ical diagnostic practices vary from country to country, producing the different rates. A cross-national study conducted by diagnosticians trained to follow the same procedures in each country is needed.

Etiology of Somatoform Disorders Other Than Conversion Disorder

Now that we've considered conversion disorder in depth, let's look at various perspectives on the etiology of the other somatoform disorders. We begin by briefly discussing genetic research on somatoform disorders and neurobiological risk factors for body dysmorphic disorder. Then we consider cognitive behavioral models used to explain a range of somatoform disorders, since these reflect the dominant perspective on somatoform disorders other than conversion disorder.

Genetic Research on Somatoform Disorders Torgersen (1986) reported the results of a twin study of somatoform disorders that included 10 cases of conversion disorder, 12 of somatization disorder, and 7 of pain disorder. No co-twin had the same diagnosis as his or her proband! Genetic factors, then, from the studies so far, seem to be of no importance for most somatoform disorders, with the possible exception of body dysmorphic disorder, discussed next.

Neurobiology of Body Dysmorphic Disorder Above, we described some parallels in the symptoms of body dysmorphic disorder and obsessive-compulsive disorder. These two disorders are often comorbid as well. Some researchers have suggested that body dysmorphic disorder may be a special form of obsessive-compulsive disorder. For example, the rates of obsessive-compulsive disorder are higher than average in the family members of people with body dysmorphic disorder (Gustad & Phillips, 2003), and both disorders are associated with changes in the volume of the caudate nucleus region of the brain (Rauch et al., 2003). Thus, some of the neurobiological risk factors for obsessive-compulsive disorder may be involved in the genesis of body dysmorphic disorder.

Cognitive Behavioral Models Cognitive behavioral models suggest that a number of different mechanisms contribute to somatoform disorders. Figure 6.3 illustrates one model of how these mechanisms could fit together for somatoform disorders other than body dysmorphic disorder. The process is believed to start with a physiological symptom, as a result of either a medical illness or a nonpathological change in physiological functioning (e.g., increased heart rate from effort). For body dysmorphic disorder, the process is thought to begin when a person notices a physical anomaly. Because physical symptoms are so common, these models tend to focus on cognitive and behavioral processes that amplify responses to physical symptoms.

Everybody experiences physical sensations and pays a certain amount of attention to their appearance, but people who are prone to somatoform disorders appear to have a cognitive style characterized by paying much greater attention to these things. In one study, researchers used a version of the emotion Stroop task (see p. 46) to examine attention to cues of physical health problems among patients with somatoform disorder, depression, or panic disorder (Lim & Kim, 2005). Patients with somatoform disorder attended more to words that were related to physical health, whereas other patients did not. Several researchers have replicated these findings (cf. Lecci & Cohen, 2007). Hence, people with somatoform disorder may automatically focus on cues of physical health problems.

Once people with somatoform disorder notice physical symptoms, they also seem to make more negative attributions about them. (An attribution is a person's idea about why something is happening.) The specific attributions will vary with the somatoform disorder. For example, a woman with hypochondriasis might interpret a red blotch as a sign of skin cancer (Marcus et al., 2007). Or a man with body dysmorphic disorder might focus

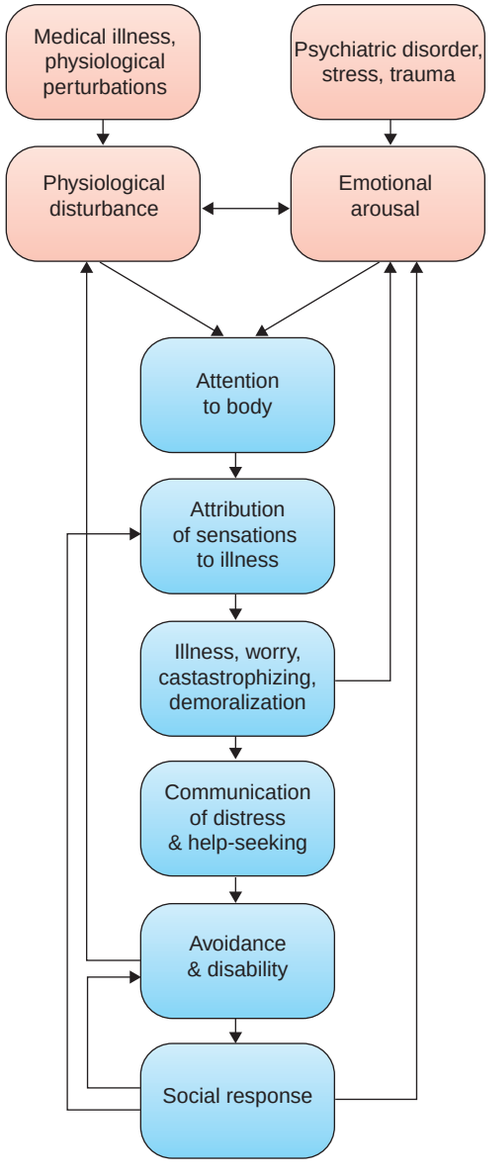


Figure 6.3 Mechanisms involved in somatoform disorders. From Looper and Kirmayer (2002).

obsessively on unnoticeable facial wrinkles, convinced that they make him unlovable. A person with somatoform disorder might overestimate the odds that a symptom is a sign of a disease (Rief et al., 2006). The exact form of the cognitive bias may vary, but most somatoform disorders seem to be characterized by worry about health and a tendency to catastrophize the symptoms (i.e., to interpret them in the worst possible way). Once these negative thoughts begin, elevated anxiety and cortisol reactivity may exacerbate somatic symptoms and distress over those symptoms (Rief & Auer, 2001).

In Chapter 5, we described a very similar cognitive process as part of panic disorder (see p. 138). That is, people with panic disorder are likely to overreact to physiological symptoms. In panic disorder, the person often believes that the symptoms are a sign of an immediate threat (e.g., a heart attack), whereas in hypochondriasis, the person believes the symptoms are a sign of an underlying long-term disease (e.g., cancer or AIDS). The types of physical cues that a person focuses on also differ for people with panic disorder compared to hypochondriasis. The person with panic disorder often focuses on symptoms that will actually become worse as they become more anxious—for example, a fast heart rate, shortness of breath, or sweaty palms. In contrast, a patient with hypochondriasis cannot, for example, increase the size of a spot on the skin by misconstruing it as cancer.

The tendency to believe that one is physically ill may have evolved from early experiences of medical symptoms or from family attitudes that became internalized. Consistent with the idea of developmental influences on cognitive biases, people with somatoform disorders report that, as children, they often missed school because of illness (Barsky et al., 1995).

Fear that a bodily sensation signifies illness (or one's physical appearance signifies ugliness) is likely to have two behavioral consequences. First, the person may assume the role of being sick and avoid work and social tasks, and this can intensify symptoms by limiting exercise and other healthy behaviors. Second, the person may seek reassurance, both from doctors and from family members, and this help-seeking behavior may be reinforced if it results in the person getting attention or sympathy. Often, people with these disorders tend to have trouble eliciting socially reinforcing interactions in other ways. For example, people with the symptoms of somatoform disorders often have trouble identifying their emotions and describing them directly (Bankier, Aigner, & Bach, 2001), so they may find attention and sympathy for health concerns particularly reinforcing. Beyond the attention, people may receive other types of behavioral reinforcers for somatoform symptoms—for example, people receive disability payments based on the amount that symptoms interfere with their daily activities.

Check Your Knowledge 6.4

True or false?

1. Conversion disorder is highly heritable.
2. The two-stage psychodynamic model of conversion disorder emphasizes unconscious perceptions and motivation for having symptoms.
3. The risk factors for pain disorder may overlap substantially with the risk factors for obsessive-compulsive disorder.

Treatment of Somatoform Disorders

One of the major obstacles to treatment is that most people with somatoform disorders do not want to consult mental health professionals. They resent referrals from their physician to "shrinks" because they interpret such a referral as a sign that the doctor thinks the illness is "all in their head." Innovative programs in which general practitioners help people think less negatively about symptoms, or insurance companies work with comprehensive teams to offer psychological and medical care, are likely to be needed.

Psychodynamic treatment has been found to be effective in alleviating the symptoms of somatoform disorders in one study (Junkert-Tress et al., 2001) and the symptoms of pain disorder in one other study (Monsen & Monsen, 2000), but there have been few other controlled studies comparing psychodynamic treatment to other treatments. In this section, therefore, we focus mainly on cognitive behavioral treatments and on the use of antidepressant medications.

Cognitive behavioral therapists have applied many different techniques in trying to help people with somatoform disorders. As illustrated with the clinical case of Louis described earlier, these include helping people (1) identify and change the emotions that trigger their somatic concerns, (2) change their cognitions regarding their somatic symptoms, and (3) change their behaviors so they stop playing the role of a sick person and gain more reinforcement for engaging in other types of social interactions (Looper & Kirmayer, 2002).

The negative emotions that accompany anxiety and depressive disorders often trigger physiological symptoms and intensify the symptoms of somatoform disorders (Simon, Goreje, & Fullerton, 2001). Indeed, as shown in Chapters 5 and 8, concern about physical health is common among people suffering from anxiety or depression. It should therefore come as no surprise that treating anxiety and depression often reduces somatoform symptoms (Phillips, Lì, Zhang, 2002; Smith, 1992).

Cognitive strategies involve training people to pay less attention to their body. Alternatively, cognitive strategies might help people identify and challenge negative thoughts about their bodies.

Behavioral techniques might involve helping people resume healthy activities and decrease their reliance on playing the sick role. For example, a therapist might use operant conditioning approaches with family or friends to reduce the amount of attention they give the person who is displaying somatic symptoms, or they might help the person rebuild a lifestyle that has been damaged by too much focus on illness-related concerns.

Doctors' responses may help shape whether initial concerns about symptoms intensify or diminish. For example, in one study, patients with medically unexplained gastrointestinal symptoms were randomly assigned to receive high or low levels of warmth, attention, and reassurance from doctors. Those who received high levels of support showed more improvement in symptoms and quality of life over the next six weeks compared to those who received low levels of support (Kaptchuk et al., 2008). Hence, a new focus of research is on how to help primary care doctors best respond to somatoform disorders. With these general principles in mind, we turn to treatments that have been tested for specific somatoform disorders.

Pain Disorder There is evidence from a number of double-blind experiments that low doses of some antidepressant drugs, most especially imipramine (Tofranil), are superior to a placebo in reducing chronic pain and distress (Fishbain et al., 2000). Interestingly, these antidepressants reduce pain even when, in the low dosages given, they don't alleviate the associated depression (Simon, 1998). Increasing attention has been given to the high rates of addiction to opioid medications among people treated for pain (Streltzer & Johansen, 2006).

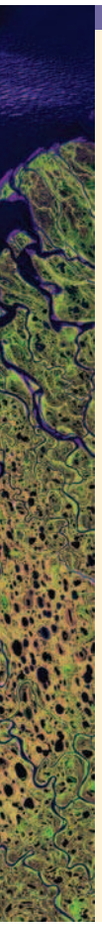
Current thinking suggests that, in conducting psychotherapy, it is fruitless to make a sharp distinction between psychogenic pain and medically caused pain, such as pain resulting from injury to muscle tissue. Typically, clinicians assume that pain has physical and psychological components. Effective treatments for pain disorder tend to include the following ingredients:

- Validating that the pain is real, not just "in the patient's head"
- Rewarding the person for less focus on pain and more focus on life
- Relaxation training

In general, it is advisable to focus less on what the patient cannot do because of pain and more on teaching the patient how to deal with stress, encouraging the patient to engage in more activities, and helping the patient gain a greater sense of control. The cognitive behavioral techniques used to do this are described in greater detail in Focus on Discovery 6.4.

Body Dysmorphic Disorder At least four trials have been conducted in which people with body dysmorphic disorder were randomly assigned to receive either a cognitive behavioral treatment or a control treatment. In each study, cognitive behavioral treatment was shown to produce a major decrease in symptoms of the disorder (Looper & Kirmayer, 2002).

Given the parallels between body dysmorphic disorder and obsessive-compulsive disorder, cognitive behavioral treatment for obsessive-compulsive disorder—exposure and response prevention (ERP, see p. 149)—has been modified to address the symptoms of body dysmorphic disorder. For example, for response prevention, people may be asked to avoid checking their appearance in mirrors and other reflective surfaces. Cognitive behavioral approaches that include response prevention have been found to reduce symptoms of body dysmorphic disorder (Veale et al., 1996).



FOCUS ON DISCOVERY 6.4

The Management of Pain

There is no one-to-one relationship between a stimulus that is capable of triggering pain, referred to as nociceptive stimulation, and the actual sensation of pain. Soldiers in combat can be wounded by a bullet and yet be so involved in their efforts to survive that they do not feel any pain until later. This well-known fact hints at ways of controlling pain: if one is distracted from a nociceptive stimulus, one may not experience as much pain as when one attends to the stimulation (Turk, 2001). It is also well-known that pain can be increased by anxiety, depression, and stress hormones (Gatchel et al., 2007).

Brain regions activated by physical pain overlap with the brain regions activated by psychological pain (such as the pain of remembering a relationship loss). That is, both types of pain increase activity in the region of the brain called the medial anterior cingulate. Researchers have begun to examine whether persons with unexplained physical pain demonstrate overreactivity in the medial anterior cingulate to physical sensations. Indeed, people with gastrointestinal pain that is unexplained by medical causes show more activity of the medial anterior cingulate in response to gastric sensations than do people with gastrointestinal pain due to medical illnesses (Mayer et al., 2005). One of the fascinating aspects of this finding is that attention and mood have strong influences on the activity of the medial anterior cingulate.

Because it is so hard to regulate pain, researchers have drawn on basic research to develop treatment programs that teach people specialized skills for managing pain and its consequences for that their lifestyle. These programs typically begin by providing information about the nature of pain, including the fact that being in a negative mood can make the pain worse (Morley, 1997). Often, treatment programs include training in the use of distraction and cognitive techniques for controlling pain, as illustrated by the following:

> *The patient may be encouraged to alter the focus of their attention to the pain without switching attention directly away from the pain. In this instance, the subject may be asked to focus on the sensory qualities of the pain and transform it to a less threatening quality. For example, a young man with a severe "shooting" pain was able to reinterpret the sensory quality into an image which included him shooting a goal in a soccer match. As a result of this transformation, the impact of the pain was greatly reduced. (Morley, 1997, p. 236)*

Treatment programs also tend to distinguish between pain per se—that is, the perception of nociceptive stimulation—and suffering and pain behaviors. Suffering refers to the emotional response to nociception. Pain behaviors refer to observable behaviors associated with pain or suffering; examples include moaning, clenching teeth, irritability, and avoidance of activity (Turk, Wack, & Kerns, 1985). Pain programs often focus on reducing suffering and pain behaviors. The emphasis is on restoring a lifestyle rather than allowing pain to destroy a person's lifestyle. The goal is increased activity and function, which can sometimes even reduce the actual experience of pain.

Hypochondriasis Cognitive behavioral treatment for people with hypochondriasis aims at reducing their excessive attention to bodily sensations, challenging their negative thoughts about those sensations, and discouraging them from seeking reassurance from doctors (Warwick & Salkovskis, 2001). The behavioral components of the treatment focus on keeping people from repetitively checking on their health, increasing their engagement in activities, and decreasing their focus on seeking treatment. Cognitive behavioral approaches have proven effective in reducing health concerns, symptoms of depression and anxiety, and health care utilization compared to no treatment conditions (Thomson & Page, 2007). For example, in one study, patients who received cognitive behavioral treatment reported being much less concerned about physical symptoms when they were present (Barksy & Ahern, 2004). In one study, cognitive behavioral treatment was as effective as an antidepressant in reducing the health anxiety symptoms of hypochondriasis (Greeven et al., 2007).

Somatization Disorder In a widely accepted approach to somatization disorder, the physician does not dispute the validity of the person's physical complaints but minimizes the use of diagnostic tests and medications, as well as maintaining contact with the person regardless of whether he or she is complaining of illness (Monson & Smith, 1983). A study of this approach found that it resulted in less frequent use of health care services (Rost, Kashner, & Smith, 1994).

Several studies have found that cognitive behavioral treatment can reduce somatic symptoms compared to control conditions, although effects have tended to be small (Deary et al., 2007). Available cognitive behavioral treatments have included a range of specific techniques. For Maria described on page 172, cognitive treatment could help her monitor her negative cognitions about her physical symptoms and help her evaluate the accuracy of those cognitions. She could also practice focusing less on physical symptoms. One goal of treatment is to help the person reduce the anxiety and depression that may underlie unexplained somatic symptoms. Techniques such as relaxation training and various forms of cognitive treatment have proven useful in this regard (Payne & Blanchard, 1995).

Cognitive behavioral therapists often focus on addressing the social concerns associated with somatization disorder. Maria, the woman described earlier, revealed that she was extremely anxious about her shaky marriage and about situations in which other people might judge her. Techniques such as exposure and cognitive restructuring could address her interpersonal fears, which might help lessen her somatic complaints. Assertion training and social skills training—for example, coaching Maria in effective ways to approach and talk to people, to maintain eye contact, to give compliments, to accept criticism, and to make requests—could be useful in helping her to develop healthier interpersonal interactions that do not focus on her physical illness.

Behavioral approaches could help change her reliance on playing the role of a sick person. If the people who live with Maria have adjusted to her illness by reinforcing her avoidance of normal adult responsibilities, family therapy might help. Maria and the members of her family might be able to change relationships to support her movement away from a focus on physical complaints.

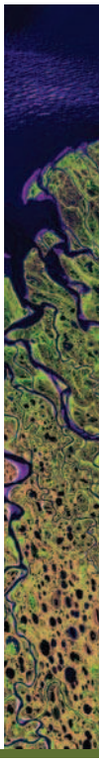
Conversion Disorder There have been no psychological treatments that have reduced symptoms of conversion disorder in controlled trials. Traditional long-term psychoanalysis, psychodynamic psychotherapy, and hypnosis, have not been demonstrated to be useful with conversion disorder (Kroenke, 2007; Simon, 1998). Case studies suggest that it is usually not a good idea to try to convince people with conversion disorder that their symptoms are related to psychological factors; rather, it is better to offer gentle support. Case studies also have suggested that reinforcing a person for being able to improve his or her functioning can be helpful.

Quick Summary

The somatoform disorders are defined by physical symptoms that are believed to be related to psychological causes. Somatoform disorders include body dysmorphic disorder, pain disorder, hypochondriasis, somatization, and conversion disorder. In pain disorder, psychological factors are believed to create or intensify pain. In body dysmorphic disorder, a person experiences distress and impairment over imagined ugliness or physical flaws. Hypochondriasis is characterized by belief in a severe disease, despite evidence to the contrary. Conversion disorder is characterized by sensory and motor dysfunctions suggesting neurological impairments that cannot be explained by medical tests. In somatization disorder, multiple physical complaints, not adequately explained by physical disorder or injury, eventuate in frequent visits to physicians, hospitalization, and even unnecessary surgery. The symptoms of somatoform disorders may arise suddenly in stressful situations.

Psychodynamic theories of conversion disorder have focused on the idea that people can be unaware of their perceptions and abilities and that some people may be motivated to have symptoms. Body dysmorphic disorder is often comorbid with obsessive-compulsive disorder, and these two disorders may be related to some of the same genetic and neurobiological risk factors. Cognitive behavioral models of somatoform disorders focus on cognitive beliefs that promote negative responses to bodily sensations and appearance. Little systematic research is available on the etiology of somatoform disorders.

One problem in treating somatoform disorders is that few people will want to see a mental health provider for their physical symptoms. Nonetheless, cognitive behavioral techniques have been found to be helpful, including strategies to help people address emotions more directly, change their cognitive responses to physical symptoms, and shift from assuming the sick role. Beyond these cognitive behavioral approaches, some other techniques have been found to be helpful for specific somatoform disorders. For pain disorder, cognitive behavioral theropy (CBT) and low levels of antidepressant medication may be helpful. For body dysmorphic disorder, CBT techniques that are highly parallel with those used to treat obsessive-compulsive disorder are helpful. For somatization disorder, physicians can reduce health care utilization by minimizing the use of diagnostic tests.



Summary

Dissociative Disorders

- Dissociative disorders are defined by disruptions of consciousness, memory, and identity.
- The dissociative disorders include dissociative amnesia, dissociative fugue, depersonalization disorder, and dissociative identity disorder.
- Most of the writing about the causes of dissociative disorders focuses on dissociative identity disorder. People with dissociative identity disorder very often report severe physical or sexual abuse during childhood. One model, the posttraumatic model, suggests that extensive reliance on dissociation to fend off overwhelming feelings from abuse puts people at risk for developing dissociative identity disorder. The sociocognitive model, though, raises the question of whether these symptoms are elicited by treatment. Proponents of the sociocognitive model point out that abuse in childhood may result in heightened suggestibility, that some therapists use strategies that suggest such symptoms to people, and that most people do not recognize the presence of any alters until after they see a therapist.
- Regardless of theoretical orientation, all clinicians focus their treatment efforts on helping a clients cope with anxiety, face fears more directly, and operate in a manner that integrates their memory and consciousness.
- Psychodynamic treatment is perhaps the most commonly used treatment for dissociative disorders, but some of the techniques involved, such as hypnosis and age regression, may make symptoms worse.

Somatoform Disorders

- In somatoform disorder, biological explanations for physical symptoms cannot be found. The major somatoform diagnoses include pain disorder, body dysmorphic disorder, hypochondriasis, conversion disorder, and somatization disorder.
- Psychodynamic theory proposes that in conversion disorder, repressed impulses are converted into physical symptoms. Sackeim has proposed a two-stage model of conversion disorder that focuses on lack of conscious awareness of perceptions as well as motivation for symptoms. Cultural factors that influence how people think about and express distress may shape the rates of disorder as well.
- Data regarding other somatoform disorder are less available. Most somatoform disorders do not appear to be inherited. There may be some neurobiological overlap between body dysmorphic disorder and obsessive-compulsive disorder. Cognitive behavioral models emphasize that some people may have a cognitive style that leads them to be overly attentive to physical concerns and to make negative attributions about these symptoms and their implications. The form of the cognitive bias may differ for the various somatoform disorders. Behavioral reinforcement may maintain help-seeking behavior.
- Antidepressants have been shown to be effective for some somatoform disorders. Cognitive behavioral treatments, which have received a great deal of support, try to address the maladaptively negative cognitions about physical symptoms, to reduce anxiety, and to reinforce behavior that is not consistent with the sick role.

Answers to Check Your Knowledge Questions

6.1 1. a, b; 2. c; 3. b
6.2 1. T; 2. T; 3. T
6.3 1. b; 2. e; 3. c; 4. d
6.4 1. F; 2. T; 3. F

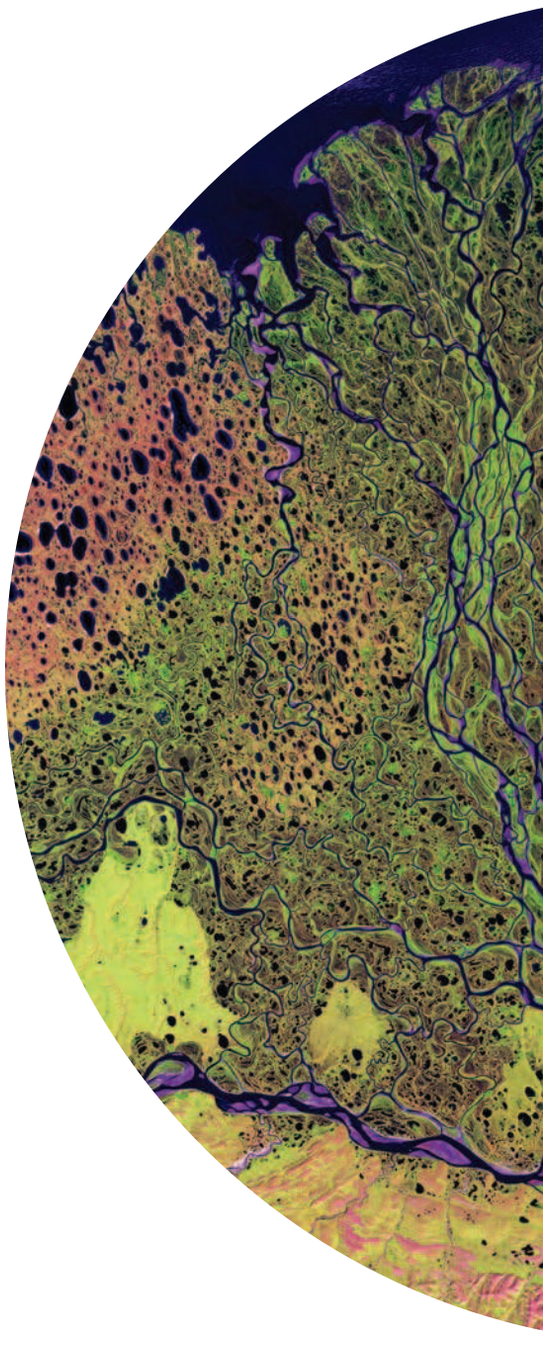
Key Terms

anesthesia
blindsight
body dysmorphic disorder
conversion disorder
depersonalization disorder
dissociation
dissociative amnesia
dissociative disorders
dissociative fugue
dissociative identity disorder (DID)
explicit memory
factitious disorder
hypochondriasis
implicit memory
la belle indifférence
malingering
pain disorder
posttraumatic model (of DID)
sociocognitive model (of DID)
somatization disorder
somatoform disorders

7 Stress and Health

LEARNING GOALS

1. Be able to distinguish the definitions of *stress, coping,* and *social support.*
2. Be able to understand the theories of the stress–illness link as well as the concept of allostatic load and the basic components of the immune system.
3. Be able to describe how psychological factors impact cardiovascular disorders, asthma, and AIDS.
4. Be able to delineate the importance of gender, socioeconomic status, and ethnicity in health.
5. Be able to describe the major psychological treatments for psychological factors affecting medical conditions, including psychological approaches to reduce anger, anxiety, and depression as well as the concept of stress management.



Clinical Case: Mark

Mark Howard was 38. After earning an M.B.A., he had joined the marketing division of a large corporation and had worked his way up in the company. His talent and long hours of work had recently culminated in a promotion to head of his division. The promotion had left him with mixed feelings. On the one hand, it was what he had been working so hard to achieve; but on the other hand, he had never been comfortable giving orders to others and he especially dreaded the staff meetings he would have to run.

Soon after the promotion, during a routine checkup, Mark's physician discovered that Mark's blood pressure had moved into the borderline hypertension range, around 150 over 100. Before implementing any treatment, the physician asked Mark to wear an ambulatory monitor for a few days so that his blood pressure could be assessed as he went about his usual routine. The device was programmed to take blood pressure readings 20 times a day.

On the first day of monitoring, Mark had a staff meeting scheduled for 10 o'clock. While he was laying out the marketing plans for a new product, the cuff inflated to take his blood pressure. A couple of minutes later, he checked the reading. It was 195 over 140—not a borderline reading, but seriously high blood pressure. The next day he signed up for a yoga class and consulted a therapist for help with stress management. He also went to a trainer to help redesign his exercise plan and diet. He hoped that with these behavioral changes, he could continue in his managerial role and reduce the stress associated with the position.

DSM-IV-TR Criteria for Psychological Factors Affecting Medical Conditions

- A medical illness is present.
- Psychological factors have influenced the course of the condition, interfered with treatment, increased health risks, or exacerbated symptoms.

Experiencing major life events such as starting school is associated with increased risk of illness. (Will McIntyre/Photo Researchers.)

A NUMBER OF DISORDERS characterized by genuine physical symptoms are worsened by stress. These disorders include those that we focus on in this chapter—cardiovascular disorders (including essential hypertension and coronary heart disease), asthma, and AIDS—as well as other, perhaps less serious but extremely common conditions such as headache and gastritis.

DSM-IV-TR approaches this topic under the rubric **psychological factors affecting medical conditions**, in the broad section "Other Conditions That May Be a Focus of Clinical Attention." The psychological factors that may be involved include Axis I and II disorders; personality traits, such as neuroticism; coping styles, expression and experience of emotions such as anger; and behavioral issues, such as failing to exercise regularly.

The many demonstrations of the pervasive role of psychological factors in health form the basis for the fields of **behavioral medicine** and **health psychology**. Since the 1970s these fields have dealt with the role of psychological factors in all facets of health and illness. Beyond examining the role of stress in the exacerbation or maintenance of illness, researchers in these fields study psychological treatments (e.g., stress management) and the health care system itself (e.g., how better to deliver services to underserved populations) (Appel et al., 1997; Stone, 1982).

Prevention is also a major focus of health psychology. As the twentieth century progressed and infectious diseases were brought under better control, people were dying more often from such illnesses as coronary heart disease (CHD). The causes of CHD involve behavior—people's lifestyles—such as smoking, eating too much, and excessive alcohol use. Thus, it is believed that changing unhealthy lifestyles can prevent many cases of CHD. Health psychologists are at the forefront of these preventative efforts, some of which we describe later in this chapter.

Health psychology and behavioral medicine are not restricted to a set of techniques or to particular principles of changing behavior. Clinicians in the field employ a wide variety of techniques—from contingency management, to stress reduction, to cognitive behavioral approaches—all of which share the goal of altering unhealthy living habits, distressed psychological states, and aberrant physiological processes in order to bring about health benefits.

We begin our discussion by reviewing general findings on the relationship among stress, health, and illness. Then we turn to an in-depth examination of three disorders—cardiovascular disorders, asthma, and AIDS. Next we look at the relationships between health and gender, socioeconomic status, and ethnicity. Finally, we consider various approaches to the treatment of psychological factors affecting medical conditions.

What Is Stress?

To understand the role of stress, we must first be able to define and measure it. Neither task is simple, as we discussed in Chapter 3. Stress has been defined in many ways. Perhaps one of the more influential antecedents to our current conceptualizations of stress was the work by the physician Hans Selye. He introduced the term *general adaptation syndrome* (GAS) to describe the biological response to sustained and high levels of stress. In Selye's model there are three phases of the response (see Figure 7.1):

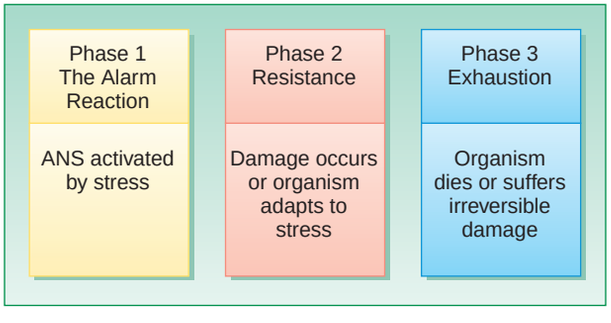


Figure 7.1 Selye's general adaptation syndrome.

1. During the first phase, the alarm reaction, the autonomic nervous system is activated by the stress.
2. During the second phase, resistance, the organism tries to adapt to the stress through available coping mechanisms.
3. If the stressor persists or the organism is unable to adapt effectively, the third phase, exhaustion, follows, and the organism dies or suffers irreversible damage (Selye, 1950).

In Selye's syndrome, the emphasis was on the body's response, not the environmental events that trigger that response. Psychological researchers later broadened Selye's concept to account for the diverse stress responses that people exhibited, including emotional upset, deterioration of performance, or physiological changes such as increases in the levels of certain hormones. The problem with these response-focused definitions of stress is that the criteria are not clear-cut. Physiological changes in the body can occur in response to a number of things that we would not consider stressful (e.g., anticipating a pleasurable event).

Other researchers defined stress as a stimulus, often referred to as a stressor, rather than a response, and identified stress with a long list of environmental conditions, such as electric shock, boredom, catastrophic life events, daily hassles, and sleep deprivation. Stimuli that are considered stressors can be major (the death of a loved one), minor (daily hassles, such as being stuck in traffic), acute (failing an exam), or chronic (a persistently unpleasant work environment). For the most part, they are experiences that people regard as unpleasant, but they can also be pleasant events.

Like response-based definitions of stress, stimulus-based definitions present problems. It is important to acknowledge, that people vary widely in how they respond to life's challenges. A given event does not elicit the same amount of stress in everyone. For example, a family that has lost its home in a flood but has money enough to rebuild and strong social support from a network of friends nearby will experience less stress from this event than will a family that has neither adequate money to rebuild nor a network of friends to provide social support.

As a way of addressing the limitations associated with defining stress as either a response or a stimulus, researchers have emphasized that how we perceive or *appraise* the environment determines whether a stressor is present. Stress is perhaps most completely conceptualized as the subjective experience of distress in response to perceived environmental problems. A final exam that is merely challenging to some students may be highly stressful to others who do not feel prepared to take it (whether their concerns are realistic or not).

Daily hassles can be upsetting and increase risk for illness. (Herve Donnezan Photo Researchers.)

Studies Using the Assessment of Daily Experience (ADE)

As discussed in Chapter 3, one of the currently effective methods for measuring stress and its relationship to health is the Assessment of Daily Experience (ADE). Recall that the ADE does not rely on retrospective reports as much as other measures of stress do. Studies using the ADE ask people for responses at the end of the day for several days.

Researchers used the ADE to study the relationship between events in daily life and the onset of respiratory infection (Stone, Reed, & Neale, 1987). After reviewing the data, the researchers identified 30 people who had experienced episodes of respiratory infection during the assessment period. Next, they examined the daily frequency of undesirable and desirable events during a period of 1 to 10 days before the start of an episode. For each person, they also selected a set of control days without an episode, matching that set to the days examined before the start of an episode, to control for the higher ratio of desirable versus undesirable events typically reported on weekends. Results of this analysis are shown in Figure 7.2 and Figure 7.3. As expected, prior to the onset of illness, there were significant decreases in the number of desirable events and significant increases in the number of undesirable events.

These results, which have been replicated (Evans & Edgerton, 1990), were the first to show a relationship between life events and health with both factors measured daily over a period of several days. By carefully controlling for relevant factors, this study lets us come much closer to asserting with confidence that negative life events can make people more vulnerable to episodes of infectious illness.

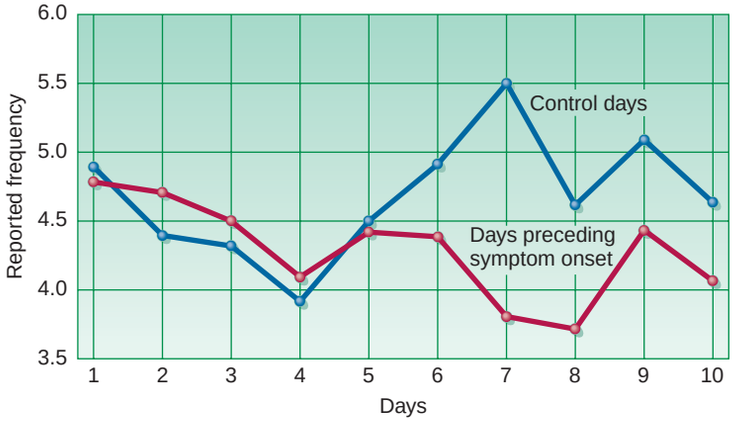


Figure 7.2 Mean number of desirable events in the 10 days preceding an episode of respiratory infection. After Stone et al. (1987).

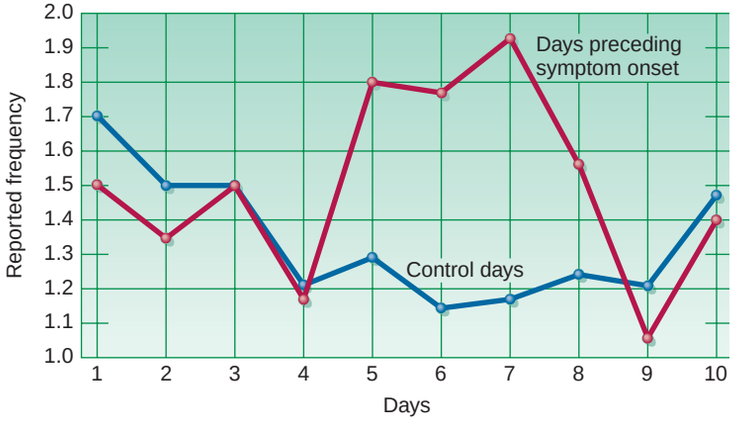


Figure 7.3 Mean number of undesirable events in the 10 days preceding an episode of respiratory infection. After Stone et al. (1987).

An exam may be appraised as an interesting challenge or as an event that is extremely stressful. (Borrfdon/Photo Researchers.)

Several experimental studies have confirmed the relationship between stress and respiratory infection. In these studies, volunteers took nasal drops containing a mild cold virus and also completed a battery of measures of recent stress. The advantage of this method was that exposure to the virus was under experimental control. Researchers found that stress was clearly linked to developing a cold (Cohen, Tyrell, & Smith, 1991; Stone et al., 1992) and that the stressors most often implicated were interpersonal problems and work difficulties (Cohen et al., 1998).

Coping and Health

Relevant to individual differences in perceptions of potentially stressful situations is the concept of **coping**, or how people try to deal with problems, including the problem of handling the typically negative emotions stress produces. Even among those who perceive a situation as stressful, the effects of the stress may vary depending on how the person copes with it. Researchers have identified two broad dimensions of coping (Lazarus & Folkman, 1984):

- *Problem-focused coping* involves taking direct action to solve the problem or seeking information that will be relevant to the solution. An example would be developing a study schedule covering an entire semester in order to reduce end-of-semester pressure.
- *Emotion-focused coping* involves efforts to reduce the negative emotional reactions to stress—for example, by distracting oneself from the problem, relaxing, or seeking comfort from others.

Problem-focused and emotion-focused coping are more effective depending upon the situation. For example, distraction may be an effective way of dealing with the emotional upset produced by impending surgery, for example, but it would be a poor way to handle the upset produced by the discovery of a lump on the breast (Lazarus & Folkman, 1984). Similarly, persistently trying to solve a problem that is unsolvable leads to increases in frustration, rather than providing any psychological benefit (Terry & Hynes, 1998).

Coping researchers also refer to *avoidance coping,* which involves aspects of both problem-focused and emotion-focused coping (Carver & Scheier, 1999). The essence of avoidance coping is either attempting to avoid admitting that there is a problem to deal with (e.g., by denial) or neglecting to do anything about the problem (e.g., by giving up or just wishing the problem would go away). In some cases, avoidance coping (e.g., giving up) may be a practical approach—for example, a person who has repeatedly been denied admission to graduate school may do best by giving up on this endeavor and instead focusing on pursuing a different career path. In general, however, evidence indicates that avoidance coping is the least effective method of dealing with most problems (Roesch & Weiner, 2001).

Researchers have found that changes in the frequency of daily life events precede the onset of episodes of respiratory infection. (Michael P. Gadomski/Photo Researchers.)

The role of positive emotions in coping is an area of current interest to researchers (Folkman & Moskowitz, 2000). Positive emotions may co-occur with negative emotions during stressful situations, and they can provide some benefit. For example, positive emotions can "undo" some of the ill effects of negative emotions, particularly the physiological effects (Fredrickson & Levenson, 1998). In one study, people who expressed genuine smiling and laughter when talking about their relationship with a spouse who had died 6 months earlier had fewer grief-related symptoms and better relationships with others 2 years after the loss (Keltner & Bonanno, 1997). Another study found that people who were able to find positive meaning, such as spiritual growth or an appreciation of life, following a traumatic event were able to respond to a laboratory stressor in a more adaptive fashion (Epel, McEwen, & Ickovics, 1998). A prospective study found that men with AIDS who reported experiencing more positive affect lived longer than men with AIDS who experienced less positive affect (Moskowitz, 2003). A review of several studies reported that older people in the community who experience higher levels of positive affect were more likely to live longer (Pressman & Cohen, 2005). In addition, all people, regardless of age, who reported experiencing more positive affect had better health in general.

Studies Using the COPE Scale

The relationship between health and coping is most often assessed by means of questionnaires that ask respondents to indicate how much they used various ways of coping to handle a recent stressor. One such measure, the COPE (Coping Orientations to Problems Experienced) scale, is presented in Table 7.1.

As with the relationship between stress and health, the best way to examine links between coping and health is by means of longitudinal study. Breast cancer, which strikes about one woman in nine and is the second most deadly cancer among women (behind lung cancer), has been investigated in this way. Breast cancer is a major stressor on many levels: it is life-threatening; surgical interventions are often disfiguring and thus have serious implications for psychological well-being; and both radiation therapy and chemotherapy often have very unpleasant side effects.

In one study, the coping methods of women who had just been diagnosed with breast cancer were assessed several times during the year following the diagnosis (Carver et al., 1993). Women who accepted their diagnosis and retained a sense of humor had lower levels of distress. Avoidant coping methods, such as denial and behavioral disengagement (see Table 7.1), were associated with higher levels of distress, and this negative relationship between denial and adjustment to breast cancer has been replicated (Heim, Valach, & Schaffner, 1997). Another longitudinal study of several types of cancer found that avoidant coping ("I try not to think about it") predicted greater progression of the disease at a 1-year follow-up (Epping-Jordan, Compas, & Howell, 1994). These results show that how a person reacts to a stressor is as crucial as the stressor itself in predicting its physical and emotional effects. In the case of cancer, reducing stress by ignoring the problem is not a good idea.

Table 7.1 Scales and Sample Items from the COPE

Active Coping
I've been concentrating my efforts on doing something about the situation I'm in.

Suppression of Competing Activities
I've been putting aside other activities in order to concentrate on this.

Planning
I've been trying to come up with a strategy about what to do.

Restraint
I've been making sure not to make matters worse by acting too soon.

Use of Social Support
I've been getting sympathy and understanding from someone.

Positive Reframing
I've been looking for something good in what is happening.

Religion
I've been putting my trust in God.

Acceptance
I've been accepting the reality of the fact that it happened.

Denial
I've been refusing to believe that it has happened.

Behavioral Disengagement
I've been giving up the attempt to cope.

Use of Humor
I've been making jokes about it.

Self-Distraction
I've been going to movies, watching TV, or reading, to think about it less.

Source: From Carver et al., 1993.

Social Support and Health

Another factor that can significantly reduce the negative effects of stress is social support. **Structural social support** refers to a person's basic network of social relationships, which includes factors such as marital status and number of friends. **Functional social support** refers more to the quality of a person's relationships—for example, whether a woman believes she can call on friends in a time of need (Cohen & Wills, 1985).

Structural support is a well-established predictor of mortality (i.e., death). People with few friends or relatives tend to have a higher mortality rate than those with a higher level of structural support (Kaplan et al., 1994). Similarly, unmarried people have a higher mortality rate than married people, and this is particularly true for men (N. J. Johnson et al., 2000). In one study, people with more diverse social networks were found to be less likely to develop a cold following exposure to a virus (S. Cohen et al., 1997). Higher levels of functional support have been found to be related to lower rates of atherosclerosis (clogging of the arteries) (Seeman & Syme, 1987), to an increased ability among women to adjust to chronic rheumatoid arthritis (Goodenow, Reisine, & Grady, 1990), and to less distress among women following surgery for breast cancer (Alferi et al., 2001).

How does social support exert its beneficial effects? One possibility is that people with higher levels of social support are more likely to have healthy lifestyles—for example, eating right, not smoking, and not drinking too much alcohol. Another possibility is that social support (or the lack of it) could have a direct effect on physiological processes (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). For example, low levels of social support are related to an increase in negative emotions (Kessler & McLeod, 1985), which may affect some hormone levels and the immune system (S. Cohen et al., 1997; Kiecolt-Glaser et al., 1984). In fact, both psychological and physiological mechanisms are at work, and theories that take both into account are able to account for the link between social support and health most completely.

