that they have killed or hurt someone. They may have auditory hallucinations in which voices accuse them of having committed an atrocity or instruct them to kill themselves.

The Diagnosis of Unipolar Depressive Disorders

Depression takes several forms. The DSM-IV-TR recognizes two categories of unipolar depression: **major depression** and **dysthymic disorder.** The diagnosis of major depression requires that a person experience either depressed mood or loss of interest in usual activities, plus at least four other symptoms of depression chronically for at least two weeks. In addition, these symptoms have to be severe enough to interfere with the person's ability to function in everyday life.

Dysthymic disorder is a less severe form of depressive disorder than is major depression, but it is more chronic. To be diagnosed with dysthymic disorder, a person must be experiencing depressed mood plus two other symptoms of depression for at least *two years.* During these two years, the person must never have been without the symptoms of depression for more than a two-month period. Said one woman with dysthymic disorder, "It just goes on and on. I never feel really good; I always feel kind of bad, and it seems it's never going to end."

Some unfortunate people experience both major depression and dysthymic disorder. This has been referred to as **double depression.** People with double depression are chronically dysthymic, then occasionally sink into episodes of major depression. As the major depression passes, however, they return to dysthymia rather than recover to a normal mood. As one might imagine, people with double depression are even more debilitated than are people with major depression or dysthymia. One study that followed people with double depression over about nine years found that they remained free of the symptoms of minor or severe depression only about one-third of that time (Judd

TABLE 9.2 DSM-IV-TR

Subtypes of Major Depression (and the Depressive Phase of Bipolar Disorder)

The DSM-IV-TR specifies a number of subtypes of major depression and the depressive phase of bipolar disorder.

Subtype	Characteristic Symptoms
With melancholic features	Inability to experience pleasure, distinct depressed mood, depression regularly worse in morning, early morning awakening, marked psychomotor retardation or agitation, significant anorexia or weight loss, excessive guilt
With psychotic features	Presence of depressing delusions or hallucinations
With catatonic features	Catatonic behaviors: catalepsy, excessive motor activity, severe disturbances in speech
With atypical features	Positive mood reactions to some events, significant weight gain or increase in appetite, hypersomnia, heavy or laden feelings in arms or legs, long-standing pattern of sensitivity to interpersonal rejection
With postpartum onset	Onset of major depressive episode within four weeks of delivery of child
With seasonal pattern	History of at least two years in which major depressive episodes occur during one season of the year (usually the winter) and remit when the season is over

et al., 1998). People with double depression also are less likely to respond to treatments.

Over half of the people diagnosed with major depression or dysthymia also have another psychological disorder. The most common disorders to co-occur with depression are substance abuse, such as alcohol abuse; anxiety disorders, such as panic disorder; and eating disorders (Blazer et al., 1994). Sometimes, the depression precedes and perhaps causes the other disorder. In other cases, depression follows and may be the consequence of the other disorder.

The DSM-IV-TR also recognizes several subtypes of depression—different forms the disorder can take (see DSM-IV-TR criteria in Table 9.2). These subtypes apply both to major depression and to the depressive phase of a bipolar disorder.

The first subtype of depression is *depression with melancholic features,* in which the physiological symptoms of depression are particularly prominent. Second is *depression with psychotic features*, in which people experience delusions and hallucinations during a major depressive episode. Third, people with *depression with catatonic features* show the strange behaviors collectively known as *catatonia*, which can range from a complete lack of movement to excited agitation. Fourth, there is *depression with atypical features*. The criteria for this subtype are an odd assortment of symptoms (review Table 9.2).

The fifth subtype is *depression with postpartum onset*. This diagnosis is given to women when the onset of a major depressive episode occurs within four weeks of the delivery of a child. More rarely, women develop mania postpartum and are given the diagnosis of *bipolar disorder with postpartum onset*. As many as 30 percent of women experience the *postpartum blues*—emotional lability (unstable and quickly shifting moods), frequent crying, irritability, and fatigue—in the first few weeks after giving birth. For most women, these symptoms are only annoying and pass completely within two weeks of the birth. Only about 1 in 10 women experience postpartum depressions serious enough to warrant a diagnosis of a depressive disorder (Steiner, Dunn & Born, 2003).

The final subtype of major depressive disorder is *depression with seasonal pattern*, sometimes referred to as **seasonal affective disorder,** or **SAD.** People with SAD have a history of at least two years of experiencing major depressive episodes and fully recovering from them. The symptoms seem to be tied to the number of daylight hours in a day. People become depressed when the daylight hours are short and recover when the daylight hours are long. In the northern hemisphere, this means people are depressed November through February and not depressed the remainder of the year. Some people with this disorder actually develop mild forms of mania or have full manic episodes during the summer months and are diagnosed with *bipolar disorder with seasonal pattern*. In order to be diagnosed with seasonal affective disorder, a person's mood

People in northern latitudes have higher rates of seasonal affective disorder.

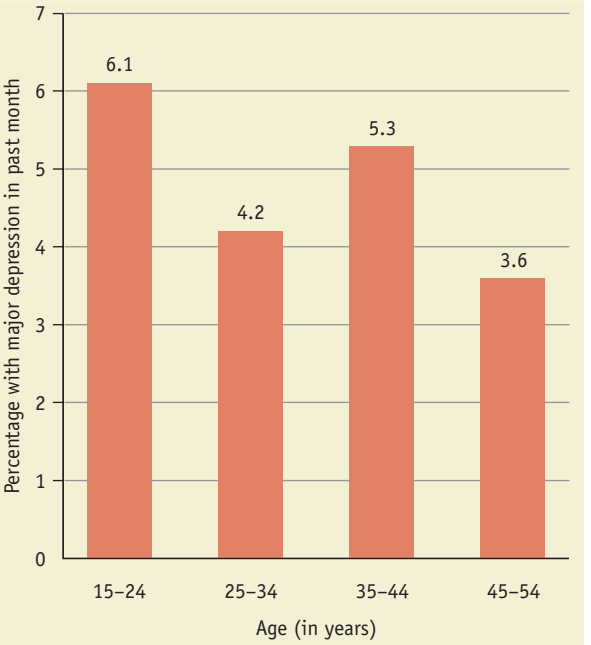
changes cannot be the result of psychosocial events, such as regularly being unemployed during the winter. Rather, the mood changes must seem to come on without reason or cause.

Although many of us may feel our mood changes with the seasons, only about 1 percent of the U.S. population experiences a diagnosable seasonal affective disorder (Blazer, Kessler, & Swartz, 1998). This disorder is more common in latitudes where there are fewer hours of daylight in the winter months (Rosen et al., 1990). For example, people in Norway and Sweden are more prone to SAD than are people in Mexico and southern Italy.

Prevalence and Course of Depression

Depression is one of the most common psychological problems. At sometime in their lives, 16 percent of Americans experience an episode of major depression (Kessler et al., 2003). Among adults, 15- to 24-year-olds are most likely to have had a major depressive episode in the past month (Blazer et al., 1994; Kessler et al., 2003) (see Figure 9.1). There are lower rates among 45- to 54-year-olds, and other studies have found even lower rates in people 55 to 70 years of age, with only about 2 percent diagnosable with a major depression (Kessler et al., 2003; Newmann, 1989; Zisook & Downs, 1998). The rates of depression go up, however, among the "old-old," those over 85 years of age. When they do occur, depressions in older people tend to be quite severe, chronic, and debilitating (Lyness, 2004).

FIGURE 9.1 **Age Differences in Depression.** Shown are the percentages, in one study, of people in each age group who were diagnosed with major depression in a one-month period. Those 15 to 24 years old have the highest rates of depression, and those 45 to 54 years old have the lowest rates.



Source: Blazer et al., 1994.

Perhaps it is surprising that the rate of depression is so low among older adults. The diagnosis of depression in older adults is complicated (Lyness, 2004). First, older adults may be less willing than younger adults to report the symptoms of depression, because they grew up in a society less accepting of the disorder.

Second, depressive symptoms in the elderly often occur in the context of a serious medical illness, which can interfere with making an appropriate diagnosis. Third, older people are more likely than younger people to have mild to severe cognitive impairment, and it is often difficult to distinguish between a depressive disorder and the early stages of a cognitive disorder.

Although these factors are important, some researchers suggest that the low rate is valid and have offered explanations. The first is quite grim: Depression appears to interfere with physical health; as a result, people with a history of depression may be more likely to die before they reach old age (Lyness, 2004). The second explanation is more hopeful: As people age, they may develop

more adaptive coping skills and a psychologically healthier outlook on life, and this may lead them to experience fewer episodes of depression (Elder, Liker, & Jaworski, 1984).

Most studies show that women are about twice as likely as men to experience both mild depressive symptoms and severe depressive disorders (Nolen-Hoeksema, 2002). This gender difference in depression has been found in many countries, in most ethnic groups, and in all adult age groups. Could it be that females are more willing to admit to depression than males? The gender difference in depression is found even in studies that use relatively objective measures of depression that do not rely much on self-reports, such as clinicians' ratings of depression, or the reports of family members or friends. As we discuss the various theories of depression in this chapter, we will explore how these theories explain this gender difference in depression.

Depression appears to be a long-lasting, recurrent problem for some people (Boland & Keller, 2002). One nationwide study found that people with major depression spent an average of 16 weeks during the previous year with significant symptoms of depression (Kessler et al., 2003). The picture one gets is of a depressed person spending much of his or her time at least moderately depressed (Judd & Akiskal, 2000). Then, even after the depressed person recovers from one episode of depression, he or she remains at high risk for relapses into new episodes. People with a history of multiple episodes of depression are even more likely to remain depressed for long periods of time.

Depression is a costly disorder to the individual and to society. A study of over 1,100 employed people found that those who had significant symptoms of depression lost an average of 5.6 hours per week in productive work time, compared with 1.5 hours per week in those not depressed. The authors suggest that depression in workers costs employers an estimated $44 billion per year in lost productivity alone (not including the costs of treatment) (Stewart et al., 2003).

The good news is that, once people undergo treatment for their depression, they tend to recover much more quickly and their risk for relapse is reduced. The bad news is that many people with depression never seek care, or they wait years after their symptoms have begun to seek care (Kessler et al., 2003). Why don't people suffering the terrible symptoms of depression seek treatment? It may be because they do not have the money or insurance to pay for care. But often it is because they feel they should be able to get over their symptoms on their own. They believe that the symptoms are just a phase they are going through, that they will pass with time and won't affect their lives in the long term.

Depression does sometimes pass without treatment, and without long-term consequences. Some people seem to be left with scars from their bouts of depression, however. Their ways of thinking, their views of themselves, their social relationships, and their academic and work histories may be changed for the worse by the depression and may remain impaired long after the symptoms of depression have passed. Even if they do not relapse into additional major depressive episodes, people with previous episodes of major depression tend to have enduring problems in many areas of their lives (Boland & Keller, 2002). Their functioning on the job tends to remain impaired even after their depression has subsided. They report that they are not interested in sex or do not enjoy sex as much as they used to, and there is chronic conflict and dissatisfaction in their intimate relationships (Joiner, 2002).

Depression in Childhood and Adolescence

Depression is less common among children than among adults. At any point in time, as many as 2.5 percent of children and 8.3 percent of adolescents can be diagnosed with major depression, and as many as 1.7 percent of children and 8.0 percent of adolescents can be diagnosed with dysthymic disorder (for reviews, see Garber & Horowitz, 2002; Lewinsohn & Essau, 2002). Between 15 and 20 percent of youth will experience an episode of major depression before the age of 20 (Lewinsohn & Essau, 2002).

Depressive symptoms that don't quite meet the diagnostic criteria for major depression are even more common in adolescents. A study of 9,863 students in grades 6, 8, and 10 in the United States found that 25 percent of the girls versus 10 percent of the boys reported elevated depressive symptoms. The highest rates were among American Indians (29 percent), followed by 22 percent of Hispanics, 18 percent of Whites, 17 percent of Asian Americans, and 15 percent of African Americans. Youth who were using substances were more likely to be depressed (Saluja et al., 2004).

The Scars of Childhood Depression

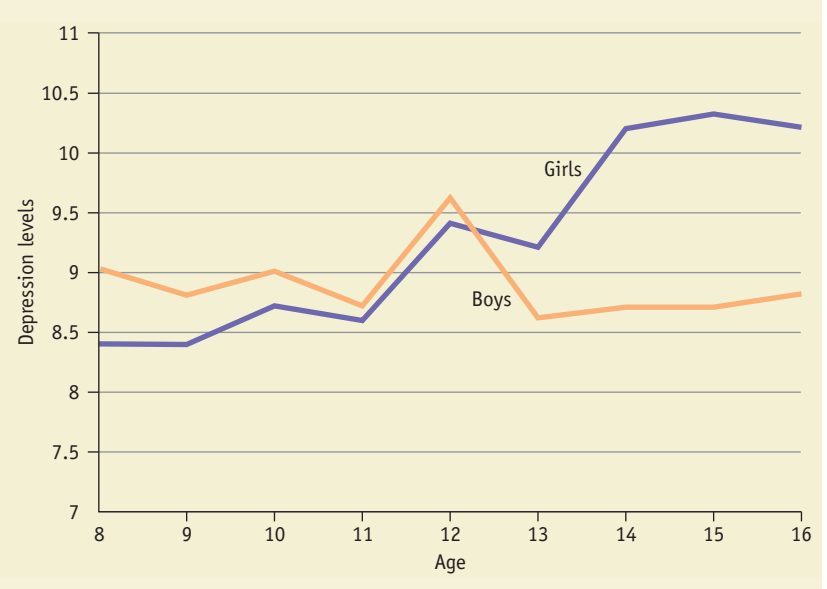
Depression may be most likely to leave psychological and social scars if it occurs initially during childhood, rather than during adulthood (Cole et al., 1998). Self-concept is still being developed in childhood and adolescence, much more so than in adulthood. A period of significant depressive symptoms while one's self-concept is undergoing substantial

change can have long-lasting effects on the content or structure of one's self-concept. Similarly, the development of skills and abilities in school is cumulative during childhood and adolescence. For this reason, a bout of depression that interferes with learning can have long-term effects on children's achievement. Finally, children and adolescents are dependent on and connected with other people to a greater extent than are adults, so a bout of depression that impairs social skills can have long-term effects on social relationships.

Depression may also increase negative thinking, because it brings with it a host of new negative events. Stress-generation models suggest that the symptoms of depression—such as low motivation, fatigue, problems in concentration, low self-esteem, and decreases in social interactions and skills—can interfere with youngsters' functioning in all domains of their lives (Hammen, 1991, 1992). Because depression affects so many domains of functioning in a youngster's life, it may lead to increases in many kinds of stressors. For example, having a depressed child in the family can cause strains on parents, which may affect their relationships, perhaps putting a fragile marriage or partnership over the edge and contributing to separation. Or the cost of treatment for a depressed child may cause significant financial strain in a family.

Depression in less common in children than in adults, but can be debilitating and have long-term consequences.

FIGURE 9.2 **Emergence of a Gender Difference in Depression.** In childhood, boys and girls have relatively equal levels of depression, but, beginning around age 13, girls' levels of depression rise, whereas boys' levels do not.



Source: Twenge & Nolen-Hoeksema, 2002.

The Effects of Puberty

Girls' rates of depression escalate dramatically over the course of puberty, but boys' rates do not (Twenge & Nolen- Hoeksema, 2002) (see Figure 9.2). Although there is some evidence that girls' increase in depressive symptoms is correlated with the hormonal changes of puberty (Angold, Costello, & Worthman, 1998), the observable physical changes of adolescence may have more to do with the emotional development of girls and boys than with hormonal development, because these characteristics affect boys' and girls' self-esteem differently. Girls appear to value the physical changes that accompany puberty much less than do boys. In particular, girls dislike the weight they gain in fat and their loss of the long, lithe look that is idealized in modern fashions. In contrast, boys like the increase in muscle mass and other pubertal changes their bodies undergo (Dornbusch et al., 1984). Body dissatisfaction appears to be more closely related to low self-esteem and depression in girls than in boys (Allgood-Merten, Lewinsohn, & Hops, 1990).

The pubertal increase in depression for girls may occur only among European American girls, however, not among African American and Latino girls (Hayward et al., 1999). It may be that African American and Latino girls do not accept the pressures to be thin as much as European American girls do, and this protects them against declines in their self-image and well-being with the onset of puberty. The social environment of African American and Latino girls may also protect them against depression in other ways, although currently there is too little research to determine what factors are important.

SUMMING UP

- Depression includes disturbances in emotion (sadness, loss of interest), bodily function (loss of sleep, appetite, and sexual drive), behavior (retardation or agitation), and thought (worthlessness, guilt, suicidality).
- The two primary categories of unipolar depression are major depression and dysthymic disorder. There are several subtypes of major depression.
- Young adults have the highest rates of depression.
- Many people who become depressed remain so for several months or more and have multiple relapses over their lifetimes.

BIPOLAR MOOD DISORDERS

VOICES

> There is a particular kind of pain, elation, loneliness, and terror involved in this kind of madness. When you're high it's tremendous. The ideas and feelings are fast and frequent like shooting stars and you follow them until you find better and brighter ones. Shyness goes, the right words and gestures are suddenly there, the power to seduce and captivate others a felt certainty. There are interests found in uninteresting people. Sensuality is pervasive and the desire to seduce and be seduced irresistible. Feelings of ease, intensity, power, well-being, financial omnipotence, and euphoria now pervade one's marrow.
>
> But, somewhere, this changes. The fast ideas are far too fast and there are far too many; overwhelming confusion replaces clarity. Memory goes. Humor and absorption on friends' faces are replaced by fear and concern. Everything previously moving with the grain is now against—you are irritable, angry, frightened, uncontrollable, and enmeshed totally in the blackest caves of the mind. You never knew those caves were there. It will never end. (Goodwin & Jamison, 1990, pp. 17–18)

TABLE 9.3 DSM-IV-TR

Symptoms of Mania

A diagnosis of mania requires that a person show an elevated, expansive, or irritable mood for at least one week, plus at least three of the other symptoms listed here.

Elevated, expansive, or irritable mood
Inflated self-esteem or grandiosity
Decreased need for sleep
More talkative than usual, a pressure to keep talking
Flight of ideas or sense that your thoughts are racing
Distractibility
Increase in activity directed at achieving goals
Excessive involvement in potentially dangerous activities

Source: Reprinted with permission from the *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition, Text Revision. Copyright © 2000 American Psychiatric Association.

This person is describing an episode of bipolar disorder. When she is manic, she has tremendous energy and vibrancy, her self-esteem is soaring, and she is filled with ideas and confidence. Then, when she becomes depressed, she is despairing and fearful, she doubts herself and everyone around her, and she wishes to die. This alternation between periods of mania and periods of depression is the classic manifestation of bipolar disorder.

Symptoms of Mania

We have already discussed the symptoms of depression in detail, so let's focus on the symptoms of mania (see the DSM-IV-TR criteria in Table 9.3). The moods of people who are manic can be *elated*, but that elation is often mixed with *irritation* and *agitation*.