Social support has also been studied in the laboratory, where cause and effect can be more readily established than is possible in the naturalistic studies already described. In one such study, college-age women were required to complete a challenging task while experiencing high or low stress with or without social support (Kamarck, Annunziato, & Amateau, 1995). Stress was created by having the experimenter behave coldly and impersonally as she told the women to improve their performance on the task. Social support was created by having a close friend "silently cheer on" each woman while sitting close to her and placing a hand on her wrist. The researchers measured each woman's blood pressure while she performed the task. As Figure 7.4 shows, high stress

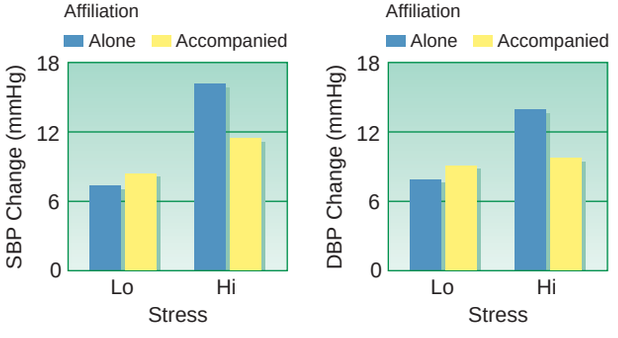


Figure 7.4 Stress led to increased blood pressure, but less so for people who experienced the stressor with a friend. DBP, diastolic blood pressure; SBP, systolic blood pressure. From Kamarck et al. (1995).

Seeking comfort or social support from others can be beneficial for health. (Bruce Ayers/Stone/Getty Images.)

led to higher blood pressure, but this effect was much greater in women who experienced the stress alone, without social support. This shows that social support can have a causal effect on a physiological process.

Research on the relationship between marriage and health has suggested that social support can affect health by impacting the married couple's relationship and each partner's emotions, cognitions, and physiology. For example, a review of laboratory studies of marital conflict discussions concluded that these interactions, which are full of negative emotions and cognitions, have a negative impact on cardiovascular reactivity, the immune system, and the endocrine system (Robles & Kiecolt-Glaser, 2004). These short-term psychological and physiological effects of marital conflict have prospectively predicted cardiovascular and blood pressure dysregulation (Baker et al., 2000) and divorce (Kiecolt-Glaser et al., 2003).

Check Your Knowledge 7.1 (Answers are at the end of the chapter.)

Answer the questions.

1. Which of the following describes the phases of Selye's general adaptation syndrome?
   - **a.** alarm, resistance, exhaustion
   - **b.** alarm, adapt, change
   - **c.** stress, react, move on
   - **d.** stress, resist, adapt
2. Problem-focused coping differs from emotion-focused in that:
   - **a.** Problem-focused coping emphasizes reducing emotions that problems cause; emotion-focused coping emphasizes acting on emotions, particularly negative emotions.
   - **b.** Problem-focused coping emphasizes taking action to solve a problem; emotion-focused coping emphasizes reducing negative feelings.
   - **c.** Both are forms of structural social support.
   - **d.** None of the above are correct.
3. Which of the following statements is incorrect regarding the relationship between social support and health?
   - **a.** People with social support may perform more healthy behaviors.
   - **b.** Social support may buffer against negative emotions, which can influence the immune system.
   - **c.** Marital conflict can negatively impact health.
   - **d.** All of the above are correct.

Understanding the Stress–Illness Link

Theories of the stress–illness link are developed to understand how psychological factors such as negative emotions impact health and disease. Theories in this domain are invariably diathesis–stress in nature—that is, theories that focus on individual vulnerabilities to stress. It is clear that not all people respond to stress in the same way, and some people are much more vulnerable. Some people are vulnerable to certain disorders and not to others. Researchers are focused on understanding psychological and neurobiological diatheses, or vulnerabilities to stress.

Before discussing these vulnerabilities, it is important to consider some indirect influences of stress on health. For example, stress may lead to health changes that are not directly due to biological or psychological factors but to changes in health-related behaviors. High stress may result in increased smoking, disrupted sleep, increased alcohol consumption, and altered diet (the opposite of what we saw with social support). These behavioral changes may then increase the risk of illness. For example, low socioeconomic status (often thought of as a stressor) has been shown to be related to greater mortality from several diseases, and this relationship is accounted for by a higher incidence among poor people of such behaviors as smoking and excessive use of alcohol (Lynch et al., 1996). In addition, high stress may result in changes in cardiovascular reactivity to stress or in changes in the immune system, such as an increase in cytokine production. These physiological changes may then increase the risk of illness. Thus, the stress–illness association is real but is likely to be mediated through changes in health behaviors or in physiology rather than to be a direct effect of stress.

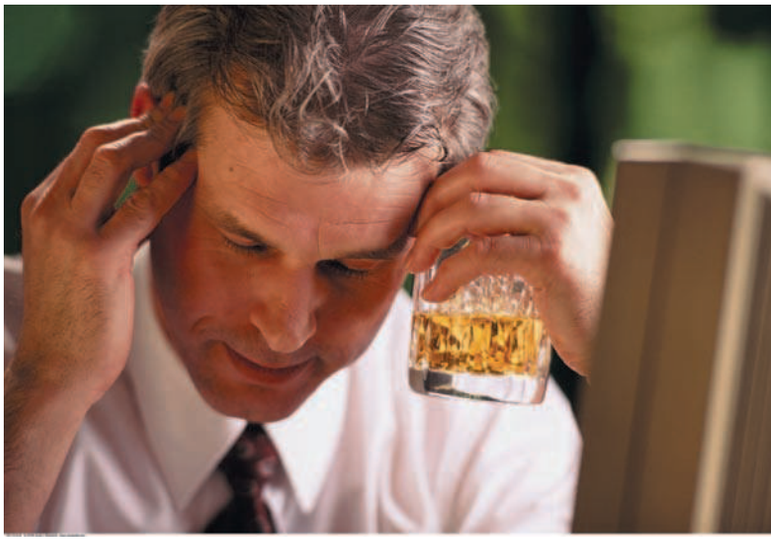
Neurobiological Perspectives

Biological responses are a healthy and regular part of responding to stress. It is only when biological responses to stress are continuously activated or when other neurobiological processes do not bring the body's systems back to their pre-stress levels within a reasonable amount of time that physiological damage can occur. The major biological responses to stress involve activation of the sympathetic nervous system and the HPA axis (see Figure 2.9, on p. 38).

Theories about the ways in which stress impacts health take specific factors into consideration, such as different kinds of stressors and differences among peoples' responses to stress, appraisals of stressful situations, and perceptions of control (Kemeny, 2003). We will discuss two neurobiological systems that have been the focus of much research—stress hormones (the endocrine system) and the immune system. Before we begin, however, we should note that these systems do not respond to stress in isolation—indeed, we now know that the autonomic nervous system, the endocrine system, and the immune system all impact one another.

Allostatic Load: Prolonged Exposure to Stress Hormones The body pays a price if it must constantly adapt to stress, and this price can be expressed in terms of what is referred to as **allostatic load**. For example, if the body is exposed to high levels of stress hormones such as cortisol and becomes susceptible to disease because of altered immune system functioning, we can view this as an effect of a high allostatic load. Furthermore, high levels of cortisol can have direct effects on the brain—for example, by damaging cells in the hippocampus, which regulates the secretion of cortisol. The result may be that, over time, the allostatic load makes the person even more susceptible to the effects of stress.

People can manifest allostatic load in different ways (McEwen & Seeman, 1999). Some people may have high levels of stress hormones simply because they experience frequent stress, whereas high levels in other people may be due to some difficulty in adapting to stress. For example, some people may have a genetically slow-to-adapt biological stress reaction. Other people may have learned behaviors that interfere with adaptation (such as poor eating habits, too little exercise, smoking, and excessive alcohol consumption). Still others may have trouble "shutting down" the biological stress response (e.g., they may exhibit an unusually high level of cortisol secretion even after the stress has abated). And some may have a weakened biological stress response, reflected by low levels of cortisol release in response to stress, which in turn causes other parts of the immune system to overrespond.



Stress may indirectly increase the risk of illness by causing people to engage in behaviors such as increased consumption of alcohol. (MTPA Stock/Masterfile.)

A real-world example of the way in which stress can interfere with our biological systems involves public speaking. Most people react to the stress of public speaking with an increase in cortisol secretion. After repeated exposure to such a stress (i.e., many public speaking experiences), most people adapt to the stress, and the amount of cortisol secreted declines. However, about 10 percent of people show no adaptation and even increase their secretion of cortisol (Kirschbaum, Prussner, & Stone, 1995), and these are the people at risk for health problems. A study measuring the presumed effects of allostatic load on the body (e.g., elevated blood pressure, cholesterol levels, and cortisol secretion) found that higher allostatic load effects predicted greater risk for cardiovascular disease $2\frac{1}{2}$ years later (Seeman et al., 1997).

Stress and the Immune System Stressors have multiple effects on the body—on the autonomic nervous system, on hormone levels, and on brain activity. One major area of current interest is the immune system, which is an important consideration in infectious diseases, cancer, and allergies as well as in autoimmune diseases, such as rheumatoid arthritis, in which the immune system attacks the body. The field that studies how psychological factors impact the immune system is called **psychoneuroimmunology**. Reviews of nearly 300 studies confirmed that a wide range of stressors produce problematic changes in the immune system, including medical school examinations, depression and bereavement, marital discord and divorce, job loss, caring for a relative with Alzheimer's disease, and the Three Mile Island nuclear disaster, among others (Kiecolt-Glaser et al., 2002; Segerstrom & Miller, 2004).

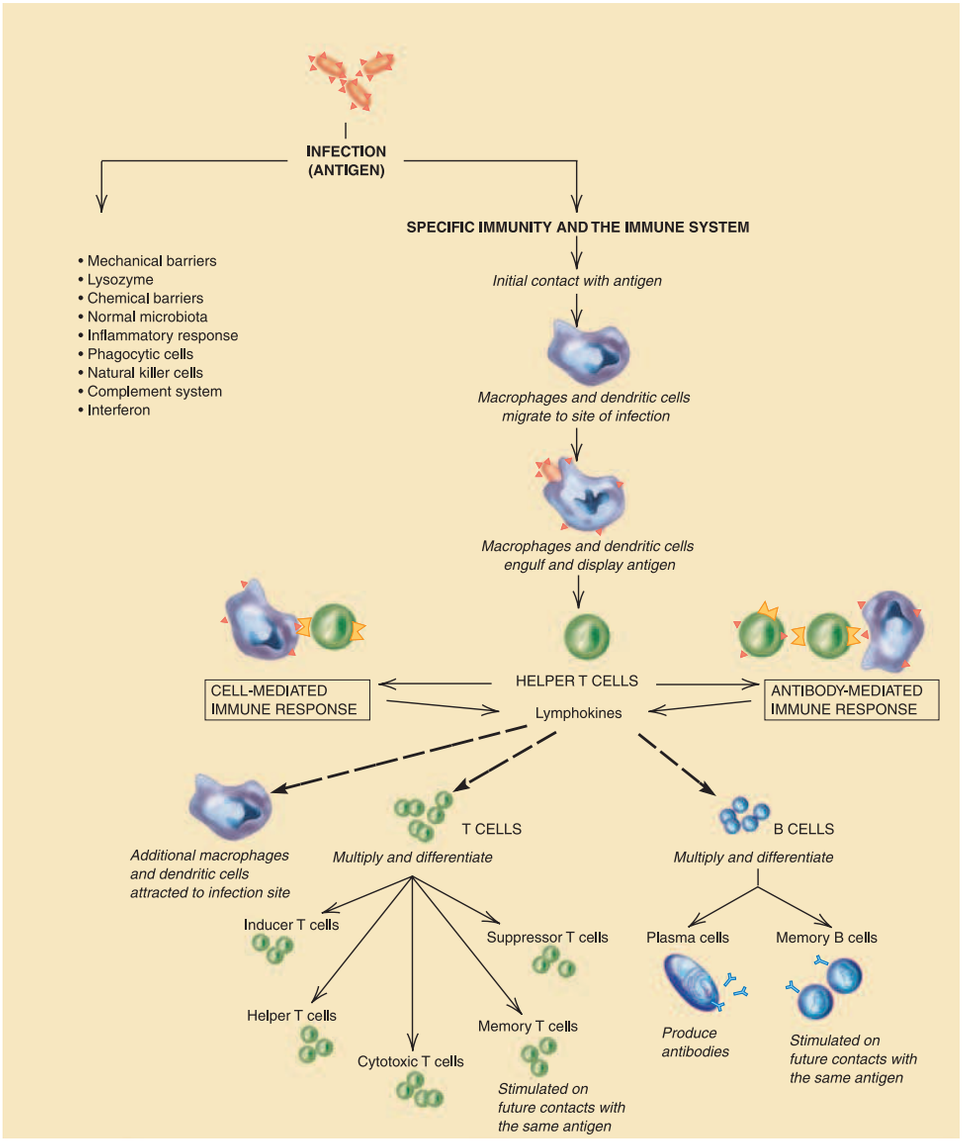


Figure 7.5 Components of the immune system.

The immune system involves a broad array of cells and proteins that respond when the body is infected or invaded. A useful way of thinking about the immune system is to consider two broad types of immunity, natural and specific (Segerstrom & Miller, 2004).

Natural immunity is the body's first and quickest line of defense against infectious microorganisms or other invaders. A number of different cells, such as macrophages and natural killer cells, are unleashed on the invaders and begin to destroy them. Inflammation or swelling is a sign of these natural immunity cells at work. Activation of macrophages in turn stimulates the release of substances called **cytokines**, which help initiate such bodily responses to infection as fatigue, fever, and activation of the HPA axis. Although unpleasant, fever is actually a sign that the body is responding as it should to an infection.

Specific immunity involves cells that respond more slowly to infection, such as lymphocytes, which are involved in responding to specific pathogens or invading agents. Lymphocytes include T-helper cells and B cells. T-helper cells promote the release of cytokines; B cells release antibodies that deal with specific pathogens.

Figure 7.5 shows the components of the immune system. In the following sections, we illustrate the role of stress and immune system changes in infectious diseases.

The effects of stress on the immune system are direct, and they can happen very early in life. In fact, a series of studies with animals has convincingly demonstrated that prenatal stress experienced by a mother can produce long-lasting changes in behavior and the immune system in her offspring (Coe & Lubach, 2005). For example, compared to infants of mothers who experienced no stress during pregnancy, the infants of rhesus monkey mothers who were exposed to chronic stress during pregnancy (exposure to loud and unpredictable noises 5 days a week for one-quarter of their pregnancy) were observed to have emotion regulation difficulties as babies and adolescents that negatively impacted their place in the social group (Coe et al., 1999; Roughten et al., 1998). In addition, these babies exhibited immune system disturbances that continued into adolescence, including a deficiency of pro-inflammatory cytokines, such as interleukin-6 (Coe et al., 2002). **Interleukin-6 (IL-6)** promotes inflammation in response to infection and is importantly linked to human diseases.

In the past 15 years, much progress has been made toward answering a key question: whether such immune system changes are great enough to actually increase the risk of disease, leading to outcomes such as early death from cancer or the onset of arthritis. For example, we now know that stress-induced changes to the immune system can hasten the

progression of AIDS (see the more detailed discussion later in this chapter). Recent studies that examine the body's response to different types of vaccines (e.g., flu and herpes vaccines) show that people exposed to stress are slower to develop antibodies in response to vaccines than those not exposed to stress. This slower response could increase the chance of illness. In addition, exposure to stress also slows the process of wound healing, which relies on the immune system (Kiecolt-Glaser et al., 2002). Across a number of studies, age appears to be a factor—that is, older adults are more likely than younger adults to show a harmful immune response to stress.

Other evidence indicates that stress can trigger the release of cytokines such as interleukin-1 and interleukin-6, as if the body were fighting off an infection (Maier & Watkins, 1998). Why is this important for health? Inflammation and higher levels of IL-6 have been linked to a number of diseases in older adults, such as coronary heart disease, arthritis, multiple myeloma, non-Hodgkin's lymphoma, osteoporosis, and type 2 diabetes. Thus, if stress increases the release of IL-6, the impact on health is likely to be negative. Based on the studies with animals reviewed above, the effects of stress on the immune system can even occur prenatally. It remains to be seen if these same effects are observed in humans. Nevertheless, the effects of stress on the immune system can be substantial.

Psychodynamic Perspectives

Psychodynamic theories propose that specific conflicts and their associated negative emotional states give rise to health problems. Of the psychodynamic theorists who studied the stress-illness link, Franz Alexander (1950) had the greatest impact. In his view, repressed emotional impulses created a chronic negative emotional state that impacted health, thus setting the stage for problems like ulcers, asthma, or essential hypertension. Alexander formulated this theory of unexpressed anger, or **anger-in theory**, on the basis of his observations of patients undergoing psychoanalysis. Although this theory has not received much empirical support, the role of anger expression as a psychological factor in essential hypertension and coronary heart disease continues to be investigated, as discussed in the sections below on those disorders.

Cognitive and Personality Perspectives

We experience stress in relation to a wide variety of conditions and situations. Physical threats obviously create stress, but so do negative emotions such as resentment, regret, and worry, which often do not easily pass and which cannot be fought against or escaped from as readily as can physical threats. Negative emotions stimulate sympathetic nervous system activity and may keep the body's stress systems aroused and the body in a continual state of emergency, sometimes for far longer than it can bear, as suggested by the notion of allostatic load.

In our general discussion of stress, we saw that the appraisal of a potential stressor is central to how it affects the person. People who continually appraise life events and experiences as exceeding their resources may be chronically stressed and at risk for the stress to adversely affect their health. We have also seen that how people cope with stress can be relevant. In addition, personality traits are implicated in several disorders, most notably cardiovascular disease. People who chronically experience high levels of negative emotions are at increased risk for the development of heart problems.

Personality traits have also been linked to immune system functioning. For example, the predisposition to experience negative emotions has been linked to slowed antibody production following a flu vaccine (Rosenkranz et al., 2003). In a study of first-year law school students, optimism predicted better mood and a stronger immune system (Segerstrom et al., 1998). This link between optimism and immune functioning was mediated in part by students' cognitive appraisals of stress—that is, optimistic students appraised law school as less aversive than did nonoptimistic students, which presumably made school less stressful and led to better immune system functioning.

Quick Summary

DSM-IV-TR covers stress and health under "Psychological Factors Affecting Medical Conditions," which can be applied to just about any illness. The fields of health psychology and behavioral medicine are dedicated to the study of how psychological factors and associated stress impact health.

Stress can be viewed as a response to a stimulus or as the stimulus itself, but neither view is without problems. Different people experience different degrees of stress in relation to the same stimuli. These differences relate to ways in which people perceive or appraise events and to people's coping styles, some of which are beneficial for health whereas others are not. Longitudinal studies using the ADE have shown a link between stress and respiratory infections, and longitudinal studies using the COPE scale have shown a link between coping styles and breast cancer. People with structural social support have better health.

Theories about the ways in which stress impacts health are diathesis–stress theories that involve both psychological and neurobiological factors. One neurobiological factor is the allostatic load that results when stress results in high levels of stress hormones. Some people may have high levels of stress hormones because they experience frequent stress, whereas high levels in other people may be due to some difficulty in adapting to stress. Another neurobiological factor is the effect of stress on the immune system. The field that studies how psychological factors impact the immune system is called psychoneuroimmunology. A wide range of stressors produce problematic changes in the immune system, and this can impact disease, for example, in hastening the progression of AIDS. Cytokines help initiate such bodily responses to infection as fatigue, fever, and activation of the HPA axis. Inflammation and higher levels of the cytokine IL-6 have been linked to a number of diseases in older adults. Psychological variables can include unexpressed anger and high levels of negative emotions, both of which can increase stress, and optimism, which then can reduce stress.

Check Your Knowledge 7.2

True or false?

1. Allostatic load refers to the body's need to adapt to stress.
2. The body's first line of defense against infection involves lymphocytes.
3. Cytokines are linked to the relationship between stress and illness.
4. Anger-in refers to expressing the experience of anger.

Cardiovascular Disorders

Cardiovascular disorders (collectively referred to as "cardiovascular disease") are diseases involving the heart and blood-circulation system. Cardiovascular disease accounts for almost half of the deaths in the United States each year, is one of the leading killers of men and women from all ethnicities, and affects nearly 65 million Americans. In 2004, the estimated costs associated with cardiovascular disease, including health care and reductions in productivity, amounted to $368.4 billion (American Heart Association, 2004).

In this section we focus on two forms of cardiovascular disease that appear to be adversely affected by stress—essential hypertension and coronary heart disease. Of all the cardiovascular disorders, coronary heart disease causes the greatest number of deaths. Experts generally agree that many of the deaths resulting from cardiovascular disorders could be prevented or delayed by dealing with behavioral risk factors. One of the American Heart Association's web slogans is "Your lifestyle is your best defense against a heart attack" (www.americanheart.org).

Essential Hypertension

Hypertension, commonly called high blood pressure, increases the risk of atherosclerosis (clogging of the arteries), heart attacks, and strokes; it can also cause death through kidney failure. Hypertension without an evident biological cause is called **essential hypertension** (or sometimes *primary hypertension*), and no more than 10 percent of all cases of hypertension in the United States have an evident biological cause—thus, essential hypertension accounts for about 90 percent of all cases. According to recent estimates, varying degrees of hypertension are found in about 20 percent of the adult population of the United States; it is twice as frequent in African Americans as in whites. As

many as 10 percent of American college students have hypertension, and most of them are unaware of their illness. Around the world, hypertension affects between 25 and 33 percent of the adult population (Kearney et al., 2004). People who do not have their blood pressure checked may go for years without knowing that they are hypertensive, which is why this disease is known as the silent killer.

Clinical Description Blood pressure is measured by two numbers, one for systolic pressure (arterial pressure when the ventricles contract and the heart is pumping) and the other for diastolic pressure (arterial pressure when the ventricles relax and the heart is resting). Normal blood pressure in a young adult is about 120 (systolic) over about 80 (diastolic). High blood pressure is defined as 140 or higher (systolic) over 90 or higher (diastolic).

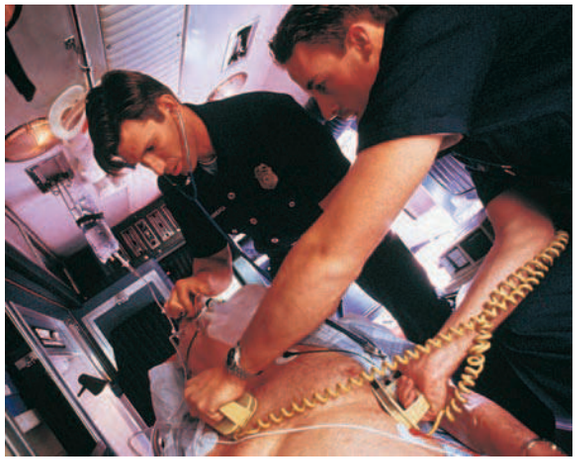
Etiology Essential hypertension is viewed as a heterogeneous condition—that is, a condition brought on by some combination of the many possible disturbances in bodily systems responsible for regulating blood pressure. Risk factors for hypertension include genes, which play a substantial role in blood pressure; obesity; excessive intake of alcohol; and excessive salt consumption. In addition, blood pressure may be elevated by increased cardiac output (the amount of blood leaving the left ventricle of the heart), by increased resistance to the passage of blood through the arteries (vasoconstriction), or by both. The physiological mechanisms that regulate blood pressure interact in an extremely complex manner—activation of the sympathetic nervous system is a key factor, but hormones, salt metabolism, and central nervous system mechanisms are all involved—and many of these physiological mechanisms can be affected by psychological stress.

The Role of Stress Various stressful real-world conditions have been examined to determine their role in the etiology of essential hypertension. Stressful interviews, natural disasters such as earthquakes, and job stress have all been found to produce short-term elevations in blood pressure (Niedhammer et al., 1998).

It is also relatively easy to produce increased blood pressure in the laboratory. The induction of various emotional states, such as anger, fear, and sadness, increases blood pressure (Caccioppo et al., 1998). Similarly, challenging tasks such as mental arithmetic, mirror drawing, putting a hand in ice water (known as the cold pressor test), and giving a speech in front of an audience all lead to increased blood pressure (Manuck, Kaplan, & Clarkson, 1983; Tuomisto, 1997).

Although the results from laboratory studies are interesting, ultimately we must understand blood pressure increases in people's natural environments. Therefore, researchers have also undertaken studies of ambulatory blood pressure, wherein participants wear a blood pressure cuff that takes readings as they go about their daily lives. Many of these studies have asked participants about their emotional state at the time a blood pressure reading is taken. The general finding has been that both positive and negative emotional states are associated with higher blood pressure (Jacob et al., 1999; Kamarck et al., 1998). Because there is evidence that anger is the negative emotion most strongly linked to elevated blood pressure (Faber & Burns, 1996; Schwartz, Warren, & Pickering, 1994), we discuss anger more later.

Other ambulatory monitoring studies have examined environmental conditions associated with blood pressure. For example, a series of studies examined the effects of stress on blood pressure among paramedics (Shapiro, Jamner, & Goldstein, 1993). In one of these analyses, ambulance calls were divided into high- and low-stress types. As expected, the high-stress calls were associated with higher blood pressure. Even more interesting were the results when the paramedics were divided into groups on the basis of personality test measures of anger and defensiveness. The groups did not differ in blood pressure during the low-stress calls. However, during the high-stress calls, paramedics high in anger and defensiveness had higher blood pressure. In another study, participants rated job strain each time blood pressure readings were taken (Kamarck et al., 1998). Blood pressure was lower at times when participants felt in control of their work environment—for example, when they felt they could exercise choice over what they were working on. Still another ambulatory blood pressure study found that, for women, the combination of job strain and family responsibilities was associated with increases in systolic and diastolic blood pressure (Brisson et al., 1999).



In a study of paramedics, high-stress ambulance calls, as when the victim had to be revived, led to greater blood pressure increases than low-stress calls. (Bruce Ayres/Stone/Getty Images.)

Among men, the expression of anger has been linked to cardiovascular disease. (Stone/Getty Images.)

In the ambulatory monitoring studies just described, the overall amount of blood pressure increase associated with emotional states or environmental conditions was rather small. But some people experience large increases, suggesting that only people who have some predisposition, or diathesis, will experience large blood pressure increases that over time may lead to sustained hypertension. Next, we discuss three possible diatheses, including anger, the Type A behavior pattern, and cardiovascular reactivity.

Anger Anger per se is not bad for our cardiovascular health; rather, it is excessive or inappropriate anger that is linked to poor health (Mayne, 2001). What is less clear is the relative importance of different aspects of anger: becoming angry easily, becoming angry and not expressing it, or having a cynical or suspicious attitude toward others. Research has not totally resolved this issue, but the evidence suggests that being easily angered may be the most important factor (e.g., Räikkönen et al., 1999).

Complicating the picture further, anger may function differently in men and women, depending on the situation. With respect to cardiovascular health, expressing anger has been related to increased blood pressure reactivity in men, whereas suppressing anger has been linked to increased blood pressure reactivity in women (Faber & Burns, 1996; Shapiro, Goldstein, & Jamner, 1995). Sex differences have also been found in the relationship between becoming angry easily and ambulatory blood pressure. Among men, but not among women, this trait is related to higher blood pressure (Guyll & Contrada, 1998). We return to this issue in our discussion of myocardial infarction.

Type A Behavior Pattern In 1958, two cardiologists, Meyer Friedman and Ray Rosenman, identified a behavior pattern called **Type A behavior pattern** (Friedman, 1969; Rosenman et al., 1975). A structured interview identifies three components of the Type A behavior pattern. *Achievement striving/competitiveness* characterizes someone with an intense and competitive drive for achievement and advancement. *Time urgency/impatience* characterizes someone with an exaggerated sense of urgency—of time passing and of the need to hurry. *Hostility* captures someone who exhibits considerable aggressiveness and hostility toward others. The Type A behavior pattern has been most often studied as a risk factor for coronary heart disease, but it has also been examined as a predisposing factor for hypertension.

One characteristic of the Type A behavior pattern is feeling under time pressure and consequently trying to do several things at once. (GoodShoot/SUPERSTOCK.)

A large, prospective, and longitudinal study called the Coronary Artery Risk Development in Young Adults Study (CARDIA) examined whether the three components of Type A could predict the development of hypertension among black and white men and women. The study began in 1985 with over 5,000 participants between the ages of 18 and 30. Fifteen years later, a team of investigators examined Type A and hypertension among 3,308 of these original participants (Yan et al., 2003). They found that the time urgency/impatience and hostility components from the Type A structured interview predicted a twofold increase in risk of developing hypertension. The findings for time urgency/impatience were stronger for men than women; the findings for hostility were just about equally strong for both men and women. Achievement striving/competitiveness was a predictor of later hypertension for white men only. These findings provide strong support for the role of psychological factors like these in hypertension.

Cardiovascular Reactivity In the past 15 years or so there has been a great deal of interest in cardiovascular reactivity as a risk factor for hypertension (and coronary heart disease as well). *Cardiovascular reactivity* refers to the extent to which blood pressure and heart rate increase in response to stress. Typically, researchers assess cardiovascular reactivity to a laboratory stressor (or, even better, a battery of stressors) among people who are not currently hypertensive and then assess the participants again some years later to determine whether the reactivity measure (usually, the amount by which the stressor causes change from a baseline condition) predicts blood pressure.

In one study, cardiovascular reactivity was measured while participants performed a laboratory reaction-time task in which they were threatened with shock if their responses were slow (Light et al., 1992). A follow-up 10 to 15 years later found that heart-rate reactivity was the

strongest predictor of high blood pressure. Of importance, these reactivity measures predicted subsequent blood pressure over and above the contribution of standard clinical predictors such as family history of hypertension. Other research has shown that cardiovascular reactivity is related to other known risk factors for hypertension, such as socioeconomic status and race (Gump, Matthews, & Räikkönen, 1999; Jackson et al., 1999).

These findings concern levels of blood pressure but not the actual disease of hypertension. A different study entailed a 4-year follow-up of 508 Finnish men whose blood pressure reactivity had been assessed as they anticipated a bicycle exercise test (Everson et al., 1996). Men whose systolic blood pressure increased by 30 points or more were almost four times more likely to have developed hypertension 4 years later. One limitation of this study is that reactivity was assessed in an unusual situation. Thus, we can't be sure that the results would generalize to the more usual tests that have been used to assess reactivity.

Further support for the importance of cardiovascular reactivity comes from high-risk research comparing people with and without a family history of hypertension (Adler & Ditto, 1998; Lovallo & Al'Absi, 1998). People with such a history show greater blood pressure reactivity to various stressors. Coupled with research showing the heritability of hypertension, these findings suggest that blood pressure reactivity is a good candidate for a genetically transmitted diathesis. But what exactly is inherited?

Research has focused on genes linked to the neurotransmitter serotonin. This evidence suggests that people with one or two long alleles in the promoter region of the serotonin transporter gene (see Chapter 2) show greater cardiovascular reactivity in response to tasks involving mental arithmetic (R. B. Williams et al., 2001) and are at greater risk for myocardial infarction (Fumeron et al., 2002). Needed next are studies that examine how stress interacts with the presence of a long allele on this gene. This type of work has been done for depression: recall from our discussions in Chapter 2 that having one or two *short* alleles in this gene was associated with an increased risk of depression, but only among those people who had experienced early life stress.

Coronary Heart Disease

Coronary heart disease (CHD) takes two principal forms, angina pectoris and myocardial infarction, or heart attack.

Clinical Description The symptoms of **angina pectoris** are periodic chest pains, usually located behind the sternum and frequently radiating into the back and sometimes the left shoulder and arm. The major cause of these severe attacks of pain is an insufficient supply of oxygen to the heart, called *ischemia*, which in turn is due to coronary atherosclerosis, a narrowing or plugging of the coronary arteries by deposits of cholesterol, a fatty material, or to constriction of these blood vessels. Some episodes of ischemia do not cause pain, so these are called episodes of silent ischemia. Both angina and episodes of silent ischemia are precipitated by physical exertion or emotional stress and are commonly relieved by rest or medication. Angina and silent ischemia rarely result in serious physical damage to the heart, because blood flow to the heart is reduced but not cut off. If, however, the narrowing of one or more coronary arteries progresses to the point of producing a total blockage, a myocardial infarction, or heart attack, is likely to occur.

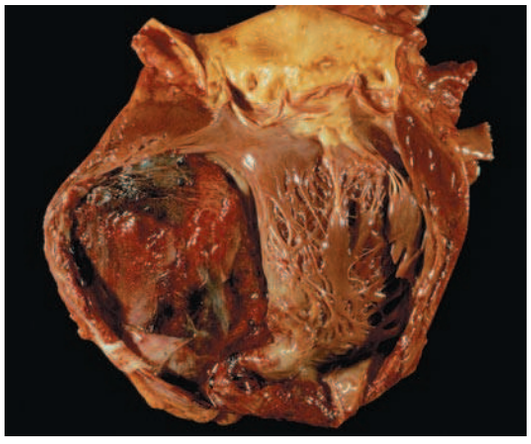
Myocardial infarction, perhaps better known as heart attack, is a much more serious disorder; it is the leading cause of death in the United States today. Like angina pectoris, it is caused by an insufficient supply of oxygen to the heart. But unlike angina, a heart attack usually results in permanent damage to the heart.

Etiology Several factors increase the risk of CHD; the risk generally increases with the number and severity of these factors. Table 7.2 lists these risk factors.

A combination of particular risk factors has been named the **metabolic syndrome**. It is defined by the presence of a number of related metabolic factors, including abdominal obesity, insulin resistance, high blood pressure, low HDL cholesterol (the "good" cholesterol), and heightened inflammation as indexed by the presence of a protein called CRP (Grundy et al., 2004). Adults with the metabolic syndrome are more likely to die from CHD than those without it (Malik et al., 2004). People who have what is referred to as "high-normal" blood pressure (i.e. systolic pressure of 130–139 and diastolic pressure of 80–85) are also at elevated risk of CHD (Vasan et al. 2001).

Table 7.2 Risk Factors for Cardiovascular Disease

Age (older people are at greater risk)
Cigarette smoking
Trans fats
Diabetes
Elevated blood pressure
Elevated serum cholesterol
Excessive use of alcohol
Increase in the size of the left ventricle of the heart
Long-standing pattern of physical inactivity
Obesity
Sex (men are at greater risk)



Myocardial infarction can do serious damage to the heart. (CNRI/ Photo Researchers, Inc.)

In the next three sections, we examine research on psychological risk factors for CHD. As with research on the links between stress and health, psychological and biological risk factors are intertwined, and a complete explanation of the etiology of CHD must include both. Results from the INTERHEART study of over 15,000 people who had had a heart attack and nearly 15,000 people who had not (the control group), which spanned 52 countries, found that psychosocial risk factors, such as stress, accounted for a third of the risk of a later heart attack (Rosengren et al., 2004; Yusuf et al., 2004).

Stress and Myocardial Infarction In the short term, physical exertion can trigger a myocardial infarction, as can episodes of anger (Mittleman et al., 1997). Acute stress is another short-term factor—the frequency of myocardial infarction, for example, increased among residents of Tel Aviv on the day of an Iraqi missile attack (NHLBI, 1998). Death from myocardial infarction did not increase following the September 11, 2001, terrorist attacks in the United States (Chi et al., 2003); however, there were almost twice as many heart arrhythmias among people who had a cardiac defibrillator implanted (Steinberg et al., 2004). Over the longer term, more chronic stressors, such as marital conflict and financial worries, are also relevant.

One of the most studied chronic stressors is job strain (Karasek, 1979), an employment situation involving too much work, too little time, a lack of control over decision making, and a lack of opportunity to make full use of skills on the job. Several studies have found that a high level of job strain is associated with increased risk for myocardial infarction. In one study, over 10,000 British workers were assessed for the degree of control they could exercise over their jobs. They were then followed for about 5 years to determine the incidence of CHD. As in earlier studies, more CHD was found at follow-up among workers in lower-status jobs (e.g., clerical work). This result, in turn, was related to these workers' reports of having little control on the job (Marmot et al., 1997). In a large-scale study conducted in Finland, a high level of job demands was related to the progression of atherosclerosis (Lynch, Kaplan, et al., 1997) and to cardiovascular disease mortality and morbidity (Lynch, Krause, et al., 1997).

Other Psychological Risk Factors Contemporary evidence linking CHD to psychological risk factors stems from the early investigations of the Type A behavior pattern. Initial support for the idea that the Type A pattern predicts CHD came from the classic Western Collaborative Group Study (WCGS) (Rosenman et al., 1975). In this double-blind, prospective investigation, 3,154 men aged 39 to 59 were followed over a period of $8\frac{1}{2}$ years. People who had been identified as Type A by interview were more than twice as likely to develop CHD as were Type B men (Type B behavior pattern is characterized by a less driven and less hostile way of life). Traditional risk factors, such as high levels of cholesterol, were also found to be related to CHD, but even when these factors were controlled for, Type A individuals were still twice as likely to develop CHD.

Hostility is linked to coronary-artery blockage. (Somos Images LLC/Alamy)

More recent research, however, has not supported the predictive power of Type A behavior (e.g., Eaker, Pinsky, & Castelli, 1992; Orth-Gomer & Unden, 1990). There are several reasons for these conflicting results. One is that later investigators used different methods of assessing Type A (e.g., questionnaires) that may not have been adequate. Second, it became apparent that not all aspects of the Type A behavior pattern were truly related to CHD. For example, in further analyses of the WCGS data, anger and hostility emerged as the major predictors of CHD (Hecker et al., 1988). A longitudinal study also found that difficulty controlling one's anger is related to higher rates of CHD (Kawachi et al., 1996). Anger and hostility are also related to several other variables that play a role in CHD. For example, high levels of anger and hostility are related to greater blood pressure reactivity to stress, higher levels of cholesterol, abnormal deposits of calcium on the walls of coronary arteries, cigarette smoking and alcohol use, the metabolic syndrome among adolescents, and greater activation of platelets, which play a major role in the formation of blockages in the coronary arteries (Fredrickson et al., 2000; Iribarren et al., 2000; Räikkönen, Matthews, & Salomen, 2003; Weidner et al., 1989).

Anger and hostility may be differentially related to CHD risk for men and women. For example, researchers found that indirect expressions of anger were associated with CHD risk for women, whereas overt expressions of anger were related to CHD risk for men (Siegman et al., 2000). These findings are similar to those discussed earlier showing that expression of anger was related to increased blood pressure reactivity in men, but the suppression of anger was related to increased blood pressure reactivity in women (Faber & Burns, 1996; Shapiro et al., 1995).

Other findings suggest that cynicism (an approach to life that involves hostility) is an important component of the Type A behavior pattern (Almada, 1991). For example, the amount of coronary artery blockage was especially high in Type A participants who had earlier given responses to MMPI items reflecting a cynical or hostile attitude (e.g., agreeing with the statement "Most people will use somewhat unfair means to gain profit or advantage, rather than lose it"). A study of medical students who had been healthy when they took the MMPI 25 years earlier found a higher rate of CHD and death in those whose answers had indicated cynicism toward others (Barefoot, Dahlstrom, & Williams, 1983). More recently, cynicism was found to predict atherosclerosis, myocardial infarctions, and death from CHD in the large Finnish study mentioned earlier (Everson et al., 1997; Kamarck et al., 1997). Cynicism also prospectively predicted a broad array of cardiovascular disorders (angina, stroke, heart attack) among older adults, and the link between cynicism and disease was accounted for in part by the presence of the metabolic syndrome among participants (Nelson, Palmer, & Pederson, 2004).

What is not yet clear is the best way to think about these results concerning anger, hostility, and cynicism. Are they the same or different? Is one more important than another in predicting cardiovascular disease? The answers to these questions remain unanswered at this point.

Research has also examined the relationship between other negative emotions—particularly anxiety and depression—and CHD. For example, anxiety has been shown to be related to the onset of CHD (Kawachi et al., 1994; Kubzansky & Kawachi, 2000) and to prospectively predict cardiac events among men with coronary artery disease (Frasure-Smith & Lesperance, 2008). Similarly, research has found that depression is related to the development of CHD (reviewed by Suls & Bunde, 2005). Two separate meta-analyses have found that depression increases the risk for CHD (Barth, Schumacher, & Hermann-Lingen, 2004; Nicholson, Kuper, & Hemingway, 2006). In addition, cardiac patients who also have a mood disorder are over five times more likely than others to die within 6 months of a heart attack (Glassman & Shapiro, 1998).

It is almost certain that these factors interact with biological factors to produce their effects on CHD. Anxiety, for example, is associated with activation of the sympathetic nervous system, which can lead to both hypertension and atherosclerosis. Research has also shown that depression is linked to a greater tendency for platelets to aggregate and thus produce obstructions in the arteries. Furthermore, depression is often associated with increases in steroidal hormones, which increase blood pressure and damage cells in arteries (Musselman, Evans, & Nemeroff, 1998).

Check Your Knowledge 7.3

Fill in the blanks.

1. _____ hypertension has no known biological cause.
2. Type A behavior pattern is linked to the disorders _____ and _____.
3. Cardiovascular reactivity is linked to changes in _____ in laboratory studies, but its link to _____ is less well established.

Asthma

Asthma, a disorder of the respiratory system (see Figure 7.6), afflicts over 22 million people in the United States (NHLBI, 2003). In 2000, the economic costs of asthma in the United States, including both medical expenditures and lost productivity, amounted to $14.5 billion (NHLBI, 2001). The prevalence of asthma increased 74 percent from 1984 to 1994, but it seems to have remained stable since 1997, perhaps due to the fact that the survey is now more reliable (NHLBI, 2003). The prevalence of asthma among children has risen 60 percent since 1980 (Eder, Ege, & von Mutius, 2006). Of the over 22 million Americans with asthma, nearly one-third are children. In California, children with asthma missed an average of one week of school in 2005 (UCLA Center for Health Policy Research, 2008). During childhood, asthma is more common among boys than among girls. But by age 18 it becomes more prevalent in women and remains so until after age 45, when men again predominate (NHLBI, 2004). Asthma is more common among smokers, people who are obese, and people of lower socioeconomic status (Gwynn, 2004).

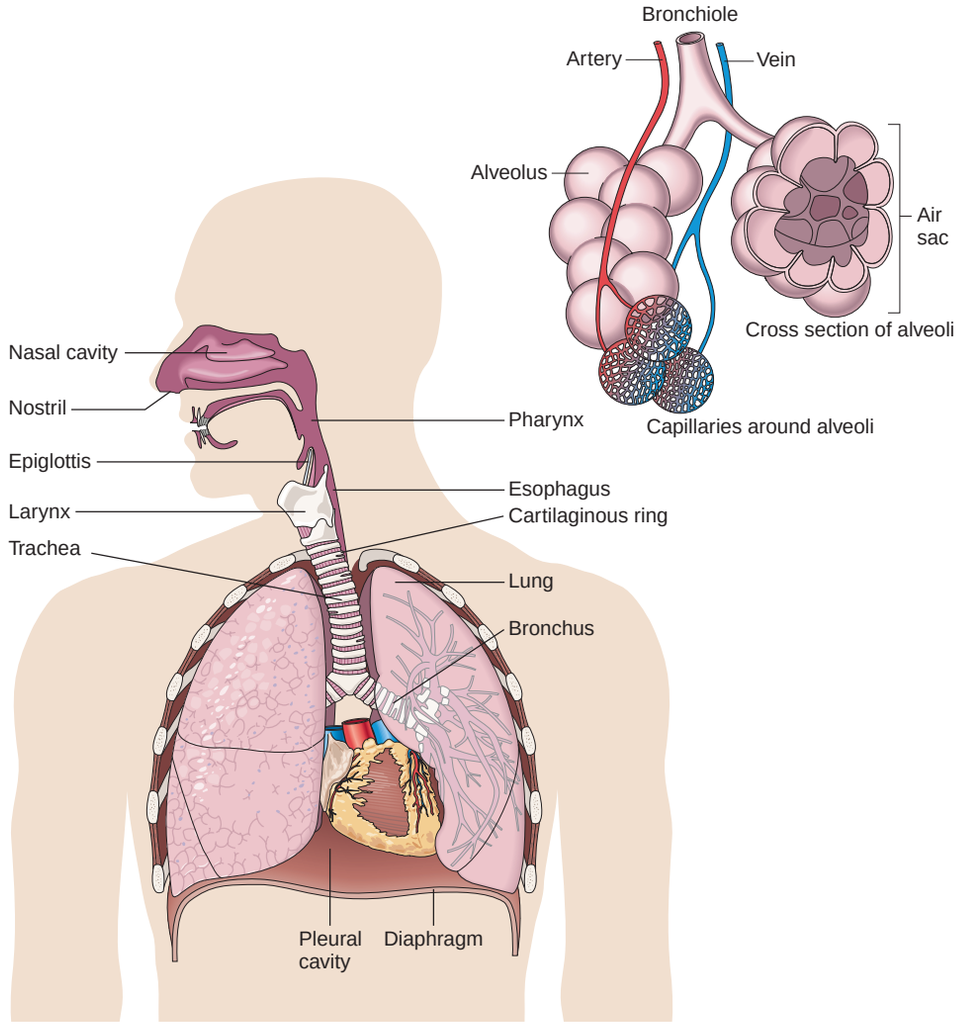


Figure 7.6 Major structures of the respiratory system—trachea, lungs, bronchi, bronchioles, and alveoli—and the ancillary organs. In asthma, the air passages, especially the bronchioles, become constricted and fluid and mucus build up in the lungs.