> First and foremost comes a general sense of intense well-being. I know of course that this sense is illusory and transient—

(*continued*)

VOICES

> Although, however, the restrictions of confinement are apt at times to produce extreme irritation and even paroxysms of anger, the general sense of wellbeing, the pleasurable and sometimes ecstatic feeling-tone, remains as a sort of permanent background of all experience during a manic period. (Goodwin & Jamison, 1990, pp. 25–26)

The manic person is filled with a *grandiose self-esteem*, meaning that his view of himself is unrealistically positive and inflated. *Thoughts* and *impulses* race through his mind. At times, these grandiose thoughts are delusional and may be accompanied by grandiose hallucinations. A manic person may *speak rapidly* and *forcefully*, trying to convey the rapid stream of fantastic thoughts he is having. He may become agitated and irritable, particularly with people he perceives as "getting in his way." He may engage in a variety of *impulsive behaviors*, such as ill-advised sexual liaisons or spending sprees. He may have *grand plans* and *goals*, which he pursues frenetically.

The Diagnosis of Mania

In order to be diagnosed with mania, an individual must show an elevated, expansive, or irritable mood for at least one week, plus at least three of the other symptoms listed in Table 9.3. These symptoms must impair the individual's ability to function in order to qualify for the diagnosis.

People who experience manic episodes meeting these criteria are said to have **bipolar I disorder.** Most of these people eventually fall into a depressive episode. For some people with bipolar I disorder, the depressions are as severe as major depressive episodes, whereas others have episodes of depression that are relatively mild and infrequent. People with **bipolar II disorder** experience severe episodes of depression that meet the criteria for major depression, but their episodes of mania are milder and are known as **hypomania** (see the DSM-IV-TR criteria in Table 9.4). Hypomania has the same symptoms as mania. The major difference is that, in hypomania, these symptoms are not severe enough to interfere with daily functioning and do not involve hallucinations or delusions.

Just as dysthymic disorder is the less severe but more chronic form of unipolar depression, there is a less severe but more chronic form of bipolar disorder, known as **cyclothymic disorder.** A person with cyclothymic disorder alternates between episodes of hypomania and moderate depression chronically over at least a two-year period. During the periods of hypomania, the person may be able to function reasonably well in daily life. Often, however, the periods of depression significantly interfere with daily functioning, although these periods are not as severe as those qualifying as major depressive episodes.

About 90 percent of people with bipolar disorder have multiple episodes or cycles during their lifetime (APA, 2000). The length of an individual episode of bipolar disorder varies greatly from one person to the next. Some people are in a manic state for several weeks or months before moving into a depressed state. More rarely, people switch from mania to depression and back within a matter of days. The number of lifetime episodes also varies tremendously from one person to the next,

TABLE 9.4 DSM-IV-TR

Criteria for Bipolar I and Bipolar II Disorders

Bipolar I and II disorders differ in the presence of major depressive episodes, episodes meeting the full criteria for mania, and hypomanic episodes.

Criteria	Bipolar I	Bipolar II
Major depressive episodes	Can occur but are not necessary for diagnosis	Are necessary for diagnosis
Episodes meeting full criteria for mania	Are necessary for diagnosis	Cannot be present for diagnosis
Hypomanic episodes	Can occur between episodes of severe mania or major depression but are not necessary for diagnosis	Are necessary for diagnosis

but a relatively common pattern is for episodes to become more frequent and closer together over time. If a person has four or more cycles of mania and depression within a year, this is known as **rapid cycling bipolar disorder.**

Prevalence and Course of Bipolar Disorder

Bipolar disorder is less common than unipolar depression. About 1 or 2 in 100 people experience at least one episode of bipolar disorder at sometime in their lives (Judd & Akiskal, 2003; Kessler et al., 1994; Lewinsohn, Klein, & Seeley, 2000). Men and women seem equally likely to develop the disorder, and there are no consistent differences among ethnic groups in the prevalence of the disorder (Weissman et al., 1996). Most people who develop bipolar disorder do so in late adolescence or early adulthood (Lewinsohn, Seeley, Klein, 2003). About half of the people who eventually develop a bipolar disorder have experienced their first episode by early adulthood (Judd & Akiskal, 2003).

Like people with unipolar depression, people with bipolar disorder often face chronic problems on the job and in their relationships between their episodes (Keck et al., 1998). One study, which followed people who had been hospitalized for an episode of bipolar disorder, found that, over the year following their hospitalization, only about one in four recovered fully from their symptoms and were able to lead a relatively normal life (Keck et al., 1998). The best predictors of recovery were full compliance with medication taking and higher social class, which may have afforded people better health care and social support. Judd and colleagues (2002) followed 146 patients with bipolar I disorder for almost 13 years and found that they experienced significant symptoms during 47 percent of the weeks. Depressive symptoms were more common, occurring 32 percent of the weeks, than manic symptoms, which occurred about 9 percent of the weeks, or cycling/mixed symptoms of depression and mania, which occurred 6 percent of the weeks. The presence of symptoms, even if they do not meet the criteria for an episode of mania or depression, is associated with deficits in both social and occupational functioning, and the symptoms appear to increase the risk for relapse (Marangell, 2004). In addition, people with bipolar disorder often abuse substances (such as alcohol and hard drugs), which also impairs their control over their disorder, their willingness to take medications, and their functioning in life (Goodwin & Ghaemi, 1998; Keck et al., 1998; van Gorp et al., 1998).

A controversial issue in research on bipolar disorder is the extent to which it exists and can be diagnosed reliably in children and young adolescents. One longitudinal study followed 86 prepubertal children who had been diagnosed with bipolar disorder using strict criteria (Geller et al., 2004). They found that, over a two-year period, these children continued to show the symptoms of mania or hypomania for an average of 57 weeks and the symptoms of depression for an average of 47 weeks. These data supported the initial diagnosis of bipolar disorder and suggest that pediatric bipolar disorder tends to be chronic.

Creativity and Bipolar Disorder

Could there possibly be anything good about suffering from a bipolar disorder? Some theorists have argued that the symptoms of mania—increased self-esteem, a rush of ideas, the courage to pursue these ideas, high energy, little need for sleep, hypervigilance, and decisiveness—can actually benefit certain people, especially highly intelligent or talented people. In turn, the melancholy of depression is often seen as inspirational for artists. Indeed, some of the most influential people in history have suffered, and perhaps to some extent benefited, from a mood disorder.

Some political leaders, including Abraham Lincoln, Alexander Hamilton, Winston Churchill, Napoleon Bonaparte, and Benito Mussolini, and some religious leaders, including Martin Luther and George Fox (founder of the Society of Friends, or Quakers), have been posthumously diagnosed by psychiatric biographers as having periods of mania, hypomania, or depression (Jamison, 1993). Although during periods of depression these leaders were often incapacitated, during periods of mania and hypomania they accomplished extraordinary feats. While manic, they devised brilliant and daring strategies for winning wars and solving domestic problems and had the energy, self-esteem, and persistence to carry out these strategies. The Duke of Marlborough, a great English military commander, was able to put his chronic hypomania to great use:

> No one can read the whole mass of the letters which Marlborough either wrote, dictated, or signed personally without being astounded at the mental and physical energy which it attests. . . . After 12 or 14 hours in the saddle on the long reconnaissances often under cannon-fire; after endless inspections of troops in camp and garrison; after ceaseless calculations about food and supplies, and all the anxieties of direct command in war, Marlborough

> would reach his tent and conduct the foreign policy of England, decided the main issues of its Cabinet, and of party politics at home. (Rowse, 1969, pp. 249–250)

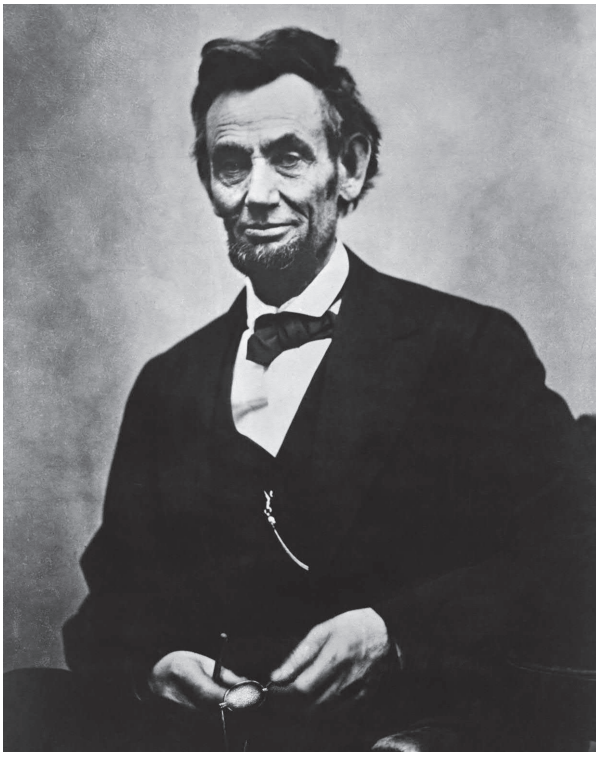
Marlborough was an ancestor of Winston Churchill, who was also able to put his cyclothymic temperament to use in his career. However, Churchill's biographer also documented how the grandiosity, scheming, and impulsiveness that are part of mania can be a liability in a leader:

> All those who worked with Churchill paid tribute to the enormous fertility of his new ideas, the inexhaustible stream of invention which poured from him, both when he was Home Secretary, and later when he was Prime Minister and director of the war effort. All who worked with him also agreed that he needed the most severe restraint put upon him, and that many of his ideas, if they had been put into practice, would have been utterly disastrous. (Storr, 1988, pp. 14–15)

Writers, artists, and composers of music have a higher than normal prevalence of mania and depression. For example, a study of 1,005 famous twentieth-century artists, writers, and other professionals found that the artists and writers experienced two to three times the rate of mood disorders, psychosis, and suicide attempts than did comparably successful people in business, science, and public life. The poets in this group were most likely to have been manic (Ludwig, 1992).

Does mania simply enhance (and depression inhibit) productivity in naturally creative people? Or is there a deeper link between creativity and bipolar disorder? This is a difficult question to answer by simply examining how many creative people are also manic. However, one group of researchers found an ingenious way to address this question (Richards et al., 1988). They hypothesized that the genetic abnormalities that cause bipolar disorder are in close proximity to the genetic abnormalities that cause great creativity. According to this hypothesis, the close relatives of patients with bipolar disorder should be more creative, even if they do not have bipolar disorder, than the close relatives of people without bipolar disorder. The participants in this study were patients with bipolar disorder or cyclothymia, their first-degree relatives (siblings, parents, and children), a control group of people with no psychiatric disorders, and their first-degree relatives. The relatives in both

Winston Churchill had periods of manic symptoms that may have been both an asset and a liability.



Abraham Lincoln suffered periods of severe depression.

these groups had no history themselves of mood disorders, so any creativity they showed was in the absence of mania or depression.

To measure creativity, the researchers examined the lives of these participants for evidence that they had used their special talents in original, and creative ways. For example, one participant who was rated as extremely creative was an entrepreneur who had advanced from a chemist's apprentice to an independent researcher of new products. He then had started a major paint manufacturing company, and, during the Danish Resistance of World War II, he had surreptitiously manufactured and smuggled explosives for the Resistance. A participant who was rated as low in creativity had been a bricklayer for 20 years and then inherited a large trust fund and retired to a passive life on a country estate. An advantage of this measure of creativity is that it did not require that a person receive social recognition to be considered creative.

The results of this study suggested that the relatives of the people with bipolar disorder or cyclothymia were more creative than the participants with no history of bipolar disorder or cyclothymia or their relatives. The people with cyclothymia and the healthy relatives of those with bipolar disorder had somewhat higher creativity scores than did the patients who had bipolar disorder. This suggests that creativity that is associated with a predisposition toward bipolar disorder is more easily expressed in people who do not suffer from full episodes of mania and depression but may suffer from milder mood swings (Richards et al., 1988).

We should not overemphasize the benefits of bipolar disorder. Although many creative people with bipolar disorder may have been able to learn from their periods of depression and to exploit their periods of mania, many also have found the highs and lows of the disorder unbearable and have attempted or completed suicide. As Wurtzel (1995, p. 295) notes,

> While it may be true that a great deal of art finds its inspirational wellspring in sorrow, let's not kid ourselves in how much time each of those people wasted and lost by being mired in misery. So many productive hours slipped by as paralyzing despair took over. This is not to say that we should deny sadness its rightful place among the muses of poetry and of all art forms, but let's stop calling it madness, let's stop pretending that the feeling itself is interesting. Let's call it depression and admit that it is very bleak.

SUMMING UP

- The symptoms of mania include elation, irritation and agitation, grandiosity, impulsivity, and racing thoughts and speech. People with bipolar disorder experience periods of both mania and depression.
- The two major diagnostic categories of bipolar mood disorders are bipolar disorder and cyclothymic disorder.
- Bipolar mood disorders are less common than unipolar depression, but they are equally common in men and women.
- The onset of bipolar disorder is most often in late adolescence or early adulthood. Most people with bipolar disorder have multiple episodes.
- There is some evidence that people with bipolar disorder are more creative.

BIOLOGICAL THEORIES OF MOOD DISORDERS

Most of the modern biological theories of the causes of mood disorders focus on genetic abnormalities or dysfunctions in certain neurobiological systems. These two types of theories complement each other: Genetic abnormalities may cause mood disorders by altering a person's neurobiology. In this section, we first review the evidence for a genetic contribution to depression and mania. Second, we review the evidence that neurotransmitters play a role in depression and mania. Third, we examine a variety of abnormalities that have been found in the brains of people with mood disorders. Fourth, we explore hypotheses that the neuroendocrine system, which regulates hormones throughout the body, becomes dysregulated in the mood disorders (see the Concept Overview in Table 9.5 on page 314).

The Role of Genetics

Family history and twin studies suggest that the mood disorders can be transmitted genetically (Southwick, Vythilingam, & Charney, 2005; Wallace, Schneider, & McGuffin, 2002).

Family History Studies

Family history studies of people with bipolar disorder find that their first-degree relatives (i.e., parents, children, and siblings) have rates of both bipolar disorder and unipolar depression at least two to three times higher than the rates of relatives of people without bipolar disorder (MacKinnon, Jamison, & DePaulo, 1997; Wallace et al., 2002) (Figure 9.3 on pae 314).

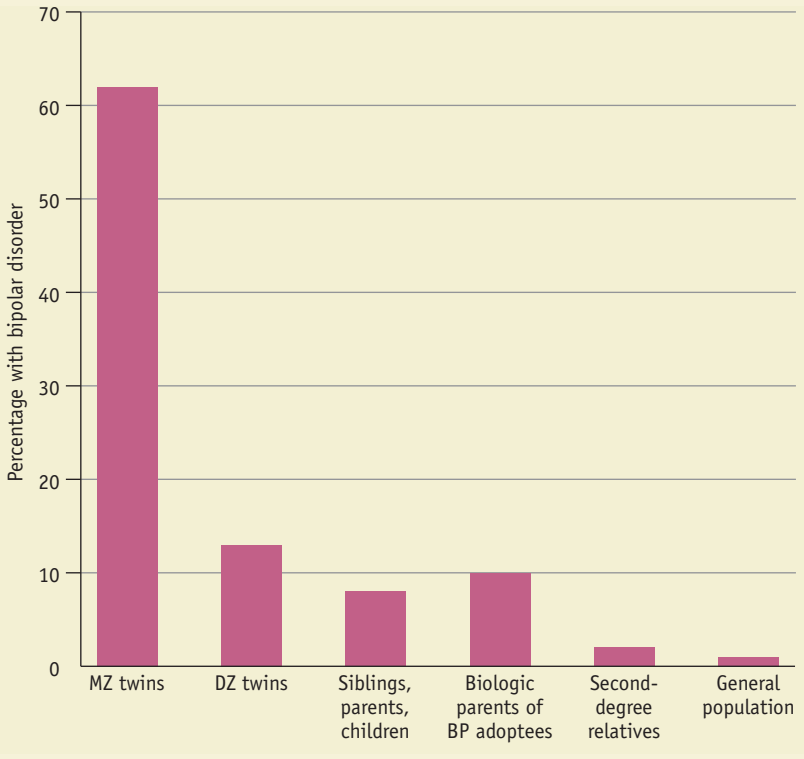
TABLE 9.5 Concept Overview

Biological Theories of Mood Disorders

A number of biological factors have been implicated in the mood disorders.

Theory	Description
Genetic theory	Disordered genes predispose people to depression or bipolar disorder.
Neurotransmitter theories	Dysregulation of neurotransmitters and their receptors causes depression and mania. The monoamine neurotransmitters—norepinephrine, serotonin, and dopamine—have been most researched.
Neurophysiological abnormalities	Abnormalities in the structure and functioning of the prefrontal cortex, hippocampus, anterior cingulate cortex, and amygdala.
Neuroendocrine abnormalities	Depressed people show chronic hyperactivity in the hypothalamic-pituitary-adrenal axis and slow return to baseline after a stressor, which affects the functioning of neurotransmitters.

FIGURE 9.3 **Risk for Bipolar Disorder in Relatives of People with Bipolar Disorder and in the General Population.** The risk of developing bipolar disorder decreases as the genetic similarity between an individual and a relative with bipolar disorder decreases.



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Does this mean that, if you have a close relative with bipolar disorder, you are destined to develop the disorder? No—most studies find that fewer than 10 percent (and often less than 5 percent) of the first-degree relatives of people with bipolar disorder develop the disorder themselves (MacKinnon et al., 1997) (review Figure 9.3). In other words, the risk is higher for people with a bipolar relative, but only a minority of them develop the disorder.

Unipolar depression also clearly runs in families. Family history studies find that the first-degree relatives of people with unipolar depression are two to three times more likely also to have depression, compared with the first-degree relatives of people without the disorder (Klein et al., 2001). Interestingly, the relatives of people with depression do *not* tend to have any greater risk for bipolar disorder than do the relatives of people with no mood disorder. This suggests that bipolar disorder has a genetic basis different from that of unipolar depression.

Twin Studies

Twin studies of bipolar disorder have shown that the probability that both twins will develop the disorder, or its *concordance rate,* is about 60 percent among monozygotic (identical) twins, compared with about 13 percent among dizygotic (nonidentical) twins (MacKinnon et al., 1997; McGuffin & Katz, 1989; Wallace et al., 2002). This finding suggests that genetics plays a substantial role in vulnerability to bipolar disorder.

Twin studies of major depression also find higher concordance rates for monozygotic twins than for dizygotic twins (e.g., Kendler et al., 2001). Some twin studies of major depression suggest that genetics plays a heavier role in this disorder for women than for men (Kendler et al., 2001). Other twin studies, however, have found no gender difference in the heritability of depression (Eaves et al., 1997; Kendler & Prescott, 1999; Rutter et al., 1999). Still other studies suggest that the types of genes responsible for depression may differ between women and men (Zubenko et al., 2002).

Specific Genetic Abnormalities

What kinds of genetic abnormalities might play a role in these disorders? One specific genetic abnormality that some studies suggest may be involved in the vulnerability to depression is on the serotonin transporter gene (Southwick et al., 2005). As we will discuss shortly, serotonin is one of the neurotransmitters implicated in depression. Abnormalities on the serotonin transporter gene could lead to dysfunction in the regulation of serotonin, which in turn could affect the stability of individuals' moods. In a longitudinal study, Caspi and colleagues (2003) found that people with abnormalities on the serotonin transporter gene were at increased risk for depression when they faced negative life events.

It is likely that there is no single location on a gene that leads to mood disorders. Many researchers believe that the genetic predisposition to mood disorders is *multifactorial*—it involves many factors. That is, a particular configuration of several disordered genes may be necessary to create a mood disorder.