Clinical Description

In an asthma attack, the air passages in the lungs become narrowed, causing extremely labored and wheezy breathing. In addition, activity of the immune system during asthma attacks leads to inflammation of lung tissue, resulting in an increase in mucus secretion and edema (accumulation of fluid in the tissues) (Moran, 1991).

Clinical Case: Tom

During his childhood, Tom had frequent asthma attacks. His asthma was triggered principally by pollen, and each year he went through a particularly bad period that included several trips to the emergency room of a local hospital. He also seemed to get more than his share of colds, which frequently developed into bronchitis. As he reached his teenage years, the attacks of asthma mysteriously vanished, and he was symptom-free for the next 20 years. But at age 34 the attacks returned with a vengeance following a bout of pneumonia. In contrast to his childhood attacks, emotional stress now appeared to be the major precipitant. This hypothesis was confirmed when his physician asked Tom to keep a diary for 2 weeks in which he recorded how he had been feeling and what had been going on before each attack. He had four attacks over the period, three preceded by unpleasant interactions with his boss at work and one by an argument with his wife over an impending visit by her parents.

Asthma attacks occur intermittently, sometimes almost daily and sometimes separated by weeks or months, and vary in severity. The frequency of attacks may increase seasonally, when certain pollens are present. The airways are not continuously blocked; rather, the respiratory system returns to normal or near normal either spontaneously or after treatment, and this differentiates asthma from chronic respiratory problems such as emphysema (Creer, 1982). Symptoms may last an hour or may continue for several hours or sometimes even for days.

For people with exercise-induced asthma, the attacks follow strenuous exercise. Although some athletes are debilitated by the attacks, others are able to perform at the highest level despite their asthma—for example, Jackie Joyner-Kersee, a six-time Olympic medalist in track and field.

Most often, asthma attacks begin suddenly. A severe attack is a frightening experience and may cause a panic attack (Carr, 1998, 1999), which exacerbates the asthma. People with asthma have immense difficulty getting air into and out of the lungs and feel as though they are suffocating; the gasping, wheezing, and coughing can compound the fear. After an attack, a person may become exhausted by the exertion and fall asleep as soon as breathing is more normal.

Olympic medalist Jackie Joyner-Kersee suffers from exercise-induced asthma. (© AP/Wide World Photos.)

Etiology

Asthma attacks seem to be brought on by a very wide variety of factors, including allergens, environmental toxins such as secondhand smoke, viral infections, cold, and exercise. Air pollution is known to be a contributing factor—for example, emergency room visits for asthma declined considerably in urban Atlanta during the 1996 Olympic games when automobile traffic was severely restricted (Friedman et al., 2001). As you will see from the discussions below, stress or negative emotions can exacerbate the impact of environmental toxins on asthma.

Biological Factors Diatheses leading to asthma might include the effects of respiratory infections, as seems to have been the case with Tom. Allergies, too, can predispose people to this disorder. When asthma is caused primarily by allergens, the cells in the respiratory tract are especially sensitive to one or more substances (allergens), such as pollen, molds, fur, air pollution, smoke, and dust mites, which bring on an attack. People whose asthma is primarily allergic may have an inherited hypersensitivity of the respiratory mucosa, which then overresponds to one or more of such usually harmless substances. Asthma runs in families, which is consistent with genetic transmission of a diathesis (Eder et al., 2006). Studies are narrowing in on how genes may interact with environmental factors to produce asthma (e.g., Cookson & Moffatt, 1997, 2000).

Stressful Life Events and Negative Emotions The importance of psychological factors in asthma attacks is a topic of debate. Psychological factors that may interfere with the functioning of the respiratory system and thus bring on an asthma attack include stressful life events, anxiety, anger, depression, and frustration. Even when asthma is originally induced by an infection or allergy, psychological stress can precipitate an attack. Because of the link between the autonomic nervous system (ANS) and the constriction and dilation of the airways, and the connection between the ANS and emotions, research has focused on heightened experience and expression of negative emotions. Asthma patients report that many attacks are precipitated by emotions such as anxiety (Rumbak et al., 1993). In both laboratory and real-life settings, people with asthma show greater constriction of the bronchial tubes in response to stressors (Affleck et al., 2000; Miller & Wood, 1994).


Asthma attacks are often treated by using a nebulizer to spray a fine mist of a bronchodilator into the bronchial tubes. (Hattye Young/Photo Researchers.)

Negative emotions have also been found to be directly related to reports of asthma symptoms and to peak expiratory flow, which is an assessment of airway obstruction obtained by taking a deep breath and then exhaling as hard as possible into a device that measures the force of the air expelled. One study assessed people with asthma over a period of several days (Smyth et al., 1999). Five times each day the participants were signaled to measure their peak expiratory flow and to record in a diary any asthma symptoms they were experiencing, their level of stress, and the mood they were in. Reports of higher levels of stress and negative emotions were

related to lower peak flow and more reports of asthma symptoms. A prospective study asked children ages 6 to 13 and their parents to keep daily records of asthma symptom reports and peak expiratory flow for 18 months (Sandberg et al., 2004). The parents and children were also interviewed about stressful life events during the period of the study. The investigators found that the children were nearly five times as likely to have an asthma attack if they experienced a stressful life event 1 or 2 days before the attack. In addition, the children who experienced stressful life events were more likely to have another attack 5 to 7 weeks later.

In interpreting these results it is probably reasonable to assume that some of this heightened negative emotional experience is a reaction to having a chronic disease. But some research also shows that negative emotions precede asthma attacks, indicating that negative emotion may play a role in precipitating attacks (Hyland, 1990). In contrast, a prospective study found that pleasant emotions predicted better lung functioning among people with asthma (Apter et al., 1997).

Role of the Family Sources of psychological stress associated with asthma include parent–child interactions and other family-related factors. One investigation looked at 150 pregnant women who had asthma (Klinnert, Mrazek, & Mrazek, 1994). The investigators intended to study the mothers' children, who were at genetic risk, and to assess parental characteristics as well. Both parents were interviewed 3 weeks after the child's birth to determine their attitudes and sensitivity toward the infant, their strategy for sharing parenting duties, and the presence of any stress. The amount of stress experienced by the mother in the past year was also assessed. The children were closely monitored over the next 3 years, and the frequency of asthma was then related to parenting problems in the family. Results showed a high rate of asthma among children whose mothers had high levels of stress and whose families were rated as having parenting problems (see Figure 7.7).

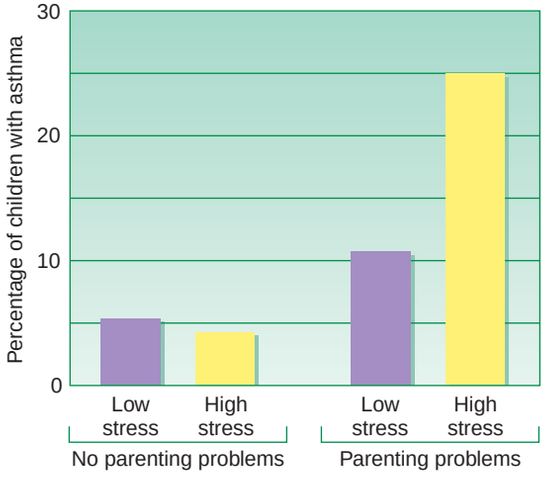


Figure 7.7 The effects of parenting problems and mothers' prior stress on the frequency of asthma in their children. Children whose mothers had been under a high level of stress and who were raised in families with parenting problems had high rates of asthma. From Klinnert et al. (1994).

Not all research, however, has found that parent–child relationships figure in asthma. One study used cross-sectional and prospective analyses to study the link between family factors and (re)hospitalization for asthma (Chen et al., 2003). The cross-sectional analyses revealed that a higher level of family conflict and strain was associated with an increased number of hospitalizations for asthma among children in the family. Although this factor did not prospectively predict rehospitalization among the children, parental reports of less mastery and less emotional concern over their child's illness were predictive of later rehospitalization. These cross-sectional findings, however, do not tell us about the direction of the effects—that is, family stress could be greater due to having a serious illness in the family, or the severity of the asthma could be greater due to family stress. Nevertheless, the longitudinal, prospective findings suggest that higher levels of family stress do not cause an exacerbation in asthma, at least not with respect to rehospitalization, whereas lower levels of parental mastery and emotional concern seem to predict a later exacerbation.

AIDS

AIDS (acquired immunodeficiency syndrome) is a significant public health concern worldwide. We discuss this invariably fatal illness here because it can be prevented by psychological means.

In the United States, AIDS was originally proclaimed a disease of gay men, but it never was exclusively a "gay disease." In Africa and parts of Latin America, AIDS is found primarily among heterosexuals, and throughout the world HIV-positive women [i.e., women who are infected with HIV (human immunodeficiency virus), which causes AIDS] give birth to HIV-positive babies. The following statistics give some sense of the scope of AIDS (CDC, 2004, 2008; Epstein, 2007; UNAIDS, 2007 at http://www.unaids.org/en):

- Since the start of the epidemic, more than 25 million people worldwide have died of AIDS.
- As of 2007, over 33 million people worldwide are now HIV-positive. Of these, nearly 23 million live in sub-Saharan Africa. In 2007, an estimated 1.6 million people died from AIDS in sub-Saharan Africa; an estimated 21,000 people died from AIDS in North America.
- In 2005, 40 percent of pregnant women in Botswana and Swaziland were HIV-positive.

- There were nearly 5 million new cases worldwide in 2003. This was the largest number of new cases in any single year since the epidemic began. The number of new cases appears to have leveled off since then.
- Worldwide, women now account for nearly 50 percent of people infected with HIV.
- In the United States, African American and Hispanic women are 17 times more likely than white women to become infected.
- In 2006, 49 percent of the new HIV diagnoses in the United States were found in African Americans.
- In 2003, AIDS was the fifth leading cause of death among adults ages 35 to 44, a remarkable statistic given that it wasn't even included as a cause of death just over 25 years ago.

Clinical Description

The medical complexities of AIDS are beyond the scope of this book, but it is important to understand a few fundamentals. AIDS is a disease in which the body's immune system is severely compromised by HIV, putting the person at high risk for fatal diseases such as Kaposi's sarcoma, rare forms of lymph cancer, and a wide range of dangerous fungal, viral, and bacterial infections. The term *opportunistic* is often used to describe these illnesses, which are seldom found in people with healthy immune systems, because we can say that these diseases take advantage of the opportunity afforded by a weakened immune system (Kalichman, 1996). Strictly speaking, people do not die of AIDS but of the diseases to which AIDS makes them vulnerable.

Spread of the Disease

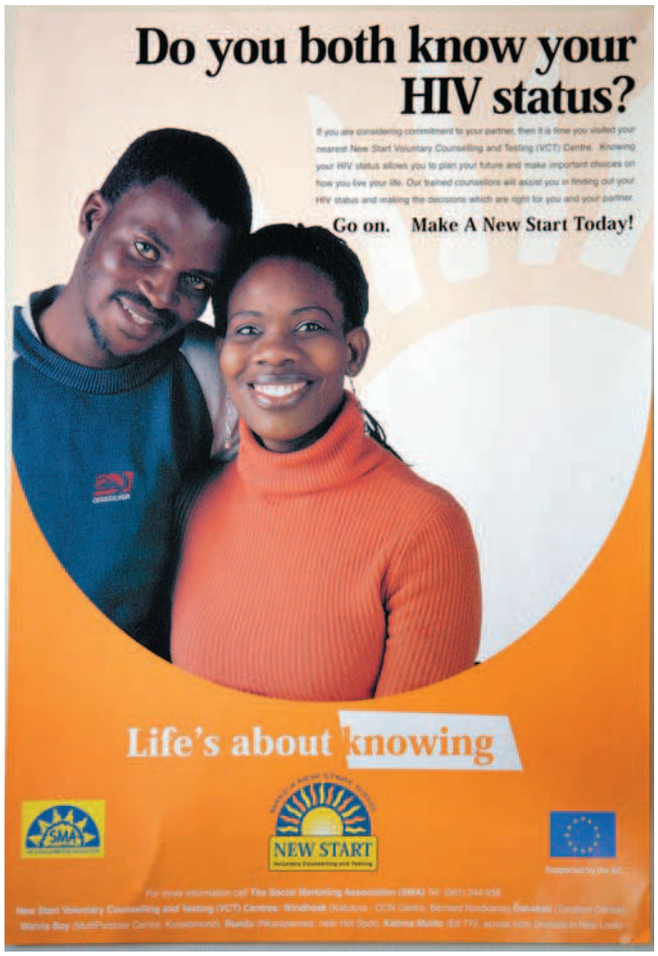
HIV is most often transmitted from one person to another through unsafe sexual practices, regardless of sexual orientation. HIV is present in blood, semen, and vaginal secretions and can be transmitted only when infected fluids get into the bloodstream. It cannot be transmitted through casual social contact. Among intravenous drug users, sharing unsterilized needles can introduce HIV-carrying blood into the bloodstream of another. Infants born to HIV-positive mothers are at risk, for the virus can cross the placental barrier and infect the developing fetus.

Risk is elevated in people who abuse drugs, including drugs that are not injected, perhaps because the effects of drugs can compromise a person's ability or willingness to consider the consequences of behavior. Another possible mechanism is a risk-taking disposition, for which frequent drug use is a marker; that is, engaging in unprotected sex may be another sign of a tendency to take risks (Chesney, Barrett, & Stall, 1998). Whatever the reasons, ample evidence shows that unsafe sex is associated with the frequent use of many kinds of drugs, including alcohol and methamphetamine (Colfax et al., 2004; Halkitis, Parsons, & Stirratt, 2001).

AIDS Prevention Efforts

Despite promising advances in drug treatment (e.g., AZT, saquinavir), there is widespread agreement that by far the best strategy is prevention through behavioral changes. For example, scientists generally agree that needle-exchange programs or the free distribution of needles to intravenous drug users reduces needle sharing and thereby the spread of infectious diseases (Gibson, 2001; Yoast et al., 2001). The primary focus in preventing sexually transmitted AIDS is on changing sexual practices. Early efforts in this regard were successful. For example, in the 1980s, when prevention efforts focused on encouraging the use of condoms during every sexual contact, new cases of HIV dropped in many large cities, from an annual infection rate among gay men of around 10 percent or more to 1 or 2 percent. But more recent data have indicated that younger gay men are engaging in more unprotected sexual behaviors than are older gay men (CDC, 2001; 2008). In 2006, the most common type of transmission of HIV in the United States was male-to-male sexual contact, with over 50 percent of new HIV infections being transmitted in this way (CDC, 2008).

Why might this be happening? Perhaps young gay men do not see as many of their age cohort infected as do older gay men, and so they don't consider AIDS as much of a threat for



Encouraging information about HIV testing is a key component to AIDS prevention efforts. This advertisement emphasizes the importance of HIV testing in the country of Namibia. (Sean sprague/Alamy)

themselves as it is for others. After the Food and Drug Administration (FDA) permitted direct marketing of AIDS medicines to consumers in 1997, ads from drug companies began to appear that portrayed HIV-positive men as healthy, active, robust, and happy and as engaging in all sorts of activities and sports; such ads may have given the impression that these drugs can cure AIDS.

Even if such ads accurately portrayed some small percentage of HIV-positive men, their relevance for women seems doubtful. Moreover, many HIV-positive men are not as healthy and active as the ads suggested. More importantly, these drugs can neither cure AIDS nor reduce its transmission. The FDA has since ordered that such ads be discontinued.

Other prevention efforts focus on encouraging monogamous relationships. One can eliminate the possibility of exposure by being in a monogamous relationship with a partner who tests negative for HIV. However, monogamous relationships are rare among young people and are not invariably found among married people or those in other committed relationships. Moreover, monogamous relationships are not the social norms in some eastern and southern African countries, such as Uganda. Instead, "concurrent long-term partnerships" are more common (Epstein, 2007). These relationships are faithful relationships, in that partners know about other partners and relationships outside these partnerships are not typically formed. Programs aimed at reducing the number of sexual partners has been shown to be effective in Uganda and Thailand (Shelton et al. 2004). Nonetheless, promoting faithful, monogamous relationships in this region of the world as a prevention strategy runs counter to the social and cultural norms of the region, making such programs more difficult to implement.

Although advocating monogamous relationships remains a prevalent approach in the public health arena, at least in Western countries, prevention is best directed at encouraging sexually active people to use condoms, which are about 90 percent effective in preventing HIV infection. Avoiding sex after using alcohol or drugs is also prudent because, as noted, use of these substances increases the tendency to engage in risky sex (e.g., Colfax et al., 2004).

Abstinence-only programs in schools do not appear to be effective HIV prevention programs among adolescents in the United States. A review of 13 different programs involving nearly 16,000 students found no evidence that abstinence-only programs decreased risky sexual behaviors, pregnancy, or the risk of developing HIV (Underhill, Montgomery, & Operario, 2007).

How can changes be brought about, especially in generations of sexually active people? Social psychological and cognitive behavior theory and research suggest strategies that can form the basis of effective preventive interventions (Chernoff, 1998; Kalichman, 1995; Kelly, 1995):

- Provide accurate information about HIV transmission. Such information is readily available online from the Centers for Disease Control and from local AIDS organizations.
- Explain clearly what the person's risks are (e.g., people with many sexual partners are at higher risk, regardless of sexual orientation; sharing needles with other intravenous drug users is very risky).
- Identify cues to high-risk situations (e.g., drinking alcohol in a sexually provocative situation is associated with higher-risk sexual behavior).
- Provide instruction in condom use (and, as appropriate, needle cleaning and exchanging).
- Provide social skills training that includes sexual assertiveness skills (e.g., resisting pressure to have sex or insisting that safer sex be practiced) and other communication skills that can help preserve relationships while reducing the risk of infection with HIV.
- Work at the community level to generate large-scale social support for making safer sex the expected thing, creating a "we're all in this together" atmosphere.

AIDS prevention efforts are more successful if they include educational information and role playing. (Keith Dannemiller/©Corbis)

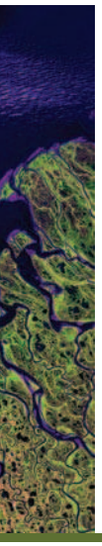
An early review (Chernoff, 1998) revealed that interventions implementing these principles often, but not always, leads to increases in sexual assertiveness, reductions in unprotected anal intercourse, and increased use of condoms in gay and bisexual men, low-income minority women, female prostitutes, and substance abusers. These programs also show some promise among high school and college-age students in the United States, but not with middle or junior high students.

A more recent meta-analysis (Albarracon et al., 2005) of over 300 prevention programs found that prevention programs were more likely to be effective if they included (1) educational information, (2) the positives associated with condom use (e.g., better relationship quality and health), (3) active (e.g., role-playing) rather than passive (e.g., lecture) interventions, and (4) behavioral skills training (e.g. how to use a condom, how to say no). Prevention programs that tried to induce fear into participants were least effective, particularly for people over the age of 21. School-based programs that included condom use skills and support for condom use by family and friends were also effective. A different meta-analysis concluded that prevention programs that included experts as group leaders (rather than peers) were more effective for some groups—gay men, partners of IV drug users, and commercial sex workers—but that laypeople were more effective than experts for other groups—IV drug users and heterosexuals with multiple sex partners (Durantini et al., 2006). Prevention programs for women were more effective when run by women; younger people responded better when group leaders were also young people; and people of color responded better when group leaders were also people of color. These findings suggest that a "one-size-fits-all" approach to HIV prevention will not work well.

To illustrate the promise of HIV prevention programs, we describe two examples in some detail. One noteworthy study involved over 3,700 heterosexual men and women at the highest risk for HIV infection in 37 sexually transmitted disease (STD) clinics across the United States (NIMH Multisite HIV Prevention Trial Group, 1998). In this study, three-quarters of the participants were African American and the rest were Latino; most were unemployed and single. All met one or more of the following high-risk criteria: having sex with multiple partners, being infected with an STD, having sex with someone known to have multiple partners, having sex with an intravenous drug user, and having sex with someone known to be HIV-positive. A 4-week behavioral intervention applying several of the principles mentioned here, compared with a one-session information-only control group, led to significantly more reduction in unprotected sex, with some indications also of fewer cases of STDs over a 12-month period following the intervention. In addition, the estimated cost per person in the 4-week behavioral intervention was less than $300, which is equivalent to the cost of about 1 week of treatment with the AIDS medications.

An additional large study, the EXPLORE study, conducted across six U.S. cities, included nearly 4,300 men who have sex with men. The men were randomly assigned to either a prevention intervention group or a standard group. Men in the standard group received counseling two times a year on the risks associated with unprotected sex, as recommended by the Centers for Disease Control. Men in the prevention intervention group received 10 individual counseling sessions that focused on individual and social factors that contribute to risky sexual behaviors, such as alcohol and drugs, mood states, communication issues, and pleasure associated with risky sexual behavior. Counselors followed a treatment manual for each of the 10 sessions. At both 12 and 18 months after the interventions, men in the prevention intervention group tended to be less likely than men in the standard group to have been infected with HIV; however, the difference between the two groups in HIV infection rates was not statistically significant. Does this mean the intervention was not successful? Not necessarily. The men in the prevention intervention group engaged in fewer risky sexual behaviors, such as unprotected anal receptive sex and unprotected sex with an HIV-positive partner (EXPLORE Study Team, 2004).

Unfortunately, prevention efforts aimed at women have been less successful. A meta-analysis of all such studies found that despite an abundance of findings on social and contextual risk factors associated with women and HIV, there are few prevention intervention studies designed to address them. The studies that have been done to date do not yield very promising results (Logan, Cole, & Leukefeld, 2002). This is clearly an area in need of research, as women account for about half of the HIV infections worldwide, and the rates of HIV infection among women of color in the United States are much higher than among men or white women.



Quick Summary

The reasons people have hypertension are not well understood, but psychological factors such as Type A behavior pattern and anger as well as behavioral factors such as diet and exercise play a key role. Laboratory and naturalistic studies have shown that stress, anger, Type A, and cardiovascular reactivity all contribute to short-term increases in blood pressure. There is less research showing how these factors are linked to hypertension.

Type A behavior pattern has predicted CHD in some studies but not others. Researchers have looked at more specific components of Type A, and evidence suggests that anger, hostility, and cynicism may be important predictors of CHD. Anxiety and depression are also related to CHD. Cardiovascular reactivity and heart-rate variability are possible biological risk factors for CHD.

Asthma involves severe constriction of the airways; the air passages in the lungs become narrowed, causing extremely labored and wheezy breathing. There are many causes of asthma, several of which involve allergens or air pollutants. Psychological factors, such as stressful life events, negative emotions, and family conflict, may trigger attacks.

AIDS remains an epidemic worldwide, with over half of the 33 million people with HIV living in sub-Saharan Africa. Prevention has been a key focus of psychological researchers since the disease can be prevented with changes in behavior. Much work has been done to change risky sexual behaviors among gay and straight men as well as straight women. These programs have been effective in the short term, but longer-term follow-ups have not yet been conducted. Additional work is needed to develop effective prevention programs for adolescents.

Clinical Case: Juana

Juana was a 58-year-old veterinarian. She was slightly overweight but exercised regularly, did not smoke, and had only an occasional glass of wine with dinner. She had been happily married for 22 years, and her children were both in college. She enjoyed her job quite a bit, despite the long hours and the after-hours calls she often received from worried pet owners.

For several weeks, Juana had been experiencing fatigue and had found herself taking short naps during the day. She had had increasingly frequent bouts of indigestion, and occasionally she had become dizzy or lightheaded. She wondered if these were the beginning signs of menopause. She consulted with her doctor, who examined her and concluded that the symptoms would probably resolve on their own. If they didn't go away in a few weeks, the doctor said, Juana should contact him again. About a week later, Juana began to experience shortness of breath. Then, on a long hike with her husband, she had a fainting spell. Thinking it was the heat, she dismissed it. The next night, she awoke from sleep feeling nauseous and sweaty. She was sure this was a symptom of menopause until she felt a shooting pain in her arm. She awakened her husband, and he took her to the hospital. An electrocardiogram revealed that Juana had had a small heart attack. The next day, Juana had an angiogram that showed three completely occluded coronary arteries. She needed bypass surgery. Beyond her concerns about her health, Juana was quite surprised by this turn of events—didn't heart attacks and heart disease affect men more than women, and shouldn't her doctor have diagnosed her condition?

Gender and Health

At every age from birth to 85 and older, more men die than women. Men are more than twice as likely to die in automobile accidents and of homicides, cirrhosis, heart disease, lung cancer and other lung diseases, and suicide. Women, however, have higher rates of morbidity (i.e., poor heath). That is, general poor health is more frequent among women, and women have a higher incidence of several specific diseases. For example, women have higher rates of diabetes, anemia, gastrointestinal problems, and rheumatoid arthritis; and they report more visits to physicians, use more prescription drugs, and account for two-thirds of all surgical procedures performed in the United States.

What are some of the possible reasons for the differences in mortality and morbidity rates in men and women? It might be that women have some biological mechanism that protects them from certain life-threatening diseases. For example, epidemiological and observational studies suggested that estrogen might offer protection from cardiovascular disease. Based on this evidence, many women began hormone replacement therapy (HRT) following menopause (when estrogen naturally declines) in an attempt to reduce the risk of cardiovascular disease. However,

Research shows that women live longer than men but women have more health problems than men. (Thomas Langreder VISUM/The Image Works.)

the first randomized clinical trial of HRT for postmenopausal women, called the Heart and Estrogen/Progestin Replacement Study, failed to find a reduced risk with respect to one type of cardiovascular disease—coronary heart disease (CHD) (Hulley et al., 1998).

A later randomized trial, called the Women's Health Initiative (WHI), began in 1993 with a sample of over 150,000 women. It was a large, prospective study designed to examine the effects of HRT as possible protective factors against osteoporosis, CHD, and cancer. One group of women was randomly assigned to receive estrogen plus progesterone therapy (combined therapy), another group was randomly assigned to receive estrogen only, and a third group was randomly assigned to receive a placebo. The researchers originally planned to follow women prospectively for 9 years. However, the study was stopped for women in the combined therapy group after 5 years because the data suggested that the combined treatment was *increasing* the risk of CHD, stroke, and breast cancer (Writing Group for the Women's Health Initiative Investigators, 2002). A follow-up assessment of these women was conducted 2 to 3 years after the study was stopped (Heiss et al., 2008). Women who had received the combined therapy were no longer at greater risk for developing CHD and stroke, but they still had a greater risk of developing breast cancer than those who had received placebo. The estrogen-only portion of the study was similarly discontinued after 7 years because there was no evidence for a decreased risk of CHD or breast cancer, though there was a protective effect for hip fractures, which are related to osteoporosis (WHI, 2004). Other findings from this large study suggested that neither form of hormone replacement therapy reduced the risk of dementia and that the combined therapy actually seemed to increase the risk (Shumaker et al., 2003, 2004). These findings may not be the last word on HRT. The WHI study was limited in that most of the women were postmenopausal, leaving open the possibility that HRT during the period of transition to menopause could afford some health benefits.

Early evidence suggested that women are less likely than men to exhibit the Type A behavior pattern and are also less hostile than men (Waldron, 1976; Weidner & Collins, 1993). Newer evidence, however, indicates that anger is not necessarily more commonly experienced and expressed by men (Kring, 2000; Lavoie et al., 2001). Moreover, increased hostility and both the suppression and expression of anger are associated with risk factors for CHD among women (Matthews et al., 1998; Rutledge et al., 2001). In addition, anxiety and depression are more common among women than men (see Chapters 5 and 8) and are also linked to cardiovascular disease (e.g., Suls & Bunde, 2005).

Another question concerns why the gap between mortality rates in men and women is decreasing. In the early twentieth century, most deaths were due to infectious diseases, but now most deaths result from diseases that are affected by lifestyle. One possibility, then, is that lifestyle differences between men and women account for the sex difference in mortality and that these lifestyle differences are decreasing. Although men still smoke more than women and consume more alcohol, women are catching up in their use of alcohol and cigarettes. Not surprisingly then, these behavior changes in women are paralleled by increases in lung cancer and by the failure of the mortality rate for cardiovascular disease to decrease among women (lung cancer has been the leading cause of cancer death among women since 1987). In 2004, a report issued by the National Women's Law Center and the Oregon Health and Sciences University, called "Making the Grade: A National and State-by-State Report Card," showed that 39 states received a failing grade in their efforts to curb smoking among women. Only one state, Utah, received a semisatisfactory grade (Waxman et al., 2004).

Other explanations focus on the identification and treatment of disease in women. For example, even though cardiovascular disease is the number one killer of women and more women than men have died from heart disease since 1984, there is still a widespread belief that men should be more concerned with heart disease than women, as Juana believed. Furthermore, a common diagnostic procedure for heart disease risk, the so-called stress test, which involves measuring heart rate while on a treadmill, is not a good predictor of heart problems among women (Mora et al., 2003), particularly women who do not have chest pains (L.J. Shaw et al., 2006).

In addition, a low dose of aspirin does not appear to prevent myocardial infarction in women as it does in men (though it does appear to prevent stroke in women, which is not true for men) (Ridker et al., 2005). Other research shows that women are less likely to be referred to a cardiovascular rehabilitation program following a heart attack, perhaps contributing to their persistent rates of mortality (Abbey & Stewart, 2000).

There are several possible explanations for the difference in morbidity of men and women. First, because women live longer than men, they may be more likely to experience certain diseases that are associated with aging. Second, women may be more attentive to their health than are men and thus may be more likely to visit physicians and be diagnosed. Third, women are exposed to more stress than men and they rate stress as having a greater impact on them, particularly stress related to major life events (Davis, Matthews, & Twamley, 1999). Fourth, physicians tend to treat women's health concerns and complaints less seriously than men's concerns, as Juana's physician appeared to do (Weisman & Teitelbaum, 1985). Finally, evidence indicates that women's morbidity differs depending on socioeconomic and demographic variables, such as income, education, and ethnicity. For example, having more education and a higher income are associated with fewer risk factors for cardiovascular disease, including obesity, smoking, hypertension, and reduced amounts of exercise. In the United States, women tend to have lower income than men. Also, 16.5 million women in the United States do not have health insurance. However, even after controlling for differences in income level and education, Mexican American and African American women still had a greater likelihood of having more risk factors for CHD than men (Winkleby et al., 1999). Furthermore, death from cardiovascular disease is more common among African American women than European American women (Casper et al., 2000).

Socioeconomic Status, Ethnicity, and Health

Low socioeconomic status (SES) is associated with higher rates of health problems and mortality from all causes. A number of explanations have been proposed for the correlation between SES and poor health and mortality, but many of these are still in need of empirical support. Recent research attempts to trace the connections between health and SES, encompassing economic, societal, relationship, individual, and biological factors. For example, one pathway to poor health has to do with environmental factors that reinforce poor health behaviors. Poorer neighborhoods often have high numbers of liquor stores, grocery stores offering fewer healthy food choices, and fewer opportunities for exercise at health clubs or parks. Given these environmental constraints, it is perhaps not surprising to learn that lower SES people are more likely than higher SES people to engage in behaviors that increase the risk of disease, such as smoking, eating fewer fruits and vegetables, and drinking more alcohol (Lantz et al., 1998).

Other pathways include limited access to health services and greater exposure to stressors. Recall our earlier discussion of allostatic load, the bodily effects of repeated and chronic stress. In a longitudinal study, researchers found that people who were the most economically disadvantaged had the highest allostatic load (Singer & Ryff, 1999). These investigators also found that, regardless of SES, people who reported having poor relationships with parents or negative relationships with spouses had a higher allostatic load than people with positive parental and spousal relationships. Not surprisingly, the combination of lower SES and negative relationships had the greatest effect on allostatic load. These findings indicate that lower SES is a likely source of chronic stress that impacts the body. They also illustrate the complicated relationship of individual, social, and economic factors with health.

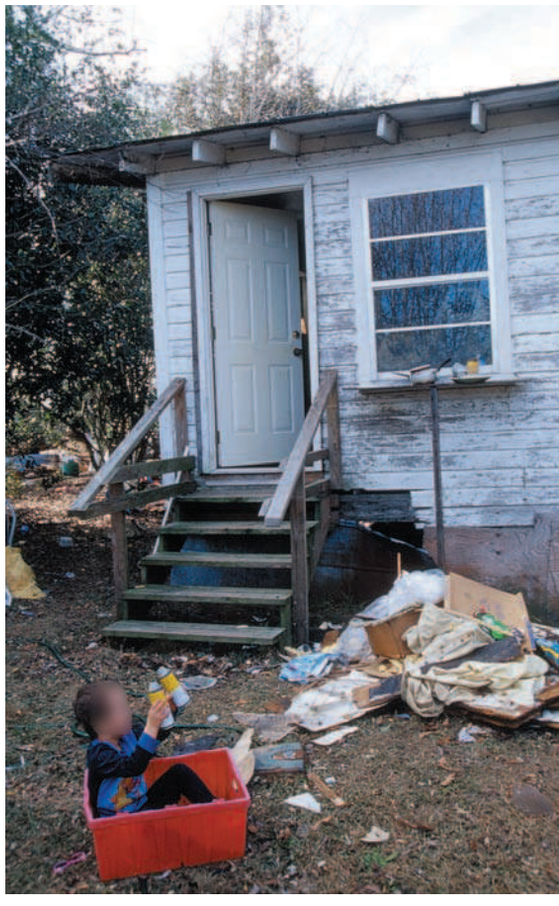
Certainly discrimination and prejudice are sources of chronic stress, and these abhorrent social conditions continue to affect people of color as well as people of lower SES and, in turn, impact health (Mayes, Cochran, & Barnes, 2007). Since people of color are found in high numbers among lower SES groups, ethnicity has also been a feature of research into the relationship of SES to health. Consider, for example, that the mortality rate for African Americans is nearly two times as high as it is for whites in the United States (Williams, 1999). Why might this be? The reasons are complex and not completely understood. Some research suggests that

certain risk factors for disease are more common in people of color. For example, risk factors for cardiovascular disease (such as smoking, obesity, hypertension, and reduced exercise) are higher among women from ethnic minorities than among white women. This finding holds even when members of the two groups are comparable in SES (Winkleby et al., 1998). The increased prevalence of some of these risk factors shows up in studies of children as young as 6 to 9 years of age. For example, African American and Mexican American girls in this age range have higher body mass indexes and higher fat intake than do non-Hispanic white girls (Winkleby et al., 1999). Other studies have found that increased stress associated with discrimination is linked to cardiovascular reactivity among African American women (Guyll, Matthews, & Bromberger, 2001). Consideration of SES at multiple levels, including the individual, family, and neighborhood, is also important (Mayes et al., 2007). For example, lower family SES and neighborhood SES were found to be associated with greater cardiovascular reactivity for African American children and adolescents, but only lower family SES was associated with greater cardiovascular reactivity among white children and adolescents (Gump et al., 1999). In sum, both SES and ethnicity are important factors in health.

Ethnicity is also an important factor in how people cope with cancer, and it is associated with detection of illness, adherence to treatment regimens, survival, and quality of life (these relationships were examined in a review by Meyerowitz et al., 1998). Among the many ethnicity-related findings based on data from the National Cancer Institute are the following:

- African Americans have the highest rates of cancer overall, as a result of very high rates of lung and prostate cancer among men.
- Although African American women have lower breast cancer rates than do white women, their mortality rates 5 years after diagnosis are the same because their survival rates are lower.
- Latino men and women have low rates of cancer in general, but Latina women have high rates of cervical cancer.
- Asian Americans have low rates for all cancers except stomach cancer. These and other findings are illustrative of ethnic differences in the incidence and outcomes of cancer. What might account for these differences? The answers appear to lie less with ethnic and biological factors than with social and psychological factors such as access to and willingness to seek out medical care (e.g., Bach et al., 1999).

As one might expect, the ability to afford health care is not uniform across ethnic and socioeconomic lines. The years of research on SES and health have clearly demonstrated that there is a linear relationship between morbidity/mortality and SES: the higher the SES, the better the health. But the reasons this is so are less clear. Taken together, the explanations posited above can account for part of the relationship between SES and health, but not all. Investigators are now proposing models that cut across these different explanations in an effort to do a better job of accounting for the SES–health link.



Poverty is stressful and is associated with poor health. (Jeff Greenberg/PhotoEdit.)

Quick Summary

Women live longer than men, yet women have poorer health than men. Researchers wondered if sex hormones might protect women from death, but controlled studies of estrogen replacement failed to show that it decreased risk for CHD. Men may be more likely than women to exhibit the Type A behavior pattern, but there are few gender differences in anger, and women are more likely to experience anxiety and depression. Even though CHD is the number one killer of women, some diagnostic procedures (e.g., stress test) and preventative measures (e.g., aspirin therapy) are not as effective for women as they are for men. Women may have poorer health than men because they live longer. Lower socioeconomic status (SES) is associated with poorer health, and women are more likely than men to have lower SES.

The reasons lower SES is linked to poorer health are many and include environmental factors such as limited resources in poorer neighborhoods, greater stress associated with lower SES, less social support, discrimination, and less access to health care. Ethnicity impacts some diseases, such as cancer, but the reasons for this are not well understood. Some differences attributed to ethnicity may have more to do with SES.

Check Your Knowledge 7.4

True or false?

1. Negative emotions don't exacerbate asthma attacks.
2. HIV prevention efforts that emphasize monogamous relationships have been highly successful in Africa.
3. HIV prevention programs have shown promise in the short term, except among young adolescents.
4. Among the reasons women may have poorer health than men is that their concerns are not taken as seriously by doctors.
5. Higher SES is associated with greater allostatic load.

Treatment of Psychological Factors Affecting Medical Conditions

Whether high blood pressure is biologically caused or, as in essential hypertension, linked to psychological stress, a number of medications can reduce it. Asthma attacks can also be alleviated by medications, taken either by inhalation or injection, which dilate the bronchial tubes. The help drugs provide in ameliorating damage and discomfort in particular bodily systems cannot be underestimated. They are frequently lifesaving. Mental health and medical professionals recognize, however, that most drug interventions do not address the fact that the person is reacting to psychological stress. Therapists of all persuasions agree that reducing anxiety, depression, and anger is important for health.

Treating Hypertension and Reducing the Risk of Coronary Heart Disease

Before the advent of effective drugs for treating hypertension in the late 1950s, physicians generally advised patients basically to "take it easy," lose weight, and restrict salt intake—all reasonably helpful measures. It is noteworthy that simple verbal reassurance was also considered important and was even demonstrated to be helpful in an early study (Reiser et al., 1950) in which non-psychiatrically-trained internists (physicians specializing in internal medicine, which includes the diagnosis and treatment of hypertension) provided what we today call nonspecific supportive psychotherapy. Clinically significant reductions in blood pressure were observed after 2 years of regular, albeit infrequent, contact.

The advent of effective drugs to lower blood pressure shifted the direction of treatment strongly toward their use from the 1960s onward. But over time the undesirable side effects of these medications—drowsiness, lightheadedness, and, in men, erectile difficulties—as well as the growth of behavioral approaches to treatment led many investigators to explore nonpharmacological treatments for borderline essential hypertension. Successful nonpharmacological efforts have been directed at losing weight, restricting salt intake, giving up cigarettes, exercising regularly, and reducing alcohol consumption. Still, those with more severe hypertension usually have to take drugs to control its deleterious long-term effects.

Two out of three Americans over the age of 60 have high blood pressure, and more than half of them take costly and sometimes risky hypertensive medication (risky because all drugs pose particularly serious risks to older people). As just mentioned, the importance of losing weight and reducing salt intake has been recognized for many years as useful in keeping blood pressure under control, but until recently there was little optimism that diet and weight loss could play a positive role among older adults, who often have had undesirable dietary habits for a lifetime.

A report from TONE, the controlled Trial of Nonpharmacologic Interventions in the Elderly (Whelton et al., 1998), indicated for the first time that significant benefits can be achieved by people between the ages of 60 and 80 who are obese and who are taking blood pressure medication. Specifically, half the overweight people in the study who reduced their salt intake by 25 percent and lost as little as 8 pounds over the course of 3 months were able to come off their antihypertensive medications and maintain normal blood pressure. The ability to maintain normal blood pressure was achieved by 31 percent of the patients who reduced their salt intake,

36 percent of those who lost weight, and more than half of those who reduced both their salt intake and their weight. Furthermore, these results—the dietary and weight changes as well as the maintenance of normal blood pressure without medication—lasted for more than 3 years.

Research has shown that increasing exercise—for example, walking up stairs rather than using an elevator, and walking short distances rather than driving—yields as much cardiovascular benefit as a structured program of aerobic exercise (Whelton et al., 2002). Other research indicates that people with essential hypertension, as well as those whose blood pressure is within the normal range, should adopt regular exercise habits, such as walking briskly almost every day for about half an hour or engaging in other aerobic exercise that raises the heart and respiration rates (Whelton et al., 2002). Most people can engage in such activity without even checking with their physician if the activity is not so strenuous that it prevents them from carrying on a conversation at the same time. In fact, the research suggests that people with high blood pressure and no other health complications should try exercise for about a year before turning to drugs to lower their blood pressure. For those already taking antihypertension drugs, a regular and not necessarily strenuous exercise regimen can sometimes reduce or even eliminate their dependence on medication. Decreases of 10 points in both systolic and diastolic blood pressure—a significant reduction—can be achieved by most people after just a few weeks. All these beneficial results may be mediated by the favorable effects that exercise has on stress, weight, and blood cholesterol. And if the sense of well-being that accompanies regular exercise and weight loss generalizes to the adoption of other health-enhancing habits, such as stopping smoking and avoiding drinking to excess, the positive effects on blood pressure will be stronger and more enduring.

Exercise, such as walking briskly, can help to reduce blood pressure. (Ariel Skelley/Corbis Images.)

Exercising regularly can also reduce mortality from cardiovascular disease (Blumenthal et al., 2002; Wannamethee, Shaper, & Walker, 1998). One study compared a stress-management intervention with an exercise intervention for men who had a history of CHD (Blumenthal et al., 2002). Patients were randomly assigned to either a weekly stress-management group, an aerobic exercise group, or a no-treatment group. The interventions lasted for 4 months. Five years after the intervention, the men who had received stress-management training or exercise were significantly less likely to have had another cardiac problem than the men who did not receive treatment.

Evidence suggesting the importance of cognitive change, as well as the role of anger, has been reported. Borderline hypertensive patients who achieved significant reductions in angry thoughts showed decreases in blood pressure—as their articulated thoughts became less angry, their blood pressure became lower (Davison, Haaga, et al., 1991). This finding is consistent with the research discussed earlier linking anger with hypertension.