Neurotransmitter Dysregulation

Most of the biochemical theories of mood disorders have focused on neurotransmitters, the biochemicals that facilitate the transmission of impulses across the synapses between neurons. Many different neurotransmitters may play a role in the mood disorders, but the neurotransmitters that have been implicated most often in the mood disorders are the **monoamines.**

The specific monoamines that have been implicated are **norepinephrine, serotonin,** and, to a lesser extent, **dopamine.** These neurotransmitters are found in large concentrations in the *limbic system,* a part of the brain associated with the regulation of sleep, appetite, and emotional processes. These neurotransmitters are thought to cause both depression and mania—imbalances in one direction may cause depression and imbalances in the other direction may cause mania.

The early theory of the roles of these neurotransmitters in mood disorders was that depression was caused by a reduction in the amount of norepinephrine or serotonin in the synapses between neurons (Glassman, 1969; Schildkraut, 1965). This depletion could occur for numerous reasons: decreased synthesis of the neurotransmitter from its precursors, increased degradation of the neurotransmitter by enzymes, or impaired release or reuptake of the neurotransmitter (see Chapter 2 to review these processes). Mania was thought to be caused by an excess of the monoamines or perhaps dysregulation of the levels of these amines, especially dopamine. Taken together, these theories are known as the **monoamine theories** of mood disorders (Bunney & Davis, 1965; Schildkraut, 1965).

More recent studies of the monoamine theories have focused on the number and functioning of receptors for the monoamines on neurons in people suffering from mood disorders (Southwick et al., 2005). Recall from Chapter 2 that neurotransmitters and their receptors interact, somewhat as locks and keys do. Each neurotransmitter fits a particular type of receptor on the nerve cell membrane. If there is the wrong number of receptors for a given type of neurotransmitter or if the receptors for that neurotransmitter are too sensitive or not sensitive enough, then the neurons do not efficiently use the neurotransmitter that is available in the synapse.

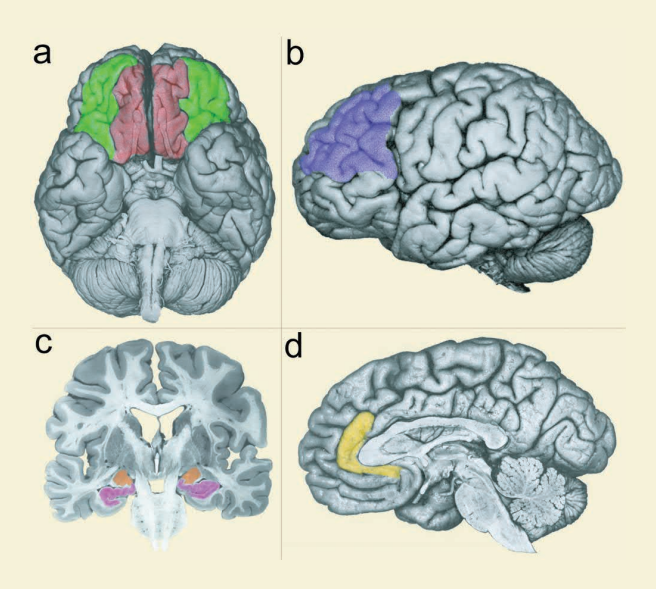
Several studies suggest that people with major depression or bipolar disorder may have abnormalities in the number and sensitivity of receptors for the monoamine neurotransmitters (Hasler et al., 2004; Southwick et al., 2005). In major depression, receptors for serotonin and norepinephrine appear to be too few or insensitive. In bipolar disorder, the picture is less clear, but it is likely that receptors for the monoamines undergo poorly timed changes in sensitivity, which are correlated with mood changes (Kujawa & Nemeroff, 2000).

Most of the neurotransmitter abnormalities found in people with mood disorders are state-dependent. That is, these differences are present when the mood disorder is present but tend to disappear when the mood disorder subsides. Certain neurotransmitter abnormalities may be correlated with, but may not necessarily cause, the mood disorders. As the technology for determining the functioning of neurotransmitter systems develops, our understanding of the relationship between neurotransmitters and mood disorders will no doubt increase.

Brain Abnormalities

Neuroimaging studies using computerized tomography (CT) scans, positron-emission tomography

FIGURE 9.4 **Brain Areas That May Be Involved in Mood Disorders.** *(a)* Orbital prefrontal cortex *(green)* and ventromedial prefrontal cortex *(red)*. *(b)* Dorsolateral prefrontal cortex *(purple)*. *(c)* Hippocampus *(pink)* and amygdala *(orange)*. *(d)* Anterior cingulated cortex *(yellow)*.



(PET), and magnetic resonance imaging (MRI) have found consistent abnormalities in at least four areas of the brain in people with mood disorders. These areas are the prefrontal cortex, the hippocampus, the anterior cingulate cortex, and the amygdala (Davidson et al., 2002; Southwick et al., 2005) (see Figure 9.4).

Both reductions in metabolic activity and a reduction in the volume of gray matter in the *prefrontal cortex*, particularly on the left side, have been found in people with serious depression or bipolar disorder (review Figure 9.4a, green and red areas, and b, purple areas) (Buchsbaum et al., 1997; Drevets, 2001; Drevets et al., 1997). Davidson, Pizzagalli, Nitschke, and Putnam (2002) have suggested that the left prefrontal cortex is more involved in approach-related goals and that inactivity in this region is associated with the lack of motivation and goal orientation in depression. The successful treatment of depression with antidepressant medications is associated with increases in metabolic activity in the left prefrontal cortex (Kennedy et al., 2001).

The *anterior cingulate* plays an important role in the body's response to stress, in emotional expression, and in social behavior, as well as in the processing of difficult information (Davidson et al., 2002) (review Figure 9.4d, yellow area). People with depression show decreased activity in the anterior cingulate relative to controls (Buchsbaum et al., 1997; Drevets et al., 1997). This lack of activity may be associated with problems in attention, in the planning of appropriate responses, and in coping, as well as with anhedonia found in depression. Again, activity increases in this region of the brain when people are successfully treated for their depression (Mayberg et al., 1997; Pizzagalli et al., 2001).

The *hippocampus* is critical in memory and in fear-related learning (review Figure 9.4c, purple area). MRI studies show a smaller volume in the hippocampus of people with major depression or bipolar disorder (Bremner et al., 2000; Noga, Vladar, & Torrey, 2001). Similarly, PET studies show lower metabolic activity in the hippocampus in people with major depression (Saxena, Brody, et al., 2001). Damage to the hippocampus could be the result of chronic arousal of the body's stress response. People with depression show chronically high levels of the hormone cortisol, particularly in response to stress, indicating that their bodies overreact to stress and do not return to normal levels of cortisol as quickly as the bodies of healthy people do. The hippocampus contains many receptors for cortisol, and chronically elevated levels of cortisol may inhibit the development of new neurons in the hippocampus (see Pariante & Miller, 2001; Sapolsky, Krey, & McEwen, 1986).

Abnormalities in the structure and functioning of the amygdala are found in several disorders involving mood (Davidson et al., 2002) (review Figure 9.4c, orange area). The amygdala helps direct attention to stimuli that are emotionally salient and have major significance for the individual. Studies of people with mood disorders show an enlargement of the amygdala (Altshuler et al., 1998; Mervaala et al., 2000) and increased activity in this part of the brain (Drevets, 2001). Activity in the amygdala has been observed to decrease to normal values in people successfully treated for depression (Drevets, 2001). The effects of overactivity in the amygdala are not yet entirely clear, but Drevets (2001) and Davidson and colleagues (2002) suggest that it may bias people toward aversive or emotionally arousing information and lead to rumination over negative memories and negative aspects of the environment.

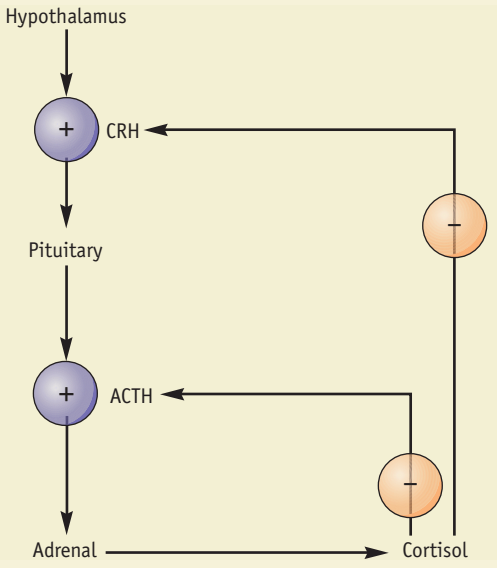
It is not known whether any of these abnormalities in the structure or functioning of the brain are causes of the mood disorders or the consequences of these disorders (Davidson, Pizzagalli, & Nitschke, 2002; Thase et al., 2002). Animal studies suggest that many of these brain abnormalities

can be caused by conditions in the environment, including chronic stress and chronic lack of control (Leverenz et al., 1999). Thus, for some people with mood disorders, the initial cause of their disorder may have been environmental, but the disorder may cause changes in the brain that increase their vulnerability to future episodes. For other people with mood disorders, brain dysfunction may be caused by abnormal genes.

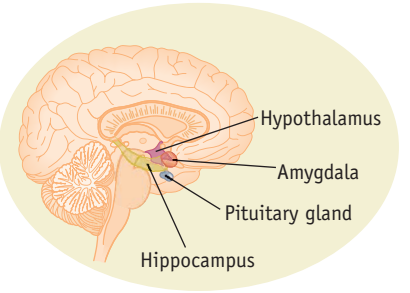
Neuroendocrine Factors

Hormones have long been thought to play a role in mood disorders, especially depression. The *neuroendocrine system* regulates a number of important hormones, which in turn affect basic functions, such as sleep, appetite, sexual drive, and the ability to experience pleasure (to review the neuroendocrine system, see Chapter 2). These hormones also help the body respond to environmental stressors.

FIGURE 9.5 **The Hypothalamic-Pituitary-Adrenal Axis.** The hypothalamus synthesizes corticotropin-releasing hormone (CRH). CRH is transported to the pituitary gland, where it stimulates the synthesis and release of adrenocorticotropic hormone (ACTH), which then circulates to the adrenal glands, producing cortisol. Cortisol then inhibits the production of further ACTH and CRH. Normally, this process prevents too much or too prolonged physiological arousal following a stressor. In major depression, however, people often show abnormal cortisol functioning, suggesting that there is dysregulation in the hypothalamic-pituitary-adrenal (HPA) axis.



Three key components of the neuroendocrine system—the hypothalamus, pituitary, and adrenal cortex—work together in a biological feedback system that is richly interconnected with the limbic system and the cerebral cortex. This system is often referred to as the **hypothalamic-pituitary-adrenal axis,** or **HPA axis**, and is involved in the fight-or-flight response, as discussed in Chapter 6.



The hypothalamic-pituitary-adrenal (HPA) axis and structures of the limbic system may be involved in the development of depression.

Normally, when we are confronted with a stressor, the HPA axis becomes more active (see Figure 9.5). It increases the body's levels of major stress hormones, such as **cortisol**, which help the body respond to the stressor by making it possible to fight the stressor or flee from it. Once the stressor is gone, the HPA axis activity returns to its baseline levels. Thus, this biological feedback loop both helps activate the HPA system during stress and calms the system when the stress is over.

People with depression tend to show chronic hyperactivity in the HPA axis and an inability for the HPA axis to return to normal functioning following a stressor (Southwick et al., 2005; Young & Korzun, 1998). In turn, the excess hormones produced by heightened HPA activity seem to have an inhibiting effect on receptors for the monoamines. One model for the development of depression is that people exposed to chronic stress may develop poorly regulated neuroendocrine systems. Then, when they are exposed even to minor stressors later in life, the HPA axis overreacts and does not easily return to baseline. This overreaction creates change in the functioning of the monoamine neurotransmitters in the brain, and an episode of depression is likely to ensue (Southwick et al., 2005).

Women's Hormonal Cycles as a Factor

Many people have argued over the years that women's greater vulnerability to depression is tied to hormones—specifically, the so-called ovarian hormones, estrogen and progesterone. The main fuel for this idea comes from evidence that women are more prone to depression during the premenstrual period of the menstrual cycle, the postpartum period, and menopause. These are times when estrogen and progesterone levels change dramatically.

Research over the past several decades has shown that most women do not experience significant changes in their moods during times of hormonal change (Nolen-Hoeksema, 2002; Young &

Korszun, 1998). However, there is a small group of women, about 3 percent of the population, who frequently experience increases in depressive symptoms during the premenstrual phase. Many of these women also have a history of frequent major depressive episodes or anxiety disorders with no connection to the menstrual cycle or of other psychiatric disorders (Steiner et al., 2003). This history suggests that these women have a general vulnerability to depression or anxiety, rather than a specific vulnerability to premenstrual depression.

This information has led many researchers to argue that depressions during the premenstrual period should not be given a separate diagnosis, such as **premenstrual dysphoric disorder,** but, rather, should be considered only exacerbations of major depression or dysthymia. Others argue that premenstrual depression should be recognized separately, with its own diagnosis, because it is different from depression that has no link with the menstrual cycle and therefore should be studied separately. The authors of the DSM-IV-TR dealt with this controversy by putting diagnostic criteria for premenstrual dysphoric disorder in an appendix, rather than in the main body of its text with other officially recognized diagnoses.

Even among women who clearly do have premenstrual symptoms (PMS), there is little evidence that their symptoms are due to changes in estrogen or progesterone levels across the menstrual cycle (Steiner et al., 2003; Young & Korzun, 1998). Many studies have found no differences in estrogen or progesterone levels between women with PMS and those without PMS. There clearly is something about the menstrual cycle that is worsening mood in women with PMS, but it appears that estrogen or progesterone does not have consistent direct effects on mood.

About 1 in 10 women experience a severe postpartum depression in the first few months after giving birth. This might seem like strong evidence that hormonal changes play a role in women's depressions, because this is a period of great hormonal change in women's bodies. However, studies comparing rates of depression in women who are and are not postpartum have tended not to find differences in rates of depression (O'Hara & Swain, 1996). Even among women who do become seriously depressed during the postpartum period, depressions do not seem to be linked to any specific imbalances in hormones (Hendrick, Altshuler, & Suri, 1998).

Postpartum depressions are often linked to severe stress in women's lives, such as financial strain, marital difficulties, lack of social support, and fussy babies (Brugha et al., 1998; Hendrick et al., 1998; O'Hara & Swain, 1996). In addition, women who have a history of depression clearly are at increased risk for postpartum depression (Steiner et al., 2003). These women may carry a general vulnerability to depression, which is triggered by either the physiological or the environmental changes of the postpartum period.

About 1 in 10 women suffer postpartum depression.

Menopause marks the cessation of menstrual periods, and circulating ovarian hormones decrease dramatically at menopause (Young & Korzun, 1998). Twenty years ago, the belief that women were more prone to depression during menopause was so strong among clinicians that there was a separate diagnostic category in the DSM for this type of depression. Several studies have found, however, that women are no more likely to show depression around the time of menopause than at any other time in their lives (Matthews et al., 1990; Nicol-Smith, 1996). In addition, there are no consistent mood effects of taking estrogen replacement drugs for menopausal women (Young & Korzun, 1998).

In sum, the evidence that women's moods are tied to their hormones is mixed, at best. Some women clearly do experience more depression during the postpartum period, menopause, and other times when their hormone levels change rapidly.

The extent to which these experiences of depression account for the generally higher rates of depression among women compared with men is less clear.

Early Stress as a Cause of Neurobiological Vulnerability to Depression

There is increasing evidence that early traumatic stress, such as being the victim of incest, severe neglect, or other serious, chronic stress, can lead to some of the neurobiological abnormalities that may predispose people to depression (Southwick et al., 2005). Studies of children who have been abused or neglected show that their biological responses to stress, particularly the response of their HPA axis, often are either exaggerated or blunted (Cicchetti & Toth, 2005). Heim and colleagues (Heim & Nemeroff, 2002; Heim, Plotsky, & Nemeroff, 2004) have found that women who were sexually abused as children show altered HPA responses to stress as adults, even when they are not depressed. Similarly, animal studies show that early stress (such as separation from their mothers) promotes exaggerated neurobiological stress reactivity and vulnerability to depression-like responses to future stressors (see Southwick et al., 2005). These neurobiological vulnerabilities can be reduced in animals by providing them with subsequent supportive maternal care and/or pharmacological interventions.

SUMMING UP

- Genetic factors clearly play a role in bipolar disorder, although it is somewhat less clear what role genetics plays in many forms of unipolar depression.
- The neurotransmitter theories suggest that imbalances in levels of norepinephrine or serotonin or the dysregulation of receptors for these neurotransmitters contribute to depression, and dysregulation of norepinephrine, serotonin, or dopamine is involved in bipolar disorder.
- Neuroimaging studies have shown abnormalities in the structure and functioning of the prefrontal cortex, hippocampus, anterior cingulate cortex, and amygdala in people with mood disorders.
- People with depression have chronic hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis, which helps regulate the body's response to stress.
- Abnormalities in the biological stress response may result from early stressors in some people and contribute to depression.

PSYCHOLOGICAL THEORIES OF MOOD DISORDERS

Psychological theories have focused almost exclusively on depression, because the evidence that bipolar disorder is caused by biological factors is strong. However, new episodes of bipolar disorder may be triggered by experiencing stressful events or living in an unsupportive family (Frank, Schwartz, & Kupfer, 2000; Hlastsala et al., 2000). This pattern suggests a diathesis-stress model of bipolar disorder, in which the *diathesis*, or vulnerability, is a biological one, such as a genetic predisposition to the disorder, and stressors, such as the loss of a job, can trigger new episodes. In this section, however, we focus on depression and the psychological theories that have tried to explain it (see the Concept Overview in Table 9.6 on page 320).

Behavioral Theories

Depression often arises as a reaction to stressful negative events, such as the breakup of a relationship, the death of a loved one, a job loss, or a serious medical illness (Hammen, 2005). Sixty-five percent of people with depression in one study reported a negative life event in the six months prior to the onset of their depression (Frank et al., 1994). People with depression are more likely than nondepressed people to have chronic life stressors, such as financial strain or a bad marriage. People who suffer depression also tend to have a history of traumatic life events, particularly events involving loss (Hammen, 2005).

The Reduction of Positive Reinforcers

Peter Lewinsohn's **behavioral theory of depression** suggests that life stress leads to depression because it reduces the positive reinforcers in a person's life (Lewinsohn & Gotlib, 1995). The person begins to withdraw, which only results in a further reduction in reinforcers, which leads to more withdrawal, and a self-perpetuating chain is created.

For example, imagine that a man is having difficulty in his relationship with his wife. Interactions with her are no longer as positively reinforcing as they formerly were, so he stops initiating these interactions as often. This only worsens the communication between him and his wife, so the relationship becomes even worse. He withdraws further and becomes depressed about this area of his life. Lewinsohn suggests that such a pattern is especially likely in people with poor social skills, because they are more likely to experience rejection by others and to withdraw in response to this rejection, rather than to find ways to overcome the rejection (Lewinsohn, 1974). In addition, once a person begins engaging in

TABLE 9.6 Concept Overview

Psychological Theories of Mood Disorders

The psychological theories of depression have focused on aspects of the environment, of thinking, and of a person's past.