Reducing Anger and Hostility, Depression, and Social Isolation

Reducing anger and hostility has been a focus of behavioral medicine interventions for many years, especially in hypertension and coronary heart disease. These studies generally involved men who had suffered a heart attack, and the focus was on reducing the likelihood that they would have a second one.

Early programs focused on reducing Type A behavior (Friedman et al., 1982). As evidence now shows that the hostility component of Type A behavior is most predictive (Williams, 2001), researchers have targeted hostility more specifically. In one study, men who had experienced myocardial infarction were randomly assigned to a hostility reduction treatment or an information-only treatment. Men who received the hostility treatment reported less hostility, were rated as less hostile, and evidenced a decrease in diastolic blood pressure (Gidron, Davidson, & Bata, 1999).

The focus on anger and hostility has been widened to include depression and social isolation, also discovered to be risk factors in cardiovascular disorders as well as in other illnesses. The Enhancing Recovery in Coronary Heart Disease (ENRICHD) project targeted depression and social isolation among people recovering from myocardial infarction. Although the treatment was successful in reducing depression and social isolation, those in the treatment group did not have better heart health at the end of the study compared to the control group (Writing Committee for the ENRICHD Investigators, 2003). See Focus on Discovery 7.1 for a discussion of coping with cancer.

FOCUS ON DISCOVERY 7.1

Coping with Cancer

An optimistic, upbeat attitude can be important in combating illness, including illnesses as serious as cancer (Carver et al., 1993) and HIV-positive status (Taylor et al., 1992). The mechanism by which an optimistic attitude helps people with life-threatening illnesses may be its link to adaptive coping. Optimistic people may be more likely to engage in risk-reducing behaviors such as avoiding risky sex or engaging in prescribed regular exercise following coronary bypass surgery (Scheier & Carver, 1987).

Psychological Interventions to Help People Cope

Problem-solving therapy (PST) has shown its value in helping cancer patients cope with the myriad life challenges facing them, from daily hassles to dealing with isolation and depression (Nezu et al., 1997). An important component of PST is that it gives patients an enhanced sense of control, which seems to be particularly important for people with a life-threatening illness who are experiencing the side effects of treatment. PST has also been shown to be helpful for caregivers, who have to cope with the patient's many cancer-related problems, including fears about death, severe fatigue, and medication side effects such as hair loss (Bucher et al., 1999).

The stressfulness of a cancer diagnosis and its treatment makes stress-management approaches relevant, like those described earlier. Clear information about the treatment procedures themselves, including what the patient is likely to experience during and following treatment, as well as training in relaxation and hypnosis, can be helpful in reducing anxiety both prior to and following various cancer treatments. It is especially important that patients understand that fatigue is a natural accompaniment of many treatments for cancer, especially radiation and chemotherapy. A common side effect is conditioned food aversions that result from associating certain foods with nausea from chemotherapy or radiation (Chambers & Bernstein, 1995).

Interventions to Encourage Prevention

Psychological interventions also focus on preventing cancer by encouraging healthy behaviors and discouraging unhealthy ones. For example, logical programs to help people stop smoking are discussed in Chapter 10. Other interventions are aimed at getting women to perform breast self-examination (BSE). The main hurdle in accomplishing this is that BSE significantly raises the probability of an aversive consequence, that is, finding a lump. Logically, it is better to take this risk than not, but the fact is that the fear of learning something unpleasant is a major deterrent to doing the exam (Mahoney, 1977). For the same reason, many high-risk women (those with first-degree relatives who had breast cancer) do not have regular mammograms (Vogel et al., 1990).

In an effort to develop ways to help women perform BSE regularly, researchers compared two pamphlets on BSE (Meyerowitz & Chaiken, 1987). One contained persuasive arguments for performing BSE while emphasizing the negative consequences of not performing BSE; the other emphasized the positive consequences of performing BSE. Both pamphlets also contained factual information about breast cancer and instructions about how to do BSE. In the following examples from these pamphlets, words in parentheses were included in the positive condition, those in brackets in the negative.

> *By [not] doing BSE now, you (can) [will not] learn what your normal healthy breasts feel like so that you will be (better) [ill] prepared to notice any small, abnormal changes that might occur as you get older.*
>
> *Research shows that women who (do) [do not do] BSE have (an increased) [a decreased] chance of finding a tumor in the early, more treatable stage of the disease. (Meyerowitz & Chaiken, 1987, p. 504)*

Stress Management

Stress management is a set of techniques for helping people who are seldom labeled as patients (e.g., hospital personnel, factory workers, and students) to cope with the challenges that life poses for all of us. Stress management has also been used successfully for several specific diseases, including tension headaches, cancer, hypertension, AIDS, coronary heart disease, and chronic pain (e.g., Antoni et al., 2000). For CHD, stress management is most effective when the intervention focuses on behavioral targets, such as quitting smoking, exercising regularly, and reducing blood pressure (Dusseldorp et al., 1999).

Stress management encompasses a variety of techniques, and more than one is typically used in any given instance (Davison & Thompson, 1988; Lehrer & Woolfolk, 1993; Steptoe, 1997). Techniques include the following:

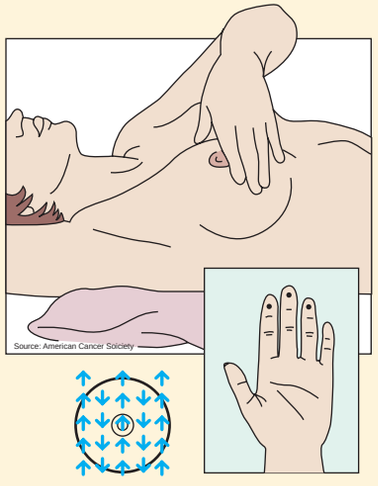
- *Relaxation training.* The most common form of relaxation training is progressive muscle relaxation, which involves systematically tensing and then relaxing each major muscle group

A community psychology approach to stress management focuses on the environment rather than on the person. To reduce stress, an office can be redesigned with partitions to provide some privacy for employees. (Dick Luria/Photo Researchers, Inc.)

The groups given the different pamphlets did not differ in their attitudes toward BSE immediately after reading the pamphlets. However, four months later, women who had received the negatively framed information were more likely to have engaged in BSE. This effect may have occurred because those who do not engage in regular BSE take an ignorance-is-bliss attitude, but by making the possible negative consequences of not doing BSE more salient, the pamphlet made doing the exam more acceptable. This finding is particularly important because most information intended to help women do BSE stress the positive rather than the negative.

Other researchers extended this work by asking whether messages of the kind that were found effective in encouraging BSE—sometimes referred to as "loss-frame" communications—might under certain circumstances be less effective than messages that emphasize the benefits of a certain behavior (referred to as "gain-frame" messages). The results from two experiments suggest that loss-frame communications are superior when people are being encouraged to detect signs of illness, while gain-frame communications are better when people are being encouraged to prevent illness (Rothman & Salovey, 1997).

What is the difference? BSE and mammography are designed to detect signs of illness. The person lacks information about whether she has an illness and has to decide whether to engage in behavior that could provide information on her status, in this case, whether she might have breast cancer. In contrast, a person going to the beach has a choice about putting on sunscreen with a protection factor of 15 or greater, the strength recommended to reduce the chances of getting skin cancer in the future. Such behavior is preventive in nature, that is, it is designed to have a direct effect on reducing the chances of getting skin cancer.



Encouraging women to perform breast self-examination (BSE) can lead to earlier detection of cancer and better treatment outcomes. Shown here is an advertisement from the American Cancer Society demonstrating how to perform BSE.

In a study that replicated the findings of Meyerowitz and Chaiken, women told of the risks arising from not taking a mammography exam—the loss-frame approach—were more likely to have a mammogram than were women told of the benefits of doing so (Banks et al., 1995). In a later study, sunbathers at a beach who were told of the benefits of using sunscreen—a gain-frame communication—were more likely to use sunscreen after reading a communication that emphasized the benefits of such preventive health behavior than were those who read something that focused on the risks of not using sunscreen (Detweiler et al., 1999).

The implications of these studies are important. If one wants to foster healthful detection-related behaviors, one should emphasize the risks associated with not taking a certain action that could detect a problem. But if one wants to foster healthful preventive behaviors, it is best to emphasize the benefits that arise from doing something that can forestall a problem.

in the body. Teaching people to relax deeply and to apply these skills to real-life stressors can be helpful in lowering their stress levels. There is also evidence that the immune function can be improved by relaxation training (Jasnoski & Kugler, 1987; Kiecolt-Glaser et al., 1985), although enduring benefits are doubtful unless relaxation is practiced regularly over a long period of time (Davison & Thompson, 1988; Goldfried & Davison, 1994).

- *Cognitive restructuring*. Cognitive restructuring includes approaches to alter people's belief systems and reduce the negativity of their interpretations of experience. Providing information to reduce uncertainty and enhance the person's sense of control has also been helpful in reducing stress.
- *Behavioral skills training*. Because it is natural to feel overwhelmed if one lacks the skills to execute a challenging task, stress management often includes instruction and practice in skills such as time management and effective prioritizing. Also included under this rubric is training in assertion skills—expressing likes and dislikes without encroaching on the rights of others.
- *Environmental-change approaches*. Whereas the other individual strategies aim at helping the person deal with a particular environment, environmental-change approaches take the position that sometimes the environment is the problem and that efforts are best directed at altering it. One kind of environmental approach draws on research on the positive role of

social support on health. If social support helps keep people healthy or helps them cope with illnesses, then it is reasonable to assume that enhancing such support can only be beneficial. Another kind of environmental change involves the workplace. Altering management practices or providing greater privacy and fewer interruptions can reduce stress in the world where people work and live for a significant portion of their waking hours (Murphy et al., 1995).

Cognitive Behavioral Interventions

Cognitive behavioral interventions have shown some success. For asthma sufferers, behavioral treatments that emphasize breathing training are associated with taking fewer medications, but they do not really improve lung functioning significantly (Ritz & Roth, 2003). For men and women with AIDS, group-based cognitive behavioral treatments have been effective in reducing distress, increasing coping and perceived social support, and reducing depression (Lechner et al., 2003; Lutgendorf et al., 1997). Bereavement group therapy for HIV-positive gay men who had lost a partner or close friend to AIDS was beneficial in decreasing distress and viral load, a biological predictor of the progression to AIDS (Goodkin et al., 2001).

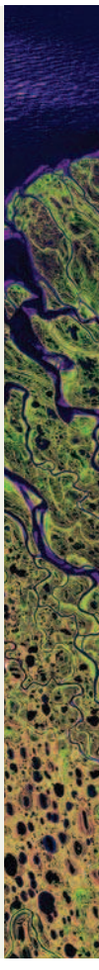
Check Your Knowledge 7.5

True or False?

1. Behavioral interventions aimed at reducing salt intake and increasing exercise are effective in reducing CHD risk.
2. Stress management involves arousal reduction, cognitive restructuring, behavioral skills training, and environmental change.
3. Cognitive behavioral approaches have been shown to be effective with AIDS and, to a lesser extent, asthma.

Summary

- Psychological factors affecting a medical condition refers to diseases produced or influenced in part by psychological factors, including stress, social support, and negative emotions.
- In attempting to understand the complex stress–illness relationship, researchers have focused on precisely defining what stress is, on assessing differences in how people cope with perceived stress, and on how social support impacts the stress–illness relationship.
- Theories of the stress–illness relationship are diathesis–stress in nature but differ in whether the diathesis is described in psychological or neurobiological terms. Theories emphasizing a neurobiological diathesis emphasize the effects of allostatic load or changes in the immune system that are caused by stress. Theories emphasizing a psychological diathesis focus on such factors as emotional states, personality traits, cognitive appraisals, and specific styles of coping with stress. The most successful accounts of etiology are those that integrate psychological and neurobiological factors.
- Cardiovascular disorders, which involve the heart and circulatory system, include essential hypertension and coronary heart disease (CHD). While both conditions are complex and multifaceted, their etiologies appear to include a tendency to respond to stress with increases in blood pressure or heart rate. Anger, hostility, cynicism, anxiety, and depression are linked to these conditions.
- People with asthma tend to have respiratory systems that overrespond to allergens or that have been weakened by prior infection. Psychological factors such as anxiety, anger, depression, stressful life events, and family conflict may trigger an asthma attack.
- Acquired immunodeficiency syndrome (AIDS) has psychological elements in that it can be preventable by psychological means. The primary focus of prevention is to change people's behavior—specifically, to encourage safer sex and to discourage the sharing of needles in intravenous substance abuse.



- Treatment for CHD, hypertension, asthma, and AIDS usually includes medication. The general aim of psychotherapies for these disorders is to reduce stress, anxiety, depression, or anger.
- Researchers in the field of behavioral medicine try to find psychological interventions that can improve patients' physiological state by changing unhealthy behaviors and reducing stress. They have developed ways of helping people relax, smoke less, eat fewer unhealthy foods, and engage in behaviors that can prevent or alleviate illnesses, such as encouraging breast self-examination and adhering to medical treatment recommendations.
- Stress management interventions help teach people techniques to cope with stress and thereby ameliorate the toll that stress can take on the body.

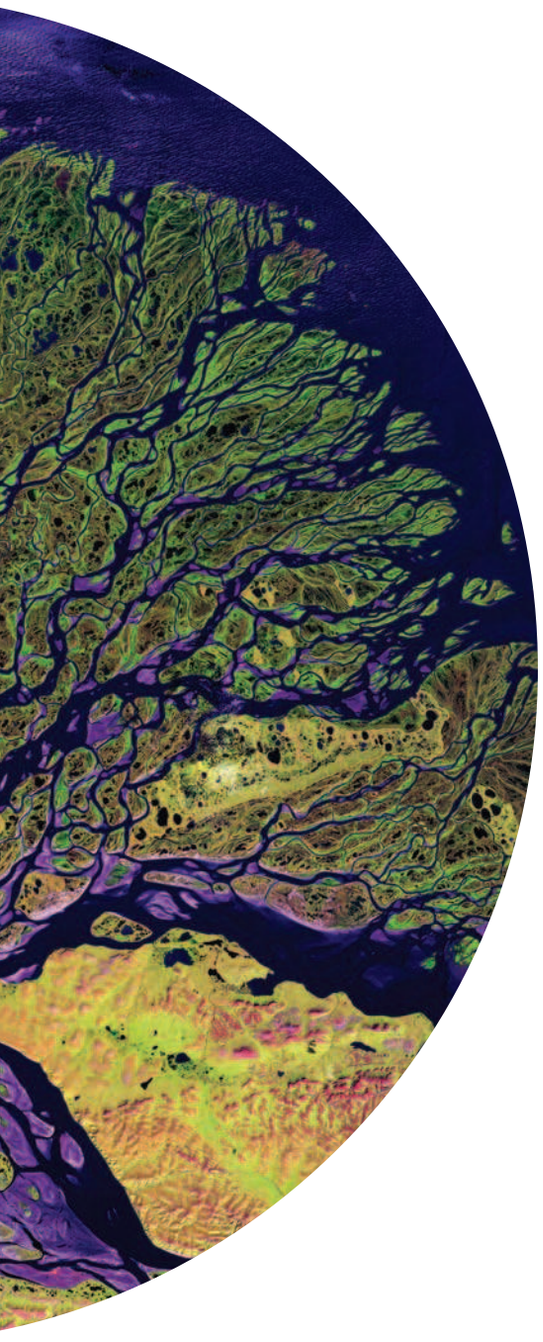
Answers to Check Your Knowledge Questions

7.1 1. a; 2. b; 3. d
7.2 1. T; 2. F; 3. T; 4. F
7.3 1. Essential or primary; 2. essential hypertension, CHD; 3. blood pressure, hypertension
7.4 1. F; 2. F; 3. T; 4. T; 5. F
7.5 1. T; 2. T; 3. T

Key Terms

AIDS (acquired immuno deficiency syndrome)
allostatic load
anger-in theory
angina pectoris
asthma
behavioral medicine
cardiovascular disorders
coping
coronary heart disease (CHD)
cytokines
essential hypertension
functional social support
health psychology
interleukin-6 (IL-6)
metabolic syndrome
myocardial infarction
psychological factors affecting medical condition
psychoneuroimmunology
stress management
structural social support
Type A behavior pattern

8 Mood Disorders



LEARNING GOALS

1. Be able to describe the symptoms of depression and mania, the diagnostic criteria for depressive disorders and bipolar disorders, and the epidemiology of these disorders.
2. Be able to discuss the genetic, neurobiological, social, and psychological factors that contribute to the mood disorders.
3. Be able to identify the medication and psychological treatments of depressive and manic symptoms as well as the current views of electroconvulsive therapy.
4. Be able to explain the epidemiology of suicide as well as the neurobiological, social, and psychological risk factors associated with suicide and the methods for preventing suicide.

Clinical Case: Mary

Mary M., a 38-year-old mother of four children, had been deeply depressed for about 2 months when she first went to see a psychologist. Three years earlier, she had returned to work when health care bills made it hard for her family to get by on her husband's income as a high school teacher. About 7 months before her visit to the psychologist, she was laid off from her job as an administrative assistant, which was a serious blow to the family's finances. She felt guilty about the loss of her job and became preoccupied with signs of her overall incompetence. Each night, she struggled for more than an hour to fall asleep, only to wake up frequently throughout the night. She had little appetite and as a result had lost 10 pounds. She also had little energy for and no interest in activities that she had enjoyed in the past. Household chores became impossible for her to do, and her husband began to complain. Their marriage had already been strained for two years, and her negativity and lack of energy contributed to further arguments. Finally, realizing that something serious had happened to his wife, Mr. M. cajoled her into making an appointment with a psychologist. (You will read about the outcome of Mary's treatment later in this chapter.)

MOOD DISORDERS INVOLVE DISABLING disturbances in emotion—from the extreme sadness and disengagement of depression to the extreme elation and irritability of mania. In this chapter, we begin by discussing the clinical description and the epidemiology of the different mood disorders. Next, we consider various perspectives on the etiology of these disorders, and then we consider approaches to treating them. We conclude with an examination of suicide, an action far too often associated with mood disorders.

Clinical Descriptions and Epidemiology of Mood Disorders

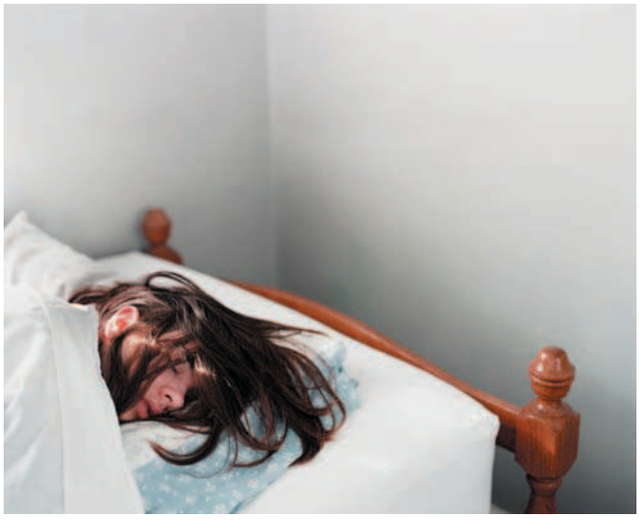
DSM-IV-TR recognizes two broad types of **mood disorders**: those that involve only depressive symptoms and those that involve manic symptoms (bipolar disorders). We begin by considering the signs of depression, the formal criteria for diagnosis of the depressive disorders (including major depressive disorder and dysthymic disorder), and the epidemiology and consequences of depressive disorders. Then we turn to bipolar disorders. There, we describe the signs of mania, followed by the formal criteria for diagnosing bipolar I disorder, bipolar II disorder, and cyclothymic disorder, and then the epidemiology and consequences of bipolar disorders. Table 8.1 presents a summary of the symptoms of each of these disorders. After covering the basic diagnostic categories, we describe DSM-IV-TR subtypes that are used to further define depressive disorders and bipolar disorders.

Depressive Disorders

The cardinal symptoms of depression include profound sadness and/or an inability to experience pleasure. Most of us experience sadness during our lives, and most of us say that we are "depressed" at one time or another. But most of these experiences do not have the intensity and duration to be diagnosable. The author William Styron (1992) wrote about his depression, "Like anyone else I have always had times when I felt deeply depressed, but this was something altogether new in my experience—a despairing, unchanging paralysis of the spirit beyond anything I had ever known or imagined could exist."

When people develop a depressive disorder, their heads may reverberate with self-recriminations. Like Mary, described in the clinical case, they may become focused on their flaws and deficits. Paying attention can be so exhausting that they have difficulty absorbing what they read and hear. They often view things in a very negative light, and they tend to lose hope.

Physical symptoms of depression are also common, including fatigue and low energy as well as physical aches and pains. These symptoms can be profound enough to convince afflicted persons that they must be suffering from some serious medical condition, even though the symptoms have no apparent physical cause (Simon et al., 1999). Although people with depression typically feel exhausted, they may find it hard to fall asleep and may wake up frequently. Other people sleep throughout the day. They may find that food



Some people with depression have trouble falling asleep and staying asleep. Others find themselves sleeping for more than 10 hours but still feeling exhausted. (Shannon Fagan/Stone/Getty Images.)

Table 8.1 A Summary of Mood Disorder Diagnoses

Depressive Disorders	Diagnostic Criteria	Bipolar Disorders	Diagnostic Criteria
Major depressive disorder	Sad mood or loss of pleasure for 2 weeks, along with at least four other symptoms	Bipolar I disorder	At least one lifetime manic or mixed episode
		Bipolar II disorder	At least one lifetime episode of hypomania and episodes of major depression
Dysthymic disorder	Mood is down and other symptoms are present at least 50 percent of the time for at least 2 years	Cyclothymic disorder	Recurrent mood changes from high to low, without manic episodes for at least 2 years

DSM-IV-TR Criteria for Major Depressive Disorder

Sad mood or loss of pleasure in usual activities.
At least four of the following:

- Sleeping too much or too little
- Psychomotor retardation or agitation
- Poor appetite and weight loss, or increased appetite and weight gain
- Loss of energy
- Feelings of worthlessness
- Difficulty concentrating, thinking, or making decisions
- Recurrent thoughts of death or suicide

Symptoms are present nearly every day, most of the day, for at least 2 weeks.
The symptoms are not due to normal bereavement.

tastes bland or that their appetite is gone, or they may experience an increase in appetite. Sexual interest disappears. Some may find their limbs feel heavy. Thoughts and movements may slow for some (*psychomotor retardation*), but others cannot sit still—they pace, fidget, and wring their hands (*psychomotor agitation*). Beyond these cognitive and physical symptoms, initiative may disappear. Social withdrawal is common; many prefer to sit alone and be silent. Some people with depression neglect their appearance. When people become utterly dejected and hopeless, thoughts about suicide are common.

Major Depressive Disorder The DSM-IV-TR diagnosis of **major depressive disorder (MDD)** requires depressive symptoms to be present for at least 2 weeks. These symptoms must include either depressed mood or loss of interest and pleasure. As shown in the DSM-IV-TR criteria, at least four additional symptoms must be present, such as changes in sleep, appetite, concentration or decision-making, feelings of worthlessness, suicidality, or psychomotor agitation or retardation.

MDD is called an **episodic disorder**, because symptoms tend to be present for a period of time and then clear. Even though episodes tend to dissipate over time, an untreated episode may stretch on for 5 months or even longer. For a small percentage of people, the depression becomes chronic—the person does not completely snap back to the prior level of functioning. Even among those who improve enough that they no longer meet the criteria for diagnosis of MDD, some people experience subclinical depression for years (Judd et al., 1998).

Major depressive episodes tend to recur—once a given episode clears, a person is likely to experience another episode. About two-thirds of people with an episode of major depression will experience at least one more episode during their lifetime (Solomon et al., 2000). The average number of episodes is about four (Judd, 1997). With every new episode that a person experiences, his or her risk for experiencing another episode goes up by 16 percent (Solomon et al., 2000).

There is controversy about whether a person with five symptoms lasting two weeks (i.e., someone who meets the criteria for diagnosis with MDD) is distinctly different from someone who has only three symptoms for 10 days (i.e., someone who meets the criteria for so-called *subclinical depression*). A study of twins found that subclinical depression predicted the occurrence of future episodes of MDD and even the diagnosis of MDD in a co-twin. That is, when one twin had subclinical depression, both twins were likely to have future episodes of major depression (Kendler & Gardner, 1998). Another study found that subclinical depression resulted in as much trouble functioning in everyday activities as did a diagnosis of MDD (Gotlib, Lewinsohn, & Seeley, 1995).

Kirsten Dunst has described her problems with major depressive disorder (MDD). One out of every five women will experience an episode of depression during her lifetime. (Allstar Picture Library/Alamy.)

Dysthymic Disorder People with **dysthymic disorder** (also called *dysthymia*) are chronically depressed—more than half of the time for at least two years, they feel blue or derive little pleasure from usual activities and pastimes. In addition, they have at least two of the other symptoms of depression. DSM-IV-TR distinguishes dysthymia from MDD by the initial duration, type, and number of symptoms. Compare the diagnostic criteria for MDD and dysthymic disorder—note that one of the diagnostic criteria for dysthymia is that the person does not have enough symptoms to warrant a diagnosis of MDD. Over time, though, symptoms of dysthymic disorder tend to worsen if people do not receive treatment; in a 10-year follow-up study, 95 percent of patients with dysthymic disorder developed MDD (Klein, Shankman, & Rose, 2006). The key difference between dysthymic disorder and MDD is chronicity.

DSM-IV-TR Criteria for Dysthymic Disorder

Depressed mood more than half of the time for two years.
At least two of the following during that time:

- poor appetite or overeating
- sleeping too much or too little
- poor self-esteem
- trouble concentrating or making decisions
- hopelessness

The symptoms do not clear for more than two months at a time.
No major depressive episode was present during the first two years of symptoms.

Epidemiology and Consequences of Depressive Disorders MDD is one of the most prevalent psychiatric disorders. One large-scale epidemiological study in the United States estimated that 16.2 percent of people meet the criteria for diagnosis of MDD at some point in their lives (Kessler Berglund, et al., 2005). Dysthymia is rarer than MDD: about 2.5 percent of people meet criteria for dysthymia during their lives (Kessler et al., 2005).

MDD is approximately two times more common among women than among men (see Focus on Discovery 8.1 for a discussion of possible reasons for this gender difference in rates of MDD). Socioeconomic status also matters—that is, MDD is three times as common among people who are impoverished compared to those who are not (Kessler et al., 2005).

The prevalence of depression varies considerably across cultures. In a major cross-cultural study using the same diagnostic criteria and structured interview in each country, prevalence of MDD varied from a low of 1.5 percent in Taiwan to a high of 19 percent in Beirut, Lebanon (Weissman et al., 1996). Similar findings have emerged in a study of depression rates among 26,000 people receiving care through primary care doctors in 14 countries (Simon et al., 2002). Another study yielded the intriguing result that people who have moved to the United States from Mexico have lower rates of MDD and other psychiatric disorders than do people of Mexican descent who were born in the United States (Vega et al., 1998). Why? People have speculated that traditional Mexican

FOCUS ON DISCOVERY 8.1

Gender Differences in Depression

Major depression occurs about twice as often in women as in men. Similar gender ratios in the prevalence of depression have been documented in many countries around the world, including the United States, France, Lebanon, and New Zealand (Weissman & Olfson, 1995). Intriguingly, the ratio does not hold in some cultural groups. For example, this gender difference does not hold between Jewish adults, because depression is more common among Jewish men than among other men (Levav et al., 1997). But for most ethnic and cultural groups, a clear gender difference in MDD begins to emerge during early adolescence and is documented consistently by late adolescence. Some of you might wonder if these findings just reflect a tendency for men to be less likely to describe symptoms. So far, evidence does not support that idea (Kessler, 2003). Although a fair amount of research has focused on hormonal factors that could explain the vulnerability of women, findings have been mixed for this idea, too (Brems, 1995). Several social and psychological factors may help explain this gender difference (Nolen-Hoeksema, 2001):

- Twice as many girls as boys are exposed to childhood sexual abuse.
- During adulthood, women are more likely than men to be exposed to chronic stressors such as poverty and caretaker responsibilities.
- Acceptance of established social roles among girls may intensify self-critical attitudes about appearance. Adolescent girls worry more than adolescent boys about their body image, a factor that does appear tied to depression (Hankin & Abramson, 2001).
- Social roles may interfere with pursuit of some potentially rewarding activities that are not considered "feminine."
- Exposure to childhood and chronic stressors, as well as the effects of female hormones, could change the reactivity of the HPA axis, a biological system guiding reactions to stress.
- A focus on gaining approval and closeness within interpersonal relationships, which is more commonly endorsed by women, may intensify reactions to interpersonal stressors (Hankin, Mermelstein, & Roesch, 2007).
- Social roles promote emotion-focused coping among women, which may then extend the duration of sad moods after major stressors. More specifically, women tend to spend more time ruminating about sad moods or wondering about the reasons why unhappy events have occurred. Men tend to spend more time using distracting or action-focused coping, such as playing a sport or engaging in other activities that shake off the sad mood. A fair amount of research suggests that rumination will intensify and prolong sad moods (Nolen-Hoeksema, Morrow, & Fredrickson, 1993).

The gender difference in depression does not emerge until adolescence. At that time, young women encounter many stressors and more pressure concerning social roles and body image, and they tend to ruminate about the resulting negative feelings. (Jeff Greenberg/Photo Researchers.)

In all likelihood, gender differences in depression are related to multiple factors. In considering these issues, bear in mind that men are more likely to demonstrate other types of disorders, such as alcohol and substance abuse as well as and antisocial personality disorder (Kessler et al., 1994). Hence, understanding gender differences in psychopathology is likely to require attending to many different risk factors and syndromes.

FOCUS ON DISCOVERY 8.2

Seasonal Affective Disorder: The Winter Blues

Criteria for the seasonal subtype of MDD specify that a person experiences depression during two consecutive winters and that the symptoms clear during the summer. These winter depressions appear to be much more common in northern climates than southern climates; while less than 2 percent of people living in sunny Florida report these patterns, about 10 percent of people living in New Hampshire report **seasonal affective disorder** (Rosen et al., 1990).

For mammals living in the wild, a slower metabolism in the winter could have been a lifesaver during periods of scarce food. For some unlucky humans, though, this same mechanism might contribute to seasonal affective disorder. It is believed that seasonal affective disorder is related to changes in the levels of melatonin in the brain. Melatonin is exquisitely sensitive to light and dark cycles and is only released during dark periods. People with seasonal affective disorder show greater changes in melatonin in the winter than do people without seasonal affective disorder (Wehr et al., 2001).

This woman is having light therapy, which is an effective treatment for patients with seasonal depression. (David White /Alamy.)

Fortunately, several treatment options are available for seasonal affective disorder. Like other subtypes of depression, seasonal affective disorder responds to antidepressant medications and cognitive behavioral therapy (Rohan et al., 2007). Winter blues, though, are as likely to remit with 30 minutes of bright light each morning as with fluoxetine (Prozac) (Lam et al., 2006). At least eight high-quality studies have examined bright light as a treatment for seasonal affective disorder (Golden et al., 2005), and it is established as a first-line recommendation in the American Psychiatric Association treatment guidelines for depression.

In one surprising finding, researchers were trying to find a good control condition to compare to bright light. To have a similar apparatus, they used negative air ionizers. Researchers were surprised when the control condition actually worked! That is, patients who received 30-minute intense doses of negative air ions each morning also demonstrated significant improvements in mood (Terman, Terman, & Ross, 1998). Researchers are continuing to examine the use of negative air ionizers (Terman & Terman, 2006).

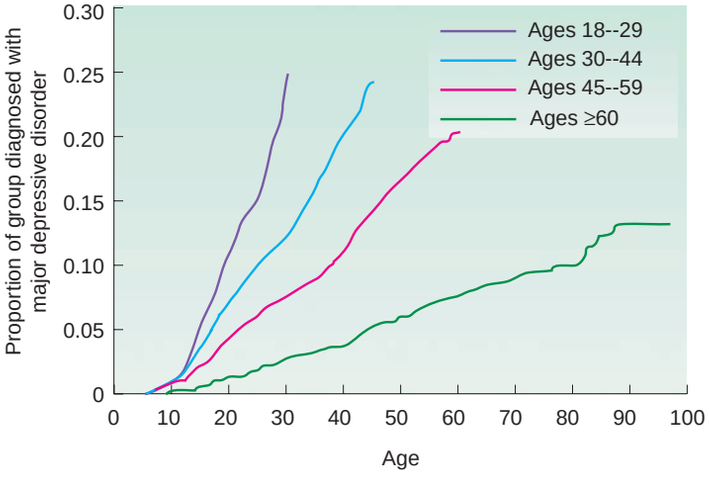


Figure 8.1 With each generation, the median age of onset for major depression gets younger. Adapted from Kessler et al. (2003). *JAMA, 289*, 3095–3105.

values may help protect against depression or that the resiliency of people who are able to immigrate could be protective. Symptoms of depression also show some cross-cultural variation, probably resulting from differences in cultural standards of acceptable expressions of emotional distress. For example, people in South Korea are less likely to describe a sad mood or suicidal thoughts than are people in the United States (Chang et al., 2008). Complaints of nerves and headaches are common in Latino culture, and reports of weakness, fatigue, and poor concentration are common in some Asian cultures. On the other hand, these symptom differences do not appear to be major enough to explain the differing rates of depression across countries (Simon et al., 2002).

It is tempting to assume that differences in prevalence rates by country indicate a strong role for culture. It turns out that differences between countries in rates of depression may be fairly complex to understand. As noted in Focus on Discovery 8.2, one factor may be distance from the equator. Rates of winter depression, or seasonal affective disorder, are higher farther from the equator, where days are shorter. There is also a robust correlation of per capita fish consumption with depression; countries with more fish consumption, such as Japan and Iceland, have much lower rates of MDD and bipolar disorder (Hibbeln et al., 2006). Undoubtedly, cultural and economic factors, such as wealth disparity and family cohesion, play an important role in rates of depression as well.

In most countries, the prevalence of MDD increased steadily during the mid to late twentieth century (Klerman, 1988); at the same time, the age of onset decreased. Figure 8.1 shows that the age of onset has become lower for each recent generation of people in the United States: among people in their sixties, less than 5 percent reported that they had experienced an episode

of MDD by age 20, whereas among people ages 18–29, almost 10 percent reported that they had experienced an episode of MDD by age 20. The median age of onset is now the late teens to early twenties. One possible explanation for the increasing depression rates lies in the social changes that have occurred over the past 100 years. Support structures—such as a tightly knit extended families and marital stability, which were a more central part of society in the past—are often absent for people today. Yet there are no clear data about why depression seems to strike earlier and earlier. Beyond the prevalence rates, the symptoms of depression vary somewhat across the life span. Depression in children often results in somatic complaints, such as headaches or stomachaches (see p. 433). In older adults, depression is often characterized by distractibility and complaints of memory loss (see p. 478).

Both MDD and dysthymia are often associated, or comorbid, with other psychological problems. The most common comorbid conditions include anxiety disorders, substance-related disorders, sexual dysfunctions, and personality disorders. As many as two-thirds of people who meet the criteria for diagnosis of MDD during their lifetime also will meet the criteria for diagnosis of an anxiety disorder at some point (Mineka, Watson, & Clark, 1998).

Depression has many serious consequences. As we will discuss later, suicide is a real risk. MDD is also one of the world's leading causes of disability (Murray & Lopez, 1996); it is estimated that MDD is associated with $31 billion per year in lost productivity in the United States (Stewart et al., 2003). MDD is also related to a high risk of other health problems, particularly cardiovascular disease (Osby et al., 2001).

Although the diagnostic criteria for dysthymia require fewer symptoms than for MDD, do not make the mistake of thinking that dysthymia is a less severe disorder than MDD. Unlike MDD, dysthymia is chronic. One study found that the average duration of dysthymic symptoms was more than five years (Klein et al., 2006). The chronicity of these symptoms takes a toll. Indeed, a study following patients for five years found that people with dysthymia were more likely to require hospitalization, to attempt suicide, and to be impaired in their functioning than were people with MDD (Klein et al., 2000).

Margaret Trudeau, the former first lady of Canada, has become an advocate for better mental health services since her own diagnosis with bipolar disorder. (Neil Burstyn/NewsCom.)

Bipolar Disorders

DSM-IV-TR recognizes three forms of bipolar disorders: bipolar I disorder, bipolar II disorder, and cyclothymic disorder. Manic symptoms are the defining feature of each of these disorders. The bipolar disorders are differentiated by how severe and long-lasting the manic symptoms are.

These disorders are labeled "bipolar" because most people who experience mania will also experience depression during their lifetime (mania and depression are considered opposite poles). An episode of depression is not required for a diagnosis of bipolar I, but it is required for a diagnosis of bipolar II disorder. Most people seeking treatment for bipolar I disorder are likely to have experienced depressive episodes (Johnson & Kizer, 2002).

Mania is a state of intense elation or irritability accompanied by other symptoms shown in the diagnostic criteria. During a manic episode, people may become louder and make an incessant stream of remarks, sometimes full of puns, jokes, rhymes, and interjections about nearby stimuli that have attracted their attention (like Wayne in the clinical case). These remarks may be difficult to interrupt and may shift rapidly from topic to topic, reflecting an underlying **flight of ideas**. During mania, people may become sociable to the point of intrusiveness. They can also become excessively self-confident. Unfortunately, they can be oblivious to the potentially disastrous consequences of their behavior, which can include imprudent sexual activities, overspending, and reckless driving. They may stop sleeping but stay incredibly energetic. Attempts by others to curb such excesses can quickly bring anger and even rage. Mania usually comes on suddenly over a period of a day or two.

Most manic episodes are "purely" manic, but sometimes, people experience **mixed episodes**, characterized by severe symptoms of both mania and depression within the same week. DSM-IV-TR also includes criteria for **hypomania** (see diagnostic criteria for mania and hypomania). *Hypo-* comes from the Greek for "under"; hypomania is "under"—less extreme than—mania. Although mania involves significant impairment, hypomania does not. Rather, hypomania involves a change in functioning that does not cause serious problems.

DSM-IV-TR Criteria for Manic and Hypomanic Episodes

Distinctly elevated or irritable mood.
At least three of the following (four if mood is irritable):

- Increase in goal-directed activity or physical restlessness
- Unusual talkativeness; rapid speech
- Flight of ideas or subjective impression that thoughts are racing
- Decreased need for sleep
- Inflated self-esteem; belief that one has special talents, powers, or abilities
- Distractibility; attention easily diverted
- Excessive involvement in pleasurable activities that are likely to have undesirable consequences, such as reckless spending, sexual behavior, or driving.

For a manic episode:

- Symptoms last for 1 week or require hospitalization
- Symptoms cause significant distress or functional impairment.

For a hypomanic episode:

- Symptoms last at least 4 days
- Clear changes in functioning that are observable to others, but impairment is not marked.

Clinical Case: Wayne

Wayne, a 32-year-old insurance appraiser, had been married for 8 years. He and his wife and their two children lived comfortably and happily in a middle-class neighborhood. He had not experienced any clear symptoms until age 32. One morning, Wayne told his wife that he was bursting with energy and ideas, that his job was unfulfilling, and that he was just wasting his talent. That night he slept little, spending most of the time at his desk, writing furiously. The next morning he left for work at the usual time but returned home at 11:00 A.M., his car overflowing with aquariums and other equipment for tropical fish. He had quit his job, then withdrawn all the money from the family's savings account and spent it on tropical fish equipment. Wayne reported that the previous night he had worked out a way to modify existing equipment so that fish "won't die anymore. We'll be millionaires." After unloading the paraphernalia, Wayne set off to canvass the neighborhood for possible buyers, going door-to-door and talking to anyone who would listen.

Wayne reported that no one in his family had been treated for bipolar disorder. But his mother had gone through periods when she would stop sleeping and become extremely adventurous. For the most part, the family had regarded these episodes as unproblematic, but during one period, she had set off across the country without the children and had returned only after spending a major amount of money.

The following bit of conversation indicates Wayne's incorrigible optimism and provocativeness:

Therapist: Well, you seem pretty happy today.
Wayne: Happy! Happy! You certainly are a master of understatement, you rogue! [Shouting, literally jumping out of his seat.] Why, I'm ecstatic! I'm leaving for the West Coast today, on my daughter's bicycle. Only 3,100 miles. That's nothing, you know. I could probably walk, but I want to get there by next week. And along the way I plan to contact a lot of people about investing in my fish equipment. I'll get to know more people that way—you know, Doc, "know" in the biblical sense [leering at the therapist seductively]. Oh, God, how good it feels.

DSM-IV-TR Criteria for Cyclothymic Disorder

For at least 2 years:

- Numerous periods with hypomanic symptoms that do not meet criteria for a manic episode
- Numerous periods with depressive symptoms that do not meet criteria for a major depressive episode.

The symptoms do not clear for more than 2 months at a time.

Symptoms cause significant distress or functional impairment.

Bipolar I Disorder In DSM-IV-TR, the criteria for diagnosis of **bipolar I disorder** (formerly known as manic-depressive disorder) include a single episode of mania or a single mixed episode during the course of a person's life. Note, then, that a person who is diagnosed with bipolar I disorder may or may not be experiencing current symptoms of mania. In fact, even if they experienced only one week of manic symptoms years ago, they are still diagnosed with bipolar I disorder. Even more than MDD, bipolar disorders tend to recur. Over 50 percent of people with bipolar I disorder have four or more episodes (Goodwin & Jamison, 1990).

Bipolar II Disorder DSM-IV-TR also includes a milder form of bipolar disorder, called **bipolar II disorder**. To be diagnosed with bipolar II disorder, a person must have experienced at least one major depressive episode and at least one episode of *hypomania.*

Cyclothymic Disorder (also called *cyclothymia*) is a second chronic mood disorder (the other is dysthymic disorder). As with diagnosis of dysthymic disorder, DSM-IV-TR requires that symptoms be present for at least two years (see diagnostic criteria). In cyclothymic disorder, the person has frequent but mild symptoms of depression, alternating with mild symptoms of mania. Although the symptoms do not reach the severity of full-blown manic or depressive episodes, people with the disorder and those close to them typically notice the ups and downs. During lows, a person may be sad, feel inadequate, withdraw from people, and sleep for 10 hours a night. During highs, a person may be boisterous, overly confident, socially uninhibited and gregarious, and need little sleep.

Epidemiology and Consequences of Bipolar Disorders Bipolar I disorder is much rarer than MDD—about 1 percent of people will meet the criteria for bipolar I disorder (Weissman et al., 1996). It is hard to know with certainty, but researchers estimate that about 2 percent of people experience bipolar II disorder (Merikangas et al., 2007) and perhaps another 4 percent experience cyclothymic disorder (Regeer et al., 2004). The average age of onset of bipolar disorders is in the early twenties, but these conditions are being seen with increasing frequency among children and adolescents (Kessler et al., 2005). Bipolar disorders occur equally often in men and women, but women experience more episodes of depression than do men (Leibenluft, 1996). Most people with bipolar disorder also meet diagnostic criteria for anxiety disorders (Merikangas et al., 2007).

Bipolar I disorder is among the most severe forms of mental illnesses. One-third of people remain unemployed a full year after hospitalization for mania (Harrow et al., 1990). In one study, researchers interviewed patients every month for 15 years. People with bipolar I disorder reported that symptoms led to an inability to work about 30 percent of the time (Judd et al., 2008). Suicide rates are high for both bipolar I and bipolar II disorders (Angst et al., 2002). People with bipolar disorders are at high risk for a range of other medical conditions, including cardiovascular disease, diabetes mellitus, obesity, and thyroid disease (Kupfer, 2005). Not only are medical problems present, they are often quite severe. People who have been hospitalized for bipolar I disorder are twice as likely to die from medical illnesses in a given year as are people without mood disorders (Osby et al., 2001). The sad consequences of bipolar disorders are not offset by evidence that hypomania is associated with creativity and achievement (see Focus on Discovery 8.3).

People with cyclothymia are at elevated risk for developing episodes of mania and major depression. Even if full-blown manic episodes do not emerge, the chronicity of cyclothymic symptoms takes a toll.