Theory	Description
Behavioral theories	
Lewinsohn's theory	Depressed people experience a reduction in positive reinforcers and an increase in aversive events, which leads to their depression.
Learned helplessness theory	Depressed people lack control, which leads to the belief that they are helpless, which leads to depressive symptoms.
Cognitive theories	
Aaron Beck's theory	Depressed people have a negative cognitive triad of beliefs about the self, the world, and the future, which is maintained by distorted thinking.
Reformulated learned helplessness theory	Depressed people have the tendency to attribute events to internal, stable, and global factors, which contributes to depression.
Ruminative response styles theory	Depressed people tend to ruminate about their symptoms and problems.
Psychodynamic theory	Depressed people are unconsciously punishing themselves because they feel abandoned by another person but cannot punish that person; dependency and perfectionism are risk factors for depression.
Interpersonal theories	Depressed people have poor relationships with others.

depressive behaviors, these behaviors are reinforced by the sympathy and attention they engender in others.

Learned Helplessness Theory

Another behavioral theory—the **learned helplessness theory**—suggests that the type of stressful event most likely to lead to depression is uncontrollable negative events (Seligman, 1975). Such events, especially if frequent or chronic, can lead people to believe that they are helpless to control important outcomes in their environment. In turn, this belief in helplessness leads people to lose their motivation, to reduce actions that might control the environment, and to be unable to learn how to control situations that are controllable. These deficits, known as **learned helplessness deficits,** are similar to the symptoms of depression: low motivation, passivity, and indecisiveness (Seligman, 1975).

The initial evidence for the learned helplessness theory came from studies with animals, as described in Chapter 2. A group of researchers conducted a series of studies in which dogs were given controllable shock, uncontrollable shock, or no shock (Overmier & Seligman, 1967; Seligman & Maier, 1967). The dogs in the controllable shock group could turn off the shock by jumping a short barrier, and they quickly learned how to do so (as did the dogs that had previously received no shock). The dogs in the uncontrollable shock group could not turn off or otherwise escape the shock. The dogs in the controllable and uncontrollable shock conditions received the same total amount of shock. However, when the dogs in the uncontrollable shock group were put into a situation in which they could control the shock, they seemed unable to learn how to do so. They just sat in the box, passive and whimpering, until the shock went off. Even when the experimenter dragged these dogs across the barrier in an attempt to teach them how to turn off the shock, the dogs did not learn the response. The researchers argued that the dogs in the uncontrollable shock group had learned they were helpless to control the shock, and their passivity and inability to learn to control the shock were the result of this learned helplessness.

In turn, the researchers argued that many human depressions are *helplessness depressions*, result-

Children who lose a parent may come to believe that important areas of their lives are not under their control and, thus, develop a helplessness depression.

ing when people come to believe they are helpless to control important outcomes in their environment. For example, children who lose their mothers may come to believe that important areas of their lives are not under their control. The loss of a mother may mean not only the loss of the person to whom the child is most closely attached but also years of disruption and instability as the child is moved from one set of relatives to another, if the father is not able to care for the child. Such chronic instability might persuade the child that life truly is uncontrollable, and this may be why childhood bereavement is a predisposing factor for depression. Similarly, women who are frequently battered by their husbands may develop the belief that there is nothing they can do to control their beatings or other parts of their lives, and this may explain the high rates of depression among battered women (Koss & Kilpatrick, 2001).

Cognitive Theories

> "Good morning, Eeyore," shouted Piglet.
> "Good morning, Little Piglet," said Eeyore.
> "If it *is* a good morning," he said.
> "Which I doubt," said he.
> "Not that it matters," he said.
> (Milne, 1961, p. 54)

Like poor Eeyore, some people have a chronically gloomy way of interpreting the things that happen to them. According to the cognitive theories of depression, these gloomy ways of thinking are a cause of depression.

Aaron Beck's Theory

One of the first cognitive theories of depression was developed by psychiatrist Aaron Beck. Beck (1967) argued that people with depression look at the world through a **negative cognitive triad:** They have negative views of themselves, of the world, and of the future. People with depression then commit many types of errors in thinking—such as jumping to negative conclusions on the basis of little evidence, ignoring good events, focusing only on negative events, and exaggerating negative events—that support their negative cognitive triad (see Table 9.7 on page 322).

People with depression may not be aware that they hold these negative views or that they make these errors in thinking. Often, these negative thoughts are so automatic that people with depression do not realize how they are interpreting situations. A wide range of studies have supported the hypothesis that people with depression show these negative ways of thinking, and some longitudinal studies have shown that these thinking styles predict depression over time (Abramson et al., 2002). Beck's theory led to one of the most widely used and successful therapies for depression, cognitive-behavioral therapy.

Reformulated Learned Helplessness Theory

Another influential cognitive theory of depression, the **reformulated learned helplessness theory,** was proposed to explain how cognitive factors might influence whether a person becomes helpless and depressed following a negative event (Abramson, et al., 1978; Peterson & Seligman, 1984). This theory focuses on people's causal attributions for events. A **causal attribution** is an explanation of why an event happened. According to this theory, people who habitually explain negative events by causes that are internal, stable, and global blame themselves for these negative events, expect negative events to recur in the future, and expect to experience negative events in many areas of their lives. In turn, these expectations lead them to experience long-term learned helplessness deficits plus self-esteem loss in many areas of their lives.

TABLE 9.7 Errors or Distortions in Thinking in Depression

Error	Description
All-or-nothing thinking	You see things in black-and-white categories. If your performance falls short of perfect, you see yourself as a total failure.
Overgeneralization	You see a single negative event as a never-ending pattern of defeat.
Mental filter	You pick out a single negative detail and dwell on it exclusively, so that your vision of all reality becomes darkened, like a drop of ink that discolors an entire beaker of water.
Disqualifying the positive	You reject positive experiences by insisting they "don't count" for some reason. In this way, you can maintain a negative belief that is contradicted by your everyday experiences.
Jumping to conclusions	You make a negative interpretation, even though there are no definite facts that convincingly support your conclusion: (a) *Mind Reading*. You arbitrarily conclude that someone is reacting negatively to you, and you don't bother to check this out. (b) *The Fortune Teller Error*. You anticipate that things will turn out badly, and you feel convinced that your prediction is an already established fact.
Magnification (catastrophizing) or minimization	You exaggerate the importance of things (such as your goof-up or someone else's achievement), or you inappropriately shrink things until they appear tiny (your own desirable qualities or another's imperfections). This is also called the "binocular trick."
Emotional reasoning	You assume that your negative emotions necessarily reflect the way things really are: "I feel it; therefore, it must be true."
"Should" statements	You try to motivate yourself with "shoulds" and "shouldn'ts", as if you had to be whipped and punished before you could be expected to do anything. "Must" and "oughts" are also offenders. The emotional consequence is guilt. When you direct "should" statements toward others, you feel anger, frustration, and resentment.
Labeling and mislabeling	This is an extreme form of overgeneralization. Instead of describing your error, you attach a negative label to yourself: "I'm a *loser*." When someone else's behavior rubs you the wrong way, you attach a negative label to that person. Mislabeling involves describing an event with language that is highly colored and emotionally loaded.
Personalization	You see yourself as the cause of a negative external event, which, in fact, you were not primarily responsible for.

Source: Burns, 1980.

For example, consider a student who becomes depressed after failing a psychology exam. The reformulated learned helplessness theory would suggest that she has blamed her failure on internal causes—she didn't study hard enough—rather than external causes—the exam was too hard. Further, she has assumed that the failure was due to stable causes, such as a lack of aptitude in psychology, rather than unstable causes, such as the instructor's not allowing enough time, and she can expect to fail again. Finally, she has attributed her failure to a global cause, such as her difficulty in learning the material for this particular test. This global attribution would lead to failure in other academic areas.

Again, researchers equate learned helplessness deficits with depression and argue that an internal-stable-global attributional style for negative events puts people at risk for depression. Abramson et al. (1989) argued that hopelessness depression develops when people make pessimistic attributions for the most important events in their lives and perceive that they have no way of coping with the consequences of these events. The reformulated learned helplessness theory and the hopelessness theory have motivated a great deal of research (Abramson et al., 2002).

One of the most definitive studies of this theory of depression was a long-term study of college

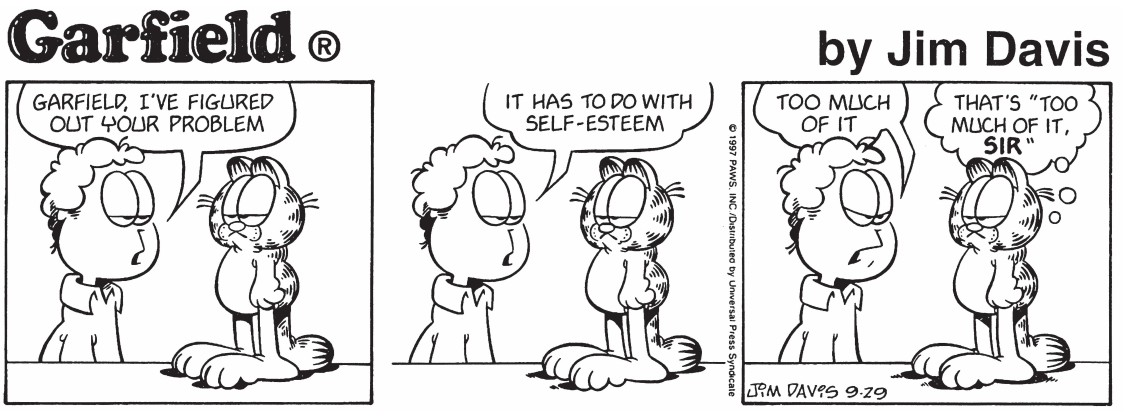
students (Alloy, Abramson, & Francis, 1999). Researchers interviewed first-year students at two universities and identified those with hopeless attributional styles and those with optimistic attributional styles. They then tracked these students for the next 2½ years, interviewing them every 6 weeks. Among the students with no history of depression, those with a hopeless cognitive style were much more likely to develop a first onset of major depression than were those with an optimistic attributional style (17 percent versus 1 percent). In addition, among those who had a history of depression, students with a hopeless style were more likely to have a relapse of depression than those with an optimistic style (27 percent versus 6 percent). Thus, a pessimistic attributional style predicted both first onsets of depression and relapses of depression.

Is it possible that people with depression are not distorted in their negative views of the world but actually are seeing the world realistically for the terrible place that it is? Researchers began investigating this possibility when they stumbled on a phenomenon now referred to as **depressive realism:** When asked to make judgments about how much control they have over situations that are actually uncontrollable, people with depression are quite accurate. In contrast, nondepressed people greatly overestimate the amount of control they have, especially over positive events. For example, in one study (Alloy & Abramson, 1979), depressed and nondepressed people were asked to judge to what degree they could control the onset of a green light by pushing a button on a display panel. In truth, none of the subjects had control over the onset of the light. In conditions in which the subjects were rewarded whenever the green light came on, the nondepressed people grossly overestimated their control over the onset of the light. In contrast, the depressed subjects accurately judged that they had no control over the onset of the light.

Subsequently, a long line of research has shown that nondepressed people have a robust illusion that they can control all sorts of situations that truly are out of their control and that they have superior skills, compared with most people (Taylor & Brown, 1988). For example, nondepressed people believe they can control games of chance, such as the lottery; that they are more likely than the average person to succeed in life; that they are more immune to car accidents than other people; and that their social skills are better than most people's. In contrast, people with depression do not seem to hold these illusions of control and superiority. Indeed, people with depression seem amazingly accurate in judging the amount of control they have over situations and their skills at various tasks. This research on illusion of control calls into question the notion that depression results from unrealistic beliefs that one cannot control one's environment or from negative errors in thinking about oneself and the world. Perhaps it is not accurate, realistic thinking that prevents people from becoming depressed but, rather, hope and optimism.

Ruminative Response Styles Theory

Another cognitive theory, the **ruminative response styles theory,** focuses more on the process of thinking, rather than the content of thinking, as a contributor to depression (Nolen-Hoeksema, 2003). Some people, when sad and upset, focus intently on how they feel—their symptoms of fatigue and poor concentration and their sadness and hopelessness—and can identify many possible causes of these symptoms. They do not attempt to do anything about these causes, however, and continue to engage in **rumination** about their depression.



Several studies have shown that people with this more ruminative coping style are more likely to develop major depression and may remain depressed longer than people with a more action-oriented coping style (Nolen-Hoeksema, 2000; Nolen-Hoeksema, Larson, & Grayson, 1999; Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Parker, & Larson, 1994). Rumination is not just another symptom of depression, although people who are more depressed have more to ruminate about. People with depression differ in the extent to which they ruminate, and those who ruminate more become more severely depressed over time and remain depressed longer than those who do not.

Women are more likely than men to ruminate when they are depressed (Nolen-Hoeksema, 2002; Nolen-Hoeksema et al., 1999). This may be because women are exposed to more circumstances that make them ruminate—more negative events and circumstances over which they feel they have no control. Regardless of the reasons for this gender difference in rumination, women's tendency to ruminate appears to contribute to their higher rates of depression, compared with men (Nolen-Hoeksema et al., 1999).

Psychodynamic Theories

Some people seem to find themselves in unhealthy, destructive relationships over and over again. Each time these relationships end, they vow never to get into similar relationships again. However, they do and then find themselves depressed over the problems in the new relationships or when the relationships inevitably end.

Psychodynamic theorists suggest that such patterns of unhealthy relationships stem from people's childhood experiences that prevented them from developing a strong and positive sense of self reasonably independent of others' evaluations (Arieti & Bemporad, 1980; Bibring, 1953; Blatt & Zuroff, 1992; Freud, 1917). As adults, these people are constantly searching for approval and security in their relationships with others. They are anxious about separation and abandonment and may allow others to take advantage and even abuse them, rather than risk losing the relationship by complaining. They are constantly striving to be "perfect," so that they will be loved. Even when they accomplish great things, they do not feel secure or positive about themselves. Eventually, a problem in a close relationship or a failure to achieve perfection occurs, and they plunge into depression.

Many modern psychodynamic theorists still rely on the groundbreaking work Freud published in his paper *Mourning and Melancholia* to describe just how depression develops when a person perceives he or she has been abandoned or has failed. Freud pointed out that people who are depressed have many of the symptoms of people who are grieving the death of a loved one: They feel sad, alone, unmotivated, and lethargic. Unlike grieving people, people with depression display severe self-hate and self-blame. Indeed, said Freud, people with depression appear to want to punish themselves, even to the point of killing themselves.

Freud argued that people with depression are not actually blaming or punishing themselves. Instead, they are blaming or punishing those who they perceive have abandoned them. People with depression are so dependent on the approval and love of others that much of their ego or sense of self is made up of their images of these others—what Freud called the "love objects." When they believe others have rejected them, people with depression are too frightened to express their rage outwardly. Instead, they turn their anger inward on the parts of their own egos that have incorporated the love objects. Their self-blame and punishment is actually blame and punishment of the others who have abandoned them. This is Freud's **introjected hostility theory** of depression. The case of Giselle illustrates the processes described by the psychodynamic theories of depression.

CASE STUDY

Giselle was raised by two well-meaning but emotionally inhibited parents. The parents had emigrated to the United States from Eastern Europe in the 1970s, fleeing persecution for their anticommunist beliefs. Even after settling in the United States, Giselle's parents remained paranoid about the family's security and constantly told Giselle she had to be "good" or the family would be in danger. Thus, from an early age, Giselle suppressed any childhood willfulness or exuberance. She was not allowed to play with other children; she spent most of her time with the family maid, who had followed them to the United States. Her parents were preoccupied with their uncertain circumstances and unnecessarily belittled Giselle's childhood concerns. For example, when there was an epidemic of flu at Giselle's school, her mother told her not to worry, because only the smart and pretty girls were getting sick. The mother doted on the father when he was in the house, ignoring Giselle.

(*continued*)

The father paid attention to Giselle only when she was deferential or complimentary.

As an adult, Giselle chose to become a nurse, because she felt it would gain her acceptance and love by patients. Giselle married a man who was somewhat solitary and hypercritical. He was prone to periods of depression and always preoccupied with his own concerns. Giselle became the major source of financial support during her marriage, often taking on extra shifts to earn more money. She had done remarkably well in her career because of her hard work and her repeated efforts to please others. She was also the emotional mainstay in her family, being responsible for taking care of the children and for fulfilling the usual responsibilities of running a household. Giselle rarely complained, however. She needed to be certain that everyone liked her and thought well of her, and she went to extremes of self-sacrifice to ensure the high regard of others.

After several years, her husband left her, telling her that he did not love her any longer and that she no longer gave him any pleasure in his life. In the first few days after her husband announced he was going to leave, Giselle desperately tried to win back his love by indulging his every whim. Eventually, however, they had a violent confrontation, during which he walked out. Later that evening, Giselle emptied her medicine cabinet of all drugs, drove to a secluded area, and ingested the drugs in an effort to kill herself. (Adapted from Bemporad, 1995)

Some research has supported elements of the psychodynamic perspective on depression. For example, people with depression tend to display many of Giselle's personality traits: They are dependent on others, believe that they must be perfect, have poor self-esteem, and are unable to express anger openly (Klein et al., 2002). In addition, many people with depression describe their parents as having characteristics similar to those of Giselle's parents: They are cold and neglectful, excessively moralistic and demanding of perfection, or requiring of complete devotion and dependency from their children in exchange for their love (Blatt & Zuroff, 1992). Most of these studies are cross-sectional, however, so it is not known whether these characteristics and views are symptoms of the depression or actual causes of it. A few longitudinal studies support elements of psychodynamic theories. For example, one study of middle-aged women found that those who tended to inhibit any expression of anger and who were unassertive in interpersonal interactions were more likely to become depressed over a three-year period (Bromberger & Matthews, 1996).

Traditional psychodynamic perspectives on depression have been adapted by modern theorists to develop the interpersonal theories of depression and therapies based on these theories. We turn now to the interpersonal theories.

Interpersonal Theories

Like psychodynamic theories, **interpersonal theories of depression** are concerned with people's close relationships and their roles in those relationships (Klerman et al., 1984). Disturbances in these roles are thought to be the main source of depression. These disturbances may be recent, as when a woman who believes that her marriage has been successful for years suddenly finds that her husband is having an affair. Often, the disturbances are rooted in long-standing patterns of interactions the people with depression typically have with important others. Drawing from attachment theory (Bowlby, 1982), interpersonal theorists argue that children who do not experience their caregivers as reliable, responsive, and warm develop an insecure attachment to their caregivers, which sets the stage for all future relationships (see Chapter 2). These problematic relationships become represented mentally as negative working models of others and of the self in relation to others. These models are essentially operating rules and expectations about the availability of support from others and the implications of others' lack of support for one's self-worth.

Children with insecure attachments develop expectations that they must be or do certain things in order to win the approval of others, which have been called **contingencies of self-worth** (Kuiper & Olinger, 1986; Kuiper, Olinger, & MacDonald, 1988). These are "if-then" rules concerning self-worth, such as "I'm nothing if a person I care about doesn't love me." If these contingencies of self-worth sound like the dysfunctional beliefs that Beck and other cognitive theories describe, they are—the interpersonal theorists argue that dysfunctional beliefs are the result of insecure attachments in childhood. As long as an individual meets the contingencies of self-worth set up in his or her working model, then he or she will maintain positive self-esteem and remain nondepressed. Failures to meet

these contingencies are inevitable, however, and plunge the person into depression.

According to the interpersonal theories, people who are so insecure in their relationships with others engage in **excessive reassurance seeking**—constantly looking for assurances from others that they are accepted and loved (Joiner, 2002). They never quite believe the affirmations other people give, however, and anxiously keep going back for more. After a while, their family members and friends can become weary of this behavior and become frustrated or hostile. The insecure person picks up on these cues of annoyance and becomes panicked over them, leading him or her to feel even more insecure and to engage in even more excessive reassurance seeking. Eventually, the person's social support may withdraw altogether, leading him or her to develop even more depression, as is illustrated in the following case study (from Nolen-Hoeksema, 2006).

CASE STUDY

Rachel was a 48-year-old homemaker from the Bronx, married to her husband, Phil, for 15 years. Phil never really gave Rachel good cause to doubt that he loved her. He was attentive and loving. He was at her side when she had medical problems a few years ago and when her mother died last year. He supported her decision to stay home to raise their two children and was sincerely interested in what she and the kids did during the day while he was working at his law firm.

But still Rachel doubted, and these doubts had grown stronger in the last few months as another one of her depressive periods had set in. How could he love her, when she was so boring? Surely he was just being nice when he asked about her day—he couldn't really be interested, given how exciting his own work was. She had gained weight over the years and felt she was no longer attractive to him. She wondered what he would do if one of the young women lawyers in his firm expressed interest in him.

Rachel tried to keep these concerns to herself, but they leaked out, in little comments to Phil. When he came home in the evening and said, "How was your day?" she sometimes responded, "Oh, boring as usual; you wouldn't be interested." Then, she waited to see what his response was. Phil would usually say something like, "Sure, I'm interested; tell me what you did." Then Rachel would tell him a few incidents from the day but label each one as "nothing" and "silly" as she went along. She listened intently for his response, wanting him to deny that her activities were nothing or silly and becoming anxious and disappointed if he didn't explicitly do so.

When Rachel mentioned that she felt fat or unattractive, Phil would usually respond that she was still his beautiful bride. She responded, "Oh, you have to say that; you're stuck with me." Phil felt frustrated and put off by this but tried to stay calm. "I don't have to say that; I mean it. I love you and I love how you look." But Rachel would not be satisfied: "You love me now, but will you always love me no matter what happens?"

Rachel set up all sorts of other tests of Phil's love for her and ploys to gain assurance of his devotion. If they disagreed with each other about something in the morning, Rachel would ruminate about it after Phil left for work. In her mind, she implicitly believed that, if he hadn't called to talk with her about the disagreement by 10 A.M. this meant he was really angry and their relationship was in trouble. Sometimes, if he hadn't called or e-mailed her by 11 A.M. she'd call or e-mail him with a neutral message, just to see whether he'd mention the morning's disagreement. If he didn't respond almost immediately, Rachel took this as further evidence that he was angry with her, even though she knew he was probably just very busy. If he did respond to her message, but didn't mention the disagreement and how sorry he was for it, Rachel would ruminate about this for the rest of the afternoon. By the time Phil got home in the evening, she was ripe with fear and anger, when he hadn't thought about the disagreement all day because it had been so minor.

A number of influential theories have suggested that women are socialized to base most of their self-concept and self-worth on their relationships with others, and this is what makes them more prone than men to depression. Jack (1991) and Helgeson (1994) both argue that females are

Difficulties in close relationships are often tied to depression.

more likely than males to silence their own wants and needs in a relationship in favor of maintaining a positive emotional tone in the relationship and to feel too responsible for the quality of the relationship. This leads females to have less power and to obtain less benefit from relationships. Some support has been found for this perspective on the gender difference in depression (Baron & Peixoto, 1991; Nolen-Hoeksema & Jackson, 2001).

The interpersonal theories of depression have been supported in several studies (Joiner, 2002). For example, one longitudinal study of college students found that those with an anxious, insecure attachment style had more dysfunctional negative beliefs and subsequently developed lower self-esteem and more depressive symptoms (Roberts, Gotlib, & Kassel, 1996). Most of the research on the interpersonal models of depression has focused on evaluating the therapy that was developed based on this model.

SUMMING UP

- The behavioral theories of depression suggest that stress can induce depression by reducing the number of reinforcers available to people.
- The learned helplessness theory of depression says that uncontrollable events can lead people to believe that important outcomes are outside of their control and thus can lead them to develop depression.
- The cognitive theories of depression argue that people with depression think in distorted and negative ways, and this leads them to become depressed, particularly in the face of negative events. In addition, people who ruminate about their depressive symptoms and the causes and consequences of those symptoms appear more prone to develop severe levels of depression.
- Psychodynamic theories posit that people with depression are overly dependent on the evaluations and approval of others for their self-esteem, as a result of poor nurturing by parents.
- The interpersonal theories of depression suggest that poor attachment relationships early in life can lead children to develop expectations that they must be or do certain things in order to win the approval of others, which puts them at risk for depression. They may also engage in excessive reassurance seeking, which drives away their social support.

SOCIAL PERSPECTIVES ON MOOD DISORDERS

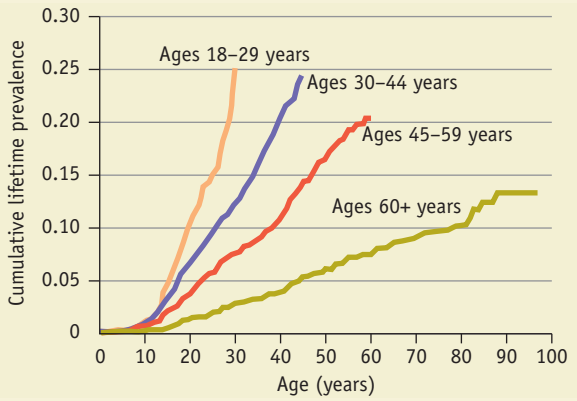
Sociologists have focused on the large age, gender, and cross-cultural differences in rates of depression, and they have tried to understand these disorders in light of those differences.

The Cohort Effect in Depression

Recall that the rates of depression appear to be lower in people over the age of 65 than in younger people and that there are several explanations for this age difference. One further explanation highlights sociocultural changes over history that may have resulted in more recent generations' being at higher risk for depression than people who were born a few generations ago (Klerman & Weissman, 1989). This type of explanation is called a **cohort effect:** People born in one historical period are at different risk for a disorder than are people born in another historical period. For example, fewer than 15 percent of people born before 1940 have experienced episodes of major depression at some time in their lives, whereas over 25 percent of people born after 1970 have already experienced major depression by the age of 30 (see Figure 9.6 on page 328). Proponents of the cohort explanation suggest that more recent generations are more at risk for depression because of the rapid changes in social values that began in the 1960s and the disintegration of the family unit (Klerman & Weissman, 1989).

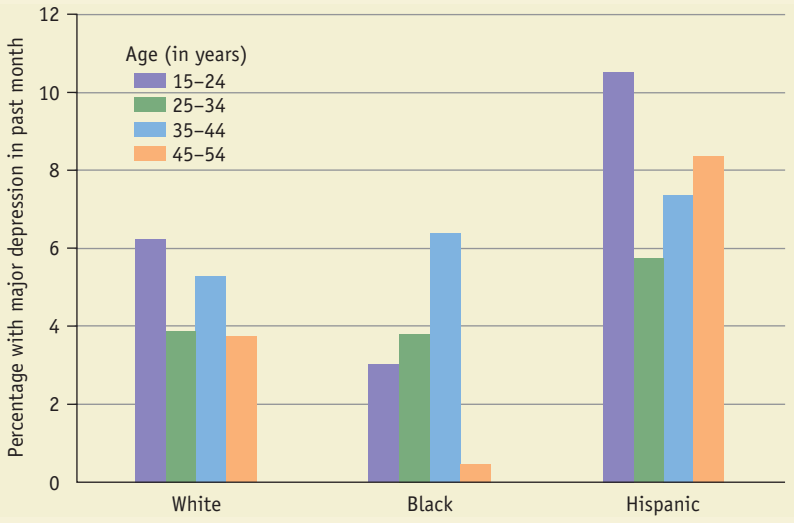
This decrease in social support and in identification with common social values may have put younger generations at higher risk for depression than older generations were. Another possible explanation is that younger generations have higher expectations for themselves than did older generations, but these expectations are too high to be met.

FIGURE 9.6 **Cumulative Lifetime Prevalence of Major Depressive Disorder by Birth Cohort.** More recent generations appear more prone to major depression.



Source: Kessler et al., 2003.

FIGURE 9.7 **Ethnic Differences in Major Depression.** These are the percentages of people in each age group and by ethnicity diagnosed with major depression in the previous month in one study. Hispanic Americans showed the highest rates across all age groups.



Source: Blazer et al., 1994.

Social Status

People who have lower status in society generally tend to show more depression. For example, in one large study done in the United States, people of Hispanic origin had a higher prevalence of major depression in the previous year than European Americans (Blazer et al., 1994) (see Figure 9.7). This may reflect the higher rate of poverty, unemployment, and discrimination that Hispanics suffer, compared with European Americans.

Figure 9.7 also suggests, however, that African Americans of most ages have even lower rates of major depression than European Americans. This may seem puzzling, given the disadvantaged status of African Americans in U.S. society. However, African Americans have high rates of anxiety disorders, suggesting that the stress of their social status may make them especially prone to anxiety disorders rather than depression. Other studies have found extremely high rates of depression among Native Americans, especially the young (Saluja et al., 2004). Depression among these Native American youth is tied to poverty, hopelessness, and alcoholism.

One of the most compelling social explanations for women's higher rates of depression is that women's lower social status puts them at high risk for physical and sexual abuse, and these experiences often lead to depression. Women are much more likely than men to be the victims of rape, incest, battering, and sexual harassment (Koss & Kilpatrick, 2001). The rates of these types of violence against women are staggering. Most studies of rape estimate that between 14 and 25 percent of women are raped in their lives, most often before the age of 30 (Koss, 1993). One in eight women reports that she has been physically assaulted by her husband in the past year, and 1.8 million women report having been severely assaulted (punched, kicked, choked, or threatened with a gun or knife) (Straus & Gelles, 1990). Survivors of physical and sexual assault show high rates of major depression, anxiety disorders, and substance abuse. Thus, it seems likely that at least some of the difference between women's and men's rates of depression may be tied to the higher rates of abuse of women than of men and the resulting depression in female abuse survivors (Nolen-Hoeksema, 2002).

Cross-Cultural Differences

One cultural group within the United States that has an especially low prevalence of unipolar depression is the Old Order Amish of central Pennsylvania. As noted in Chapter 3, the Amish are a religious community of people who maintain a very simple lifestyle oriented around farming and the church and who reject modern conveniences, such as automobiles, electricity, and telephones. Essentially, the Amish live as people did in nineteenth-century rural America. Extensive research on the mood disorders among the Amish has suggested that their prevalence of major depression is only one-tenth of that of mainstream groups in the

United States (Egeland et al., 1987). Perhaps the simple, agrarian lifestyle of the Amish, with its emphasis on family and community, helps protect its members against depression.

Similarly, cross-national studies have suggested that the prevalence of major depression is lower among less industrialized and less modern countries than among more industrialized and more modern countries (Cross-National Collaborative Group, 1992; Lepine, 2001). Again, it may be that the fast-paced lifestyles of people in modern, industrialized societies, with their lack of stable social support and community values, are toxic to mental health. In contrast, the community- and family-oriented lifestyles of less modern societies may be beneficial to mental health, despite the physical hardships that many people in these societies face because of their lack of modern conveniences.

Alternately, some researchers have suggested that people in less modern cultures may tend to manifest depression with physical complaints, rather than psychological symptoms of depression, such as sadness, loss of motivation, and hopelessness about the future (Tsai & Chentsova-Dutton, 2002). For example, a study of refugees in Somalia found that they had a concept similar to the concept of sadness, which they called *murug* (Carroll, 2004). *Murug* arises when an individual has lost a loved one or another major negative life event has occurred. The symptoms of *murug*, however, are headaches and social withdrawal.

Similarly, in China, people facing severe stress often complain of *neurasthenia*, a collection of physical symptoms such as chronic headaches, pain in the joints, nausea, lack of energy, and palpitations, as illustrated in the following case study (adapted from Kleinman & Kleinman, 1985, pp. 454–455).

CASE STUDY

Lin Hung is a 24-year-old worker in a machine factory in China who complains of headaches, dizziness, weakness, lack of energy, insomnia, bad dreams, poor memory, and a stiff neck. Pain, weakness, and dizziness, along with bouts of palpitations are his chief symptoms. His symptoms began 6 months ago, and they are gradually worsening. His factory doctors believe he has a heart problem, but repeated electrocardiograms have been normal. He believes he has a serious bodily disorder that is worsened by his work and that interferes with his ability to carry out his job responsibilities. Until his father retired from the job Lin now occupies, he was a soldier living not far from home. He didn't want to leave the army, but his father was anxious to retire so he could move to a new apartment owned by his factory in another city. Fearing that his son would not be able to stay in the army and thereafter would not find work, Lin's father pressured him to take over his job, a job the younger Lin never liked or wanted for himself. Lin Hung reluctantly agreed but now finds he cannot adjust to the work. He did not want to be a machinist and cries when he recounts that this is what he must be for the rest of his life. Moreover, he is despondent and lonely living so far away from his parents. He has no friends at work and feels lonely living in the dormitory. He has a girlfriend, but he cannot see her regularly anymore, owing to the change in work sites. They wish to marry, but his parents, who have a serious financial problem because of a very low pension, cannot provide the expected furniture, room, or any financial help. The leaders of his work unit are against the marriage because he is too young. They also criticize him for his poor work performance and frequent days missed from work owing to sickness.

On questioning Lin Hung, psychiatrists trained in Western medicine diagnosed major depression. Like many Chinese, Lin rejected the psychological diagnosis, believing firmly that he was suffering solely from a physical disorder. A psychological diagnosis would not have garnered any sympathy from Lin's coworkers or family, but a physical diagnosis could provide him with an acceptable reason to leave his job and return to his family.

Indeed, the very concept of depression may be unique to Western cultures (Tsai & Chentsova-Dutton, 2002). Symptoms such as decreases in self-esteem and lack of interest in pleasurable activities are only abnormal in cultures that expect people to have high self-esteem and to seek out positive emotions. These are expectations in Western culture, but not in many other cultures of the world.

SUMMING UP

- More recent generations appear to be at higher risk for depression than earlier generations, perhaps because of historical changes in values and social structures related to depression.

- People of lower social status tend to have higher rates of depression. Women's greater vulnerability to depression may be tied to their lower social status and the risks of abuse that accompany this social status.
- Less industrialized cultures may have lower rates of depression than more industrialized cultures. Some studies suggest that the manifestation of depression and mania may be different across cultures.

MOOD DISORDERS TREATMENTS

The mood disorders have a tremendous impact on individuals and on society. In the United States alone, between $3 billion and $6 billion per year is spent on the treatment of depression, and over $40 billion per year goes to cover losses in productivity plus the health care costs of people with mood disorders (Rost et al., 1998). By the year 2020, depression is expected to be the fourth leading cause of disability in the world (Murray & Lopez, 1996).

In any given year, about 60 percent of people suffering from bipolar disorder and about half of people suffering from major depression will seek out treatment for their disorder (Regier et al., 1993; Rost et al., 1998). The rest will suffer through their symptoms without any care. People who do seek treatment tend to be more severely impaired by their symptoms than those who do not seek treatment (Angst, 1998). Most often, the people who eventually seek treatment wait a number of years after their symptoms begin to obtain any help (Kessler et al., 1998).

Fortunately, many forms of treatment are now available for mood disorders, particularly depression. Most of these types of treatment have been shown to work for the majority of people. Thus, although there are many pathways into a mood disorder, there now are many pathways by which people can overcome or control mood disorders as well.

Biological Treatments for Mood Disorders

Most of the biological treatments for depression and mania are drug treatments (see the Concept Overview in Table 9.8). Several classes of antidepressant drugs are used to treat depression. In addition to being treated with drugs, some people with depression are treated with electroconvulsive therapy (ECT). Two new treatments for mood disorders, repetitive transcranial magnetic stimulation (rTMS) and vagus nerve stimulation, hold hope for many people. People with seasonal affective disorder (SAD) seem to benefit from a unique type of therapy: exposure to bright lights. Lithium is the treatment of choice for bipolar disorder, but anti-

TABLE 9.8 Concept Overview

Biological Treatments for Mood Disorders

Type of Treatment	Description and Mode of Action
Medication: antidepressants (tricyclics, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors), lithium, anticonvulsants, calcium channel blockers, antipsychotics	Alter the levels of neurotransmitters or sensitivity of receptors for them
Electroconvulsive therapy	Apply electrical current to the brain; may increase permeability of the blood-brain barrier, cause release of neurotransmitters, stimulate the hypothalamus, increase sensitivity of receptors
Repetitive transcranial magnetic stimulation	Expose patients to repeated, high-intensity magnetic pulses focused on particular brain structures; may change the functioning of neurotransmitters
Vagus nerve stimulation	Stimulate by a small electronic device much like a cardiac pacemaker, which is surgically implanted under a patient's skin in the left chest wall; may increase activity in the hypothalamus and amygdala
Light therapy	Expose the individual to bright light; may "reset" circadian rhythms

convulsants, antipsychotics, and calcium channel blockers are also used.

Drug Treatments for Depression

Effective drug treatments for depression have been around since the 1960s. The late twentieth century, however, saw a rapid growth in the number of drugs available for depression and in the use of these drugs by large numbers of people.

Tricyclic Antidepressants The **tricyclic antidepressants** help reduce the symptoms of depression apparently by preventing the reuptake of norepinephrine and serotonin in the synapses or by changing the responsiveness of the receptors for these neurotransmitters. These drugs are reasonably effective, leading to the relief of acute symptoms of depression in about 60 percent of people with depression (Gijsman et al., 2004; Nemeroff, 2000). Some of the most commonly prescribed tricyclic antidepressants are imipramine, amitriptylene, and desipramine.

Unfortunately, however, the tricyclic antidepressants have a number of side effects. The most common ones are dry mouth, excessive perspiration, blurring of vision, constipation, urinary retention, and sexual dysfunction. Another problem with the tricyclic antidepressants is that they can take four to eight weeks to show an effect (Fava & Rosenbaum, 1995). This is an excruciatingly long time to wait for relief from depression. Finally, the tricyclics can be fatal in overdose, which is only three to four times the average daily prescription for the drug. For this reason, physicians are wary of prescribing these drugs, particularly for people with depression who might be suicidal.

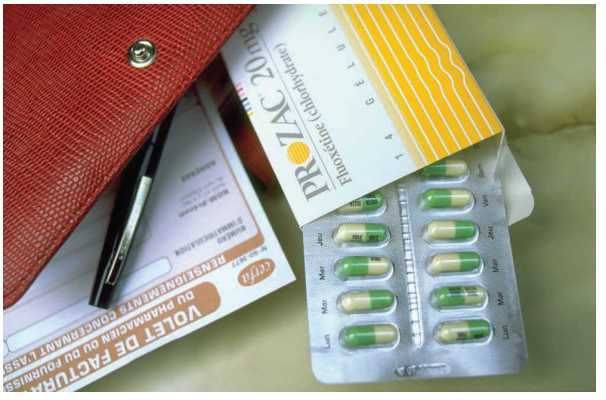
Monoamine Oxidase Inhibitors A second class of drugs used to treat depression is the **monoamine oxidase inhibitors (MAOIs).** MAO is an enzyme that causes the breakdown of the monoamine neurotransmitters in the synapse (Stahl, 1998). MAO inhibitors decrease the action of MAO and thus bring about increases in the levels of the neurotransmitters in the synapses.