FOCUS ON DISCOVERY 8.3

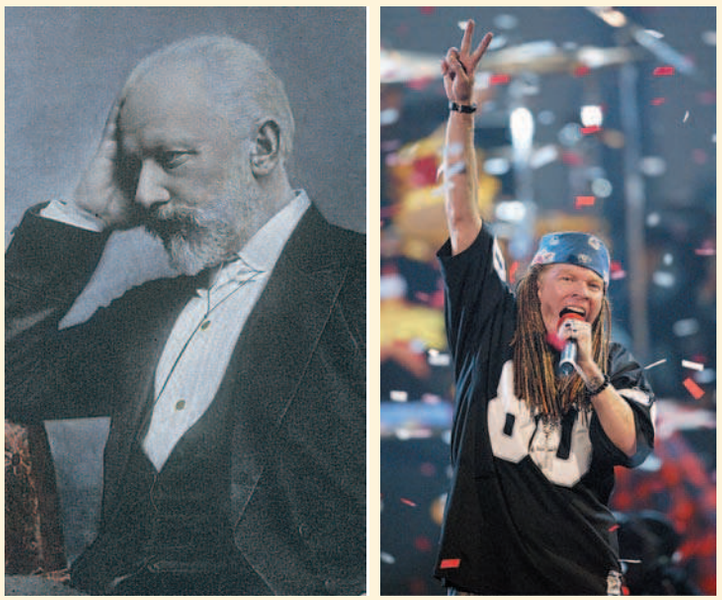
Creativity and Mood Disorders

Noted psychologist Kay Redfield Jamison has written extensively about creativity and mood disorders. (Courtesy of Kay Redfield Jamison.)

In her book *Touched with Fire: Manic-Depressive Illness and the Artistic Temperament* (1992), Kay Jamison, an expert on bipolar disorders and herself a longtime sufferer from bipolar I disorder, assembled much evidence linking mood disorders, especially bipolar disorder, to artistic creativity. Of course, most people with mood disorders are not particularly creative, and most creative people do not have mood disorders—but the list of visual artists, composers, and writers who seem to have experienced mood disorders is impressive, including Michelangelo, van Gogh, Tchaikovsky, Schumann, Gauguin, Tennyson, Shelley, and Whitman, among others.

Many people assume that the manic state itself fosters creativity through elated mood, increased energy, rapid thoughts, and a heightened ability to make connections among seemingly unrelated events. Extreme mania, however, lowers creative output, and even if people produce more work during a manic period, the quality of that work might suffer, as seems to have been the case for the composer Robert Schumann (Weisberg, 1994). Moreover, studies have shown that people who have experienced episodes of mania tend to be less creative than those who have had the milder episodes of hypomania, and both groups tend to produce less creative output than do non-ill family members (Richards et al., 1988). These findings are important, because many people with bipolar disorder worry that taking medications may limit their creativity. Rather, reducing manic symptoms should help, rather than hurt, creativity.

Self-portrait by Paul Gauguin. He is one of the many artists and writers who apparently suffered from a mood disorder. (Paul Gauguin/SUPERSTOCK.)

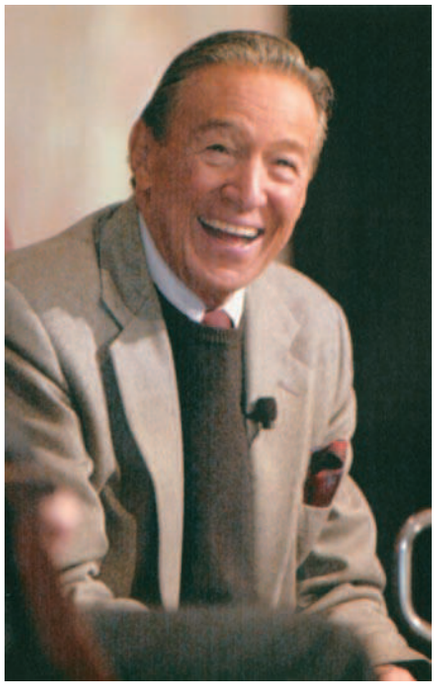


Mood disorders are common among artists and writers. Tchaikovsky was affected, and so is Axl Rose. (Left: Photo Researchers; right: Timothy A. Clary/AFP/Getty Images News and Sport Services.)

Check Your Knowledge 8.1 (Answers are at the end of the chapter.)

Fill in the blanks.

1. Major depressive disorder is diagnosed based on at least _____ symptoms lasting at least _____ weeks.
2. Approximately _____ percent of people will experience depression during their lifetime.
3. Depressive symptoms must last for at least _____ years to qualify for a DSM-IV-TR diagnosis of dysthymia.
4. Approximately _____ out of every 100 people will experience a manic episode during their lifetime.
5. Bipolar I disorder is diagnosed on the basis of _____ or _____ episodes, and bipolar II disorder is diagnosed on the basis of _____ episodes.



Mike Wallace, an internationally recognized reporter, has talked openly about his struggles with major depressive disorder. (AP/Wide World Photos.)

Subtypes of Depressive Disorders and Bipolar Disorders

The mood disorders are highly heterogeneous—that is, people who have been diagnosed with the same disorder may show very different symptoms. DSM-IV-TR deals with this by providing criteria for dividing MDD and bipolar disorders into a number of subtypes, based on either specific symptoms or the pattern of symptoms over time.

DSM-IV-TR states that MDD, bipolar I disorder, and bipolar II disorder qualify as **seasonal** if episodes happen regularly at a particular time of the year (see Focus on Discovery 8.2 for a discussion of seasonal affective disorder). Bipolar I disorder and bipolar II disorder qualify as **rapid cycling** if the person has experienced at least four episodes within the past year (see Figure 8.2). Other subtypes are applied to a given episode of MDD or mania. These include subtypes to label the presence of **psychotic features** (delusions or hallucinations), **catatonic features** (extreme physical immobility or excessive peculiar physical movement), or a **postpartum onset** (onset within 4 weeks postpartum).

Although psychotic, catatonic, and postpartum subtypes can be applied to both depressed and manic episodes, the term **melancholic** is used only for episodes of depression. As described in DSM-IV-TR, a person suffering from depression with melancholic features finds no pleasure in any activity, does not feel better even temporarily when good things happen, and also experiences at least three other symptoms of depression, such as a distinct quality of mood, depressive symptoms that are worse in the morning than at other times of day, waking at least 2 hours too early, loss of appetite, psychomotor retardation or agitation, or guilt. Studies of the distinction between depressions with or without melancholic features have not always supported the validity of this subtype. One study, for example, suggested that MDD with melancholic features may just be a more severe type of depression—that is, people with melancholic features have more comorbidity (e.g., with anxiety disorders), more frequent episodes of depression, and more impairment in everyday activities (Kendler, 1997).

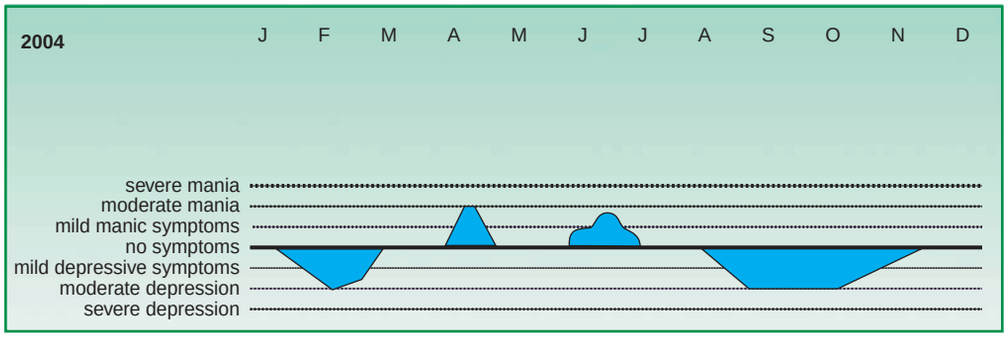
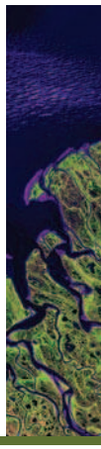


Figure 8.2 The rapid cycling subtype of bipolar disorder is defined by at least four mood episodes per year, as shown in this case.



Quick Summary

DSM-IV-TR contains two broad types of mood disorders: depressive disorders and bipolar disorders. Depressive disorders include major depressive disorder and dysthymic disorder, and bipolar disorders include bipolar I disorder, bipolar II disorder, and cyclothymic disorder. Major depression is characterized by severe episodes lasting at least 2 weeks, whereas dysthymia is characterized by milder symptoms that last at least 2 years. Bipolar I disorder is diagnosed on the basis of a single lifetime manic or mixed episode, and bipolar II disorder is diagnosed on the basis of hypomania and major depression. Cyclothymia is defined by frequent shifts between mild depressive and manic symptoms that last at least 2 years. Subtypes of mood disorders are used to differentiate different patterns of symptoms. These subtypes include distinctions based on a seasonal pattern, rapid cycling, psychotic features, catatonic features, postpartum onset, and, for depression, melancholia. MDD is one of the most common psychological disorders, whereas bipolar I disorder affects approximately 1 percent of the population. Most people with MDD will experience another episode. Bipolar disorder is even more recurrent—about 50 percent of people with bipolar I disorder experience four or more episodes.

Etiology of Mood Disorders

When we think of the profound extremes embodied in the mood disorders, it is natural to ask why these happen. How can we explain Mary sinking into the depths of depression? What factors combined to drive Wayne into his frenzied state of unrealistic ambitions? Studies of etiology focus on why these disorders unfold. No single cause can explain mood disorders. A number of different factors combine to explain their onset.

While the diagnostic criteria specify several different depressive disorders and bipolar disorders, the research on etiology and treatment has tended to focus on major depressive disorder and bipolar I disorder. For simplicity, we refer to these conditions as depression and bipolar disorder through the remainder of this chapter.

We begin by discussing neurobiological factors involved in depression and bipolar disorder. We then discuss psychosocial predictors of depression, then turn to psychosocial models of bipolar disorder.

Neurobiological Factors in Mood Disorders

As Table 8.2 shows, there are many different approaches to understanding the neurobiological factors involved in mood disorders. Here, we will discuss genetic, neurotransmitter, brain imaging, and neuroendocrine research.

Genetic Factors A meta-analysis found that, on average, the more careful studies of MZ (identical) and DZ (fraternal) twins yield heritability estimates of 37 percent for MDD (Sullivan, Neale, & Kendler, 2000). That is, about 37 percent of the variance in depression is explained

Table 8.2 Summary of Neurobiological Hypotheses about Major Depression and Bipolar Disorder

	Genetic Contribution	Neurotransmitter Dysfunction	Cortisol	Brain Imaging
Major depressive disorder	Modest	Serotonin receptor dysfunction Possible change in the dopamine receptors within the reward system	High	Changes in the dorsolateral prefrontal cortex, amygdala, hippocampus, and dorsal anterior cingulate
Bipolar disorder	High	Serotonin receptor dysfunction Possible involvement of dopamine receptors in the reward system Possible deficits in cell membranes and neuronal functioning	High	Changes in the dorsolateral prefrontal cortex, amygdala, hippocampus, and dorsal anterior cingulate Increased activity in the basal ganglia during mania

by genes. Heritability estimates are higher when researchers study more severe samples (e.g., when the people in the study are recruited in inpatient hospitals rather than outpatient clinics). Beyond the twin studies, several small-scale adoption studies also support the modest heritability of MDD (Wender et al., 1986). Genes appear to be more important among women than among men, in that heritability estimates are higher for women than for men (Kendler, Gatz et al., 2006a).

Bipolar disorder is among the most heritable of disorders. Much of the evidence for this comes from studies of twins. The most careful twin studies involve community studies where a representative sample is interviewed (rather than focusing only on people who seek treatment, who may have more severe cases of the disorder than those who are not treated). A Finnish community-based twin sample that used structured interviews to verify diagnoses obtained a heritability estimate of 93 percent (Kieseppa et al., 2004). Adoption studies also confirm the importance of heritability in bipolar disorder (e.g., Wender et al., 1986). Bipolar II disorder is also highly heritable (Edvardsen et al., 2008). Genetic models, however, do not explain the timing of manic symptoms. Other factors must be considered as the immediate triggers of symptoms.

There is a huge amount of interest in finding the specific genes involved in mood disorders through molecular genetics research (see Chapter 4 for a review of these methods). You should be aware of the large number of nonreplications within this field. For example, in a meta-analysis of bipolar disorder and MDD, Kato (2007) identified 166 genetic loci (i.e., locations on specific chromosomes) that had been linked with bipolar disorder and with MDD in initial studies. Of those 166 loci, only 6 have been studied multiple times and replicated in more than 75 percent of relevant studies.

These inconsistencies are even more troubling because positive results are much more likely to be published than null findings. Segurado and colleagues (2003) took an extra step to avoid these publication biases. Whereas previous analyses had compiled only published data, they gathered 18 original data sets, each with more than 20 probands affected by bipolar disorder, to be able to analyze even the negative, unpublished findings for genetic regions associated with bipolar disorder. Their meta-analysis provided the strongest support for three out of 120 regions implicated in bipolar disorder: 9p22.3-21.1, 10g11.21-22.1, and 14q24.1-32.12. Even these 4 regions have not been replicated in more than 10 of the 18 studies. Positive findings should be taken with a grain of salt, as disconfirmation appears to be the rule rather than the exception.

Despite the complexities of this area, there are some consistent patterns emerging across studies. We will discuss findings below that suggest that a polymorphism of the serotonin transporter gene may influence vulnerability to depression when life stress occurs. In addition, there is evidence that a gene that influences dopamine function, the DRD4.2, is related to MDD. A meta-analysis of 917 patients and 1,164 controls revealed that MDD was more common among people with a polymorphism in the DRD4.2 gene (Lopez Leon et al., 2005). These findings help provide an understanding of how the neurotransmitter deficits associated with mood disorders might develop.

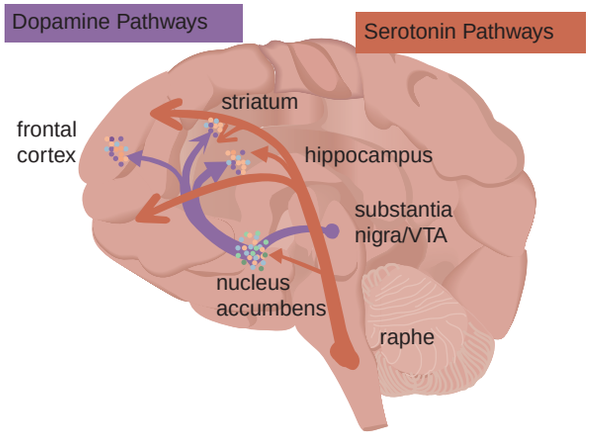


Figure 8.3 Serotonin and dopamine pathways are widespread in the brain.

Because the mood disorders are characterized by so many different symptoms, most researchers think that these disorders will eventually be related to a set of genes rather than to any single gene. Even if we can identify the genes involved in mood disorders, many questions still remain about how they will work. It is unlikely that genes will simply control whether or not a person develops depression. Rather, as we will discuss later, genes may guide the way people regulate emotions or respond to life stressors (Kendler, Gatz, Gardner, & Pedersen, 2006a). As such, they may set the stage for mood disorders to occur when other conditions are present.

Neurotransmitters Three neurotransmitters have been studied the most in terms of their possible role in mood disorders: **norepinephrine**, **dopamine**, and **serotonin**. Each of these neurotransmitters is present in many different areas of the brain. Figure 8.3 illustrates how widespread serotonin and dopamine pathways are in the brain.

Original models suggested that depression would be tied to low levels of norepinephrine and dopamine, and mania would be tied to high levels of norepinephrine and dopamine. Mania and depression were also both posited to be tied

to low levels of serotonin, a neurotransmitter that is believed to help regulate norepinephrine and dopamine (Thase, Jindal, & Howland, 2002). Researchers initially believed that mood disorders would be explained by absolute levels of neurotransmitters in the synaptic cleft that were either too high or too low. Emerging evidence, however, did not support the idea that levels of neurotransmitters were important in the mood disorders.

Studies of antidepressants were one source of contradictory evidence. On the one hand, these studies do suggest that depression is related in some way to these neurotransmitters. For example, effective antidepressants promote an immediate increase in levels of serotonin, norepinephrine, and/or dopamine. See Figure 8.4 for an overview of these immediate effects. But when researchers studied the time course of how antidepressants change neurotransmitter levels, they began to realize that depression could not be explained just by the absolute level of the neurotransmitters. Antidepressants take 7 to 14 days to relieve depression; by that time, the neurotransmitter levels have already returned to their previous state. It would seem, then, that a simple change in norepinephrine, dopamine, or serotonin levels is not a good explanation for why the drugs alleviate depression.

Other evidence also indicates that absolute levels of neurotransmitters did not tell the whole story. For decades, researchers studied the metabolites of neurotransmitters as an index of how much of a neurotransmitter was being released into the synaptic cleft. Recall that after a neurotransmitter is released into the synaptic cleft, enzymes begin to break down the neurotransmitter that is not reabsorbed by the cell. Metabolite studies, then, assess how much neurotransmitter has been broken down and carried into the cerebrospinal fluid, the blood, or the urine. Findings from metabolite studies were not consistent, suggesting that many people with depression did not have disturbances in the absolute levels of neurotransmitters; similarly, many people with mania did not have disturbances in the absolute levels of neurotransmitters (Placidi et al., 2001; Ressler & Nemeroff, 1999).

Given this inconsistent evidence, researchers began to focus on the idea that mood disorders might be related to the sensitivity of postsynaptic receptors that respond to the presence of neurotransmitter in the synaptic cleft. How can researchers test models of high or low receptor sensitivity? If receptors are more or less sensitive, one might expect people to react differently to drugs that influence the level of a given neurotransmitter. For example, receptors that are overly sensitive may respond to even the smallest amount of a neurotransmitter in the synaptic cleft. Researchers have focused more on dopamine and serotonin in these studies than on norepinephrine. People with depression respond differently from other people to drugs that increase dopamine levels, and it is thought that the functioning of the dopamine might be lowered in depression (Naranjo, Tremblay, & Busto, 2001). Among people with bipolar disorder, several different drugs that increase dopamine levels have been found to trigger manic symptoms, suggesting that dopamine receptors may be overly sensitive in bipolar disorder (Anand et al., 2000; Strakowski et al., 1997). Some theories suggest that we will need to look at dopamine receptors within specific regions of the brain, a topic we will return to when we discuss brain imaging studies below.

In addition to links between dopamine and mania, studies focused on depression and receptor sensitivity to serotonin. Researchers have conducted a set of studies that involve experimentally lowering serotonin levels. By raising or lowering serotonin levels, researchers can check how sensitive receptors are to fluctuations. A person who has insensitive receptors is expected to experience depressive symptoms as levels drop. To lower serotonin levels, researchers deplete levels of **tryptophan**, the major precursor of serotonin. Tryptophan can be depleted with a drink that contains high levels of 15 amino acids but no tryptophan. Within hours, serotonin levels are lowered, an effect that lasts for several hours. As a control condition, people can be given a similar-tasting drink that has no effect on tryptophan. Studies show that depleting tryptophan (and so lowering serotonin levels) causes temporary depressive symptoms among people with a history of depression or a family history of depression (Benkelfat et al., 1994; Neumeister et al., 2002). Current thinking is that people who are vulnerable to depression may have less sensitive serotonin receptors, so that they respond more dramatically to lower levels of serotonin.

Researchers have also examined the effects of tryptophan depletion in bipolar disorder. These studies have focused on family members of people with bipolar disorder. By studying relatives who

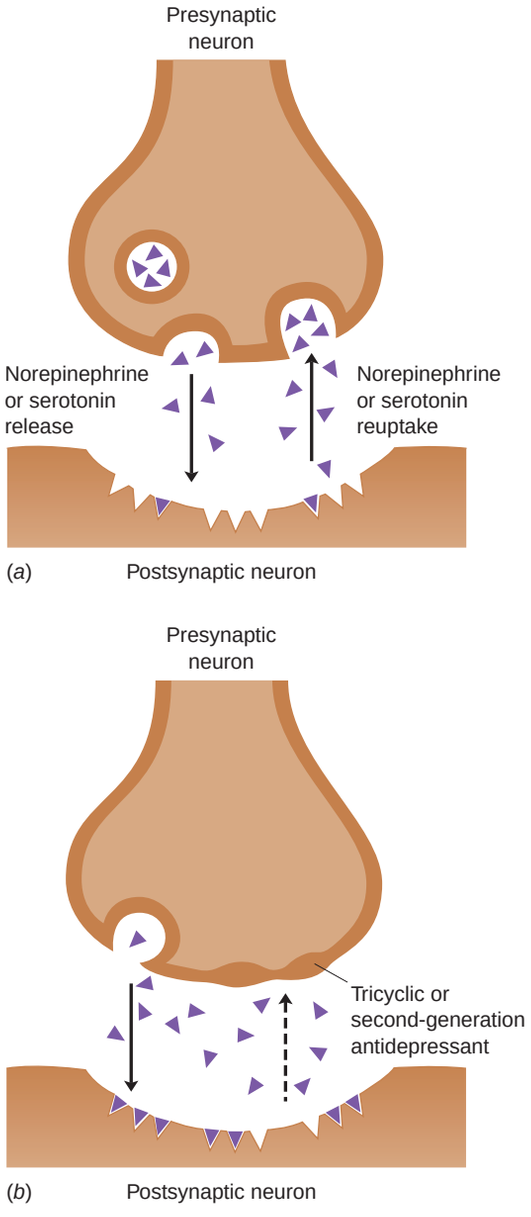


Figure 8.4 (*a*) When a neuron releases norepinephrine or serotonin into a synapse, a pumplike reuptake mechanism immediately begins to recapture some of the neurotransmitter molecules before they are received by the postsynaptic (receptor) neuron. (*b*) Tricyclic drugs block this reuptake process, enabling more norepinephrine or serotonin to reach and stimulate the postsynaptic (receptor) neuron. Selective serotonin reuptake inhibitors act more selectively on serotonin. Adapted from Snyder (1986), p. 106.

do not have the disorder, researchers can be certain that any effects are not scars from having had manic episodes or from taking medications for the disorder. Like people diagnosed with MDD and their family members, relatives of those with bipolar disorder demonstrate elevated mood reactions after tryptophan depletion compared to matched controls (Sobczak, Honig, Nicolson, Riedel et al., 2002).

Researchers are also beginning to study how medications influence receptor sensitivity. These types of studies are being conducted for both mania and depression. For example, one line of research is examining whether antidepressants alter chemical messengers called **second messengers** (see Figure 2.5), which then adjust postsynaptic receptor sensitivity. Another area of current research focuses on **G-proteins** (guanine nucleotide-binding proteins), which play an important role in modulating activity in the postsynaptic cell. High levels of G-proteins have been found in patients with mania and low levels in patients with depression (Avissar et al., 1997, 1999). Some have argued that the therapeutic effects of lithium, the most effective pharmacological treatment for mania, may result from its ability to regulate G-proteins (Manji et al., 1995).

Brain Imaging Studies Two different types of brain imaging studies are commonly used in research on mood disorders. *Structural studies* focus on whether there are fewer cells or connections within a given region of the brain. *Functional activation studies* focus on whether there is a change in the activity of a brain region. Structural studies can indicate whether a person has lost connections between brain cells; functional studies are used to gain information on how people use the cells they have.

Brain imaging studies suggest that episodes of MDD are associated with changes in many of the brain systems that are activated when a person without symptoms of depression experiences strong emotions (Davidson, Pizzagalli, & Nitschke, 2002). As one might expect, many different brain structures become involved when a person experiences emotion: the person needs to attend to and interpret the stimuli that are causing the emotion and then must make plans to deal with those stimuli (Phillips et al., 2003). Because these brain systems are so complex, researchers are still working to understand how depression relates to brain activity. We will provide a brief overview of current theory (see Davidson et al., 2002 for more detail).

Table 8.3 shows the four primary brain structures involved in depression: the **amygdala**, the **hippocampus**, the **prefrontal cortex**, and the **subgenual anterior cingulate** (Figure 8.5). The amygdala helps a person to assess how emotionally important a stimulus is. For example, animals with damage to the amygdala fail to react with fear to threatening stimuli and also fail to respond positively to food. In humans, the amygdala has been shown to respond when people are shown pictures of threatening stimuli. The other structures help a person retrieve previous memories of this type of stimulus (hippocampus) and then focus on the situation and execute appropriate plans (the prefrontal cortex and the subgenual anterior cingulate). Taken together, then, these brain structures function to assess how emotionally important a stimulus is, to focus effectively, and to make plans based on emotionally relevant cues.

Functional activation studies show elevated activity of the amygdala among people with MDD. For example, when shown sad or angry faces, people with current MDD have a more intense and sustained reaction in the amygdala than do people with no MDD (Sheline et al., 2001). Similarly, when shown negative words, people with current MDD have a more sustained reaction in the amygdala than do people with no MDD (Siegle et al., 2002). This pattern of amygdala overreactivity to emotional stimuli does not look like a medication effect or even a consequence of being in a depressed state, because it can be shown even when people are not taking medications (Siegle et al., 2007) and among relatives of people with depression who have no personal history of MDD (van der Veen et al., 2007). These findings suggest that amygdala hyperreactivity to emotional stimuli in depression

Table 8.3 Brain Structures Involved in Major Depression

Brain Structure	Functional Activation Studies
Prefrontal cortex (dorsolateral portion)	Diminished
Anterior cingulate (subgenual portion)	Diminished
Hippocampus	Diminished
Amygdala	Elevated

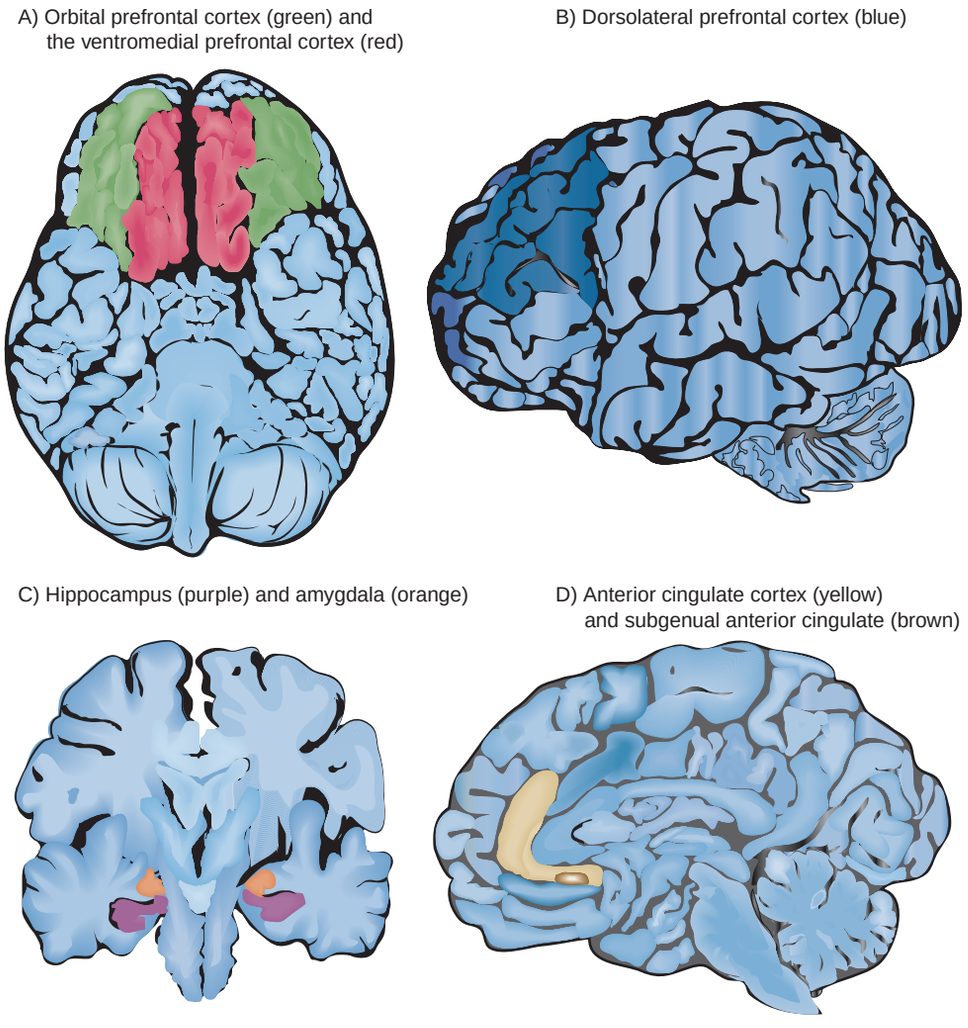


Figure 8.5 Key brain structures involved in major depressive disorder. (Reprinted with permission from the *Annual Review of Psychology*, Vol. 53, © 2002 by Annual Reviews, www.annualreviews.org.)

might be part of the vulnerability to depression rather than just part of the aftermath of being depressed. Functional studies show that MDD is also associated with diminished activation of the subgenual anterior cingulate, the prefrontal cortex, and the hippocampus during exposure to emotional stimuli (Davidson et al., 2002; Schaefer et al., 2006).

Structural studies show many parallels with the findings of the functional studies. That is, structural studies of people with depression also find diminished volume of the subgenual anterior cingulate (Drevets et al., 1997), the prefrontal cortex, and, for people who have had depression for many years, the hippocampus (Sheline, 2000).

How might these findings fit together? One theory is that the overactivity in the amygdala during depression causes oversensitivity to emotionally relevant stimuli. At the same time, systems involved in weighing rewards and costs, making decisions, and systematically pursuing goals in the face of emotions appear less active (the subgenual anterior cingulate, the hippocampus, and the prefrontal cortex). In response to emotionally relevant stimuli, then, a person with depression may react with increased emotion but decreased ability to plan (Davidson et al., 2002).

Given this pattern of findings related to greater emotional reactivity, one might expect that people who are vulnerable to depression would show a robust shift in brain activity in the context of a sad mood. One study found just that (Liotti et al., 2002). That is, people with current major depression, with a history of major depression, and with no current or previous depression were asked to think about sad events in their lives and to try to place themselves in a sad mood. All three groups reported a similar level of sadness after this induction. Before and after the sad mood induction, PET scans were taken to measure brain activity. Among people with either current or previous major depression, activity of the orbitofrontal cortex decreased after the sadness induction compared to baseline; activity of the orbitofrontal cortex did not change among people with no current or previous depression. Stated differently, as people with current depression become sad, they display

less activity in brain regions that are involved with planning and executing goals. The findings of this study indicate that these patterns of brain activity can be seen even after the depressive episode has cleared; such patterns may be part of the vulnerability to depression.

Beyond brain systems involved in emotion, another set of studies have examined whether depression is related to the sensitivity of the reward system in the brain. Remember that depressive symptoms include decreased pleasure, motivation, and energy, and manic symptoms include increased pleasure, motivation, and energy. Some researchers think that symptoms like these could be explained by changes in the sensitivity of the **reward system** in the brain (Depue & lacono, 1989). That is, changes in this brain structure could help explain why people seem less motivated to pursue rewards during depression and overly focused on possible rewards during mania. Although findings are not entirely consistent, many researchers are studying one part of the reward system, the basal ganglia, to see if dopamine receptors in this area are less sensitive during depression (Meyer et al., 2001; Neumeister et al., 2001; Tremblay et al. 2005).

Many of the same brain structures implicated in MDD also appear to be involved in bipolar disorder. In functional studies, bipolar I disorder is associated with elevated responsiveness in the amygdala, along with diminished activity of the hippocampus and prefrontal cortex (cf. Green, Cahill, & Malhi, 2007; Kruger et al., 2003). In structural studies, bipolar disorder is associated with a loss of volume in the prefrontal cortex (Rajkowska, Halaris, & Selemon, 2001).

There is a great deal of controversy about whether the volume of other brain regions is increased or decreased in bipolar disorder (Sheline, 2003). The mixed pattern of findings might be because medications for bipolar disorder can change the volume of these brain regions (Harrison, 2002).

To date, brain imaging research tells us little about what differentiates people with bipolar disorder from those with MDD. Many of the neuroimaing findings for bipolar disorder are very similar to those seen among patients with MDD. One clue might emerge from studying what happens in the brain during manic periods. Many of the brain patterns that are shown during mania are very similar to those shown during depression. On the other hand, one difference emerges. During mania, it appears that a brain region that is very involved in reactions to reward, the basal ganglia, is overly active (Blumberg et al., 1999; Caligiuri et al., 2003). These findings, though, are quite tentative, and more research is needed.

Another set of promising findings suggest that MDD and bipolar disorder might be differentiated by changes in the way that neurons throughout the brain function. People with bipolar disorder often have deficits in the membranes of their neurons (Looney & el-Mallakh, 1997). These deficits seem to operate across the brain, and they influence how readily neurons can be activated. These cellular membrane deficits are not seen in people with MDD (Thiruvengadam & Chandrasekaran, 2007). Similar research is focused on a protein involved in the functioning of many aspects of the neuron, protein kinase C. Protein kinase C has a major role in how receptors function and how messages are sent between neurons. Protein kinase C activity appears to be abnormally high among people with mania (Yildiz et al., 2008). Although these findings for neuronal function are less well established than are other brain findings, they suggest intriguing differences between bipolar disorder and MDD.

The Neuroendocrine System: Cortisol Dysregulation and Depression The **HPA axis** (hypothalamic–pituitary–adrenocortical axis; see Figure 2.9 on p. 38), the biological system that manages reactivity to stress, may be overly active during episodes of MDD. As described above, there is evidence that the amygdala is overly reactive among people with MDD. The HPA axis receives input from structures related to the amygdala and so thus may also be overly active in people with MDD. The HPA axis triggers the release of **cortisol**, the main stress hormone. Cortisol is secreted at times of stress and triggers changes that help the body prepare for threats.

Various findings link depression to high cortisol levels. For example, people with **Cushing's syndrome**, which causes oversecretion of cortisol, frequently experience depressive symptoms. A second line of research with animals has shown that when chemicals that trigger cortisol release are injected into the brain, many of the classic symptoms of depression are produced, including decreased interest in sex, decreased appetite, and sleep disturbances (Gutman & Nemeroff, 2003). In animals and humans, then, too much cortisol seems to produce depressive symptoms.

Even among people who are depressed but do not have Cushing's syndrome, cortisol levels are often poorly regulated—that is, the system does not seem to respond well to signals to decrease cortisol levels (Garbutt et al., 1994). The dexamethasone suppression test (dex/CRH) is used to measure

cortisol regulation. Among people who do not have a mood disorder, dexamethasone suppresses cortisol secretion over the course of the night. In contrast, for some people with mood disorders, dexamethasone does not suppress cortisol secretion, particularly among people whose disorder has psychotic features (Nelson & Davis, 1997). This lack of cortisol suppression is seen as a sign of poor regulation of the HPA axis during episodes of MDD. Challenging the HPA system even further by administering both dexamethasone and corticotropin-releasing hormone (which increases cortisol levels) results in deficits in the regulation of cortisol in 80 percent of people with depression (Heuser, Yassouridis, & Holsboer, 1994). These abnormal responses to dexamethasone, though, normalize when the depressive episode ends for most people. People who continue to show elevated cortisol responses to the dex/CRH test are more likely to relapse within the next year (e.g., Aubry et al., 2007).

Although cortisol helps mobilize beneficial short-term stress responses, prolonged high levels of cortisol can cause harm to body systems. For example, long-term excesses of cortisol have been linked to damage to the hippocampus—studies have found smaller-than-normal hippocampi among people who have experienced depression for years (e.g., Dunman et al., 1997).

Like people with MDD, people with bipolar disorder fail to demonstrate the typical suppression of cortisol after the dex/CRH test. This suggests that bipolar disorder is also characterized by a poorly regulated cortisol system (Watson et al., 2006). Like those with MDD, people with bipolar disorder who continue to show abnormal responses to cortisol challenge tests after their episode clears are at high risk for more episodes in the future (Vieta et al., 1999).

In sum, both bipolar disorder and MDD are characterized by problems in the regulation of cortisol levels. Dysregulation in cortisol levels also predicts a worse course of illness for bipolar disorder and MDD.

Quick Summary

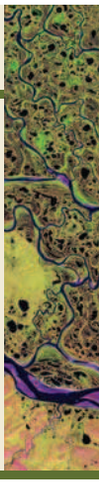
Bipolar disorder is highly heritable, and major depression is modestly heritable. Neurotransmitter models focus on serotonin, dopamine, and norepinephrine. Researchers have begun to focus on receptor sensitivity rather than levels of neurotransmitters. Consistent with this idea, studies that involve experimentally manipulating levels of neurotransmitters provide support for the role of poor serotonin sensitivity in depression and in bipolar disorder. It appears that people with depression may be less sensitive to dopamine and those with mania may be more sensitive to dopamine.

Neuroimaging studies suggest that depression is associated with changes in regions of the brain that are involved in emotion. These changes seem consistent with a greater emotional reactivity (heightened activity of the amygdala) but less planful thinking in the face of emotion (diminished activity of the prefrontal cortex, hippocampus, and subgenual anterior cingulate).

The neurobiological dysfunctions associated with depression are also observed in bipolar disorder. Both disorders are characterized by changes in the amygdala, prefrontal cortex, hippocampus, and anterior cingulate; by low functioning of the serotonin system; and by high cortisol levels. Compared to major depressive disorder, bipolar disorder appears to be uniquely related to increased activity in a region of the brain called the basal ganglia and to changes in the membranes of neurons.

Major depressive disorder and bipolar disorder are both related to poor regulation of cortisol.

Check Your Knowledge 8.2



Answer the questions.

1. Estimates of heritability are approximately _____ percent for MDD and _____ percent for bipolar I disorder.
   a. 60, 93 **b.** 20, 100
   c. 37, 93 **d.** 10, 59
2. Which of the following neurotransmitters is **not** believed to be involved in depression and mania:
   a. acetylcholine **b.** serotonin
   c. dopamine **d.** norepinephrine
3. Recent models suggest that depression is
   a. related to absolute levels of neurotransmitters
   b. related to changes in receptor sensitivity for neurotransmitters
   c. unrelated to neurotransmitter systems
4. In depression, dysregulation of the HPA axis is shown by:
   a. hypersensitivity of the pituitary gland
   b. failure to suppress cortisol by dexamethasone
   c. too little cortisol
   d. elevated parasympathetic nervous system activity
5. One brain region that appears to be overly active among people with mood disorders is the:
   a. hippocampus **b.** prefrontal cortex
   c. cerebellum **d.** amygdala

Social Factors in Depression: Life Events and Interpersonal Difficulties

Data indicate that neurobiological factors influence whether or not a person develops a mood disorder. Does this mean that social and psychological theories are useless? Not in the least. For example, neurobiological theories are consistent with vulnerability to stressors among people with mood disorders. Neurobiological factors, then, may be *diatheses* (p. 53) that increase risk for mood disorders in the context of other triggers or stressors.

The role of stressful life events in triggering episodes of depression is well established (Kendler, Karkowski, & Prescott, 1999). A great deal of research has focused on cause–effect relationships: do life events cause depression, or does depression cause life events? Prospective studies have been particularly important, because they have shown that life events typically happen before the depressive episode begins. Even with a prospective study, though, it remains possible that some life events are caused by early symptoms of depression that have not yet developed into a full-blown disorder. Remember the case of Mary, who developed symptoms after she was laid off from her job. Maybe Mary lost her job because she was sleeping too much and then arriving at work late; trouble sleeping can be a sign of depression.

Even when researchers exclude stressful life events that were caused by mild depressive symptoms, there is much evidence that stress can cause major depressive disorder. In careful prospective studies, 42 to 67 percent of people report that they experienced a very serious life event (that was not caused by symptoms) in the year before their depression began. Common events include losing a job, a key friendship, or a romantic relationship. These findings have been replicated in at least 12 studies, conducted in six different countries (Brown & Harris, 1989b). Certain types of life events, such as loss and humiliation, appear particularly likely to trigger depressive episodes (Kendler, Hettema, et al., 2003). Above and beyond the people who report stressful life events that happened suddenly, many people with depression report that they had been experiencing long-term chronic stressors before the depression, such as poverty (Brown & Harris, 1989b). Life events appear to be particularly important in the first episode of depression but less likely to be involved in later episodes (Monroe & Harkness, 2005).

Although many depressions follow a stressful life event, most people do not become depressed after such an event. Why do some people, but not others, become depressed after stressful life events? The obvious answer is that some people must be more vulnerable to stress than others. In the previous section, we described neurobiological systems involved in depression; many of these systems could be involved in reactivity to stress. Psychological and cognitive vulnerabilities also appear important. The most common models, then, are diathesis–stress models—that is, models that consider both preexisting vulnerabilities (diatheses) and stressors. Diatheses could be biological, social, or psychological.

One diathesis may be a lack of social support. People who are depressed tend to have sparse social networks and to regard those networks as providing little support (Keltner & Kring, 1998). Low social support may lessen a person's ability to handle stressful life events and make the person vulnerable to depression. One study showed that women experiencing a severely stressful life event without support from a confidant had a 40 percent risk of developing depression, whereas those with a confidant's support had only a four percent risk (Brown & Andrews, 1986). Social support, then, seems to minimize the effects of major stressors.

There is also some evidence that interpersonal problems within the family are particularly likely to trigger depression. A long line of research has focused on **expressed emotion (EE)**, defined as a family member's critical or hostile comments toward or emotional overinvolvement with the person with depression. High EE strongly predicts relapse in depression. Indeed, one review of six studies found that 69.5 percent of patients in families with high EE relapsed within 1 year, compared to 30.5 percent of patient in families with low EE (Butzlaff & Hooley, 1998). In a community study, marital discord also predicted the onset of depression (Whisman & Bruce, 1999).

Clearly, interpersonal problems can trigger the onset of depressive symptoms, but it is also important to consider the flip side of the coin. Once depressive symptoms emerge, they can create interpersonal problems—that is, depressive symptoms seem to elicit negative reactions from others (Coyne, 1976). For example, roommates of college students with depression rated social contacts with them as less enjoyable, and they reported feeling aggressive and rejecting toward them (Joiner, Alfano, & Metalsky, 1992).

What is it about the person who is depressed that elicits these negative reactions? Researchers have studied this aspect of depression by coding how people with depression interact with their spouses and with strangers, and then doing studies in which people mimic key depressive behaviors. The following signs of depression seem to elicit negative reactions in others: slow speech, silences, negative self-disclosures, negative affect, poor eye contact, and fewer positive facial expressions (Gotlib & Robinson, 1982; Gottman & Krokoff, 1989). People who are depressed also tend to make hostile comments more frequently to their spouses than do people without depression (Biglan, Hops, & Sherman, 1988).

Other research has explored the interpersonal effects of constant reassurance seeking (Joiner, 1995). More than most, people who are depressed seek reassurance that others truly care about them. But even when others express support, they are only temporarily satisfied. Their negative self-concept causes them to doubt the positive feedback, and their constant efforts to obtain reassurance come to irritate others. Ultimately, people experiencing depression actually elicit negative feedback (e.g., by asking questions like "How do you *truly* feel about me?" after the other person has already given support); eventually, other people's responses typically confirm the person's negative self-concept. Ultimately, the person's excessive reassurance seeking can lead to rejection (Joiner & Metalsky, 1995).

Many of the negative social behaviors, such as excessive reassurance seeking, could be the result of depression. If some of these same social problems are present before symptoms appear, can the problems increase the risk for depression? Research suggests that the answer is yes. Among a group of undergraduates who were not initially depressed, those who were high in reassurance seeking were more likely to develop depressive symptoms over a 10-week period (Joiner & Metalsky, 2001). Similarly, research using high-risk samples, identified before the onset of depression, suggests that interpersonal problems may precede depression. For example, the behavior of elementary school children of parents with depression was rated negatively by both peers and teachers (Weintraub, Prinz, & Neale, 1978); low social competence predicted the onset of depression among elementary school children (Cole et al., 1990); and poor interpersonal problem-solving skills predicted increases in depression among adolescents (Davila et al., 1995). It seems clear that interpersonal problems are one risk factor for depression.