The MAOIs are as effective as the tricyclic antidepressants, but physicians are more cautious in prescribing MAOIs, because their side effects are potentially more dangerous (Gitlin, 2002). When people taking MAOIs ingest food rich in an amino acid called *tyramine,* they can experience a rise in blood pressure that can be fatal. The foods that can interact with MAOIs include aged or ripened cheeses, red wine, beer, and chocolate. The MAOIs can also interact with several drugs, including antihypertension medications and over-the-counter drugs such as antihistamines. Finally, MAOIs can cause liver damage, weight gain, severe lowering of blood pressure, several of the same side effects of the tricyclic antidepressants.

Selective Serotonin Reuptake Inhibitors and Related Drugs A newer class of antidepressant drugs consists of the **selective serotonin reuptake inhibitors,** or **SSRIs.** These drugs are similar in structure to the tricyclic antidepressants, but they work more directly to affect serotonin than do the tricyclics. These drugs have become extremely popular in the treatment of depression. The SSRIs are not more effective in the treatment of depression than the antidepressants we have already discussed—about the same percentage of people respond to an SSRI as respond to a tricyclic or an MAOI (Gitlin, 2002; Montgomery et al., 2004).

These drugs have several advantages over the other antidepressants, however, which have made them extremely popular. First, many people begin experiencing relief from their depression after a couple of weeks of using these drugs, whereas it often takes four weeks or more for the other drugs to show significant effects. Second, the side effects of the SSRIs tend to be less severe than the side effects of the other antidepressants. Third, these drugs do not tend to be fatal in overdose and thus are safer than the other antidepressants (Nemeroff & Schatzberg, 1998). Fourth, the SSRIs appear to be helpful in a wide range of symptoms in addition to depression, or those often associated with depression, such as anxiety symptoms, binge eating, and premenstrual symptoms. The SSRIs may be useful in the treatment of the most chronic and persistent types of depression (Frank, Grochocinski, et al., 2000; Keller et al., 1998).

The SSRIs do have side effects, however (Gitlin, 2002). One of the most common is increased agitation or nervousness. People on SSRIs often report



The selective serotonin reuptake inhibitors have become the widest selling antidepressant drugs.

feeling "jittery" or "hyper" and that they cannot sit still. They may have mild tremors and increased perspiration and feel weak. Some find themselves becoming angry or hostile more often. Nausea and stomach cramps or gas are common side effects, as is a decrease in appetite. Sexual dysfunction and decreased sexual drive are reported by some. Finally, there appears to be an increase in risk for suicide among people on SSRIs.

A number of other drugs that have been introduced for the treatment of depression in the past decade share some similarities with the SSRIs or the older antidepressants but cannot be classified in the same categories. Some of these antidepressants were designed to affect the levels of norepinephrine as well as serotonin and thus are referred to as selective serotonin and norepinephrine reuptake inhibitors (SSNRIs). Some examples are mirtazapine (Remeron), nefazodone (Serzone), venlafaxine (Effexor), and duloxetine (Cymbalata).

Bupropion (which goes by the trade names Wellbutrin and Zyban) affects the norepinephrine and dopamine systems. It may be especially useful in people suffering from psychomotor retardation, anhedonia, hypersomnia, cognitive slowing, inattention, and craving; for example, bupropion can help people stop craving cigarettes. In addition, bupropion appears to overcome the sexual side effects of the SSRIs and thus is sometimes used in conjunction with them. The side effects of bupropion include agitation, insomnia, nausea, and seizures.

Although a large selection of drug therapies is now available for the treatment of depression, there are no consistent rules for determining which of the antidepressant drugs to try first with a person with depression. Many clinicians begin with the selective serotonin reuptake inhibitors because their side effects tend to be less significant. As we discuss in *Taking Psychology Personally: Primary Care Physicians Treating Depression*, most people with depression in treatment are being treated by their primary care physicians. People with depression often must try several drugs before finding one that works well for them and has tolerable side effects. When they find the drug that works for them, it is often as if they have regained their lives (Wurtzel, 1995, p. 329):

> VOICES
>
> And then something just kind of changed in me. Over the next few days, I became all right, safe in my own skin. It happened just like that. One morning I woke up, and I really did want to live, really looked forward to greeting the day, imagined errands to run, phone calls to return, and it was not with a feeling of great dread, not with the sense that the first person who stepped on my toe as I walked through the square may well have driven me to suicide. It was as if the miasma of depression had lifted off me, gone smoothly about its business, in the same way that the fog in San Francisco rises as the day wears on.

Electroconvulsive Therapy for Depression

Perhaps the most controversial of the biological treatments for depression is **electroconvulsive therapy (ECT).** ECT was introduced in the early twentieth century, originally as a treatment for schizophrenia. Italian physicians Ugo Cerlettii and Lucio Bini decided to experiment with the use of ECT to treat people with schizophrenia, reasoning that ECT could calm them much as experiencing an epileptic seizure would calm and sedate epileptics. Eventually, clinicians found that ECT is not effective for schizophrenia, but it is effective for depression.

ECT consists of a series of treatments in which a brain seizure is induced by passing electrical current through the brain. Patients are first anesthetized and given muscle relaxants, so that they are not conscious when they have the seizure and so that their muscles do not jerk violently during the seizure. Metal electrodes are taped to the head, and a current of 70 to 130 volts is passed through one side of the brain for about one-half of a second. Patients typically go into a convulsion, which lasts about one minute. The full ECT treatment consists of 6 to 12 sessions. ECT is most often given to people with depression who have not responded to drug therapies, and it relieves depression in 50 to 60 percent of these people (Fink, 2001).

Neuroimaging studies show that ECT results in decreases in metabolic activity in several regions of the brain, including the frontal cortex, and the anterior cingulate (Henry et al., 2001; Oquendo et al., 2001) It is not entirely clear, however, how ECT lifts depression.

ECT is controversial for several reasons. First, there were reports in the past of ECT being used as a punishment for patients who were unruly, as was depicted in the movie *One Flew over the Cuckoo's Nest*. Second, ECT can lead to memory loss and difficulties in learning new information. When ECT was first developed, it was administered to both sides of the brain, and the effects on memory and learning were sometimes severe and permanent.

Taking Psychology Personally

Primary Care Physicians Treating Depression

In most of the research on the efficacy of treatments for depression that we've discussed in this chapter, the treatment was administered by a trained clinical psychologist or psychiatrist. The majority of people treated for depression, however, never see a psychologist or psychiatrist (Rost et al., 1998). Instead, most consult their primary care physician (e.g., their "family doctor," internist, or gynecologist).

Actually, patients rarely state that they are depressed. They are more likely to report physical symptoms, such as fatigue, loss of appetite, problems sleeping, or general aches and pains. This is one reason that depression is never detected by primary care physicians in 40 to 50 percent of patients who would qualify for a diagnosis of major depression (Katon et al., 2001). Other reasons include the rushed nature of interactions between physicians and patients in today's world of managed care and the competing demands for that time—if the patient has other, more pressing or obvious medical problems, the physician is likely to attend to those instead of to complaints of malaise (Rost et al., 2000). Many physicians also report that they feel uncomfortable asking patients about depressive symptoms or other mental-health problems, and they worry about offending a patient with such questions.

When a physician does detect depression in a patient, about three-quarters of the time he or she prescribes an antidepressant medication (Williams et al., 1999). At best, only about one-third of patients are given a referral to a mental-health specialist. This may be because many patients do not have insurance benefits that cover mental-health care, and many patients refuse to see a mental-health specialist because of stigmas.

Unfortunately, the care that many primary care physicians provide for patients with depression is inadequate (Simon et al., 2001). The dosages of antidepressants prescribed often are less than what research suggests is necessary for an effective response. Side effects are not monitored systematically. Over 20 percent of patients never fill their prescriptions for antidepressant medications, and as many as 50 percent stop taking the medication without consulting their doctor (Greden, 2001).

Fortunately, studies in primary care settings have shown that the quality of care given to patients with depression can be increased significantly by collaborative care programs, in which primary care physicians work with psychiatrists and psychologists (Katon et al., 2001; Simon et al., 2001). In these programs, the primary care physician continues to be responsible for the care of the patient, but the patient is given educational materials about depression, referrals to psychotherapy or community social services if necessary, and a relatively small number of visits (two to four) with a psychiatrist specializing in depression care. Patients receiving this collaborative care are more likely to take prescribed antidepressant medications, show greater reductions in depression in the short term, and are less likely to relapse over the long term.

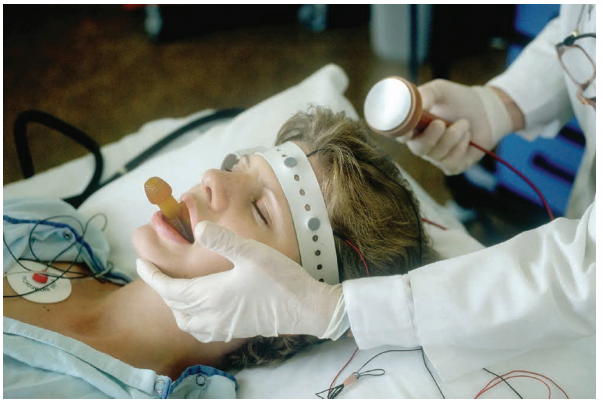
If you or someone you know seeks care from a primary care physician for depression, what can you do to ensure that you receive adequate care? First, be honest with your doctor. Talk about your symptoms, even if it makes you uncomfortable. In particular, if you have been having suicidal thoughts, tell your doctor explicitly. If your doctor seems to ignore your reports of depressive symptoms, find another doctor.

Second, ask for a referral to a mental-health specialist if your health insurance covers this. If you don't have insurance for mental-health care, ask your doctor for recommendations for community services that are low-cost or free.

Third, if your doctor prescribes antidepressant medications for you, make sure he or she knows about any medications you are taking, including herbal remedies, such as St. John's wort, to prevent possible interactions between medications. (See Chapter 5 for a discussion of the dangers of mixing herbal remedies with prescription medications.)

Fourth, if you have no intention of taking the antidepressants prescribed for you, tell your doctor—don't just accept the prescription, then ignore it. Ask your doctor about alternative treatments.

Finally, if you begin taking an antidepressant and it doesn't give you serious side effects, continue taking it for a few weeks before you judge whether it's effective. Many antidepressants take awhile to begin to work. If after a few weeks you still are not experiencing any benefits from the antidepressant, or if your symptoms of depression get worse, tell your doctor, so that he or she can change or adjust the prescription or suggest an alternative treatment.



Electroconvulsive therapy is a controversial but effective treatment for depression.

These days, ECT is usually delivered to only one side of the brain, usually the right side, because it is less involved in learning and memory. As a result, patients undergoing modern ECT do not tend to experience significant or long-term memory or learning difficulties (Glass, 2001). Because this unilateral administration is sometimes not as effective as bilateral administration, some people are still given bilateral ECT. Third, although ECT can be extremely effective in eliminating the symptoms of depression, the relapse rate among people who have undergone ECT is as high as 85 percent (Fink, 2001). Fourth, perhaps the strongest reason ECT is controversial is that the idea of having electrical current passed through a person's brain is very frightening and seems like a primitive form of treatment.

Still, ECT is sometimes the only form of treatment that works for people with severe depression. One survey found that about 10 percent of people admitted to the psychiatric wards of general hospitals in the United States with diagnoses of recurrent major depression received ECT (Olfson et al., 1998). The people most likely to receive ECT were older, White, privately insured, and more affluent. It may be that people of color and poor people do not have access to ECT in the hospitals in their neighborhoods. In addition, ECT is used more frequently in eastern and midwestern states than in western states. This may be because ECT is regulated more closely, and frowned upon more, in western states, such as California. Those people who did receive ECT early in their hospital stays had shorter stays than those who did not, suggesting they recovered more quickly from their depression.

Repetitive Transcranial Magnetic Stimulation

In recent years, researchers have been investigating new methods for stimulating the brain without the application of electric current (Sackheim & Lisanby, 2001). Scientists are using powerful magnets, such as those used in magnetic resonance imaging, to stimulate targeted areas of the brain. The procedure known as **repetitive transcranial magnetic stimulation (rTMS)** exposes patients to repeated, high-intensity magnetic pulses focused on particular brain structures, such as the left prefrontal cortex, which tends to show abnormally low metabolic activity in some people with depression. Studies have suggested that patients with depression given rTMS daily for at least a week tend to experience relief from their symptoms (Chae et al., 2001; George et al., 2003).

How does rTMS work? The electrical stimulation of neurons can result in long-term changes in neurotransmission across synapses (George et al., 2003). Neurotransmission can be enhanced or blunted, depending on the frequency of the stimulation. By stimulating the left prefrontal cortex of people with depression at particular frequencies, researchers have been able to increase neuronal activity, which in turn has had an antidepressant effect. Patients who receive rTMS report few side effects, usually only minor headaches treatable by aspirin. Patients can remain awake, rather than having to be anesthetized, as in electroconvulsive therapy (ECT), thereby avoiding the possible complications of anesthesia. There is a great deal of hope that rTMS will be an effective and safe alternative therapy, particularly for people who do not respond to drug therapies and cannot tolerate ECT.

Vagus Nerve Stimulation

Another new method that holds considerable promise in the treatment of serious depression is **vagus nerve stimulation (VNS)** (Marangell, Martinez, & Niazi, 2004). The vagus nerve is part of the autonomic nervous system; it carries information from the head, neck, thorax, and abdomen to several areas of the brain, including the hypothalamus and amygdala, which are involved in depression. In VNS, the vagus nerve is stimulated by a small electronic device much like a cardiac pacemaker, which is surgically implanted under a patient's skin in the left chest wall. Vagus nerve stimulation was originally used to control seizures in epileptic patients, and some investigators noticed that the therapy also improved mood in these patients (George et al., 2000). The mood effects of VNS occurred even in epileptic patients who were still having seizures, so researchers began studying the mood effects of VNS in patients with depression.

In one study of 38 patients with depressions that had not responded to other forms of treatment, 40 percent got substantial relief from their depression with VNS (George et al., 2000). Another 30 percent of the patients got minimal relief through

VNS. Four patients had negative side effects, including agitation, and 3 patients' surgical wounds did not heal promptly. In another study, of 59 depressed patients, 31 percent obtained significant relief from their depression with VNS; among those who had previously responded to antidepressant medications, 40 percent responded to VNS (Sackheim et al., 2001). About half of all the patients reported mild voice alteration or hoarseness as a side effect of VNS.

How does VNS work when it relieves depression? That is unknown currently, but positron-emission studies show that VNS increases activity in the hypothalamus and amygdala, which may have antidepressant effects (George et al., 2000). Additional research is currently being done on VNS in hopes that it will provide a relatively safe alternative treatment for some people with depression.

Light Therapy

Recall that seasonal affective disorder (SAD) is a form of mood disorder in which people become depressed during the winter months, when there are the fewest hours of daylight. Their moods then brighten in the summer months, when there is more daylight each day. It turns out that many people with SAD who are exposed to bright lights for a few hours each day during the winter months experience complete relief from their depression within a couple of days (Koorengevel et al., 2001; Wileman et al., 2001).

Light therapy may help reduce seasonal affective disorder by resetting *circadian rhythms,* natural cycles of biological activities that occur every 24 hours. The production of several hormones and neurotransmitters varies over the course of the day, according to circadian rhythms. These rhythms are regulated by internal clocks but can be affected by environmental stimuli, including light. People with depression sometimes show dysregulation of their circadian rhythms. Light therapy may work by resetting circadian rhythms and thereby normalizing the production of hormones and neurotransmitters (Koorengevel et al., 2001).

Light therapy can be helpful to people with seasonal affective disorder.

Another theory is that light therapy works by decreasing levels of the hormone melatonin, secreted by the pineal gland (Wehr et al., 2001). Decreasing melatonin levels can increase levels of norepinephrine and serotonin, thereby reducing the symptoms of depression. Finally, studies suggest that exposure to bright lights may directly increase serotonin levels, thereby decreasing depression (Rosenthal, 1995).

Drug Treatments for Bipolar Disorder

Many fewer drugs are available to treat bipolar disorder than to treat unipolar depression, because this disorder is understood less well than depression and because it is more rare. Fortunately, however, recent years have seen an increase in the number of drugs designed to treat bipolar disorder.

Lithium **Lithium** is the most common treatment for bipolar disorder. A number of controlled trials show that lithium is effective in preventing relapses of bipolar disorder (Geddes et al., 2004; Ghaemi, Pardo, & Hsu, 2004).

Lithium seems to stabilize a number of neurotransmitter systems, including serotonin, dopamine, and glutamate (Dixon & Hokin, 1998; Lenox & Manji, 1995). It appears to be more effective in reducing the symptoms of mania than the symptoms of depression. People with bipolar disorder are often prescribed lithium to help curb their mania and an antidepressant drug to curb their depression (Nemeroff, 2000).

Most people with bipolar disorder take lithium even when they have no symptoms of mania or depression, in order to prevent relapses. People maintained on adequate doses of lithium have significantly fewer relapses than those not maintained on lithium (Maj et al., 1998; Tondo, Jamison, & Baldessarini, 1997). Up to 55 percent of patients develop a resistance to lithium within three years, however, and only about one-third remain symptom-free on lithium (Nemeroff, 2000).

Although lithium has been a lifesaver for many people with bipolar disorder, it poses some problems. First, there are enormous differences among people in their rates of lithium absorption, so the proper dosage varies greatly from one person to the next. Second, the difference between an effective dose of lithium and a toxic dose is small, leaving a

very narrow window of therapeutic effectiveness. People who take lithium must be monitored carefully by physicians, who can determine whether the dosage of lithium is adequate to relieve the symptoms of bipolar disorder but not too large to induce toxic side effects. The side effects of lithium range from annoying to life-threatening. Many patients experience abdominal pain, nausea, vomiting, diarrhea, tremors, and twitches (Jamison, 1995, p. 93):

VOICES

> I found myself beholden to a medication that also caused severe nausea and vomiting many times a month—I often slept on my bathroom floor with a pillow under my head and my warm, woolen St. Andrews gown tucked over me. I have been violently ill more places than I choose to remember, and quite embarrassingly so in public places.

People on lithium complain of blurred vision and problems in concentration and attention that interfere with their ability to work. Lithium can cause diabetes, hypothyroidism, and kidney dysfunction. It can also contribute to birth defects if taken during the first trimester of a woman's pregnancy.

It is not surprising that many people with bipolar disorder will not take lithium or will go on and off of it, against their physicians' advice. In addition to experiencing side effects, many patients complain that they miss the positive symptoms of their mania—the elated moods, flowing ideas, and heightened self-esteem—and feel washed-out on lithium. Especially during periods of calm, they feel they can manage their illness without lithium and that they can detect when a new episode is coming and go back on the medication then. Usually, however, as a new episode of mania becomes more and more severe, their judgment becomes more impaired, and they do not go back on the lithium.

Anticonvulsants, Antipsychotics, and Calcium Channel Blockers Sometimes, lithium does not overcome mania and, even if it is effective, some people cannot tolerate its side effects. Three other classes of drugs, **anticonvulsants, antipsychotic drugs,** and **calcium channel blockers,** are alternatives to lithium for the treatment of mania.

The most commonly prescribed anticonvulsants are carbamazepine (trade name Tegretol), valproic acid (trade names Depakene and Valproate), and divalproex sodium (trade name Depakote). These drugs can be effective in reducing the symptoms of severe and acute mania, although it is not clear if they are as effective as lithium in the long-term treatment of bipolar disorder. For this reason, lithium is still usually used first, before trying the anticonvulsants (Ghaemi et al., 2004; Grunze & Walden, 2002). The side effects of carbamazepine include blurred vision, fatigue, vertigo, dizziness, rash, nausea, drowsiness, and liver disease (Nemeroff, 2000). Valproic acid and divalproex sodium seem to induce many fewer side effects and are now used more often than carbamazepine (Frances et al., 1998). But the anticonvulsants can cause birth defects if women take them while pregnant. The anticonvulsants have effects on a multitude of neurotransmitters, but the way in which the anticonvulsants reduce mania is not yet clear (Nemeroff, 2000).

The antipsychotic drugs, which are described in more detail in Chapter 11, are also used to quell the symptoms of severe mania (Nemeroff, 2000). These drugs reduce functional levels of dopamine and seem especially useful in the treatment of psychotic manic symptoms. They have many neurological side effects, however, the most severe of which is an irreversible condition known as *tardive dyskinesia*. People with tardive dyskinesia have uncontrollable tics and movements of their face and limbs. Newer drugs, such as clozapine, olanzapine, and risperidone, do not induce these neurological side effects and are being investigated for use in bipolar disorder (Post, Frye, et al., 2000).

Most recently, drugs known as calcium channel blockers, such as verapamil and nimodipine, have been shown to be effective in treating mania in some, but not all, studies (Keck et al., 2000). The calcium channel blockers are safe for women to take during pregnancy. They seem to induce fewer side effects than lithium and perhaps the anticonvulsants, but they can create dizziness, headache, nausea, and changes in heart rate. It is not currently known how these drugs work to lower mania.

Psychological Treatments for Depression

Each of the psychological theories of depression has led to a treatment designed to overcome the factors that the theory asserts causes depression. Behavior therapy focuses on changing the depressed person's schedule of reinforcements and punishments. Cognitive-behavioral therapy focuses on changing both negative cognitions and maladaptive behaviors. Interpersonal therapy works on dysfunctional relationship patterns, and psychodynamic therapy focus on uncovering the unconscious hostility toward others that is the source of the person's self-punishment (see the Concept Overview in Table 9.9).

TABLE 9.9 Concept Overview

Psychological Treatments for Depression

Each of the psychological treatments for depression aims to reverse the processes contributing to depression.

Type of Treatment	Proposed Mechanism of Action
Behavior therapies	Increase positive reinforcers and decrease aversive events by teaching the person new skills for managing interpersonal situations and the environment and engaging in pleasant activities
Cognitive-behavioral therapy	Challenges distorted thinking and helps the person learn more adaptive ways of thinking and new behavioral skills
Interpersonal therapy	Helps the person change dysfunctional relationship patterns
Psychodynamic therapies	Help the person gain insight into unconscious hostility and fears of abandonment to facilitate change in self-concept and behaviors

Behavior Therapies

Behavior therapies for depression focus on increasing the number of positive reinforcers and decreasing the number of aversive experiences in an individual's life by helping the depressed person change his or her ways of interacting with the environment and other people (Hollon, Haman, & Brown, 2002). Behavior therapies are designed to be short-term, about 12 weeks long.

The first phase of behavior therapies requires a *functional analysis* of the connections between specific circumstances and the depressed person's symptoms. When does the depressed person feel the worst? Are there any situations in which he or she feels better? The therapist may visit the depressed client's home to observe his or her interactions with family members. The client may fill out questionnaires to assess what events he or she finds pleasant or unpleasant. This analysis helps the therapist pinpoint the behaviors and interaction patterns that need to be the focus of therapy. It also helps the client understand the intimate connections between his or her symptoms and daily activities or interactions. This understanding challenges the client's belief that he or she is the helpless victim of uncontrollable forces and sets the stage for the therapist's suggestions for changes in behavior.

Once the therapist and client identify the circumstances that precipitate the client's depressive symptoms, a variety of strategies can be used to make the necessary changes in the client's life. These generally fall into three categories (Thorpe & Olson, 1997):

1. *Change the aspects of the environment that are related to the depressive symptoms.* The depressed person may be encouraged to engage in specific rewarding activities and to avoid depressing activities. For example, a depressed man who typically spends all evening in front of the television, being bored and depressed, might be encouraged to take a half-hour walk around his neighborhood every evening and to limit his television watching to one hour.
2. *Teach the depressed person skills to change his or her negative circumstances, particularly negative social interactions.* For example, a depressed woman who feels her relationship with her child is out of control might be taught parenting skills, so that she is able to interact more effectively and pleasantly with her child.
3. *Teach the client mood-management skills that can be used in unpleasant situations.* It is inevitable that people with depression will find themselves in unpleasant situations some of the time. The therapist may teach the person to use strategies, such as relaxation techniques (see Chapter 7), to reduce negative symptoms even while an unpleasant event is happening. These strategies must be woven together to meet the specific needs of an individual client. For example, consider the following case (adapted from Yapko, 1997).

> Mark worked constantly. When he was not actually at work, he was working at home. He had a position of considerable responsibility and was convinced that, if he didn't stay focused on his job, he'd miss something that

(*continued*)

CASE STUDY

would result in his being fired or kicked off the career ladder. Mark had not taken a vacation in several years. Although he wanted to continue to get pay raises and promotions, as he has each year, he was also painfully aware that life was passing him by. He felt stressed, depressed, and hopeless about ever having a "normal" life.

Mark clearly felt rewarded for his one-dimensional life with praise, pay raises, promotions, and the absence of mistakes for which he might get punished. Mark's behavior was governed by his work focus. He engaged in no social activities, lived alone, and did not organize his time to include anything but his work. The behavior therapist suggested that, if he wanted to improve his quality of life, and his outlook on life, he must learn some very specific new behaviors. Mark was encouraged to organize his schedule so that he'd have time for social and recreational opportunities. He learned he needed to actively and deliberately do things that are fun and pleasurable. The therapist practiced with him new ways to meet people and form social relationships (friendships, dating). The therapist also taught him relaxation skills to reduce his stress. Eventually, Mark felt a new sense of control over his life and his depression lifted.

Cognitive-Behavioral Therapy

Cognitive-behavioral therapy represents a blending of cognitive and behavioral theories of depression (Beck et al., 1974; Ellis & Harper, 1961; Lewinsohn et al., 1986; Rehm, 1977). There are two general goals in this therapy. First, it aims to change the negative, hopeless patterns of thinking described by the cognitive models of depression. Second, it aims to help people with depression solve concrete problems in their lives and develop skills for being more effective in their worlds, so that they no longer have the deficits in reinforcers described by behavioral theories of depression.

Like behavior therapy, cognitive-behavioral therapy is designed to be brief. The therapist and client usually agree on a set of goals they wish to accomplish in 6 to 12 weeks. These goals focus on specific problems that clients believe are connected to their depression, such as problems in their marriage or dissatisfaction with their job. From the very beginning of therapy, the therapist urges clients to take charge of the therapy as much as possible, setting goals and making decisions themselves, rather than relying on the therapist to give them all the answers.

Cognitive Techniques The first step in cognitive-behavioral therapy is to help clients discover the negative, automatic thoughts they habitually have and to understand the link between those thoughts and their depression. Often, the therapist will assign clients the homework of keeping track of times when they feel sad or depressed and writing down on sheets, such as the one in Figure 9.8, what is going through their minds at such times. Clients often report that they did not realize the types of thoughts that went through their heads when certain types of events happened. For example, the client whose automatic thought record is shown in Figure 9.8 did not realize that she had catastrophic thoughts about losing her job every time her boss was a little cross with her.

The second step in cognitive-behavioral therapy is to help clients challenge their negative thoughts. People with depression often believe that there is only one way to interpret a situation—their negative way. Therapists will use a series of questions to help clients consider alternative ways of thinking about a situation and the pros and cons of these alternatives, such as "What is the evidence that you are right in the way you are interpreting this situation?" "Are there other ways of looking at this situation?" and "What can you do if the worst-case scenario comes true?" Of course, these questions don't always move the client toward more positive ways of thinking about the situation. It is important for the therapist to be flexible in pursuing a line of questions or comments, dropping approaches that are not helpful and trying new approaches to which the client might respond better.

The third step in cognitive-behavioral therapy is to help clients recognize the deeper, basic beliefs or assumptions they hold that are feeding their depression. These basic beliefs might be ones such as "If I'm not loved by everyone, I'm a failure" or "If I'm not a complete success at everything, my life is worthless." The therapist will help clients question these beliefs and decide if they truly want to live their lives according to these beliefs. The case of Susan illustrates some of the cognitive components of cognitive-behavioral therapy (adapted from Thorpe & Olson, 1997, pp. 225–227):

FIGURE 9.8 **An Automatic Thoughts Record Used in Cognitive-Behavioral Therapy.** In cognitive-behavioral therapy, patients keep records of the negative thoughts that arise when they feel negative emotions. These records are then used in therapy to challenge the patients' depressive thinking.

Date	Event	Emotion	Automatic thoughts
April 4	Boss seemed annoyed.	Sad, anxious, worried	Oh, what have I done now? If I keep making him mad, I'm going to get fired.
April 5	Husband didn't want to make love.	Sad	I'm so fat and ugly.
April 7	Boss yelled at another employee.	Anxious	I'm next.
April 9	Husband said he's taking a long business trip next month.	Sad, defeated	He's probably got a mistress somewhere. My marriage is falling apart.
April 10	Neighbor brought over some cookies.	A little happy, mostly sad	She probably thinks I can't cook. I look like such a mess all the time. And my house was a disaster when she came in!

CASE STUDY

Susan was seen for 14 sessions of psychotherapy. She was a young, single, 24-year-old woman. Her goals for therapy were to learn how to overcome chronic feelings of depression and to learn how to deal with temptations to overeat. Susan was unemployed and living with her aunt and uncle in a rural area. She had no means of personal transportation. Hypersensitivity to the reactions of significant others and the belief that they could control her feelings seemed to be central to her low self-concept and feelings of helplessness. Susan described her mother as knowing which "buttons to push." This metaphor was examined and challenged. She was questioned as to how her mother controlled her emotions: Where were these buttons? Did they have a physical reality? Once again, the principle was asserted that it is not the actions of others that cause emotions, but one's cognitions about them.

Then the cognitions she had concerning certain looks or critical statements were examined. When her aunt was looking "sickly and silent," Susan believed that it was because she was displeased with her for not helping enough. The evidence for this belief was examined, and there was none. Alternative explanations were explored, such as the aunt might be truly ill, having a bad day, or upset with her spouse. Susan admitted that all explanations were equally plausible. Furthermore, it was noted that, in ambiguous social situations, she tended to draw the most negative and personalized conclusions.

Her consistent tendency to evaluate her self-worth in terms of her family's approval

(*continued*)

was examined. Susan still had fantasies of her family becoming like the "Walton" family (e.g., a "normal" family that was loving and accepting of one another; instead, her own family was distant and argumentative with one another). Susan began to let go of this fantasy and grieved over this loss. Once this had been done, she began to gain a better understanding of how her current cognitive distortions could be related to overconcern with familial approval. As she began to let go of her desire to live up to imagined expectations, she stopped seeing herself as a failure.

During the last stage of therapy, Susan's mother visited. This provided a real test of the gains she had made, as it was her mother's criticism that Susan feared the most. At first, she reported feeling easily wounded by her mother's criticism. These examples were used as opportunities to identify and challenge self-defeating thoughts. Soon, Susan was able to see her mother's critical statements as her mother's problem, not her own. She also discovered that, as she became better at ignoring her mother's critical remarks and not taking them to heart, her mother began to be more relaxed and open around her and criticized her less.

Behavioral Techniques Cognitive-behavioral therapists also use behavioral techniques to train clients in new skills they might need to cope better in their life. Often, people with depression are unassertive in making requests of other people or in standing up for their rights and needs. This unassertiveness can be the result of their negative automatic thoughts. For example, a person who often thinks, "I can't ask for what I need, because the other person might get mad and that would be horrible," is not likely to make even reasonable requests of other people. The therapist will first help clients recognize the thoughts behind their actions (or lack of action). Then, the therapist may work with clients to devise exercises or homework assignments in which they practice new skills, such as assertiveness, between therapy sessions.

The Effectiveness of Cognitive-Behavioral Therapy Cognitive-behavioral therapy has proven quite effective in treating depression, including major depression. About 60 to 70 percent of people with depression experience full relief from their symptoms with 12 weeks of cognitive therapy (Hollon et al., 2002; Lewinsohn & Clarke, 1999). Cognitive-behavioral therapy has been successfully adapted for the treatment of depressed children, adolescents, and older persons (Futterman et al., 1995; Garber & Horowitz, 2002; Lewinsohn & Clarke, 1999; Treatment for Adolescents with Depression Study [TADS] Team, 2004).

Interpersonal Therapy

In **interpersonal therapy (IPT),** therapists look for four types of problems in depressed patients (see the Concept Overview in Table 9.10). First, many depressed patients truly are grieving the loss of loved ones, perhaps not from death but from the breakup of important relationships. Interpersonal therapists help clients face such losses and explore their feelings about the losses. Often, clients idealize the people they have lost, feeling as if they will never have relationships as good. Therapists help clients reconstruct their relationships with the lost loved ones, recognizing both the good and bad aspects of the relationships and developing more balanced views of the relationships. Therapists also help clients let go of the past relationships and begin to invest in new relationships.

The second type of problem interpersonal therapy focuses on is interpersonal role disputes. Such disputes arise when people do not agree on their roles in a relationship. For example, a husband and wife may disagree on the proper roles each should play in relation to their children. Or a college student and a parent may disagree on the extent to which the student should follow the parent's wishes in choosing a career. Interpersonal therapists first help the clients recognize the disputes and then guide clients in making choices about concessions they are or are not willing to make to the other people in the relationships.

Therapists may also need to help clients modify and improve their patterns of communicating with others in relationships. For example, a student who resents his parents' intrusions into his private life may tend to withdraw and sulk rather than directly confront his parents about their intrusions. He would be helped in developing more effective ways of communicating his distress over his parents' intrusions.

The third type of problem addressed in interpersonal therapy is role transitions, such as the transition from college to work or from work to full-time motherhood. Sometimes, people become depressed out of grief over the roles they must leave behind. Therapists help clients develop more realistic perspectives toward roles that are lost and help clients regard new roles in a more positive manner. If clients feel unsure about their capabili-

TABLE 9.10 Concept Overview

Interpersonal Therapy

Interpersonal therapists focus on four types of interpersonal problems as sources of depression.

Type of Problem	Therapeutic Approach
Grief, loss	Help the client accept feelings and evaluate a relationship with a lost person; help the client invest in new relationships
Interpersonal role disputes	Help the client make decisions about concessions willing to be made and learn better ways of communicating
Role transitions	Help the client develop more realistic perspectives toward roles that are lost and regard new roles in a more positive manner
Interpersonal skills deficits	Review the client's past relationships, helping the client understand these relationships and how they might be affecting current relationships; directly teach the client social skills, such as assertiveness

ties in new roles, therapists help them develop a sense of mastery in the new roles. Sometimes, clients need help in developing new networks of social support within their new roles, to replace the support systems they left behind in old roles.

The fourth type of problem people with depression take to interpersonal therapy involves deficits in interpersonal skills. Such skill deficits can be the reason that people with depression have inadequate social support networks. Therapists review with clients past relationships, especially important childhood relationships, helping clients understand these relationships and how they might be affecting current relationships. Therapists might also directly teach clients social skills, such as assertiveness.

Interpersonal therapy has been shown to be highly effective in the treatment of depression, with 60 to 80 percent of people with depression recovering during this form of therapy (Weissman & Markowitz, 2002). Like cognitive-behavioral therapy, interpersonal therapy has been successfully adapted for the treatment of children and older adults with depression. It can be used both in individual therapy and in group therapy settings.

An interesting application of interpersonal therapy in a group setting occurred in a study conducted in rural Uganda (Bolton et al., 2003). The people of Uganda have suffered terrible trauma over the decades, and the rate of depression in this country is high. A group of researchers conducted a randomized clinical trial to test the effectiveness of group interpersonal psychotherapy in treating villagers in rural Uganda who were depressed. Their intervention resulted in significant decreases in depression in the villagers who received it, compared with villagers in the control group.

Interpersonal therapy for depression focuses on problems in interpersonal relationships that lead to depression.

Psychodynamic Therapies

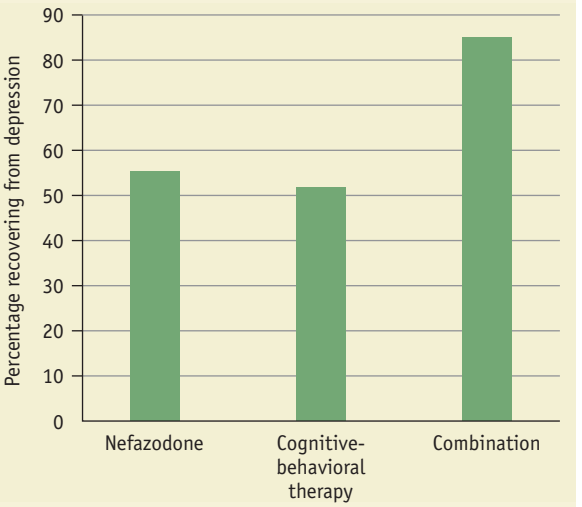
In **psychodynamic therapies,** the therapist will closely observe a depressed client's behavior to analyze the sources of his or her depression, just as a behavior or cognitive therapist will. The types of behavior the psychodynamic therapist examines, and the therapist's assumptions about the potential causes of that behavior, are very different from those that concern the behavior or cognitive therapist.

The psychodynamic therapist will closely observe the client's *transference* to the therapist—the ways in which the client treats the therapist as though the therapist were someone else, such as a parent—with the assumption that the client's transference represents unconscious conflicts and concerns with important people in his or her life. The therapist will also observe the client's recollections of both recent events and distant events,

searching for themes of abandonment, hostility, and disappointment. The therapist may listen to the client's recounting of dreams for further clues as to the unconscious concerns behind the depression. The therapist will acknowledge and interpret the themes he or she observes in the client's behaviors and recollections, to help the client gain insight, accept these unconscious concerns, and move beyond them.

Although it may seem necessary to have insight to fully gain control over one's depression, long-term psychodynamic therapies have not proven very effective in the treatment of depression (Robinson, Berman, & Neimeyer, 1990). The nature of depression may make it particularly unsuitable for long-term psychodynamic therapies. Many people with depression are too overcome by symptoms of lethargy, poor attention and concentration, and a sense of hopelessness to participate in these therapies. They may not have the energy or motivation to engage in the long process of uncovering and exploring old psychological wounds. They may be so acutely depressed that they need more immediate relief, particularly if they are suicidal.