Psychological Factors in Depression

Many different psychological factors may play a role in depressive disorders. In this section, we discuss Freud's psychoanalytic views, which emphasize the unconscious conflicts associated with grief and loss; personality factors, such as neuroticism and positive and negative affectivity; and cognitive factors, such as thoughts about the self and life events. These theories describe different diatheses to answer the question, "what are the characteristics of people who respond to negative life events with a depressive episode."

Freud's Theory In his celebrated paper "Mourning and Melancholia," Freud (1917/1950) drew from clinical observations to develop a model of depression. He theorized that the potential for depression is created early in childhood, during the oral period. If the child's needs are insufficiently or overly gratified, the person becomes fixated in the oral stage (see p. 19). This arrest in development may cause the person to become excessively dependent on other people for the maintenance of self-esteem.

Why do people with this childhood history come to suffer from depression? Freud hypothesized that after the loss of a loved one—whether by death, separation, or withdrawal of affection—the mourner identifies with the lost one—perhaps in a fruitless attempt to undo the loss. Freud asserted that the mourner unconsciously resents being deserted and feels anger toward the loved one for the loss. In addition, the mourner feels guilt for real or imagined sins against the lost person. According to the theory, the mourner's anger toward the lost one becomes directed inward, developing into ongoing self-blame and depression. In this view, depression can be described as anger turned against oneself. Overly dependent persons are believed to be particularly susceptible to this process, and, as noted above, people fixated in the oral stage are overly dependent on others.

Not much research has been carried out to test this theory, but the little information available does not strongly support it. Contrary to the idea that depression is a result of anger turned inward, people with depression express much more anger than do people without depression (Biglan et al., 1988). Despite this, some of Freud's ideas continue to influence

more recent models of depression. For example, Freud maintained that depression could be triggered by the loss of a loved one. As we have seen, a large body of evidence indicates that episodes of MDD are precipitated by stressful life events, which often involve losses. Researchers have consistently shown that people who are high in dependency are prone to depressive symptoms after a rejection (Nietzel & Harris, 1990), a finding that also is congruent with Freud's theory. Although some of Freud's ideas still influence theories of depression, researchers have gone far beyond the clinical observations that were the foundation of his ideas.

Affect and Neuroticism Current personality research has focused on the propensity to experience negative and positive affect as a risk factor for mood disorders. To understand the personality research, though, it is helpful to understand a bit about how depression and anxiety disorders relate to negative and positive affect.

As we have noted, major depression and anxiety disorders frequently co-occur. How can we differentiate anxiety and depression? One model conceptualizes depression and anxiety along three broad dimensions (see Table 8.4): **negative affect** (distress and worry), **positive affect** (happiness and contentment), and **somatic arousal** (sweaty palms, fast heart rate, etc.). Anxiety and depression are both expected to involve negative affect. Anxiety, but not depression, is expected to involve somatic arousal (Clark, Watson, & Mineka, 1994). And depression, but not anxiety, is expected to involve low levels of positive affect. People who show high negative affect, low positive affect, and high somatic arousal may be at risk for comorbid anxiety and depressive disorders.

Table 8.4 Affective Dimensions in Depressive Disorders and Anxiety Disorders

	Negative Affect	Positive Affect	Somatic Arousal
Depressive disorders	High	Low	Moderate
Anxiety disorders	High	Moderate	High
Comorbid anxiety disorders and depressive disorders	High	Low	High

According to this model, depressive disorders should be differentiated from anxiety disorders by the lack of positive affect. Many of the symptoms of depression seem closely related to lack of positive affect—a loss of interest in pleasurable activities and even symptoms like lack of appetite and lack of interest in sex. Other than the clinical symptoms, what is the evidence? Studies of responses to positive pictures and films suggest that people with MDD show fewer positive facial expressions, report less pleasant emotion, show less motivation, and demonstrate less psychophysiological activity in response to positive stimuli than do people without depression (Berenbaum & Oltmanns, 1992; Henriques & Davidson, 2000, Shestyuk et al., 2005; Sloan, Strauss, & Wisner, 2001). Research, then, does support the idea that episodes of MDD are characterized by high negative affect and low positive affect.

Can high negative affect and low positive affect be used to predict depression? Studies of personality help address this question. Several longitudinal studies suggest that **neuroticism**, a personality trait that involves the tendency to react to events with greater than average negative affect, predicts the onset of depression (Jorm et al., 2000). (As you would expect, neuroticism is associated with anxiety, too—see p. 132). A major study of twins suggests that neuroticism explains at least part of the genetic vulnerability to depression (Fanous, Prescott, & Kendler, 2004). In fact, neuroticism is the personality trait most strongly associated with depression. Thus, there is good evidence that people who tend to experience negative affect are at elevated risk for developing depression.

The evidence for positive affect as a predictor is not as clear. For example, **extraversion** is a personality trait associated with frequent experiences of positive affect. Some, but not all, studies suggest that low extraversion predicts the onset of depression (Klein et al., 2002). How would this fit with the idea that once a person is depressed, he or she experiences less positive affect? Some people might be fairly happy until the depressive symptoms kick in, at which point their level of happiness might decrease. In this way, depression may suppress positive affect.

In sum, neuroticism predicts both anxiety and depression. But low extraversion doesn't always precede depression. Once people are depressed, though, they seem to experience less positive affect than do people with no disorder.

Cognitive Theories In some theories, negative thoughts and beliefs are seen as major causes of depression. One can easily think of people who interpret life events differently; some people seem to see the downside of events much more easily than others do. Because cognitive theories are the most common focus of research on depression, we discuss two of them: Beck's theory and hopelessness theory.

Beck's Theory The most important cognitive theory of depression is that of Aaron Beck (1967). His thesis is that people develop depression because their thinking is negative (see Figure 8.6). That is, Beck proposed that depression is associated with the **negative triad**: negative views of the self, the world, and the future. The "world" part of the depressive triad refers to the person's own corner of the world—the situations he or she faces. For example, the person might think "I cannot possibly cope with all these demands and responsibilities" as opposed to worrying about problems in the broader world outside of their life.

According to this model, in childhood, people with depression acquired negative **schemata** through experiences such as loss of a parent, the social rejection of peers, or the depressive attitude of a parent. Schemata are different from conscious thoughts—they are an underlying set of beliefs that operate outside of a person's awareness to shape the way a person makes sense of his or her experiences. The negative schema is activated whenever the person encounters situations similar to those that originally caused the schema to form.

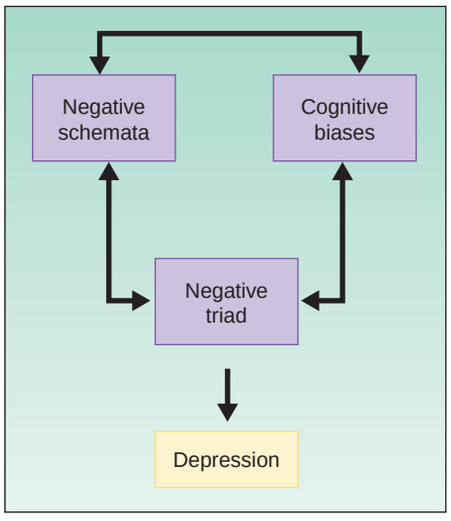


Figure 8.6 The interrelationships among different kinds of cognitions in Beck's theory of depression.

Once activated, negative schemata are believed to cause **cognitive biases**, or tendencies to process information in certain negative ways (Kendall & Ingram, 1989). That is, Beck suggested that people with depression might be overly attentive to negative feedback about themselves and more likely to remember such negative information than other people are. Likewise, they might fail to notice or to remember positive feedback about themselves. People with an underlying ineptness schema might readily notice signs that they are inept and remember feedback that they are inept. Signs that they are competent, though, are not noted or remembered. Overall, people who are depressed make certain cognitive errors to arrive at biased conclusions. Their conclusions are consistent with the underlying schema, which then maintains the schema (a vicious cycle).

How has Beck's theory been tested? One widely used instrument in studies of Beck's theory is a self-report scale called the Dysfunctional Attitudes Scale (DAS), which includes items concerning whether people would consider themselves worthwhile or lovable. Hundreds of studies have shown that people do demonstrate negative thinking on scales like the DAS during depression (Haaga, Dyck, & Ernst, 1991). In studies of how people process information, depression is associated with a tendency to pay more attention to negative stimuli than to positive stimuli (Gotlib & Krasnoperova, 1998). Once people with depression notice negative information, they tend to dwell on or ruminate about that negative information (Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Not surprisingly, then, they tend to remember negative information more than positive information (Matt, Vazquez, & Campbell, 1992).

Despite the clear evidence that thinking is negative during a depressive episode, the greatest challenge for cognitive theories of depression is to resolve questions of cause and effect. That is, can certain cognitive styles cause depression, or do depressive symptoms cause those cognitive styles? Some studies suggest that people with negative cognitive styles are at elevated risk for developing depression. For example, in a study of 1,507 adolescents, very high scores on the DAS in combination with negative life events predicted the onset of MDD (Lewinsohn, Joiner, & Rohde, 2001). Other researchers found that high scores on the DAS predicted relapse for several years after treatment for depression (Segal et al., 2006). On the other hand, in a study of 770 women followed for 3 years, the DAS did not predict first episodes of depression, nor did the DAS scores predict recurrent episodes of depression once history of depression was controlled (Otto et al., 2007). Hence, findings are not consistent regarding the DAS.

Being rejected by peers may lead to the development of the negative schema that, according to Beck's theory, plays a key role in depression. (Richard Hutchings/ Photo Researchers.)

Other studies have examined related cognitive variables as a way to predict depression. For example, cognitive biases in the way people

Check Your Knowledge 8.3

True or False?

1. As many as 42 to 67 percent of people experience a stressful life event before an episode of major depression.
2. Most people who experience a stressful life event develop MDD.
3. Depressive episodes are differentiated from anxiety by low levels of positive affect.
4. Extraversion is more predictive of depression than neuroticism is.

process positive and negative information were found to predict depression over a 12- to 18-month period in a large sample of undergraduate students (Rude et al., 2003). Other studies of cognition have focused on hopelessness theory, described next.

Hopelessness Theory In this section we discuss the evolution of an influential cognitive theory of depression—the hopelessness theory. The initial version of this theory was called learned helplessness; it was then modified to incorporate attributions and then modified again to emphasize hopelessness (see Figure 8.7).

Martin Seligman (1974) formulated **learned helplessness theory** to explain the behavior of dogs given electric shocks. He compared two groups of dogs that both received repeated shocks: one group could escape from the shocks, and the other could not. The dogs that received inescapable shocks seemed to give up. Later, when the experimental conditions were changed and the shocks could be avoided, these dogs were less likely to learn an avoidance response than dogs that had been able to escape from shocks. Rather, after a shock, most of them would lay down in a corner and whimper. Seligman proposed that animals acquire a sense of helplessness when confronted with uncontrollable aversive situations. This sense of helplessness then impairs their performance even when aversive situations are controllable. Animals exposed to uncontrollable shocks also developed symptoms that look like depressive symptoms, such as decreased appetite. On the basis of neurobiological and behavioral studies on the effects of uncontrollable stress, Seligman concluded that learned helplessness in animals could provide a model for human depression.

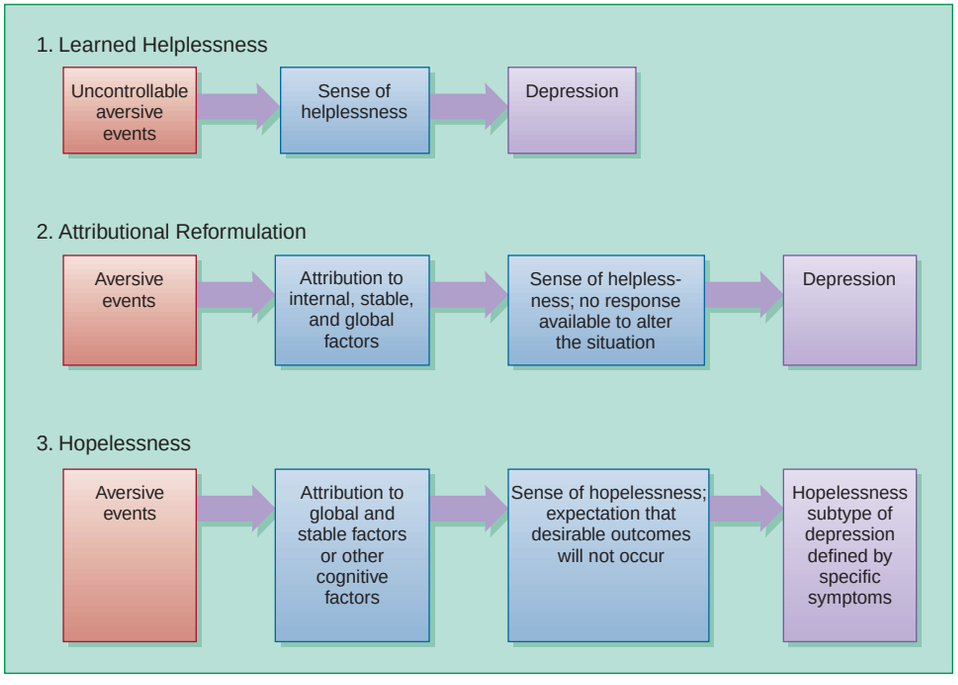


Figure 8.7 The three helplessness/ hopelessness theories of depression.

As researchers began to apply this research to humans, they found that aversive experiences triggered helplessness in some people but not others. With a moment's reflection, you can probably think of people who went through terrible stressors and still did not become depressed. Others seem to react with depression after even minor stressors. To deal with this problem, researchers revised the learned helplessness model to incorporate cognition (Abramson, Seligman, & Teasdale, 1978). The revised theory focused on three key dimensions of **attributions**—the explanations a person forms about why a stressor has occurred (Weiner et al., 1971):

1. Internal (personal) versus external (environmental) causes
2. Stable (permanent) versus unstable (temporary) causes
3. Global (relevant to many life domains) versus specific (limited to one area) causes

Table 8.5 illustrates these dimensions by considering how different people might explain their low score on the Graduate Record Examination (GRE). This revised model suggests that people whose **attributional style** leads them to believe that negative life events are due to internal, stable, and global causes are likely to become depressed.

Attributional style predicts increases in depressive symptoms (Peterson, Maier, & Seligman, 1993), but it is unclear whether attributional style predicts full diagnoses of MDD. For instance, attributional style has predicted the onset of MDD among children (Nolen-Hoeksema, Girgus, & Seligman, 1986), but some studies have found that attributional style did not predict the onset of diagnosable MDD in adolescents (Lewinsohn, Joiner, & Rohde, 2001) or adults (Barnett & Gotlib, 1988).

The current version of the theory, **hopelessness theory** (Abramson, Metalsky, & Alloy, 1989), suggests that cognitive processes explain only one type of depression (hopelessness depression). Symptoms of hopelessness depression include decreased motivation, sadness, suicidality, decreased energy, psychomotor retardation, sleep disturbances, poor concentration, and negative cognitions. In this view, the most important trigger of this type of depression is hopelessness, which is defined as an expectation that (1) desirable outcomes will not occur and that (2) the person has no responses available to change this situation. As in the revised model incorporating attributions, hopelessness can be triggered by stable and global attributions about the causes of stressors. But the model also suggests that there are other ways in which a person can become hopeless, including through low self-esteem or through the sometimes accurate recognition that life events will have severe negative consequences.

Gerald Metalsky and colleagues conducted the first test of hopelessness theory. Early in the semester, college students completed the Attributional Style Questionnaire (ASQ), as well as questionnaires to assess their grade aspirations, their depressive symptoms, hopelessness, and self-esteem. These measures were used to predict the persistence of depressive symptoms after a test among the students whose grades were below their expectations. Those who attributed poor grades to global and stable factors experienced more hopelessness, but this pattern was found only among students whose self-esteem was low. Hopelessness predicted depressive symptoms. Clearly, these results support the hopelessness theory. Also, a similar study conducted with children in the sixth and seventh grades yielded almost identical results (Robinson, Garber, & Hillsman, 1995).

One study has assessed several different aspects of cognitive theories of depression. In the Temple-Wisconsin Cognitive Vulnerability to Depression study, both the DAS and the ASQ were used to predict the development of first episodes of MDD, recurrent episodes of MDD, and also the hopelessness subtype of MDD. In this study, high- and low-risk groups were defined based on scores on the DAS and the ASQ and then followed for $2\frac{1}{2}$ years. The 173 students in the

Table 8.5 An Example of Attributions: Why I Failed My GRE Math Exam

	Internal (Personal)		External (Environmental)	
	Stable	**Unstable**	**Stable**	**Unstable**
Global	I lack intelligence.	I am exhausted.	These tests are all unfair.	It's an unlucky day, Friday the 13th.
Specific	I lack mathematical ability.	I am fed up with math.	The math tests are unfair.	My math test was numbered "13."

upper 25 percent of the distributions for both measures were classified as high risk; the 176 students in the bottom 25 percent of the distributions were classified as low risk. Findings from this study provided support for cognitive theories: students in the high-risk group were more likely to develop first episodes of MDD as well as recurrent MDD than were students in the low-risk group (Alloy et al., 2006). Unfortunately, though, because both the DAS, a measure used to test Beck's theory, and the ASQ, the measure used in tests of the hopelessness theory, were used to define the high-risk group, we do not know whether this finding supports the hopelessness theory, Beck's theory, or both. Findings did provide support for one part of the hopelessness model, in that the cognitive measures predicted the specific symptoms defined as hopelessness depression (Alloy et al., 2000).

Fitting Together the Etiological Factors in Depressive Disorders

Research integrating the neurobiological and psychosocial etiology of depressive disorders is increasingly common. One example of this integration is the growing attention to the serotonin transporter gene. In rhesus monkeys, the presence of a polymorphism (at least one short allele) in this gene is associated with poor serotonergic function. A study found that people with this polymorphism were at greater risk for depression after a stressful life event than those without the polymorphism (Caspi et al., 2003). That is, having at least one short allele was associated with elevated reactivity to stress (see Figure 8.8). Thus, some people seem to inherit a propensity for a weaker serotonin system, which is then expressed as a greater likelihood to experience depression after a major stressor. This finding has been replicated in other large-scale studies (Kendler et al., 2005). This type of neurobiological vulnerability could set the stage for depressive disorder after major negative life events. Intriguingly, a polymorphism in the serotonin transporter gene has also been related to elevated activity of the amygdala (Hariri et al., 2005). This type of work, drawing together genetic and neurobiological risk factors with social and psychological variables, is increasingly common. By considering the set of variables together, researchers can begin to develop more precise models of who is likely to become depressed under what circumstances.

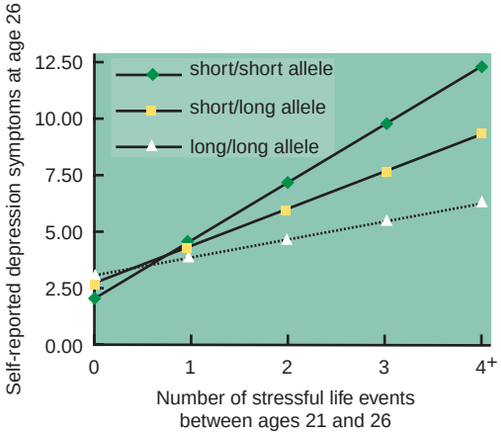


Figure 8.8 Life events interact with the serotonin transporter gene to predict symptoms of depression. Adapted from Caspi et al. (2003). *Science, 301,* 387. Reprinted with permission from AAAS.

Social and Psychological Factors in Bipolar Disorder

Most people who experience a manic episode during their life will also experience a major depressive episode—but not everyone will. Given this, researchers often study the triggers of manic and depressive episodes separately within bipolar disorder.

Depression in Bipolar Disorder The triggers of depressive episodes in bipolar disorder appear similar to the triggers of major depressive episodes (Johnson & Kizer, 2002). As in MDD, negative life events appear important in precipitating depressive episodes in bipolar disorder. Similarly, neuroticism, negative cognitions (Reilly-Harrington et al., 1999), expressed emotion (Yan et al., 2004), and lack of social support predict depressive symptoms in bipolar disorder.

Mania One psychological model hypothesizes that mania reflects a disturbance in the reward system of the brain (Depue, Collins, & Luciano, 1996). Researchers have demonstrated that people with bipolar disorder describe themselves as highly responsive to rewards on a self-report measure (Meyer et al., 2001). In addition, a particular kind of life event predicts increases in manic symptoms in people with bipolar I disorder over a 2-year period (Johnson et al., 2000, 2008)—specifically, life events that involved attaining goals, such as gaining acceptance to graduate school or getting married.

How could successes like these promote increases in symptoms? In studies that have provided (false) success feedback, people with a history of mild hypomanic symptoms seem to develop more confidence after an initial success compared to those with no history of hypomanic symptoms (Stern & Berrenberg, 1979). Thus, researchers have proposed that life events involving success may trigger cognitive changes in confidence, which then spiral into excessive goal pursuit (Johnson, 2005). This excessive goal pursuit may help trigger manic symptoms among people with bipolar disorder.

Quick Summary

Research strongly supports the role of life events as a trigger for MDD. Because many people do not become depressed after a life event, researchers have studied diatheses that could explain vulnerability to life events. Interpersonal research highlights the role of low social support, high expressed emotion, high need for reassurance, and poor social skills as risk factors for depression. Once a person becomes depressed, increases in negativity and reassurance-seeking may lead to more negativity and rejection from other people, potentially prolonging the episodes.

Beyond social factors, researchers have focused on psychological factors. Freud's theory—that depression is due to anger turned inward—has not obtained much support. But other psychological risk factors can help explain why some people become depressed. Evidence suggests that neuroticism, which involves high negative affect, predicts the onset of depression. Cognitive factors include a negative schema; negative beliefs about the self, world, and future; biases to attend to and recall negative rather than positive information; stable, global, and internal attributions for stressors; and hopelessness.

Less psychological research is available on bipolar disorder. Nonetheless, many of the variables that predict MDD also appear to predict depressive symptoms within bipolar disorder. For mania, one model suggests that mania may arise after life events involving goal attainment and excess involvement in pursuing goals.

Treatment of Mood Disorders

Most episodes of depression end after a few months, but the time may seem immeasurably longer to people with depression and to those close to them. With mania, even a few days of acute symptoms can create troubles for relationships and jobs. Moreover, suicide is a risk for people with mood disorders. Thus, it is important to treat mood disorders. Indeed, recent research suggests that it pays to treat depression. In one study, researchers ran a program at 16 major U.S. companies to identify depression, provide referrals for people with depression, and even offer therapy by phone (Wang, Simon, et al., 2007). Although the program cost several hundred dollars per worker, it saved about $1,800 per employee in lost time at work, employee turnover, and other costs.

A major public health goal is to increase the number of people who receive adequate treatment. Certainly, many people try to obtain treatment; more than 180,000,000 prescriptions per year are filled for antidepressants in the United States (IMS Health, 2006; see Figure 8.9). Despite this, surveys suggest that three-quarters of people do not receive effective treatments for MDD (Young et al., 2001). The first treatment with either psychotherapy or antidepressant medications is likely to help about 50–70 percent of people with MDD. Why do up to half of people not get relief? Many people stop treatment early. For example, among patients who are prescribed an antidepressant, 40 percent stop taking the medication within the first month (Olfson et al., 2006). Others are not provided with enough medication or therapy.

In this section, we cover psychological treatments of depressive disorders and bipolar disorder. Then we turn to biological treatments of depressive disorders and bipolar disorder.

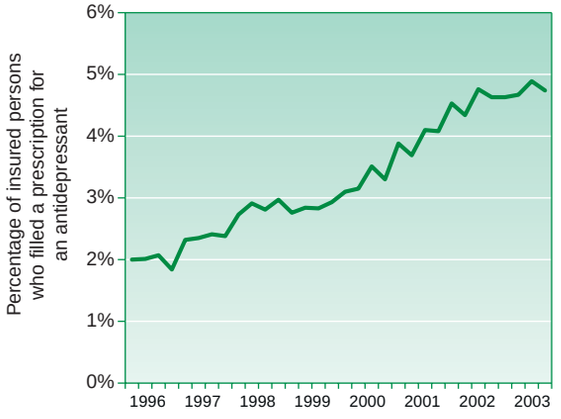


Figure 8.9 More than 180,000,000 prescriptions per year are filled for antidepressants in the United States. The percentage of people who filled a prescription for an antidepressant more than doubled between 1996 and 2003. [Drawn from Meyerhoefer & Zuvekas (2008).]

Psychological Treatment of Depression

Several different forms of psychological treatment have been shown to help relieve depression. As with studies of etiology, most of the research has focused on MDD. We note when treatments have been shown to be effective in the treatment of dysthymia.

Here, we review treatments that have been shown to perform well in comparison with placebo. For this reason, we do not discuss psychoanalytic therapy—a report from the American Psychiatric Association (1993) concluded that there is no reliable evidence indicating that long-term psychoanalysis is effective in treating depression.

Interpersonal Psychotherapy A treatment known as **interpersonal psychotherapy (IPT)** has fared well in clinical trials. As we described in Chapter 2, IPT builds on the idea that depression is closely tied to interpersonal problems (Klerman et al., 1984). The core of the therapy is to examine major interpersonal problems, such as role transitions, interpersonal conflicts,

bereavement, and interpersonal isolation. Typically, the therapist and the patient focus on one or two such issues, with the goal of helping the person identify his or her feelings about these issues, make important decisions, and make changes to resolve problems related to these issues. Like cognitive behavioral treatments, IPT is typically brief (e.g., 16 sessions). Techniques include discussing interpersonal problems, exploring negative feelings and encouraging their expression, improving both verbal and nonverbal communications, problem solving, and suggesting new and more satisfying modes of behavior.

Several studies have found that IPT is effective in relieving MDD (Elkin et al., 1989) and that it appears to prevent relapse when treatment is continued after recovery (Frank et al., 1990). In addition, studies indicate that IPT can be effective in treating MDD among adolescents (Mufson et al., 1999) and postpartum women (Zlotnick et al., 2001). In a study among villagers in Uganda, group sessions of IPT provided relief from depressive symptoms (Bolton et al., 2003). IPT has also been found to be effective in the treatment of dysthymia (Markowitz, 1994). In recent studies of elderly patients, though, IPT did not perform better than a placebo and appeared less helpful than an antidepressant medication (Dombrovsky et al., 2007; Lesperance et al., 2007). In most studies, though, IPT appears helpful.

Cognitive Therapy In keeping with their theory that depression is caused by negative schemata and cognitive biases, Beck and associates devised a **cognitive therapy** aimed at altering maladaptive thought patterns. The therapist tries to help the person with depression to change his or her opinions about the self. When a person states that he or she is worthless because "nothing goes right, and everything I try to do ends in a disaster," the therapist helps the person look for evidence that contradicts this overgeneralization, such as abilities that the person is overlooking or discounting. The therapist also teaches the person to monitor private monologues and to identify thought patterns that contribute to depression. The therapist then teaches the person to challenge negative beliefs and to learn strategies that promote making realistic and positive assumptions. Beck's emphasis is on cognitive restructuring (i.e., persuading the person to think less negatively).

Clinical Case: An Example of Challenging a Negative Thought in Cognitive Therapy

The following dialogue is an example of one way that a therapist might begin to challenge a person's negative thoughts in cognitive therapy, although it would take several sessions to help a client learn the cognitive model and to identify overly negative thoughts. It should be noted that the therapist and client are likely to challenge thoughts in several different ways over the course of treatment. Another example of thought restructuring with this client is described in Chapter 16.

Therapist: *You said that you are a "loser" because you and Roger got divorced. Now we already defined what it is to be a loser—not to achieve anything.*
Patient: *Right. That sounds really extreme.*
Therapist: *OK. Let's look at the evidence for and against the thought that you have achieved something. Draw a line down the center of the page. On the top I'd like you to write, "I have achieved some things."*
Patient: *[draws line and writes statement]*
Therapist: *What is the evidence that you have achieved some things?*
Patient: *I graduated from college, I raised my son, I worked at the office, I have some friends, and I exercise. I am reliable. I care about my friends.*
Therapist: *OK. Let's write all of that down. Now in the right column let's write down evidence against the thought that you have achieved some things.*
Patient: *Well, maybe it's irrational, but I would have to write down that I got divorced.*
Therapist: *OK. Now in looking at the evidence for and against your thought that you have achieved some things, how do you weigh it out? 50–50? Differently than 50–50?*
Patient: *I'd have to say it's 95% in favor of the positive thought.*
Therapist: *So, how much do you believe now that you have achieved some things?*
Patient: *100%.*
Therapist: *And how much do you believe that you are a failure because you got divorced?*
Patient: *Maybe I'm not a failure, but the marriage failed. I'd give myself about 10%.*
(quoted in Leahy, 2003).

Note: As is typical, this dialogue challenges some, but not all, negative thoughts. Future sessions are likely to examine other negative thoughts.

Beck also includes a behavioral technique in his therapy called **behavioral activation (BA)**, in which people are given activity assignments to provide them with successful experiences and to allow them to think well of themselves. For example, the therapist encourages patients to do things that increase the opportunity to have positive experiences, such as going for a walk and talking with friends.

Considerable research has been conducted on Beck's therapy, beginning with a widely cited study indicating that cognitive therapy was more successful than the tricyclic antidepressant imipramine (Tofranil) in alleviating depression (Rush et al., 1977). Many other studies have confirmed the efficacy of cognitive therapy for relieving symptoms of MDD and preventing subsequent bouts of depression (Hollon, Thase, & Markowitz, 2002). With modifications, early results show that cognitive therapy (CT) is promising in the treatment of dysthymia (Hollon et al., 2002). The strategies that clients learn in CT help diminish the risk of relapse even after therapy ends, an important issue in MDD (Vittengl et al., 2007). CT is particularly helpful in preventing relapse for those who need this protection the most—people with at least five episodes of previous depression gain protection from relapse through CT (Bockting et al., 2005).

An adaptation of cognitive therapy called **mindfulness-based cognitive therapy (MBCT)** focuses on relapse prevention after successful treatment for recurrent episodes of major depression (Segal, Williams, & Teasdale, 2001). MBCT is based on the assumption that a person becomes vulnerable to relapse because of repeated associations between sad mood and patterns of self-devaluative, hopeless thinking during major depressive episodes. As a result, when people who have recovered from depression become sad, they begin to think as negatively as they had when they were severely depressed. These reactivated patterns of thinking in turn intensify the sadness (Teasdale, 1988). Thus, in people with a history of major depression, sadness is more likely to escalate, which may contribute to the onset of new episodes of depression.

The goal of MBCT is to teach people to recognize when they start to become depressed and to try adopting what can be called a "decentered" perspective, viewing their thoughts merely as "mental events" rather than as core aspects of the self or as accurate reflections of reality. For example, the person might say to himself or herself such statements as "thoughts are not facts" and "I am not my thoughts" (Teasdale et al., 2000, p. 616). In other words, using a wide array of strategies, including meditation, the person is taught over time to develop a detached relationship to depression-related thoughts and feelings. This perspective, it is believed, can prevent the escalation of negative thinking patterns that may cause depression.

In one multisite study (Teasdale et al., 2000) people who formerly had depression were randomly assigned to MBCT or to "treatment as usual" (e.g., patients were instructed to seek help from other sources, as they normally would). Results of this study showed that MBCT was more effective than "treatment as usual" in reducing the risk of relapse among people with three or more previous major depressive episodes. MBCT does not appear to protect against relapse among people with only one or two previous major depressive episodes (Ma & Teasdale, 2004). This treatment, then, shows promise for patients with highly recurrent major depression.

Behavioral Activation (BA) Therapy Some studies have focused on a treatment that uses just the behavioral activation (BA) component of Beck's therapy (Jacobson & Gortner, 2000). Inactivity, withdrawal, and inertia are common symptoms of depression. From a behavioral activation perspective, however, the *function* of these behaviors is crucial. Proponents of BA contend that these behaviors will diminish the already low levels of positive reinforcement associated with depression. Consequently, BA seeks to increase participation in positively reinforcing activities so as to disrupt the spiral of depression, withdrawal, and avoidance (Martell, Addis, & Jacobson, 2001).

BA has received a great deal of attention after positive findings in a study of which ingredients in Beck's therapy are most effective (Gortner et al., 1998). These findings suggested that the BA component of Beck's therapy performed as well as the full package in relieving MDD and preventing relapse over a 2-year follow-up period. A replication study provided support for the efficacy of BA in a study of 214 patients with MDD (Dimidjian et al., 2006). These findings challenge the notion that people must directly modify their negative thinking to alleviate depression and suggest instead that engaging in rewarding activities may be enough. Group versions of behavioral therapy also appear to be effective (Oei & Dingle, 2008).

Behavioral Couples Therapy As described above, depression is often tied to relationship problems, including marital and family distress. Drawing on these findings, researchers have studied behavioral couples therapy as a treatment for depression. In this approach, researchers work with both members of a couple to improve communication and relationship satisfaction. Findings indicate that when a person with depression is also experiencing marital distress, **behavioral couples therapy** is as effective in relieving depression as individual cognitive therapy (Jacobson et al., 1991) or antidepressant medication (Barbato & D'Avanzo, 2008). As you might expect, marital therapy has the advantage that it relieves relationship distress more than individual therapy does.

Psychological Treatment of Bipolar Disorder

Medication is a necessary part of treatment for bipolar disorder, but psychological treatments can supplement medications. Psychological treatments show promise in dealing with many of the social and cognitive problems associated with bipolar disorder (Johnson & Leahy, 2004).

Educating people about their illness is a common component to treating many disorders, including bipolar disorder and schizophrenia. **Psychoeducational approaches** typically help people learn about the symptoms of the disorder, the expected time course of symptoms, the biological and psychological triggers for symptoms, and treatment strategies. Studies confirm that careful education about bipolar disorder can help people adhere to treatment with medications such as lithium (Colom et al., 2003). This is an important goal, because as many as half of people being treated for bipolar disorder do not take medication consistently (Regier et al., 1993). A friend of one of the authors put it like this: "Lithium cuts out the highs as well as the lows. I don't miss the lows, but I gotta admit that there were some aspects of the highs that I do miss. It took me a while to accept that I had to give up those highs. Wanting to keep my job and my marriage helped!" A drug alone does not address this kind of concern. Beyond helping people be more consistent about their medications, psychoeducational programs have been shown to help people avoid hospitalization (Morris et al., 2007).

Studies have begun to suggest that psychotherapies can also help reduce relapse in bipolar disorder. For example, cognitive therapy like that offered for MDD appears effective as a supplement to medications (Lam et al., 2000).

Expressed emotion (see p. 230) predicts faster relapse in bipolar I disorder (Miklowitz et al., 1996). This points to the need for family interventions. **Family-focused treatment (FFT)** aims to educate the family about the illness, enhance family communication, and develop problem-solving skills (Miklowitz & Goldstein, 1997). FFT leads to lower rates of relapse when added to medication treatment (Miklowitz et al., 2003). Not surprisingly, however, cognitive treatment and FFT seem to relieve symptoms of depression more than those of mania.

In a major study of therapy for bipolar disorder, researchers studied people who had bipolar disorder and who were depressed at the time they sought treatment (Miklowitz et al., 2007). To make sure that findings would generalize to different types of treatment centers, patients were recruited from 14 widely different treatment clinics across the United States. All of the patients in the trial received intensive medication treatment, because researchers were interested in whether adding psychotherapy to medication treatment for bipolar disorder is helpful. Patients were randomly assigned to receive either psychotherapy or collaborative care. The 130 patients assigned to collaborative care were offered three supportive sessions with a treatment provider. The 163 patients in the psychotherapy condition were further assigned to receive either cognitive therapy, FFT, or IPT (interpersonal psychotherapy). Psychotherapy was offered for up to 9 months. About 30 percent of the patients in the study discontinued treatment, and these rates did not differ by treatment condition. Each type of psychotherapy helped relieve depression more than the collaborative care condition did. There was no evidence that cognitive therapy, FFT, or IPT differed in their effects on depression. These findings suggest that it is important for people with bipolar disorder to receive psychotherapy when they are experiencing depression and that several types of therapy can be helpful.

Biological Treatment of Mood Disorders

A variety of biological therapies are used to treat depression and mania. The two major biological treatments are electroconvulsive therapy and drugs.

Electroconvulsive Therapy for Depression Perhaps the most dramatic and controversial treatment for MDD is **electroconvulsive therapy (ECT)** (see p. 15). For the most part now, ECT is only used to treat MDD that has not responded to medication. ECT entails deliberately inducting a momentary seizure and unconsciousness by passing a 70–130 volt current through the patient's brain. Formerly, electrodes were placed on each side of the forehead, a method known as *bilateral ECT*. Today, *unilateral ECT,* in which the current passes through only the nondominant (typically the right) cerebral hemisphere, is more common. In the past, the patient was usually awake until the current triggered the seizure, and the electric shock often created frightening contortions of the body, sometimes even causing bone fractures. Now the patient is given a muscle relaxant before the current is applied. The convulsive spasms of muscles are barely perceptible, and the patient awakens a few minutes later remembering nothing about the treatment. Typically, patients receive between 6 and 12 treatments, spaced several days apart.

Even with these improvements in procedures, inducing a seizure is drastic treatment. Why should anyone agree to undergo such radical therapy? The answer is simple. ECT is the most reliable treatment available for depression with psychotic features (Sackheim et al., 2001), even though we don't know why it works. Most professionals acknowledge that people undergoing ECT face some risks of short-term confusion and memory loss. It is fairly common for patients to have no memory of the period during which they received ECT and, sometimes, for the weeks surrounding the procedure. Unilateral ECT, produces fewer cognitive side effects than bilateral ECT does (Sackeim et al., 2001). Nonetheless, even unilateral ECT is associated with deficits in cognitive functioning 6 months after treatment (Sackeim et al., 2007). In any case, clinicians typically resort to ECT only if less drastic treatments have failed. Given that suicide is a real possibility among people who are depressed, many experts regard the use of ECT after other treatments have failed as a responsible approach.

Electroconvulsive therapy is an effective treatment for depression that has not responded to medication. Using unilateral shock and muscle relaxants has reduced its undesirable side effects. (Will & Deni McIntyre/Photo Researchers.)

Medications for Depressive Disorders Drugs are the most commonly used and best-researched treatments—biological or otherwise—for depressive disorders (and, as we will see, for bipolar disorders as well). As shown in Table 8.6, there are three major categories of antidepressant drugs: **monoamine oxidase (MAO) inhibitors**, **tricyclic antidepressants**, and **selective serotonin reuptake inhibitors (SSRIs)**. The clinical effectiveness of all three classes of drugs

Table 8.6 Medications for Treating Mood Disorders

Category	Generic Name	Trade Name	Side Effects
MAO inhibitor antidepressants	tranylcypromine	Parnate	Possibly fatal hypertension, dry mouth, dizziness, nausea, headaches
Tricyclic antidepressants	imipramine amitriptyline	Tofranil Elavil	Heart attack, stroke, hypotension, blurred vision, anxiety, tiredness, dry mouth, constipation, gastric disorders, erectile dysfunction, weight gain
Selective serotonin reuptake inhibitor (SSRI) antidepressants	fluoxetine sertraline	Prozac Zoloft	Nervousness, fatigue, gastrointestinal complaints, dizziness, headaches, insomnia, suicidality
Mood stabilizers	lithium	Lithium	Tremors, gastric distress, lack of coordination, dizziness, cardiac arrhythmia, blurred vision, fatigue, death (in rare cases)
Anticonvulsants	divalproex sodium	Depakote	Pancreatitis
Antipsychotics	olanzapine	Zyprexa	Hyperglycemia, diabetes, tardive dyskinesia, and, in elderly patients, cardiovascular problems, neuroleptic malignant syndrome

is about the same (Depression Guidelines Panel, 1993). A number of double-blind studies have shown these medications to be effective in treating depressive disorders, with 50–70 percent of people who complete treatment showing major improvement (Depression Guidelines Panel, 1993; Nemeroff & Schatzberg, 1998). These medications have been found to be effective in treating dysthymia as well as major depression (Hollon, Thase, & Markowitz, 2002).

One report, however, suggests that these published studies may overestimate how many people respond well to antidepressant medications. When pharmaceutical companies conduct studies to apply for either initial approval to market a medication or to support a change in the use of a medication, the data must be filed with the Food and Drug Administration (FDA). One research team recently examined what happened to the data from antidepressant studies conducted between 1987 and 2004 (Turner et al., 2008). The FDA rated 38 studies as having positive findings (e.g., supported the use of the antidepressant) and 36 as having neutral or negative findings. Of the studies that the FDA deemed to have positive findings, 37 out of 38 studies were eventually published. Of the 36 studies that the FDA deemed to have either neutral or negative findings, only 14 were published; in 11 of these 14 published studies, the published version of the findings seemed positive even though the FDA had rated findings as neutral or negative. Overall, then, published findings may be biased to be positive. These types of findings are difficult to interpret. Journals are less likely to publish an article with negative results, because they are interested in conveying news to practitioners about strategies that work. But it is also the case that some negative results emerge because a study is flawed, and so some studies may not have merited publication. One way to get around this issue is to create public databases that allow practitioners, scientists, and the public to have more access to the raw facts. Another strategy is to conduct very large-scale trials to help understand who does and does not respond to antidepressants and what to do when a person does not gain relief.

In an attempt to study antidepressant medication in the real world using a large sample, the STAR-D trial examined antidepressant response among 3,671 patients across 41 sites, including 18 primary care facilities (Rush et al., 2006). Many of the studies of treatment have screened for patients with "pure" depression (i.e., no other comorbid disorders) and offered treatment in specialized university clinics. In sharp contrast to the types of clean, non-comorbid depression histories reported in most medication trials, 75 percent of the patients enrolled in STAR-D suffered from chronic or recurrent depression, 61.5 percent had comorbid psychiatric conditions, and 83 percent had already received some (unsuccessful) treatment for the current episode. Rather than assessing whether antidepressant medication or psychotherapy was more helpful than a placebo treatment, the goal of the study was to consider the types of practical questions that physicians face in daily practice. For example, if initial treatment does not work, will switching to a second antidepressant work or should two antidepressants be given at the same time? What is the best treatment option if this second stage of treatment fails?

Patients were all started on citalopram (Celexa), an SSRI. If they did not respond to citaloram, they were offered (1) a choice of a different medication to replace the citalopram, (2) a chance to add a second medication to the citalopram, or (3) cognitive therapy if they were willing to pay part of the cost. Findings were generally sobering. Only about one-third of patients achieved full symptom relief when treated with citalopram (Trivedi et al., 2006). Among those who did not respond, very few wanted to pay for cognitive therapy. Among patients who did not respond to citalopram and were switched to a second round of medication treatment, about 30.6 percent achieved remission, regardless of which type of medication treatment they received. Among patients who did not respond well to either the first or second round of treatment, few responded to a third antidepressant (13.7 percent), and even fewer responded to a fourth antidepressant (13 percent). Even among those who achieved remission at one of these steps, relapse rates were high, so that even with this complex array of treatments offered, only 43 percent of people achieved sustained recovery (Nelson, 2006). Findings from this study highlight a number of important gaps in our science. First, there is a need for more careful testing of treatments in the real world, as findings may differ from findings obtained in specialty clinics. Second, there is a need for new treatments for those who do not respond well to currently available treatments.