FIGURE 9.9 **Comparison of Drug Therapy and Psychotherapy.** In one study, people with chronic major depression responded equally well to a drug therapy (nefazodone) and cognitive-behavioral therapy but responded best to the combination of the two therapies.



Source: Keller et al., 2000.

Comparisons of Cognitive-Behavioral, Interpersonal, and Drug Therapies

Which of the many treatments for mood disorders is the best? In the past few decades, several studies have compared cognitive-behavioral therapy, interpersonal therapy, and drug therapies with each other. Perhaps surprisingly, these three therapies, despite their vast differences, appear equally effective for the treatment of most people with depression (see DeRubeis et al., 1999; Hollon et al., 2002; Weissman & Markowitz, 2002). A growing number of studies suggest that the combination of psychotherapy and drug therapy is more effective in treating people with chronic depression than is either type of therapy alone (e.g., Hollon et al., 1992; Frank, Grochocinski, 2000; Thase et al., 1997; TADS Team, 2004).

For example, in one study, 681 patients with chronic major depression were randomly assigned to receive nefazodone (trade name Serzone), cognitive-behavioral therapy, or both for 12 weeks (Keller et al., 2000). About half of the people receiving nefazodone or cognitive-behavioral therapy alone experienced relief from their depression (see Figure 9.9). Eighty-five percent of the patients receiving both nefazodone and cognitive-behavioral therapy experienced relief from their depression.

The relapse rates in depression are quite high, even among people whose depression completely disappears in treatment. For this reason, many psychiatrists and psychologists argue that people with a history of recurrent depression should be kept on a maintenance dose of therapy even after their depression has been relieved (Hirschfeld, 1994). Usually, the maintenance therapy is a drug therapy, and many people remain on antidepressant drugs for years after their initial episodes of depression have passed. Studies of interpersonal therapy and cognitive-behavioral therapy show that maintenance doses of these therapies, usually consisting of once-a-month meetings with therapists, can also reduce relapse just as well as drugs (Hollon et al., 2002; Jarrett et al., 1998; Weissman & Markowitz, 2002).

Studies suggest that similar changes in the brain occur whether people with depression undergo psychotherapy or drug therapy. For example, Brody and colleagues (2001) put people with major depression through a PET scan to assess their brain functioning. Compared with control participants, the depressed participants showed abnormal activity in the prefrontal cortex and the temporal lobe. The depressed participants were then given either interpersonal therapy or a selective serotonin reuptake inhibitor for 12 weeks, and

their brain functioning was reevaluated at the end of the therapies. The interpersonal therapy group and drug therapy group both showed normalization of their brain functioning over the course of therapy, to similar levels.

In bipolar disorder, combining drug treatment with the psychological therapies may reduce the rate at which patients stop taking their medications and may lead more patients to achieve full remission of their symptoms, compared with lithium treatment alone (Miklowitz et al., 2000; Swarz & Frank, 2001). Psychotherapy can help people with bipolar disorder understand and accept their need for lithium treatment. It also can help them cope with the impact of the disorder on their lives (Jamison, 1995, pp. 88–89):

VOICES

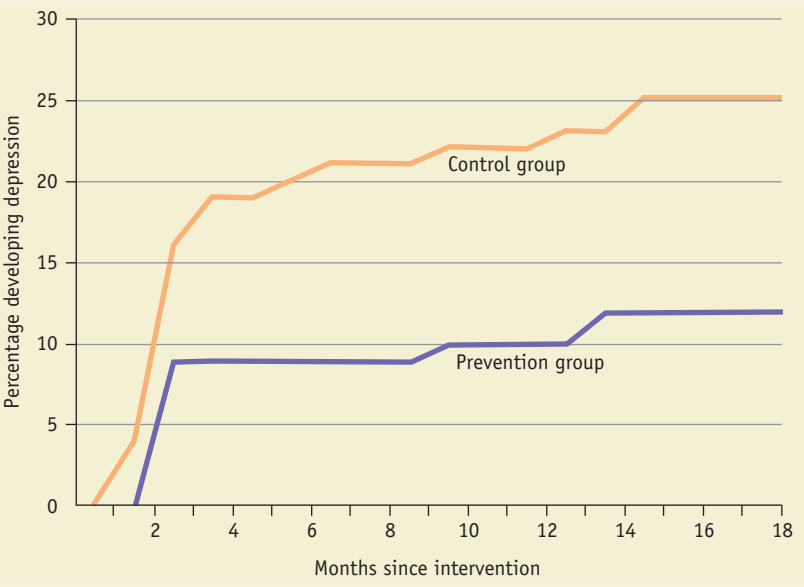
> At this point in my existence, I cannot imagine leading a normal life without both taking lithium and having had the benefits of psychotherapy. Lithium prevents my seductive but disastrous highs, diminishes my depressions, clears out the wool and webbing from my disordered thinking, slows me down, gentles me out, keeps me from ruining my career and relationships, keeps me out of a hospital, alive, and makes psychotherapy possible. But, ineffably, psychotherapy *heals*. It makes some sense of the confusion, reins in the terrifying thoughts and feelings, returns some control and hope and possibility of learning from it all. Pills cannot, do not, ease one back into reality; they only bring one back headlong, careening, and faster than can be endured at times. Psychotherapy is a sanctuary; it is a battleground; it is a place I have been psychotic, neurotic, elated, confused, and despairing beyond belief. But, always, it is where I have believed—or have learned to believe—that I might someday be able to contend with all of this.

Depression Prevention

Given the devastating effects depression can have on people's lives, an important goal for the future is to prevent depression in vulnerable people before it ever begins. Several studies using cognitive-behavioral and interpersonal therapy techniques have shown that community-based interventions can prevent first onsets of depression in people at high risk (Munoz et al., 2002). For example, cognitive-behavioral techniques can be used to prevent depression in low-income, minority people who are faced with chronic and overwhelming stressors (Munoz, 1997; Munoz et al., 1995).

Evidence that depression first arises in adolescence has led several researchers to focus on preventing depression in high-risk adolescents. In one study, adolescents at high risk to develop major depression because they already had mild to moderate symptoms of depression were randomly assigned to a cognitive-behavioral intervention or to a no-intervention control group. The students receiving the cognitive-behavioral intervention met for 15 sessions in small groups after school. They received therapy designed to help them overcome negative ways of thinking and to learn more effective coping strategies. Following the therapy, both the intervention group and the no-treatment control group were followed for up to 18 months. The children in the prevention group got some immediate benefit from this intervention; their levels of depressive symptoms declined over the course of the 15 sessions. The remarkable finding of this study was that the intervention seemed to reduce the risk for future depression in these children (Clarke et al., 1995) (see Figure 9.10). Over the year after the intervention ended, a relatively low percentage of the children who received it developed

FIGURE 9.10 **Effects of a Preventive Intervention.** In one study, adolescents undergoing 15 sessions of cognitive-behavioral therapy were less likely to develop major depression over the next 18 months than a control group who received no therapy.



Source: Clarke et al., 1995.

Studies have shown that group therapy for teenagers can prevent or reduce depression.

depression. In contrast, many of the children in the control group developed depression (for similar results, see Gillham et al., 1995; Jaycox et al., 1994). This study gives us hope that many vulnerable children can be spared from the debilitating effects of depressive episodes.

SUMMING UP

- Tricyclic antidepressants are effective in treating depression but have some side effects and can be dangerous in overdose.
- The monoamine oxidase inhibitors (MAOIs) also are effective treatments for depression but can interact with certain medications and foods.
- The selective serotonin reuptake inhibitors (SSRIs) are effective treatments for depression and have become popular because they are less dangerous and their side effects are more tolerable than are those of other drug treatments.
- Electroconvulsive therapy (ECT) involves inducing seizures in people with depression. It can be quite effective but is controversial.
- Lithium is useful in the treatment of mood disorders but requires careful monitoring to prevent dangerous side effects.
- Anticonvulsants, antipsychotics, and calcium channel blockers can help relieve mania.
- Behavioral treatment focuses on increasing positive reinforcers and decreasing aversive events by helping clients change their environments, learn social skills, and learn mood-management skills.
- Cognitive-behavioral treatment combines the techniques of behavior therapies with techniques to identify and challenge depressive thinking patterns.
- Psychodynamic therapies focus on uncovering unconscious hostility and fears of abandonment through the interpretation of transference, memories, and dreams.
- Interpersonal therapy seeks to identify and overcome problems with grief, role transitions, interpersonal role disputes, and deficits in interpersonal skills that contribute to depression.
- Cognitive-behavioral therapy, interpersonal therapy, and drug treatments seem to work equally well with most people with depression, and the combination of drug therapy and one of the psychotherapies may be the most effective.
- Some research suggests that interventions targeting high-risk groups can help prevent or delay first onsets of depression.

CHAPTER INTEGRATION

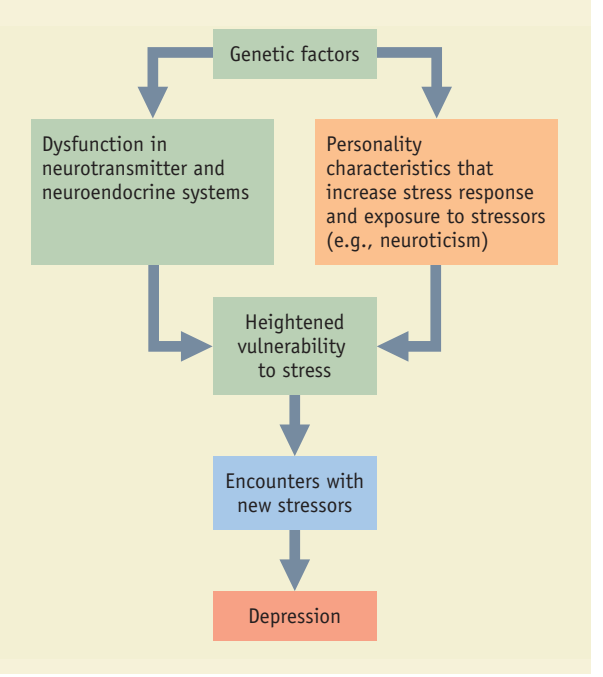
The mood disorders affect the whole person. Depression and mania involve changes in every aspect of functioning, including biology, cognitions, personality, social skills, and relationships. Some of these changes may be causes of the depression or mania, and some of them may be consequences of the depression or mania.

The fact that the mood disorders are phenomena of the whole person illustrates the intricate connections among the various aspects of functioning: biology, cognitions, personality, and social interactions. These areas of functioning are so intertwined that major changes in any one area will almost necessarily provoke changes in other areas. Many recent models of the mood disorders, particularly depression, suggest that most people who become depressed carry a vulnerability to depression for much of their lives. This may be a biological vulnerability, such as dysfunctions in neurotransmitter systems, or a psychological vulnerability, such as overdependence on others. It is not until these vulnerabilities interact with certain stressors that a full-blown depression is triggered, however.

Kendler and colleagues (Kendler, 1998; Kendler & Karkowski-Shuman, 1997) have suggested that, in major depression, genetic factors may influence vulnerability to depression by altering the individual's relationship to the environment, in addition to inducing biological abnormalities that directly cause depression (see Figure 9.11). First, genetic factors may increase the individual's biological sensitivity to stressors in the environment, by altering the neurotransmitter and neuroendocrine systems involved in the stress response. This makes it more likely that these individuals will react to a stressor with depression. In their large study of twins, they found statistical evidence that being at genetic risk for depression made twins more prone to depression in the face of negative life events. In twins with a low genetic risk for depression (e.g., a monozygotic twin whose cotwin had no history of depression), the probability of a depression, given exposure to a severe life event, was 6.2 percent. In twins with a high genetic risk for depression (a monozygotic twin whose cotwin had a history of depression), the probability of depression, given exposure to a severe life event, was 14.6 percent (Kendler & Karkowski-Shuman, 1997).

Second, genetic factors may influence the probability that individuals will select high- versus low-risk environments for the production of depression. People actively help create their environments by choosing which people they spend time with, where they live, the type of occupation they pursue, and so on. In their twin studies, Kendler and colleagues (1993) found, not surprisingly, that cotwins often shared the same life events, such as the death of a family member. This seems mostly likely due to environmental factors—specifically, having the same family members. But certain other stressors, including being robbed or assaulted or experiencing a major financial stressor, appeared to be influenced primarily by genetic factors. That is, similarities in the twins' environments could not account for their common risk of experiencing these events. In addition, these events did not seem to be solely the result of both twins' being depressed. Kendler and Karkowski-Shuman (1997) suggest that genetic factors may contribute to broad personality characteristics, such as neuroticism or impulsivity, which then lead to greater risk both for negative life events and for depression.

FIGURE 9.11 An Intergrative Model of Depression Proposed by Kendler and Colleagues



Fortunately, the interconnections among these areas of functioning may mean that improving functioning in one area can improve functioning in other areas. Improving people's biological functioning can improve their cognitive and social functioning and their personalities. Improving people's cognitive and social functioning can improve their biological functioning, and so on. Thus, although there may be many pathways into mood disorders (biological, psychological, and social), there may also be many pathways out of the mood disorders, particularly depression.

Extraordinary People: Follow-Up

We began this chapter noting the courage of one prominent theorist and researcher of abnormality, Kay Redfield Jamison, in publishing her autobiography describing her experiences with a serious mental disorder. Although many psychiatrists and psychologists have personal histories of mental disorder, they are often reluctant to let it be known, because they fear that it will bias others' attitudes toward their ideas or will affect their professional licenses or their privileges to admit patients to hospitals. They are also concerned, as was Jamison, that their revelation of mental illness would have repercussions for their families.

Why did Jamison feel the need to go public with her illness? Jamison's explanation of her decision to reveal her disorder indicates her personal triumph over fears of others' opinions and her dedication to changing cultural attitudes toward mental disorders (Jamison, 1995, pp. 7–8):

> I have no idea what the long-term effects of discussing such issues so openly will be on my personal and professional life, but whatever the consequences, they are bound to be better than continuing to be silent. I am tired of hiding, tired of misspent and knotted energies, tired of the hypocrisy, and tired of acting as though I have something to hide. One is what one is, and the dishonesty of hiding behind a degree, or a title, or any manner and collection of words, is still exactly that: dishonest. Necessary, perhaps, but dishonest. I continue to have concerns about my decision to be public about my illness, but one of the advantages of having had manic-depressive illness for more than thirty years is that very little seems insurmountably difficult. Much like crossing the Bay Bridge when there is a storm over the Chesapeake, one may be terrified to go forward, but there is no question of going back. I find myself somewhat inevitably taking a certain solace in Robert Lowell's essential question, *Yet why not say what happened?*

We can only hope that, as the public understands more about the causes of mental disorders, fewer people with these disorders will have to fear the consequences of letting it be known that they suffer. Kay Redfield Jamison, through her courageous decision to talk about her disorder and her eloquent and thoughtful writing and speaking on mental disorders, has moved us a bit closer to the fulfillment of that hope.

Chapter Summary

- There are two general categories of mood disorder: unipolar depression and bipolar disorder. People with unipolar depression experience only the symptoms of depression (sad mood, loss of interest, disruption in sleep and appetite, motor retardation or agitation, loss of energy, worthlessness and guilt, suicidality). (Review Table 9.1.) People with bipolar disorder experience both depression and mania (elated or agitated mood, grandiosity, little need for sleep, racing thoughts and speech, increase in goals and dangerous behavior). (Review Table 9.3.)
- Within unipolar depression, the two major diagnostic categories are major depression and dysthymic disorder. In addition, there are several subtypes of major depression: with melancholic features, with psychotic features, with catatonic features, with atypical features, with postpartum onset, and with seasonal pattern. (Review Table 9.2.)
- Depression is one of the most common disorders, but there are substantial age, gender, and cross-cultural differences in depression. Bipolar disorder is much less common than the depressive disorders. It tends to be a lifelong problem. The length of individual episodes of bipolar disorder varies dramatically from one person to the next and over the life course, as in depression. The expression of mania may depend on cultural norms.
- Genetic factors probably play a role in determining vulnerability to the mood disorders, especially

bipolar disorder. (Review Table 9.5.) Disordered genes may lead to dysfunction in the monoamine neurotransmitter systems. The neurotransmitters norepinephrine, serotonin, and dopamine have been implicated in the mood disorders. In addition, neuroimaging studies show abnormal structure or activity in several areas of the brain, including the prefrontal cortex, hippocampus, anterior cingulate cortex, and amygdala. There is evidence that people with depression have chronic hyperactivity in the hypothalamic-pituitary-adrenal axis, which may make them more susceptible to stress.

- Behavioral theories of depression suggest that people with much stress in their lives may have too low a rate of reinforcement and too high a rate of punishment, which then leads to depression. (Review Table 9.6.) Stressful events can also lead to learned helplessness—the belief that nothing one does can control one's environment—which is linked to depression. Most people who are faced with stressful events do not become depressed, however.
- The cognitive theories of depression argue that the ways people interpret the events in their lives determine whether they become depressed. (Review Table 9.7.) Some evidence suggests that people with depression are actually quite realistic in their negative views of life and that nondepressed people are unrealistically optimistic about life. People who ruminate in response to distress are more prone to depression.
- Interpersonal theories of depression suggest that poor attachment relationships early in life can lead children to develop expectations that they must be or do certain things in order to win the approval of others, which puts them at risk for depression. (Review Table 9.6.)
- Psychodynamic theories of depression suggest that people with depression have chronic patterns of negative relationships and tend to internalize their hostility against others.
- Social theories attribute depression to the effects of low social status, as well as changes in the social conditions that different generations face. In addition, there appear to be differences across cultures in how depression is manifested.
- Most of the biological therapies for the mood disorders are drug therapies. (Review Table 9.8.) Three classes of drugs are commonly used to treat depression: tricyclic antidepressants, monoamine oxidase inhibitors, and selective serotonin reuptake inhibitors. Each of these is highly effective in treating depression, but each has significant side effects. Electroconvulsive therapy is used to treat severe depressions, particularly those that do not respond to drugs.
- Lithium is the most effective drug for the treatment of bipolar disorder. It has a number of side effects, including nausea, vomiting, diarrhea, tremors, twitches, kidney dysfunction, and birth defects. Alternatives to lithium include anticonvulsant drugs, antipsychotic drugs, and calcium channel blockers.
- Behavior therapies focus on increasing positive reinforcers and decreasing negative events by building social skills and teaching clients how to engage in pleasant activities and cope with their moods. Cognitive-behavioral therapies focus on helping people with depression develop more adaptive ways of thinking and are very effective in treating depression. Interpersonal therapy helps people with depression identify and change their patterns in relationships and is highly effective in treating depression. Psychodynamic therapy helps people with depression uncover unconscious hostility and fears of abandonment. (Review Table 9.9.)
- Direct comparisons of drug therapies with cognitive-behavioral and interpersonal therapies show that they tend to be equally effective in the treatment of depression. The combination of drug therapy and psychotherapy may be more effective than either treatment alone for people with chronic depression.
- Effective prevention programs have been designed to reduce the risk for onset of major depression in high-risk groups.

MindMap CD-ROM

The following resources on the MindMap CD-ROM that came with this text will help you to master the content of this chapter and prepare for tests:

- Videos: Major Depression; Dysthymia; Bipolar Disorder
- Chapter Timeline
- Chapter Quiz

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