One major problem with drug treatments is that many people stop taking their medications, often because they find the side effects unpleasant (see Table 8.6) (Thase & Rush, 1997). The MAO inhibitors are the least used antidepressants because of their potentially life-threatening side effects. The SSRIs have become the most commonly prescribed antidepressants

because they tend to produce fewer side effects than the other classes of antidepressants (Enserink, 1999). In March 2004, however, the Food and Drug Administration asked manufacturers to include packaging information warning people that there have been case reports of suicidality associated with SSRIs, particularly during the early phases of treatment or after increases in dosage. There is specific concern about the potential for suicidality in children, adolescents, and young adults, and researchers continue to look at this important issue, as findings have been controversial (see Chapter 14). Such effects seem very rare, but the FDA approach is designed to protect against risk, even if that risk is faced by only a small number of people.

Although the various antidepressants hasten recovery from an episode of depression, relapse is common after the drugs are withdrawn (Reimherr et al., 2001). This is not to dismiss the advantages of temporary relief, given the potential for social problems, suicide, and hospitalization as depression continues. Results from one meta-analysis of 31 different drug trials suggest that continuing antidepressants after remission lowers the risk of recurrence from approximately 40 percent to about 20 percent (Geddes et al., 2003). Treatment guidelines recommend continuing antidepressant medications for at least 6 months after a depressive episode ends—and longer if a person has experienced several episodes. To prevent recurrence, medication doses should be as high as those offered during acute treatment.

Research Comparing Treatments for Major Depressive Disorder Combining psychotherapy and antidepressant medications bolsters the odds of recovery by more than 10–20 percent above either psychotherapy or medications alone for most people with depression, but each treatment offers unique advantages (Hollon, Thase, & Markowitz, 2002). Antidepressants work more quickly than psychotherapy, thus providing immediate relief. Psychotherapy may take longer but may help people learn skills that they can use after treatment is finished to protect against recurrent depressive episodes. Most patients are interested in knowing whether medications or therapy will be more effective in relieving symptoms. In most studies, cognitive therapy has performed as well as medication in relieving acute symptoms of depression. In one major exception, findings of the NIMH Collaborative Study on the Treatment of Depression indicated that cognitive therapy did not provide as much symptom relief as did medication for severe depression (Elkin et al., 1996; Shea et al., 1992). Some researchers questioned whether the quality of the cognitive therapy offered in the study was up to par. In a meta-analysis of other studies comparing cognitive therapy and antidepressant medications, cognitive therapy was equally effective to antidepressant medication for the treatment of people with severe depression (DeRubeis et al.,1999).

In response to the unanswered questions, researchers designed a clinical trial to compare CT versus antidepressants in the treatment of severe depression (Hollon & DeRubeis, 2003).

Clinical Case: Treatment Decisions for Mary

Mary, the woman described in the beginning of this chapter, reported increasing problems because of her depression. Given this, her therapist referred her to a psychiatrist, who prescribed Prozac (fluoxetine). Both the psychologist and the psychiatrist agreed that medication might help by quickly relieving her symptoms. But after 2 weeks, Mary decided she did not want to continue taking Prozac because she found the side effects uncomfortable and did not like the idea of taking medication over the long term. She had not gotten much relief, but she also explained that she had missed many doses because of her concerns.

With so many different types of treatment available, determining the best therapy for each client can be a challenge. Mary had experienced a major life event and transition, suggesting that interpersonal psychotherapy might fit. But she was blaming herself for her job loss and other issues, suggesting that cognitive therapy (CT) might help. Marital conflicts suggest behavioral couples therapy could be appropriate. How does a therapist choose which approach to use? Sometimes this decision reflects personal preferences and training of the therapist. Ideally, it incorporates the treatment preferences of the client as well. Her therapist began CT, in the belief that Mary's tendency to blame herself excessively when things went wrong was a key force in her depression. CT helped her learn to identify and challenge irrationally negative cognitions about herself. Therapy began by helping her identify times in day-to-day life when her sad moods could be explained by overly negative conclusions about small events. For example, when her children would misbehave, Mary would quickly assume this was evidence that she was a bad mother. Over time, Mary began to examine and challenge long-held beliefs about her lack of competence. By the end of 16 weeks of treatment, she had obtained relief from her symptoms of depression.

Researchers randomly assigned 240 patients with severe depression to receive antidepressant medication, CT, or a placebo for 4 months. Those who recovered were followed for another 12 months. The researchers found that CT was at least as effective as antidepressant medication for severe depression. They also found that experienced therapists were more effective than therapists without CT experience. Although both CT and medication helped people recover from depression more than a placebo did, CT had an advantage over the long term in that it helped protect against relapse once treatment was finished (Hollon et al., 2005). The researchers also noted that CT was less expensive than treatment with medication.

In a more recent study to compare different forms of treatment, 241 patients with MDD were randomly assigned to receive behavioral activation (BA) therapy, CT, antidepressant medication, or a placebo. Behavioral activation therapy and antidepressant medication both performed better than CT for those patients with severe depression (Dimidjian et al., 2006). In sum, comparisons of CT with antidepressant treatment have not provided consistent answers. This remains a hot topic of research.

Medications for Bipolar Disorder Medications that reduce manic symptoms are called *mood-stabilizing medications*. **Lithium**, a naturally occurring chemical element, was the first mood stabilizer identified. Up to 80 percent of people with bipolar I disorder experience at least mild benefit from taking this drug (Prien & Potter, 1993). Even though symptoms may become milder with medications, most patients continue to experience at least mild manic and depressive symptoms. The median time to relapse on lithium is approximately 1 year (Keller et al., 1992). Lithium is more effective in preventing manic episodes than depressive episodes, but it does help somewhat with depression.

Because of possibly serious side effects, lithium has to be prescribed and used very carefully. When serum levels of lithium get too high, lithium toxicity can result, so patients taking lithium must have regular blood tests. Signs of lithium toxicity range from mild symptoms like tremor, nausea, blurred vision, vertigo, and confusion, to very severe symptoms, including cardiac dysrhythmias, seizures, coma, and even death. It is recommended that lithium be used continually for the person's entire life (Bowden et al., 2000), but many patients discontinue treatment (Maj et al., 1998).

Two classes of medications other than lithium have been approved by the FDA for the treatment of acute mania: anticonvulsant (antiseizure) medications such as divalproex sodium (Depakote) and antipsychotic medications such as Olanzapine (Zyprexa). Lithium is still recommended as the first choice, but these other treatments are recommended for people who are unable to tolerate lithium's side effects. Like lithium, these medications help reduce mania and, to some extent, depression. Unfortunately, even these medications have serious side effects. Anticonvulsants have been found to be related to a twofold increase in suicidal ideation compared to rates on placebo (FDA, 2008). Beyond anticonvulsant and antipsychotic medications, several other medications show promising early results (Stahl, 2006).

Typically, lithium is used in combination with other medications. Because lithium takes effect gradually, therapy typically begins with both lithium and an antipsychotic medication, such as olanzapine, which has an immediate calming effect (Scherk, Pajonk, Leucht, 2007).

Many people continue to experience depression even when taking a mood-stabilizing medication like lithium. For these people, an antidepressant medication is often added to the regimen (Sachs & Thase, 2000). But new findings call this practice into question. Initially, concerns were raised because if administered without a mood stabilizer, an antidepressant can actually trigger manic symptoms for 25–30 percent of people with bipolar I disorder (Ghaemi, Boiman, & Goodwin, 2000; Leverich et al., 2006). Beyond these concerns about harmful effects, recent findings cast doubt on whether antidepressants actually help reduce depression among persons who are already taking a mood stabilizer (the first type of treatment provided in bipolar disorder). In one trial, patients with bipolar disorder who were already taking a mood stabilizer were randomly assigned to receive an antidepressant or placebo for 26 weeks. Findings indicated that antidepressants were not effective in combating bipolar depression when added to a mood stabilizer (Sachs et al., 2007).

A major focus of current research is on identifying the best treatments for depression in bipolar disorder. Some of these studies indicate that the anticonvulsant medication lamotrigine (Lamictal) might be effective (Calabrese et al., 1999, 2003).

Depression and Primary Care

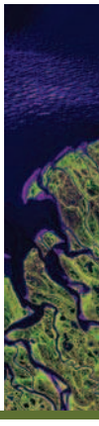
About half of all antidepressant prescriptions are written by primary care physicians. Research has shown that primary care doctors, perhaps because of time pressure, often fail to diagnose episodes of depression, and even when they offer treatment, these treatments tend to be too short, medication doses tend to be too small, and opportunities for psychotherapy tend to be limited. In health maintenance organization (HMO) settings, too, time pressures may interfere with optimal care. One group of researchers studied antidepressant treatment at an HMO and found that only about half of the patients received an adequate regimen of drug therapy (Simon et al., 2001).

Researchers are studying how to improve the quality of care offered by primary care physicians or within HMOs. Simply telling doctors to diagnose and treat depression does not work very well; studies of written treatment guidelines and workshops for doctors do not show much effect on treatment practices (Gilbody et al., 2003). More intensive programs, however, do seem to help. For example, promising results have been obtained with telephone follow-ups, increased nursing care, or specific guidelines that help physicians identify patients who should receive more intensive care (Gilbody et al., 2003). Similar programs, involving more nursing support and more patient psychoeducation, have been shown to be helpful in bipolar disorder (Simon et al., 2006).

A Final Note on the Treatment of Depression

Some of the most exciting research today is focused on how treatments work. Researchers have shown that successful treatments, whether with psychotherapy, medications, or ECT, change activity in the brain regions related to depression (Brody et al., 2001; Goldapple et al., 2004; Nobler et al., 2001). Intriguingly, antidepressant medications and ECT both stimulate growth of neurons in the hippocampus in rats (Duman, Malberg, & Nakagawa, 2001), and the effects of antidepressants, at least in animals, appear to depend on whether these neurons grow (Santarelli et al., 2003). Understanding more about how psychological and medication treatments change underlying neurobiological processes may help us refine treatments for the future.

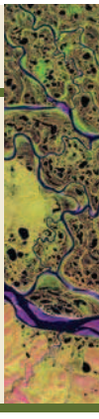
Quick Summary



Many different treatments are available for depression. Cognitive therapy, interpersonal psychotherapy, behavioral activation treatment, and behavioral couples therapy have received support. The three forms of antidepressants have been found to be similarly effective; SSRIs have become more popular because they have fewer side effects than MAO inhibitors and tricyclic antidepressants. Two studies found that cognitive therapy was not as powerful as antidepressant medication in relieving MDD, but most research has found strong support for CT in the treatment of even severe MDD.

Medication treatment is the first line of defense against bipolar disorder. The best-researched mood stabilizer is lithium, but anticonvulsants and antipsychotic medications are also used. Recent findings cast doubt on whether antidepressant medication is helpful in bipolar disorder. Some psychological treatments may help when offered as supplements to medications for the treatment of bipolar disorder. These include psychoeducational approaches, cognitive therapy, and family therapy. These treatments appear particularly helpful in improving adherence to medication and relieving depressive symptoms within bipolar disorder.

Check Your Knowledge 8.4



Circle all answers that apply.

1. Which of the following psychotherapies have obtained support in the treatment of MDD?
   - **a.** interpersonal psychotherapy
   - **b.** behavioral activation
   - **c.** psychoanalytic therapy
   - **d.** cognitive therapy
2. The most effective treatment for MDD with psychotic features is
   - **a.** Prozac
   - **b.** any antidepressant medication
   - **c.** ECT
   - **d.** psychotherapy
3. Selective serotonin reuptake inhibitors (SSRIs) are more popular than other antidepressants because
   - **a.** they are more effective
   - **b.** they have fewer side effects
   - **c.** they are cheaper

Suicide

Clinical Case: Steven

"Shannon Neal can instantly tell you the best night of her life: Tuesday, Dec. 23, 2003, the Hinsdale Academy debutante ball. Her father, Steven Neal, a 54-year-old political columnist for The Chicago Sun-Times, was in his tux, white gloves and tie. 'My dad walked me down and took a little bow,' she said, and then the two of them goofed it up on the dance floor as they laughed and laughed. A few weeks later, Mr. Neal parked his car in his garage, turned on the motor and waited until carbon monoxide filled the enclosed space and took his breath, and his life, away."

Thinking back, his wife reported that he had been under stress as he finished a book and had been hospitalized for heart problems. "Still, those who knew him were blindsided. 'If I had just 30 seconds with him now,' Ms. Neal said of her father, 'I would want all these answers.'"
(Quoted from Cohen, 2008)

Writers who killed themselves, such as Sylvia Plath, have provided insights into the causes of suicide. (Corbis-Bettmann.)

No other kind of death leaves friends and relatives with such long-lasting feelings of distress, shame, guilt, and puzzlement as does suicide (Gallo & Pfeffer, 2003). Survivors have an especially high mortality rate in the year after the suicide of a loved one.

We will focus on quantitative research on suicide, but those who study suicide learn from many different sources. Many philosophers have written searchingly on the topic, including Descartes, Voltaire, Kant, Heidegger, and Camus. In addition, novelists such as Herman Melville and Leo Tolstoy have provided insights on suicide, as have writers who have killed themselves, such as Virginia Woolf (see p. 247 for her note) and Sylvia Plath.

It is important to begin by differentiating suicidal ideation, suicide attempts, and suicide. Suicidal ideation refers to thoughts of killing oneself and is much more common than attempted successful suicide. Suicide attempts involve behaviors that are intended to cause death but do not result in death. **Suicides** involve behaviors that are intended to cause death and actually do so.

Epidemiology of Suicide and Suicide Attempts

Suicide rates may be grossly underestimated because some deaths are ambiguous—for example, seemingly accidental death may involve suicidal intentions. Nonetheless, it has been estimated that, on average, every 20 minutes someone in the United States dies from suicide (Arias et al., 2003).

Studies on the epidemiology of suicidality suggest the following:

- The overall suicide rate in the United States is about 1 per 10,000 in a given year (Centers for Disease Control and Prevention, 2006). In the United States, it is estimated that approximately 1 in 20 suicide attempts results in death (Moscicki, 1995).
- About 10–20 percent of people report suicidal ideation at least once in their lives, and 3–5 percent have made at least one suicide attempt (Weissman et al., 1999).
- Men are four times more likely than women to kill themselves (Arias et al., 2003).
- Three times as many women as men make suicide attempts that do not result in death.
- Guns are by far the most common means of suicide in the United States (Arias et al., 2003), accounting for about 60 percent of all suicides. Men usually choose to shoot or hang themselves; women are more likely to use pills, a less lethal method, which may account for their lower rate of completed suicide.
- The suicide rate increases in old age. The highest rates of suicide in the United States are for white males over age 50.
- The rates of suicide for adolescents and children in the United States are increasing dramatically but are still far below the rates of adults (see Figure 8.10). Some estimates suggest that at least 40 percent of

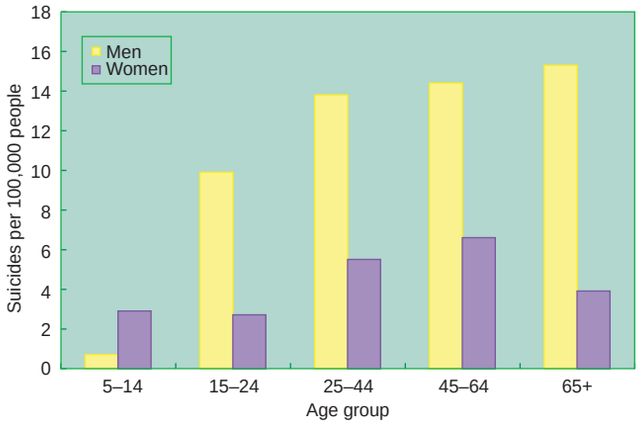


Figure 8.10 Annual deaths due to suicides per 100,000 people. From Arias et al. (2003).

children and adolescents experience suicidal ideation at least once. Because young people are less likely to die from other causes, suicide ranks as the third leading cause of death among those aged 10 to 24.

- Being divorced or widowed elevates suicide risk four- or fivefold.

Models of Suicide

Suicide is such a complex and multifaceted act that no single model can hope to explain it. Myths about suicide abound, highlighting the need for careful research (see Table 8.7). The study of suicide involves many different ethical questions and forces people to consider their own views on life and death. As an example of how complex these questions can be, Focus on Discovery 8.4 discusses physician-assisted suicide.

Psychological Disorders Suicide does not usually happen out of the blue. Rather, suicide is discussed in this chapter because many persons with mood disorders have suicidal thoughts and some engage in suicidal behaviors. More than half of those who try to kill themselves are depressed at the time of the act (Centers for Disease Control and Prevention, 2006) and as many as 15 percent of people who have been hospitalized with depression ultimately die from suicide (Angst et al., 2002). Other mental illnesses also are important in understanding suicide: as many as 90 percent of people who attempt suicide are suffering from a mental illness. Among people hospitalized for schizophrenia, bipolar I disorder, or bipolar II disorder, 10–12 percent die from suicide eventually (Angst et al., 2002; Roy, 1982). Even less severe mental disorders, such as panic disorder, eating disorders, and, among men, alcohol dependence are associated with suicide (Linehan, 1997; Schmidt, Woolaway-Bickel, & Bates, 2000).

With most disorders, suicides are most likely when a person is experiencing comorbid depression (Angst et al., 2002; Linehan, 1997; Schmidt et al., 2000). A significant number of people who are not depressed, however, make suicidal attempts or kill themselves—most notably, people who have been diagnosed with borderline personality disorder (Linehan, 1997; see p. 362). Although understanding suicide within the context of mental disorders is extremely important, most people with mental illnesses do not die from suicide.

Neurobiological Models Twin studies suggest that heritability is about 48 percent for suicide attempts (Joiner, Brown, & Wingate, 2005). Adoption studies also support the heritability of suicidality.

Research has established that, just as low levels of serotonin appear related to depression, there is a connection between serotonin and suicide. Low levels of serotonin's major metabolite, 5-HIAA, have been found among people who committed suicide (van Praag, Plutchik, & Apter, 1990). Particularly low 5-HIAA levels have been found in cases of violent and impulsive suicide (Roy, 1994; Winchel, Stanley, & Stanley, 1990). Several studies suggest that serotonin may play a role in the predisposition to suicide (Mann et al., 2000).

Table 8.7 Myths about Suicide

Common Myth	Contrary Evidence
People who discuss suicide will not actually commit suicide.	Up to three-quarters of those who take their own lives communicate their intention beforehand.
Suicide is committed without warning.	People usually give many warnings, such as saying that the world would be better off without them or making unexpected and inexplicable gifts of highly valued possessions.
Suicidal people want to die.	Most people are thankful after suicide is prevented.
People who attempt suicide by low-lethal means are not serious about killing themselves.	Many people are not well informed about pill dosages or human anatomy. Because of this, people who really want to die sometimes make nonlethal attempts.

Sources: Drawn from Fremouw, De Perzel, & Ellis (1990); Shneidman (1973).

On March 28, 1941, at the age of 59, Virginia Woolf drowned herself in the river near her Sussex home. Two suicide notes were found in the house, similar in content; one may have been written 10 days earlier, and it is possible that she may have made an unsuccessful attempt then, for she returned from a walk soaking wet, saying that she had fallen. The first was addressed to her sister Vanessa and the second to her husband, Leonard. To him, she wrote:

Dearest, I feel certain I am going mad again. I feel we can't go through another of those terrible times. And I shan't recover this time. I begin to hear voices, and I can't concentrate. So I am doing what seems the best thing to do. You have given me the greatest possible happiness. You have been in every way all that anyone could be. I don't think two people could have been happier till this terrible disease came. I can't fight it any longer. I know that I am spoiling your life, that without me you could work. And you will I know. You see I can't even write this properly. I can't read. What I want to say is I owe all the happiness of my life to you. You have been entirely patient with me and incredibly good. I want to say that—everybody knows it. If anybody could have saved me it would have been you. Everything has gone from me but the certainty of your goodness. I can't go on spoiling your life any longer. I don't think two people could have been happier than we have been. V.

Quoted from pp. 400–401, Briggs, J. (2005). Virginia Woolf: An Inner Life. Orlando, FI: Harcourt, Inc.

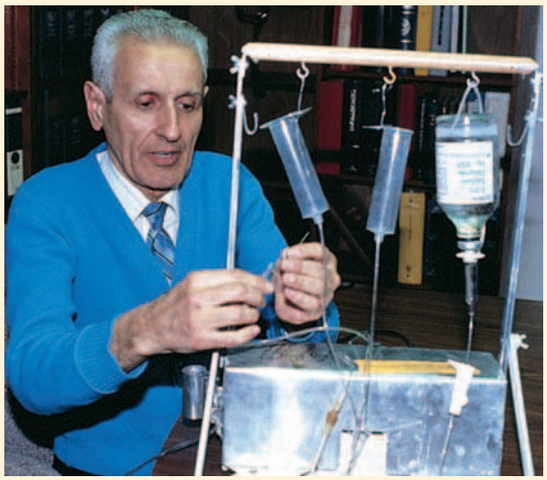
English novelist and critic Virginia Woolf (1882–1941). (Photo by George C. Beresford/Getty Images.)

FOCUS ON DISCOVERY 8.4

Physician-Assisted Suicide

Decisions not to resuscitate terminally ill patients are made every day in hospitals. About 15 percent of physicians in the United States report that they have written at least one prescription to hasten death (Meier et al., 1998). Are these examples of physician-assisted suicide? Physician-assisted suicide is a highly charged issue that came to the fore in the early 1990s when a Michigan physician, Jack Kevorkian, helped a 54-year-old Oregon woman in the early stages of Alzheimer's disease commit suicide. Kevorkian designed a machine to inject a drug that induced unconsciousness and a lethal dose of potassium chloride (Egan, 1990), and then he helped the woman press a button on the machine. Kevorkian assisted more than a hundred terminally ill people in taking their lives. At the same time, he steadfastly provoked a searching discussion about the ethical issues involved in considering whether a physician may take the life of a dying patient. Kevorkian was brought to trial several times on charges both of murder and of professional misconduct but was not convicted until 1999, when he was found guilty of murder and sentenced to prison.

Passionate arguments pro and con continue. Among powerful groups opposing assisted suicide are the American Medical Association and the Catholic Church. In contrast, others, such as the American Civil Liberties Union, believe that terminally ill people should have the right to end their suffering and that the state should not intrude in such decisions.



Jack Kevorkian, a Michigan physician, assisted many patients in taking their own lives. The controversy stimulated by his actions focused attention on the moral and ethical issues surrounding physician-assisted suicide.(Blake Discher/Corbis Sygma.)

Oregon became the first state to have a law—the Death with Dignity Act, originally approved by voters in 1994 and reaffirmed by an even greater margin in 1997—that made physician-assisted suicide legal. This law permits a patient diagnosed by two physicians as having less than 6 months to live to seek a doctor's prescription for a lethal dose of barbiturates. But the law also requires that the prescribing physician determine that the patient is not suffering from mental illness and that there be a waiting period of 15 days before the prescription can be filled. Since the law went into effect, there has been no increase in suicide rates in Oregon.

In 2001, then Attorney General John Ashcroft attempted to overturn Oregon's Death with Dignity Act, declaring that assisted suicide is not an appropriate practice for physicians. The state of Oregon responded by filing suit (*State of Oregon* v. *Attorney General John Ashcroft*), and in April 2002, a federal judge ruled that a federal agency cannot usurp powers relegated to the states without specific authority from Congress, thus leaving the Oregon law in place. The Death with Dignity Act was upheld by the Supreme Court in 2006.

Beyond the serotonin system, other research has found that among patients with MDD, those who had an abnormal dexamethasone suppression test response had a 14-fold increase in the risk of suicide over the next 14 years (Coryell & Schlesser, 2001). This is consistent with the idea that the HPA axis is overly reactive to stress among people at risk for suicide.

Sociocultural Models Some of the strongest evidence for the role of the social environment in suicide comes from the major effects of media reports of suicide. In one example of these effects, suicides rose 12 percent in the month after Marilyn Monroe's death (Phillips, 1985). A review of 293 studies found that media coverage of a celebrity suicide is much more likely to spark an increase in suicidality than coverage of a noncelebrity suicide (Stack, 2000). Media reports of natural deaths of famous people are not followed by increases in suicide, suggesting that it is not grief per se that is the influential factor (Phillips, 1974). These statistics suggest that sociocultural factors matter.

Based on patterns of suicide across different countries, Emile Durkheim (1897/1951) developed a sociological theory of suicide. He focused on three different types of suicide: egoistic, altruistic, and anomic.

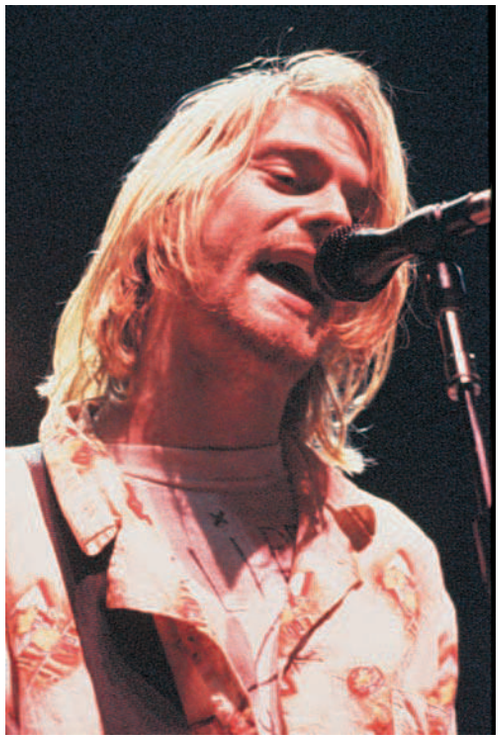
Egoistic suicide is committed by people who have few ties to family, society, or community. Social isolation is profoundly important in understanding suicide, whether measured using

social support measures or even simpler measures like whether a person is a parent or is married (Joiner, Brown, & Wingate, 2005). Consistent with the importance of social connectedness, recent widows often report suicidal ideation (Stroebe, Stroebe, & Abakoumkin, 2005).

Altruistic suicide is committed because the person believes it will be for the good of society. Examples of this type of suicide can be observed in many Eastern cultures; including the self-immolations of Buddhist monks and nuns to protest the Vietnam War and hara-kiri among the Japanese.

Anomic suicide is triggered by a sudden change in a person's relation to society. That is, societies experiencing serious economic and cultural changes can promote anomie and even suicidality. For example, as rural China has gone through massive changes in the past decade, suicide has become one of the leading causes of death (Phillips et al., 2002).

Without a doubt, when trying to understand suicide, social factors matter. But as with all sociological theories, Durkheim's hypotheses do not account for how different people react to the same societal conditions. For example, most people who are lonely do not kill themselves. Durkheim, aware of this problem, suggested that psychological factors would interact with the social causes of suicide.



The suicide of Nirvana's lead singer, Kurt Cobain, triggered an increase in suicide among teenagers. (Kevin Estrada/Retna.)

Psychological Models Suicide has been viewed as retaliation, intended to induce guilt in others; as an effort to force love from others; as an effort to make amends for wrongs; as an effort to rid oneself of unacceptable feelings; as an expression of a desire to rejoin a dead loved one; and as an expression of a desire to escape from emotional pain or an emotional vacuum. Undoubtedly, the psychological variables involved in suicide vary across people, but many researchers have attempted to identify risk factors.

Several researchers relate suicide to difficulties in problem solving (Linehan & Shearin, 1988). Problem-solving deficits do predict suicide attempts prospectively (Dieserud et al., 2003). Problem-solving deficits also relate to the seriousness of previous suicide attempts, even after controlling for depression severity, age, and intellectual functioning (Keilp et al., 2001).

One might expect that a person who has trouble resolving problems would be more vulnerable to hopelessness. Hopelessness, which can be defined as the expectation that life will be no better in the future than it is now, is strongly tied to suicidality. High levels of hopelessness are associated with a fourfold elevation in the risk of suicide (Brown et al., 2000), and hopelessness is important even after controlling for depression levels (Beck, Kovacs, & Weissman, 1975).

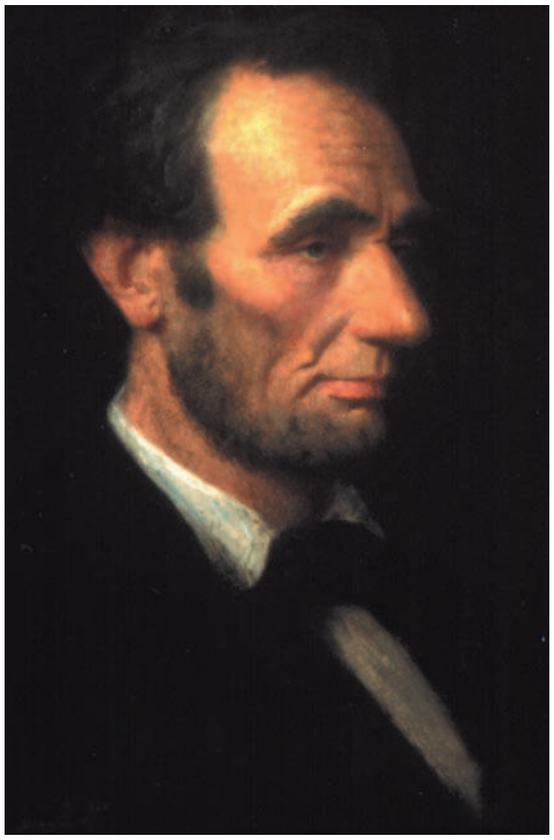
While many people become hopeless and begin to think about death as a way to ease pain, few actually hurt themselves. Among people who are experiencing suicidal thoughts, hundreds of studies document that people who are more impulsive are more likely to attempt suicide or to die from suicide (Brezo, Paris, & Turecki, 2006). While other difficulties might get a person thinking about suicide, impulsivity seems to shape whether people take the step of killing themselves rather than waiting for a better problem resolution.

Beyond these negative characteristics (e.g., poor problem solving, hopelessness, impulsivity), positive qualities may motivate a person to live and help a clinician build a case for choosing life (Malone et al., 2000). One line of research builds on Marsha Linehan's Reasons for Living (RFL) Inventory (Linehan et al., 1983). Items on this inventory tap into what is important to the person, such as responsibility to family and concerns about children. People with more reasons to live tend to be less suicidal than those with few reasons to live (Ivanoff et al., 1994).

Another factor that has been studied is life satisfaction. One prospective study in Finland found that people who expressed relatively high life satisfaction on a simple four-item questionnaire at the beginning of the study were significantly less likely to have attempted or committed suicide up to 20 years later. For example, men with the highest levels of life dissatisfaction were 25 times more likely to commit suicide than men with the lowest levels (Koivuma-Honkanen et al., 2001).

Preventing Suicide

Many people worry that talking about suicide will make it more likely to happen. Rather, clinicians have learned that it is helpful to talk about suicide openly and matter-of-factly. Giving a person permission to talk about suicide may relieve a sense of isolation.



After a broken engagement at age 31, Abraham Lincoln developed symptoms of depression that were so severe that his friends feared that he would hurt himself, and they removed any sharp objects from his room. "I am now the most miserable man living," he confessed. "Whether I shall ever be better I cannot tell; I awfully forebode I shall not. To remain as I am is impossible; I must die or be better." (Cited in Goodwin, 2003.) (Granger Collection.)

Sadly, some colleges seem to be creating policies that keep students from talking about suicidal ideas. About 10 percent of college students report that they thought about suicide in the past year. Even though suicide rates remain relatively low (about 7.5 per 100,000 students), colleges have increasingly encouraged students who endorse suicidality to withdraw (Appelbaum, 2006). More progressive schools have implemented outreach programs to allow students a chance to discuss these thoughts, offering web-based counseling that allows a student to remain anonymous.

Most people are ambivalent about their suicidal intentions, and they will communicate their intentions in some way. "The prototypical suicidal state is one in which a person cuts his or her throat, cries for help at the same time, and is genuine in both of these acts. . . . Individuals would be happy not to do it, if they didn't have to" (Shneidman, 1987, p. 170). Among those who attempt suicide but do not die, 80 percent report within the next two days that they are either glad to be alive or ambivalent about whether they want to die (Henriques et al., 2005). This ambivalence gives the clinician an important foothold.

Treating the Associated Mental Disorder One way to look at the prevention of suicide is to bear in mind that most people who kill themselves are suffering from a mental disorder. Thus, when Beck's cognitive approach successfully lessens a patient's depression, that patient's suicidal risk is also reduced. Marsha Linehan's dialectical behavior therapy with borderline patients provides another example of a treatment that is designed for a specific disorder but also provides protection from suicide (p. 377).

Studies have found that medications for mood disorders reduce the risk of suicidality three- to fourfold (Angst et al., 2002). Specifically, lithium appears effective in suicide prevention for people with bipolar disorder (Cipriani et al., 2005). Among people who have been diagnosed with depressive disorders, ECT reduces suicidality (Kellner et al., 2005), as do antidepressants among elderly people diagnosed with depression (Bruce et al., 2004). Risperidone (Clozapine), an antipsychotic medication, also appears to reduce the risk of suicide attempts among people with schizophrenia (Meltzer, 2003). Findings like these highlight the importance of helping people with mental illnesses to obtain appropriate care. Most studies of treatment though, exclude people who are highly suicidal, a fact that sadly limits our knowledge (Linehan, 1997).

Treating Suicidality Directly Cognitive behavioral approaches appear to be the most promising therapies for reducing suicidality (van der Sande, et al., 1997). Despite some findings that are not supportive (Tyrer et al., 2003), these programs have been found to reduce the risk of a future attempt among suicide attempters by 50 percent compared to treatment as usually offered in the community (Brown et al., 2005). They have also been found to reduce suicidal ideation (Joiner, Voelz, & Rudd, 2001).

Cognitive behavioral treatments include a set of strategies to prevent suicide (Brown, Henriques, Ratto, & Beck, 2002). Therapists help the clients understand the emotions and thoughts that they held just before a suicide attempt. The therapist works with the client to challenge the negative thoughts and to provide new ways to tolerate emotional distress. They also help the client problem solve about the life situations they are facing. The goal is to improve problem solving and social support and thereby to reduce the feelings of hopelessness that often precede these episodes.

Professional organizations such as the American Psychiatric Association, the National Association of Social Workers, and the American Psychological Association charge their members with protecting people from suicide even if doing so requires breaking the confidentiality of the therapist–patient relationship. Therapists are expected to take reasonable precautions when they learn a patient is suicidal (Roy, 1995). One approach to keeping such patients alive

is to hospitalize them as a short-term means of keeping them safe until they can begin to consider ways of improving their life.

Some have argued against involuntary hospitalizations and other efforts to keep people from killing themselves. Boldly and controversially, Thomas Szasz (1999) argues that it is impractical and immoral to prevent suicide. It is impractical because people who are determined to die will be able to do so (even hospitalized patients manage to take their own lives). In his view, it is immoral because people should be free to make choices. In our view, his principal omissions are that treatment and hospitalization often do deter people from suicide, and most people who are prevented from killing themselves are grateful afterward for another chance at life. There are no easy answers here, but it is important to raise the questions.

Suicide Prevention Centers There are more than 200 **suicide prevention centers** in the United States, plus others abroad (Lester, 1995). These centers typically aim to provide 24-hour phone hotline support to people in suicidal crises. It is exceedingly difficult to do controlled research on suicide prevention, and outcome studies have yielded inconsistent results. A meta-analysis of five studies on the effectiveness of suicide prevention centers failed to demonstrate that suicide rates decline after the implementation of services (Dew et al., 1987). A similarly negative finding was reported from Canada (Leenaars & Lester, 1995). Other research, however, has found that suicide rates declined in the years after suicide prevention centers were started in several cities (Lester, 1991). Thus, we are left with conflicting evidence. Human lives are precious, though, and since many people who contact prevention centers weather a suicidal crisis successfully, these efforts will continue.

Community mental health centers often provide a 24-hour-a-day hotline for people who are considering suicide. (Mark Antman/The Image Works.)

Check Your Knowledge 8.5

True or false?

1. Men have higher rates of suicide than women.
2. Men have higher rates of suicide attempts than women.
3. Adolescents have higher rates of suicide than older adults do.
4. Dopamine dysfunction is implicated in suicidality.
5. Most people with MDD will make a suicide attempt.

Summary

Clinical Descriptions and Epidemiology

- There are two broad types of mood disorders: depressive disorders and bipolar disorders.
- Depressive disorders include major depression and dysthymia, and bipolar disorders include bipolar I disorder, bipolar II disorder, and cyclothymia.
- Bipolar I disorder is defined by mania or mixed episodes. Bipolar II disorder is defined by hypomania and episodes of depression. Major depressive disorder, bipolar I disorder, and bipolar II disorder are episodic. Recurrence is very common in these disorders.
- Dysthymia and cyclothymia are characterized by low levels of symptoms that last for at least 2 years.
- Major depression is one of the most common psychiatric disorders, affecting as many as 16.2 percent of people during their lifetime. Rates of depression are twice as high in women as in men. Bipolar I disorder is much rarer, affecting about 1 percent of people.

Etiology

- Genetic studies provide evidence that bipolar disorder is strongly heritable and that depression is somewhat heritable.
- Neurobiological research has focused on the sensitivity of receptors rather than on the amount of various transmitters, with the strongest evidence for changes in serotonin receptors in depression and potential changes in the dopamine receptors within the reward system as related to mania and depression.
- Bipolar and unipolar disorders seem tied to elevated activity of the amygdala and diminished activity in regions of the prefrontal cortex, the hippocampus, and the anterior cingulate.

- Overactivity of the hypothalamic–pituitary–adrenal axis is also found among depressive patients, manifested by high levels of cortisol and poor suppression of cortisol by dexamethasone.
- Socioenvironmental models focus on the role of negative life events, lack of social support, and family criticism as triggers for episodes but also consider ways in which a person with depression may elicit negative responses from others. People with less social skill and those who tend to seek more excessive reassurance are at elevated risk for the development of depression.
- Psychological theories of depression include psychoanalytic, emotion and personality, and cognitive models. Psychoanalytic formulations focus on anger turned inward, but this idea has not been supported. Neuroticism and excess negative emotionality appear to predict the onset of depression. Beck's cognitive theory ascribes causal significance to negative schemata and cognitive biases. According to hopelessness theory, low self-esteem or beliefs that an event will have long-term meaningful consequences can instill a sense of hopelessness, which is expressed in a specific set of depressive symptoms called hopelessness depression.
- Psychological theories of depression in bipolar disorder are similar to those proposed for unipolar depression. Some researchers have proposed that manic symptoms arise because of dysregulation in the reward system in the brain. Mania can be triggered by life events involving success.

Treatment

- Several psychological therapies are effective for depression, including interpersonal therapy, cognitive therapy, behavioral activation therapy, and behavioral couples therapy.
- The major approaches that have been found to help as adjuncts to medication for bipolar disorder include psychoeducation, family therapy, and cognitive therapy.
- Electroconvulsive shock and several antidepressant drugs (tricyclics, selective serotonin reuptake inhibitors, and MAO inhibitors) have proved their worth in lifting depression. Lithium is the best-researched treatment for prevention of mania, but antipsychotic and anticonvulsant medications also help decrease manic symptoms. Antidepressant treatments have become controversial in the treatment of bipolar disorder.

Suicide

- Men, elderly people, and people who are divorced or widowed are at elevated risk for suicide. Most people who commit suicide meet diagnostic criteria for psychiatric disorders, with more than half experiencing depression. Suicide is at least partially heritable, and neurobiological models focus on serotonin and overactivity in the HPA. Social changes are common precedents to anomic suicide. Vulnerability may be tied to poor problem solving, hopelessness, impulsivity, lack of reasons to live, and low life satisfaction.
- Several approaches have been taken to prevention. For people with a mental illness, psychological treatments and medications to quell symptoms help reduce suicidality. Many people believe it is important to address suicidality more directly, though. Problem-solving therapy has shown promise in reducing suicidal behavior, but not all results have been positive. Suicide hotlines are found in most cities, but it has been hard to conduct research demonstrating that these work.

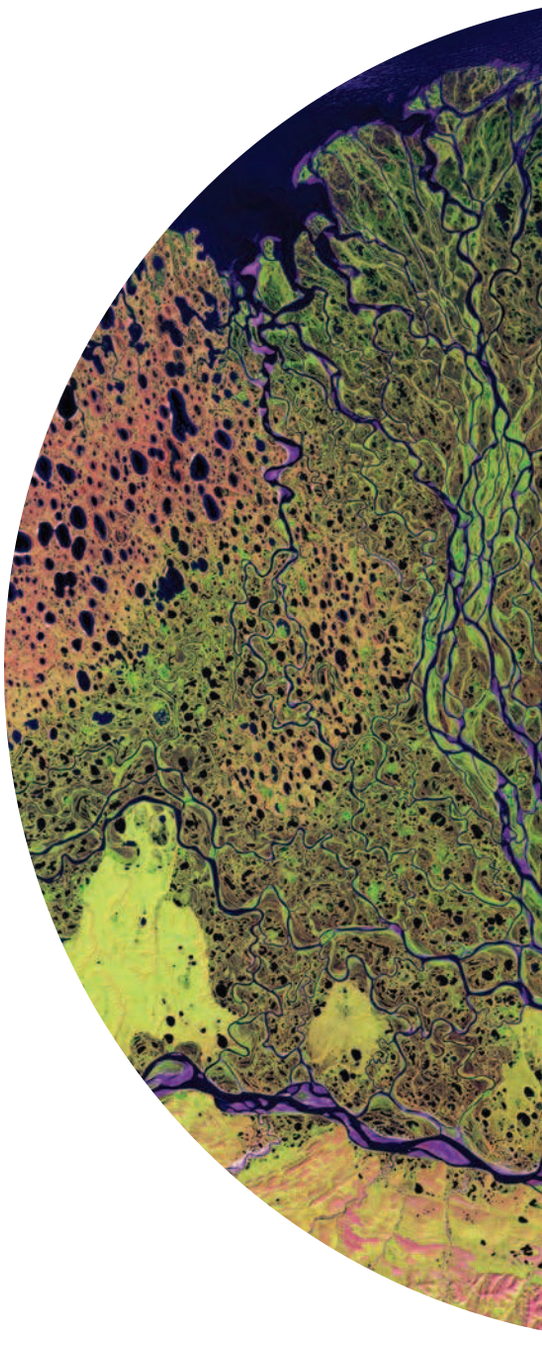
Answers to Check Your Knowledge Questions

8.1 1. five (including mood), two; 2. 16–17; 3. two; 4. one; 5. manic, mixed, hypomanic
8.2 1. c; 2. a; 3. b; 4. b; 5. d
8.3 1. T; 2. F; 3. T; 4. F
8.4 1. a, b, d; 2. c; 3. b
8.5 1. T; 2. F; 3. F; 4. F; 5. F

Key Terms

altruistic suicide
amygdala
anomic suicide
anterior cingulate
attribution
attributional style
behavioral activation (BA) therapy
behavioral couples therapy
bipolar I disorder
bipolar II disorder
catatonic features
cognitive biases
cognitive therapy
cortisol
Cushing's syndrome
cyclothymic disorder
dopamine
dysthymic disorder
egoistic suicide
electroconvulsive therapy (ECT)
episodic disorder
expressed emotion (EE)
extraversion
family-focused treatment (FFT)
flight of ideas
G-proteins
hippocampus
hopelessness theory
HPA axis
hypomania
interpersonal psychotherapy (IPT)
learned helplessness theory
lithium
major depressive disorder (MDD)
mania
melancholic
mindfulness-based cognitive therapy (MBCT)
mixed episodes
monoamine oxidase inhibitors (MAO)
mood disorders
negative affect
negative triad
neuroticism
norepinephrine
positive affect
postpartum onset
prefrontal cortex
psychoeducational approaches
psychotic features
rapid cycling
reward system
schema
seasonal affective disorder
second messengers
selective serotonin reuptake inhibitors (SSRIs)
serotonin
somatic arousal
suicide
suicide prevention centers
tricyclic antidepressants
tryptophan

9 Eating Disorders



LEARNING GOALS

1. Be able to distinguish the symptoms associated with anorexia, bulimia, and binge eating disorder and be able to distinguish among the different eating disorders.
2. Be able to describe the neurobiological, sociocultural, and psychological factors implicated in the etiology of eating disorders.
3. Be able to discuss the issues surrounding the growing epidemic of obesity in the United States.
4. Be able to describe the treatments for eating disorders and the evidence supporting their effectiveness.

Clinical Case: Lynne

Lynne, a 24-year-old Caucasian woman, was admitted to the psychiatric ward of a general hospital for treatment of anorexia nervosa. Although she didn't really think anything was wrong with her, her parents had consulted with a psychiatrist, and the three of them had confronted her with a choice of admitting herself or being committed involuntarily.

At the time Lynne was 5 feet, 5 inches and weighed only 78 pounds. She hadn't menstruated for 3 years, and she had a variety of medical problems—hypotension, irregularities in her heartbeat, and abnormally low levels of potassium and calcium.

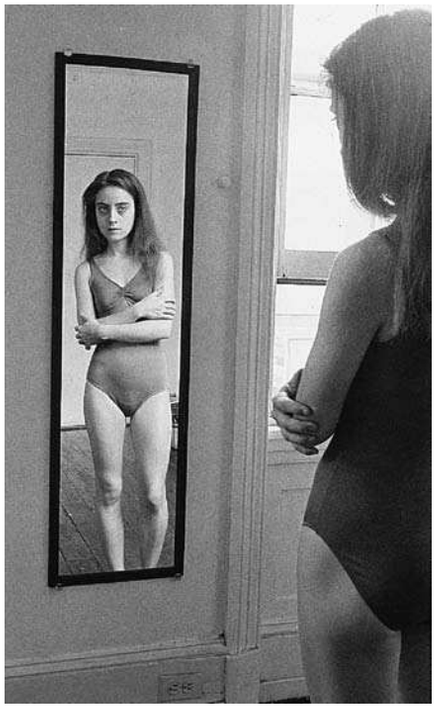
Lynne had experienced several episodes of dramatic weight loss, beginning at age 18 when she first left home for college. But none of the prior episodes had been this severe, and she had not sought treatment before. She had an intense fear of becoming fat, and although she had never really been overweight, she felt that her buttocks and abdomen were far too large. (This belief persisted even when she weighed 78 pounds.) During the periods of weight loss, she severely restricted food intake and used laxatives heavily. She had occasionally had episodes of binge eating, typically followed by self-induced vomiting so that she would not gain any weight.

MANY CULTURES ARE PREOCCUPIED with food. In the United States today, gourmet restaurants abound, and numerous magazines and television shows are devoted to food preparation. At the same time, many people are overweight. Dieting to lose weight is common, and the desire of many people, especially women, to be thinner has created a multibillion-dollar-a-year business. Given this intense interest in food and eating, it is not surprising that this aspect of human behavior is subject to disorder.

Although clinical descriptions of eating disorders can be traced back many years, particularly for anorexia nervosa, these disorders appeared in the DSM for the first time in 1980 as one subcategory of disorders beginning in childhood or adolescence. With the publication of DSM-IV, the eating disorders became a distinct category, reflecting the increased attention they have received from clinicians and researchers over the past three decades.

Clinical Descriptions of Eating Disorders

We begin by describing anorexia nervosa and bulimia nervosa. The diagnoses of these two disorders share several clinical features. We then discuss binge eating disorder, which is not yet in the current diagnostic system but has generated a good deal of attention, in part due to its association with obesity.



Despite being thin, women with anorexia believe that parts of their bodies are too fat and spend a lot of time critically examining themselves in front of mirrors. (Susan Rosenberg/Photo Researchers.)

DSM-IV-TR Criteria for Anorexia Nervosa

- Refusal to maintain normal body weight
- Body weight less than 85 percent of normal
- Intense fear of weight gain
- Body image disturbance
- In women, amenorrhea

Anorexia Nervosa

Lynne, the woman just described, had **anorexia nervosa**. The term *anorexia* refers to loss of appetite, and *nervosa* indicates that the loss is due to emotional reasons. The term is something of a misnomer because most people with anorexia nervosa actually do not lose their appetite or interest in food. On the contrary, while starving themselves, most people with the disorder become preoccupied with food; they may read cookbooks constantly and prepare gourmet meals for their families.

Lynne met all four features required for the diagnosis:

1. *Refusal to maintain normal body weight.* This is usually taken to mean that the person weighs less than 85 percent of what is considered normal for that person's age and height. Weight loss is typically achieved through dieting, although purging (self-induced vomiting, heavy use of laxatives or diuretics) and excessive exercise can also be part of the picture.
2. *Intense fear of gaining weight and being fat.* This fear is not reduced by weight loss. There is no such thing as "too thin."
3. *Distorted body image or sense of their body shape.* Even when emaciated, those with anorexia nervosa maintain that they are overweight or that certain parts of their bodies, particularly the abdomen, buttocks, and thighs, are too fat. To check on their body size, they typically weigh themselves frequently, measure the size of different parts of the body, and gaze critically at their reflections in mirrors. Their self-esteem is closely linked to maintaining thinness.
4. *Amenorrhea (loss of menstrual period).* In females, this is caused by extreme emaciation. Of the four diagnostic criteria, amenorrhea seems least important; few differences have been found between women who meet all four criteria and those who meet the other three but not amenorrhea (Garfinkel et al., 1996).

The distorted body image that accompanies anorexia nervosa has been assessed in several ways, most frequently by a questionnaire such as the Eating Disorders Inventory (Garner, Olmsted, & Polivy, 1983). Some of the items on this questionnaire are presented in Table 9.1. In another type of assessment, people with anorexia nervosa are shown line drawings of women with varying body weights and asked to pick the one closest to their own and the one that represents their ideal shape (see Figure 9.1). People with anorexia overestimate their own body size and choose a thin figure as their ideal. Despite this distortion in body size, people with anorexia nervosa are fairly accurate when reporting their actual weight (McCabe et al., 2001), perhaps because they weigh themselves frequently.

A recent study found a slightly different pattern for men with eating disorders. Men with eating disorders didn't differ from men without eating disorders when pointing to their ideal male body type. However, the men with eating disorders overestimated their own body size considerably, thus demonstrating a distortion in their own body images (Mangweth et al., 2004).

DSM-IV-TR distinguishes two types of anorexia nervosa. In the *restricting type*, weight loss is achieved by severely limiting food intake; in the *binge-eating/purging type*, as illustrated in

Table 9.1 Subscales and Illustrative Items from the Eating Disorders Inventory

Drive for thinness	I think about dieting.
	I feel extremely guilty after overeating.
	I am preoccupied with the desire to be thinner.
Bulimia	I stuff myself with food.
	I have gone on eating binges where I have felt that I could not stop.
	I have the thought of trying to vomit in order to lose weight.
Body dissatisfaction	I think that my thighs are too large.
	I think that my buttocks are too large.
	I think that my hips are too big.
Ineffectiveness	I feel inadequate.
	I have a low opinion of myself.
	I feel empty inside (emotionally).
Perfectionism	Only outstanding performance is good enough in my family.
	As a child, I tried hard to avoid disappointing my parents and teachers.
	I hate being less than best at things.
Interpersonal distrust	I have trouble expressing my emotions to others.
	I need to keep people at a certain distance (feel uncomfortable if someone tries to get too close).
Interoceptive awareness	I get confused about what emotion I am feeling.
	I don't know what's going on inside me.
	I get confused as to whether or not I am hungry.
Maturity fears	I wish that I could return to the security of childhood.
	I feel that people are happiest when they are children.
	The demands of adulthood are too great.

Source: From Garner et al. (1983).
Note: Responses use a six-point scale ranging from "always" to "never."

Lynne's case, the person has also regularly engaged in binge eating and purging. Initial research indicated a number of differences between these two subtypes, thus supporting the validity of this distinction. For example, studies have shown that people with the binge-eating/purging subtype exhibit more personality disorders, impulsive behavior, stealing, alcohol and drug abuse, social withdrawal, and suicide attempts than do people with the restricting type of anorexia (e.g., Herzog et al., 2000; Pryor, Wiederman, & McGilley, 1996). Longitudinal research, however, suggests the distinction between subtypes may not be all that useful (Eddy et al., 2002). Nearly two-thirds of women who initially met criteria for the restricting subtype had switched over to the binge-eating/purging type 8 years later. Furthermore, this study found few differences in substance abuse or personality disturbances between the two subtypes. A summary of the diagnostic criteria for anorexia nervosa appears in the margin.

Anorexia nervosa typically begins in the early to middle teenage years, often after an episode of dieting and the occurrence of a life stress. Lifetime prevalence of anorexia is less than 1 percent, and it is at least 10 times more frequent in women

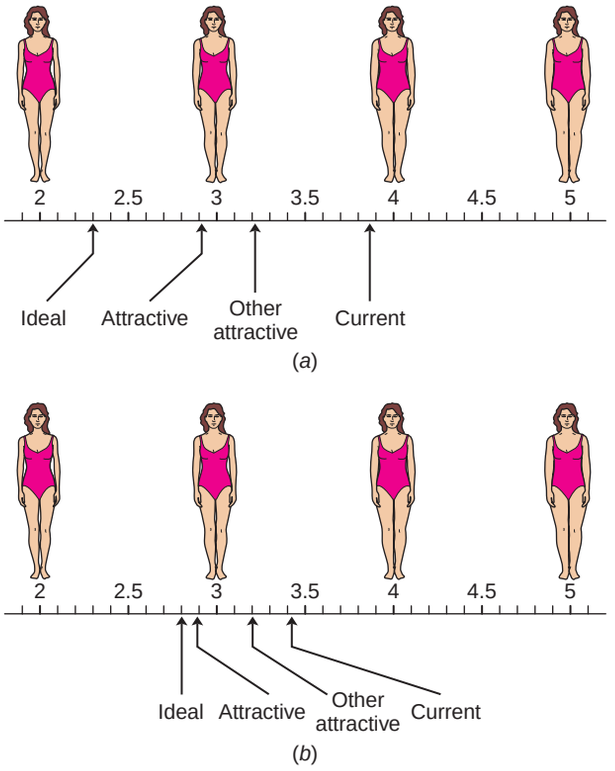


Figure 9.1 In this assessment of body image, respondents indicate their current shape, their ideal shape, and the shape they think is most attractive to the opposite sex. The figure actually rated as most attractive by members of the opposite sex is shown in both panels. Ratings of women who scored high on a measure of distorted attitudes toward eating are shown in (*a*); ratings of women who scored low are shown in (*b*). The high scorers overestimated their current size and ideally would be very thin. From Zellner, Harner, & Adler (1989).

Anorexia nervosa can be a life-threatening condition. It is especially prevalent among young women who are under intense pressure to keep their weight low. Brazilian model Ana Carolina Reston died from the condition in 2006 at age 21. (Reuters/Landov)

than in men (Hoek & van Hoeken, 2003). When anorexia nervosa does occur in men, symptomatology and other characteristics, such as reports of family conflict, are generally similar to those reported by women with the disorder (Olivardia et al., 1995). As we discuss more fully later, the gender difference in the prevalence of anorexia most likely reflects the greater cultural emphasis on women's beauty, which has promoted a thin shape as the ideal over the past several decades.

Women with anorexia nervosa are frequently diagnosed with depression, obsessive-compulsive disorder, phobias, panic disorder, alcoholism, and various personality disorders (Godart et al., 2000; Ivarsson et al., 2000). Men with anorexia nervosa are also likely to have a diagnosis of a mood disorder, schizophrenia, or substance dependence (Striegel-Moore et al., 1999b). Suicide rates are quite high for people with anorexia, with as many as 5 percent completing suicide and 20 percent attempting suicide (Franko & Keel, 2006).

Physical Consequences of Anorexia Nervosa Self-starvation and use of laxatives produce numerous undesirable biological consequences in people with anorexia nervosa. Blood pressure often falls, heart rate slows, kidney and gastrointestinal problems develop, bone mass declines, the skin dries out, nails become brittle, hormone levels change, and mild anemia may occur. Some people lose hair from the scalp, and they may develop lanugo—a fine, soft hair—on their bodies. As in Lynne's case, levels of electrolytes, such as potassium and sodium, are altered. These ionized salts, present in various bodily fluids, are essential to neural transmission, and lowered levels can lead to tiredness, weakness, cardiac arrythmias, and even sudden death.

Prognosis About 70 percent of people with anorexia eventually recover. However, recovery often takes 6 or 7 years, and relapses are common before a stable pattern of eating and weight maintenance is achieved (Strober, Freeman, & Morrell, 1997). As we discuss later, changing peoples' distorted views of themselves is very difficult, particularly in cultures that value thinness.

Anorexia nervosa is a life-threatening illness; death rates are 10 times higher among people with the disorder than among the general population and twice as high as among people with other psychological disorders. Death most often results from physical complications of the illness—for example, congestive heart failure—and from suicide (Herzog et al., 2000; Sullivan, 1995).

Clinical Case: Jill

Jill was the second child born to her parents. Both she and her brother became intensely involved in athletics at an early age, Jill in gymnastics and her brother in Little League baseball. At age 4 Jill was enrolled in gymnastics school, where she excelled. By the time she was 9, her mother had decided that Jill had outgrown the coaching abilities of the local instructors and began driving her to a nationally recognized coach several times a week. Over the next few years, Jill's trophy case swelled and her aspirations for a place on the Olympic team grew. As she reached puberty, though, her thin frame began to fill out, raising concerns about the effects of weight gain on her performance as a gymnast. She began to restrict her intake of food but found that after several days of semistarvation she would lose control and go on an eating binge. This pattern of dieting and bingeing lasted for several months, and Jill's fear of becoming fat seemed to increase during that time. At age 13, she hit on the solution of self-induced vomiting. She quickly fell into a pattern of episodes of bingeing and vomiting three or four times per week. Although she maintained this pattern in secret for a while, eventually her parents caught on and initiated treatment for her.

Bulimia Nervosa

Jill's behavior illustrates the features of **bulimia nervosa**. *Bulimia* is from a Greek word meaning "ox hunger." This disorder involves episodes of rapid consumption of a large amount of food, followed by compensatory behavior, such as vomiting, fasting, or excessive exercise, to prevent weight gain. The DSM defines a *binge* as eating an excessive amount of food within less than 2 hours. Bulimia nervosa is not diagnosed if the bingeing and purging occur only in the context of anorexia nervosa and its extreme weight loss; the diagnosis in such a case is anorexia nervosa, binge-eating/purging type. The key difference between anorexia and bulimia is weight loss: people with anorexia nervosa lose a tremendous amount of weight, whereas people with bulimia nervosa do not.

In bulimia, binges typically occur in secret; they may be triggered by stress and the negative emotions they arouse, and continue until the person is uncomfortably full (Grilo, Shiffman, & Carter-Campbell, 1994). In the case of Jill, she was likely to binge after periods of stress associated with being an elite athlete. Foods that can be rapidly consumed, especially sweets such as ice cream and cake, are usually part of a binge. One study found that women with bulimia nervosa were more likely to binge while alone and during the morning or afternoon. In addition, avoiding a craved food on one day was associated with a binge episode the next morning (Waters, Hill, & Waller, 2001). Other studies show that a binge is likely to occur after a negative social interaction, or at least the perception of a negative social exchange (Steiger et al., 1999).

Research suggests that people with bulimia nervosa sometimes ingest enormous quantities of food during binges, often more than what a person eats in an entire day; however, binges are not always as large as the DSM implies, and there is wide variation in the caloric content consumed by people with bulimia nervosa during binges (e.g., Rossiter & Agras, 1990). People report that they lose control during a binge, even to the point of experiencing something akin to a dissociative state, perhaps losing awareness of their behavior or feeling that it is not really they who are bingeing. They are usually ashamed of their binges and try to conceal them.

After the binge is over, feelings of discomfort, disgust, and fear of weight gain lead to the second step of bulimia nervosa—purging to attempt to undo the caloric effects of the binge. People with bulimia most often stick fingers down their throats to cause gagging, but after a time many can induce vomiting at will without gagging themselves. Laxative and diuretic abuse (which do little to reduce body weight) as well as fasting and excessive exercise are also used to prevent weight gain.

Although many people binge occasionally and some people also experiment with purging, the DSM diagnosis of bulimia nervosa requires that the episodes of bingeing and purging occur at least twice a week for 3 months. Is twice a week a well-established cutoff point? Probably not. Few differences are found between people who binge twice a week and those who do so less frequently, suggesting that we are dealing with a continuum of severity rather than a sharp distinction (Garfinkel, Kennedy, & Kaplan, 1995).

Like those with anorexia nervosa, people with bulimia nervosa are afraid of gaining weight, and their self-esteem depends heavily on maintaining normal weight. Whereas people without eating disorders typically underreport their weight and say they are taller than they actually are, people with bulimia nervosa are more accurate in their reports (Doll & Fairburn, 1998; McCabe et al., 2001). Yet people with bulimia nervosa are also likely to be highly dissatisfied with their bodies.

Two subtypes of bulimia nervosa are distinguished: a *purging type* and a *nonpurging type* in which the compensatory behaviors are fasting or excessive exercise. And, as with anorexia, evidence for the validity of this distinction is mixed. In some studies, people diagnosed with nonpurging bulimia were heavier, binged less frequently, and showed less psychopathology than did people with purging-type bulimia (e.g., Mitchell, 1992). But in other research, few differences emerged between the two types (e.g., Tobin, Griffing, & Griffing, 1997). Given the limited validity of these two subtypes, researchers have investigated other possible subtypes. Three different studies have now validated two different subtypes: a *dietary subtype*, which is characterized by dietary restraint, and a *dietary-depressive subtype*, which is characterized by both dietary restraint and persistent negative affect (Stice & Fairburn, 2003). Dietary restraint refers to rigid and strict patterns of eating that are very restrictive with respect to what, when, and how much to eat. People with the dietary-depressive subtype are more likely to have comorbid mood and anxiety disorders, personality disorders, more severe bulimia, social impairment, more persistent binge eating, and a poor response to cognitive behavioral treatment (Stice & Agras, 1999; Stice & Fairburn, 2003). These two subtypes appear to have greater validity than the purging/nonpurging types, and perhaps they will be included in the next version of DSM.

Bulimia nervosa typically begins in late adolescence or early adulthood. About 90 percent of cases are women, and prevalence among women is thought to be about 1 to 2 percent of the population (Hoek & van Hoeken, 2003). Many people with bulimia nervosa were somewhat overweight before the onset of the disorder, and the binge eating often started during an episode of dieting.

Bulimia nervosa is comorbid with numerous other diagnoses, notably depression, personality disorders, anxiety disorders, substance abuse, and conduct disorder (Godart et al., 2000;

DSM-IV-TR Criteria for Bulimia Nervosa

- Recurrent episodes of binge eating
- Recurrent compensatory behaviors to prevent weight gain, for example, vomiting
- Body shape and weight are extremely important for self-evaluation

Stice, Burton, & Shaw, 2004). Men with bulimia are also likely to be diagnosed with a mood disorder or substance dependence (Striegel-Moore et al., 1999). Suicide rates are much higher among people with bulimia nervosa than in the general population (Favaro & Santonastaso, 1997) but substantially lower than among people with anorexia (Franko & Keel, 2006).

A prospective study examined the relationship between bulimia and depression symptoms among adolescent girls (Stice et al., 2004). This study found that bulimia symptoms predicted the onset of depression symptoms. However, the converse was also true: depression symptoms predicted the onset of bulimia symptoms. Thus, it appears each disorder increases the risk for the other.

Physical Consequences of Bulimia Nervosa Like anorexia, bulimia is associated with several physical side effects. Although less common than in anorexia, menstrual irregularities, including amenorrhea, can occur, even though people with bulimia typically have a normal body mass index (BMI) (Gendall et al., 2000). The BMI is calculated by dividing weight in kilograms by height in meters squared and is considered a more valid estimate of body fat than many others. For women, a normal BMI is between 20 and 25. To calculate your own BMI, see Table 9.2. Bulimia nervosa, like anorexia, is a serious disorder with many unfortunate medical consequences (Garner, 1997). For example, frequent purging can cause potassium depletion. Heavy use of laxatives induces diarrhea, which can also lead to changes in electrolytes and cause irregularities in the heartbeat. Recurrent vomiting has been linked to menstrual problems and may lead to tearing of tissue in the stomach and throat and to loss of dental enamel as stomach acids eat away at the teeth, which become ragged. The salivary glands may become swollen. Death from bulimia nervosa appears to be much less common than in anorexia nervosa (Herzog et al., 2000; Keel & Mitchell, 1997).

Prognosis Long-term follow-ups of people with bulimia nervosa reveal that about 70 percent recover, although about 10 percent remain fully symptomatic (Keel et al., 1999; Reas et al., 2000). Intervening soon after a diagnosis is made (i.e., within the first few years) is linked with an even better prognosis (Reas et al., 2000). People with bulimia nervosa who binge and vomit more, and have comorbid substance abuse or a history of depression, have a poorer prognosis than people without these factors (Wilson et al., 1999).

Table 9.2 Computing Your Body Mass Index (BMI)

WEIGHT lbs	100	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215
kgs	45.5	47.7	50.0	52.3	54.5	56.8	59.1	61.4	63.6	65.9	68.2	70.5	72.7	75.0	77.3	79.5	81.8	84.1	86.4	88.6	90.9	93.2	95.5	97.7
HEIGHT in/cm	Underweight					Healthy						Overweight				Obese				Extremely obese				
5'0'' - 152.4	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
5'1'' - 154.9	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	36	37	38	39	40
5'2'' - 157.4	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	33	34	35	36	37	38	39
5'3'' - 160.0	17	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	32	32	33	34	35	36	37	38
5'4'' - 162.5	17	18	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	31	32	33	34	35	36	37
5'5'' - 165.1	16	17	18	19	20	20	21	22	23	24	25	25	26	27	28	29	30	30	31	32	33	34	35	35
5'6'' - 167.6	16	17	17	18	19	20	21	21	22	23	24	25	25	26	27	28	29	29	30	31	32	33	34	34
5'7'' - 170.1	15	16	17	18	18	19	20	21	22	22	23	24	25	25	26	27	28	29	29	30	31	32	33	33
5'8'' - 172.7	15	16	16	17	18	19	19	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	32	32
5'9'' - 175.2	14	15	16	17	17	18	19	20	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	31
5'10'' - 177.8	14	15	15	16	17	18	18	19	20	20	21	22	23	23	24	25	25	26	27	28	28	29	30	30
5'11'' - 180.3	14	14	15	16	16	17	18	18	19	20	21	21	22	23	23	24	25	25	26	27	28	28	29	30
6'0'' - 182.8	13	14	14	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28	29
6'1'' - 185.4	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28
6'2'' - 187.9	12	13	14	14	15	16	16	17	18	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27
6'3'' - 190.5	12	13	13	14	15	15	16	16	17	18	18	19	20	20	21	21	22	23	23	24	25	25	26	26
6'4'' - 193.0	12	12	13	14	14	15	15	16	17	17	18	18	19	20	20	21	22	22	23	23	24	25	25	26

Binge Eating Disorder

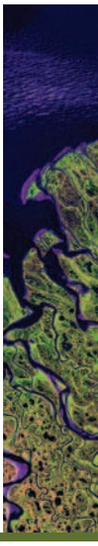
DSM-IV-TR includes **binge eating disorder** as a diagnosis in need of further study rather than as a formal diagnosis. This disorder includes recurrent binges (two times per week for at least 6 months), lack of control during the bingeing episode, and distress about bingeing, as well as other characteristics, such as rapid eating and eating alone. It is distinguished from anorexia nervosa by the absence of weight loss and from bulimia nervosa by the absence of compensatory behaviors (purging, fasting, or excessive exercise). Most often, people with binge eating disorder are **obese**. A person with a BMI of greater than 30 is considered obese. With the current explosion in the prevalence of obesity in the United States, it is perhaps not surprising that research on binge eating disorder continues to increase (Yanovski, 2003). It is important to point out, however, that not all obese people meet criteria for binge eating disorder. Indeed, only those who have binge episodes and report feeling a loss of control over their eating will qualify, which amounts to anywhere from 2 to 25 percent of obese people (Yanovski, 2003). For further discussion of obesity, see Focus on Discovery 9.1.

Though it did not meet the threshold for inclusion in the current DSM (Fairburn, Walsh, & Hay, 1993), binge eating disorder has several features that support its validity, and the bulk of evidence supports its inclusion in DSM-V (Striegel-Moore & Franco, 2008). It can be reliably defined and measured (Striegel-Moore & Franco, 2003). It is associated with obesity and a history of dieting (Kinzl et al., 1999; Pike et al., 2001). It is linked to impaired work and social functioning, depression, low self-esteem, substance abuse, and dissatisfaction with body shape (Spitzer et al., 1993; Striegel-Moore et al., 1998, 2001). Risk factors for developing binge eating disorder include childhood obesity, critical comments regarding being overweight, low self-concept, depression, and childhood physical or sexual abuse (Fairburn et al., 1998). A recent behavior genetics study (Hudson et al., 2006) found that relatives of obese people with binge eating disorder were more likely to have binge eating disorder themselves (20 percent) than were relatives of obese people without binge eating disorder (9 percent).

Binge eating disorder appears to be more prevalent than either anorexia nervosa or bulimia nervosa (Hudson et al., 2007). In the National Comorbidity Survey–Replication study, the prevalence was 3.5 percent for women and 2 percent for men. Research suggests that binge eating disorder is more common in women than men, although the gender difference is not as great as it is in anorexia or bulimia. Though only a few epidemiological studies have been done, binge eating disorder appears to be equally prevalent among Euro-, African-, Asian-, and Hispanic-Americans (Striegel-Moore & Franco, 2008).

Some researchers do not view binge eating disorder as a discrete diagnostic category but rather as a less severe version of bulimia nervosa, at least the nonpurging form of bulimia (Hay & Fairburn, 1998; Joiner, Vohs, & Heatherton, 2000; Striegal-Moore et al., 2001). On the other hand, more recent research suggests that these two conditions do differ. For example, bulimia nervosa affects women far more frequently than men, a gender difference that is not observed in binge eating disorder.

Quick Summary



Anorexia nervosa has four characteristics: refusal to maintain a normal body weight, an intense fear of gaining weight and being fat, a distorted body image, and amenorrhea. Anorexia usually begins in the early teen years and is more common in women than men. Bodily changes that can occur after severe weight loss can be serious and life threatening. About 70 percent of women with anorexia eventually recover, but it can take many years.

Bulimia nervosa involves both bingeing and purging. Bingeing often involves sweet foods and is more likely to occur when someone is alone, after a negative social encounter, and in the morning or afternoon. One striking difference between anorexia and bulimia is weight loss: people with anorexia nervosa lose a tremendous amount of weight whereas people with bulimia nervosa do not. Bulimia typically begins in late adolescence and is more common in women than men. Depression often co-occurs with bulimia, and each condition appears to be a risk factor for the other. Dangerous changes to the body can also occur as a result of bulimia, such as menstrual problems, tearing in the stomach and throat, and swelling of the salivary glands.

Binge eating disorder is characterized by several binges, and most (but not all) people are obese (defined as having a BMI greater than 30). Not all obese people meet criteria for binge eating disorder—only those who have binge episodes and report feeling a loss of control over their eating qualify. Binge eating disorder is more common than anorexia and bulimia and is more common in women than men, though the gender difference is not as great as it is in anorexia and bulimia.

FOCUS ON DISCOVERY 9.1

Obesity: A Twenty-First Century Epidemic?

Obesity is not an eating disorder, though it is an increasing public health problem, with estimated health care costs of nearly $117 billion in the year 2000 alone (U.S. Department of Health Human Services, 2001b). For example, obesity is linked to diabetes, hypertension, cardiovascular disease, and several forms of cancer. Studies have found that blood pressure is rising among children, and this may be in part due to the increase in the number of children who are overweight or obese (Muntner et al., 2004). Indeed, 90 percent of children who are overweight or obese have at least one additional heart disease risk factor such as high blood pressure or high cholesterol (*Time*, June 23, 2008).

Compared to the early 1990's, obesity among adults has increased 30 percent. Among children, it has increased 100 percent. In 2004, nearly 19 percent of children in the United States were obese and one-third of children were overweight. There is a glimmer of good news, however. Rates of obesity among adult women in the U.S. have remained steady since 1999. The news for men is less optimistic, though a recent report from the Centers for Disease Control and Prevention suggests that the rates among men may be leveling off as well (Ogden et al., 2007). Among children, the rates may have stopped increasing in 2006, though it is still too early to tell if this trend will remain (Ogden, Carroll, & Flegal, 2008). Even with prevalence rates appearing to plateau, large numbers of people in the United States are nevertheless obese or overweight. In 2006, more than a third of adult men and women in the United States were obese (Ogden et al., 2007). Obesity is also increasing in other parts of the world, from the Australian Aborigines to children in Egypt, from Siberia to Peru (Friedrich, 2002). Why are so many people overweight?

Obesity has become quite prevalent across the world in recent years. (Bourreau/Photo Researchers, Inc.)

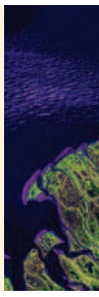
A number of factors play a role, including the environment we live in. In *Food Fight: The Inside Story of the Food Industry, America's Obesity Crisis, and What We Can Do About It*, Yale University psychologist Kelly Brownell calls our environment "toxic" with respect to the food and exercise options available to most people in the United States (Brownell & Horgen, 2003). The availability and amount of fast food have exponentially increased in the past decades. At the same time, many people, including children, have become more sedentary, spending more time working or playing at the computer and watching TV than ever before. Furthermore, physical education programs for children in schools have been declining (Critser, 2003). People eat in restaurants more than ever before, and portion sizes of foods, both in restaurants and in the grocery stores, are larger than ever. In fact, most people do not know the portion size of most foods recommended by the U.S. Department of Agriculture. A 20-ounce bottle of soda is not one serving, but two and one-half. The recommended serving of cheese is $1\frac{1}{2}$ ounces, about the size of a 9-volt battery. The ever-increasing portion sizes as well as the greater availability of unhealthy foods impact the amount we eat. In addition, the availability of healthy foods varies depending on economics. Research has shown that poorer neighborhoods have fewer grocery stores, more fast-food restaurants, and fewer healthy food selections in the stores (Moreland et al., 2002).

We are all subject to the continuing impact of advertisements, especially those promoting alluring high-calorie products such as snack foods, desserts, and meals at fast-food restaurants. For example, the advertising budget for Coke and Pepsi combined was $3 billion in 2001 (Brownell & Horgen, 2003). Compare this to the $2 million advertising campaign by the National Cancer Institute to promote eating more fruits and vegetables (Nestle, 2002). Children are particularly susceptible to advertising. A task force of the American Psychological Association concluded in 2004 that television advertisements of unhealthy foods (e.g., sugary cereals, soda) contribute to unhealthy eating habits of children under 8 years of age, largely because these children lack the requisite cognitive skills to discern truth from advertising (Kunkel et al., 2004).

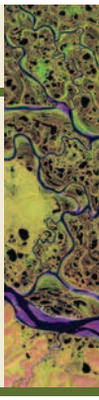
Along with the environment, heredity plays a role in obesity. In behavior genetics terms, between 25 and 40 percent of the variance in obesity can be accounted for by genetic factors (Brownell & Horgen, 2003). Adoption studies have found that children's weight is more strongly related to the weight of their biological parents than to the weight of their adoptive parents (Price et al., 1987). Similarly, 40 percent of the children of an obese parent will be obese, compared with 7 percent of the children of normal-weight parents. Heredity could produce its effects by regulating metabolic rate, impacting the hypothalamus, or influencing the production of enzymes that make it easier to store fat and gain weight. Recent molecular genetics studies have identified a number of possible genes that might contribute to obesity. A variation (polymorphism) of the Insig2 gene has sparked interest among researchers. This gene is associated with regulating fatty acids and cholesterol and is found among 10 percent of people who are obese (Herbert et al., 2006). Though genetic factors tell an important part of the story, they do not tell the entire story. Clearly, the environment plays a critical role.

Stress and its associated negative moods can induce eating in some people (Arnow, Kenardy, & Agras, 1992; Heatherton & Baumeister, 1991), and research in rats shows that foods rich in fat and sugar may actually reduce stress in the short term, giving new meaning to the term *comfort food* (Dallman et al., 2003).

The stigma associated with being overweight remains a problem. Some people think that obesity is simply a matter of personal responsibility; they believe that if people would just eat less and exercise more, obesity would not be a problem. Given the multitude of factors contributing to obesity



just noted, such a simple solution is not reasonable. Yet some members of the U.S. Congress ascribe to such beliefs. In 2004, the U.S. House of Representatives passed what became known as the "cheeseburger bill," which prevents people from suing fast-food companies for contributing to their obesity. Despite the evidence that environmental factors, including the availability and relatively unhealthy nature of a lot of fast food, some members of the House said that obesity was a matter of personal responsibility, not the responsibility of the fast-food industry. Of course, personal responsibility is important. People can and should make better choices about what and how much they eat. Nevertheless, other environmental factors can sometimes work against such choices. Other governmental agencies seem to agree that there is more to obesity than simply personal responsibility. Also in 2004, the Department of Health and Human Services announced that Medicare, the national health insurance program for the elderly or disabled, would cover treatment for obesity by removing language that had previously said obesity was not a disease (USDHHSB, 2004).



Check Your Knowledge 9.1 (Answers are at the end of the chapter.)

Answer the questions.

1. All of the following are symptoms of anorexia *except*:
   - **a.** fear of fat and gaining weight
   - **b.** unwillingness to maintain normal weight
   - **c.** perfectionism
   - **d.** distorted body image
2. Which statement is true regarding binge eating disorder?
   - **a.** It is more common in men than women.
   - **b.** It is a third type of eating disorder in DSM-IV-TR.
   - **c.** It is synonymous with obesity.
   - **d.** It includes binges but not purges.
3. Which of the following are characteristics of both anorexia and bulimia?
   - **a.** They involve a good deal of weight loss.
   - **b.** They are more common in women than men.
   - **c.** They have physical side effects (e.g., menstrual irregularities).
   - **d.** All of the above but *a* are correct.

Etiology of Eating Disorders

As with other disorders, a single factor is unlikely to cause an eating disorder. Several areas of current research—genetics, neurobiology, sociocultural pressures to be thin, personality, the role of the family, and the role of environmental stress—suggest that eating disorders result when several influences converge in a person's life.

Genetic Factors

Both anorexia nervosa and bulimia nervosa run in families. First-degree relatives of young women with anorexia nervosa are over 10 times more likely than average to have the disorder themselves (e.g., Strober et al., 2000). Similar results are found for bulimia nervosa, where first-degree relatives of women with bulimia nervosa are about four times more likely than average to have the disorder (e.g., Kassett et al., 1989; Strober et al., 2000). Furthermore, first-degree relatives of women with eating disorders appear to be at higher risk for anorexia or bulimia (Lilenfeld et al., 1998; Strober et al., 1990, 2000). Although eating disorders are quite rare among men, one study found that first-degree relatives of men with anorexia nervosa were at greater risk for having anorexia nervosa (though not bulimia) than relatives of men without anorexia (Strober et al., 2001). Finally, relatives of people with eating disorders are more likely than average to have symptoms of eating disorders that do not meet the complete criteria for a diagnosis (Lilenfeld et al., 1998; Strober et al., 2000).

Twin studies of eating disorders also suggest a genetic influence. Most studies of both anorexia and bulimia report higher MZ than DZ concordance rates (Bulik, Wade, & Kendler, 2000) and that genes account for a substantial portion of the variance among twins with eating disorders (Wade et al., 2000). On the other hand, research has shown that nonshared

environmental factors (see p. 30), like different interactions with parents or different peer groups, also contribute to the development of eating disorders (Klump, McGue, & Iacono, 2002). Research also suggests that key features of the eating disorders, such as dissatisfaction with one's body, a strong desire to be thin, binge eating, and preoccupation with weight, are heritable (Klump, McGue, & Iacono, 2000). Additional evidence suggests that common genetic factors may account for the relationship between certain personality characteristics, such as negative emotionality and constraint, and eating disorders (Klump, McGue, & Iacono, 2002). The results of these studies are consistent with the possibility that a genetic diathesis is operating, but adoption studies are also needed. Using the method of genetic linkage analysis (discussed in Chapter 4), one study reported evidence for linkage on chromosome 1 among people with anorexia (Grice et al., 2002). It will be important to replicate this finding in future studies, but all of these findings suggest that genetics do indeed play a role in eating disorders.

Neurobiological Factors

The hypothalamus is a key brain center in regulating hunger and eating. Research on animals with lesions to the lateral hypothalamus indicates that they lose weight and have no appetite (Hoebel & Teitelbaum, 1966). Thus, it is not surprising that the hypothalamus has been proposed to play a role in anorexia. The level of some hormones regulated by the hypothalamus, such as cortisol, is indeed abnormal in people with anorexia. Rather than causing the disorder, however, these hormonal abnormalities occur as a result of self-starvation, and levels return to normal after weight gain (Doerr et al., 1980; Stoving et al., 1999). Furthermore, the weight loss of animals with hypothalamic lesions does not parallel what we know about anorexia. These animals appear to have no hunger and to become indifferent to food, whereas people with anorexia continue to starve themselves despite being hungry and having an interest in food. Nor does the hypothalamic model account for body-image disturbance or fear of becoming fat. A dysfunctional hypothalamus thus does not seem highly likely as a factor in anorexia nervosa.

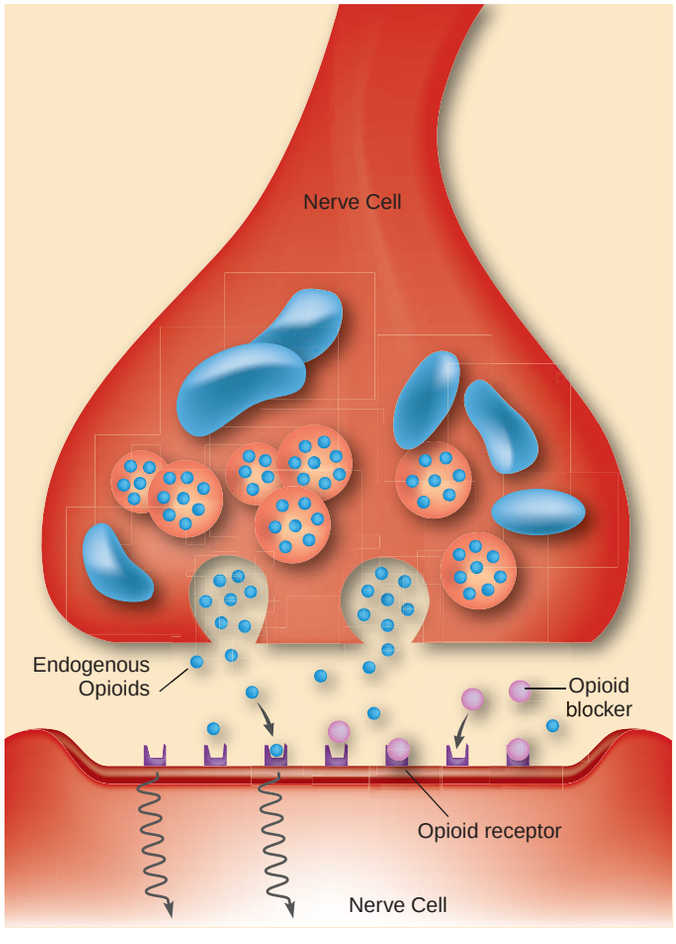


Figure 9.2 Endogenous opiod systems in the brain.

Endogenous opioids are substances produced by the body that reduce pain sensations, enhance mood, and suppress appetite. Opioids are released during starvation and have been hypothesized to play a role in both anorexia and bulimia. Starvation among people with anorexia may increase the levels of endogenous opioids, resulting in a positively reinforcing euphoric state (Marrazzi & Luby, 1986). Furthermore, the excessive exercise seen among some people with eating disorders would increase opioids and thus be reinforcing (Davis, 1996; Epling & Pierce, 1992).

Some research supports the theory that endogenous opioids play a role in eating disorders, at least in bulimia. For example, two studies found low levels of the endogenous opioid beta-endorphin (see Figure 9.2) in people with bulimia (Brewerton et al., 1992; Waller et al., 1986). In one of these studies, the researchers observed that the people with more severe cases of bulimia had the lowest levels of beta-endorphin (Waller et al., 1986). It is important to note, however, that these findings demonstrate that low levels of opioids are seen concurrently with bulimia, not that such levels are seen before the onset of the disorder. In other words, we don't know if the low levels of opioids are a cause of bulimia or an effect of changes in food intake.

Finally, some research has focused on neurotransmitters related to eating and satiety (feeling full). Animal research has shown that serotonin promotes satiety. Therefore, it could be that the binges of people with bulimia result from a serotonin deficit that causes them not to feel

satiated as they eat. Animal research has also shown that food restriction interferes with serotonin synthesis in the brain. Thus, among people with anorexia, the severe food intake restrictions could interfere with the serotonin system.

Researchers have examined levels of serotonin metabolites among people with anorexia and bulimia. With respect to anorexia, several studies have reported low levels of serotonin metabolites among people with anorexia (e.g., Kaye et al., 1984) and bulimia (e.g., Carrasco et al., 2000; Jimerson et al., 1992; Kaye et al., 1998). Lower levels of a neurotransmitter's metabolites are one indicator that the neurotransmitter activity is underactive. In addition, people with anorexia who have not been restored to a healthy weight show a poorer response to serotonin agonists (i.e., a drug that stimulates serotonin receptors) than those people who have regained a good portion of their weight, again suggesting an underactive serotonin system (Attia et al., 1998; Ferguson et al., 1999). People with bulimia also show smaller responses to serotonin agonists (Jimerson et al., 1997; Levitan et al., 1997). The antidepressant drugs that are often effective treatments for anorexia and bulimia (discussed later) are known to increase serotonin activity, adding to the possible importance of serotonin. Serotonin, though, could be also be linked to the comorbid depression often found in anorexia and bulimia.

Researchers have recently begun to more closely examine the role of the neurotransmitter dopamine in eating behavior. Studies with animals have shown that dopamine is linked to the pleasurable aspects of food that compel an animal to go after food (e.g., Szczypka et al., 2001) and brain imaging studies in humans have shown how dopamine is linked to the motivation to obtain food. In one study with healthy people, participants were presented with smells and tastes of food while undergoing a PET scan (Volkow et al., 2002). The participants also filled out a measure of dietary restraint (see Table 9.3). People who scored higher on dietary restraint exhibited greater dopamine activity in the dorsal striatum area of the brain during the presentation of food. This finding suggests that restrained eaters may be more sensitive to cues of food, since one of the functions of dopamine is to signal the salience of particular stimuli. Whether or not these findings will be relevant to people with eating disorders remains to be seen.

Though we can expect further neurotransmitter research in the future, keep in mind that much of this work focuses on brain mechanisms relevant to hunger, eating, and satiety and does little to account for other key features of both disorders, in particular the intense fear of becoming fat. Furthermore, as suggested, the evidence so far does not show that brain changes predate the onset of eating disorders.

Table 9.3 The Restraint Scale

1.	How often are you dieting? Never; rarely; sometimes; often; always.
2.	What is the maximum amount of weight (in pounds) you have ever lost within 1 month? 0–4; 5–9; 10–14; 15–19; 20+.
3.	What is your maximum weight gain within a week? 0–1; 1.1–2; 2.1–3; 3.1–5; 5.1+.
4.	In a typical week, how much does your weight fluctuate? 0–1; 1.1–2; 2.1–3; 3.1–5; 5.1+.
5.	Would a weight fluctuation of 5 pounds affect the way you live your life? Not at all; slightly; moderately; very much.
6.	Do you eat sensibly in front of others and splurge alone? Never; rarely; often; always.
7.	Do you give too much time and thought to food? Never; rarely; often; always.
8.	Do you have feelings of guilt after overeating? Never; rarely; often; always.
9.	How conscious are you of what you are eating? Not at all; slightly; moderately; extremely.
10.	How many pounds over your desired weight were you at your maximum weight? 0–1; 1–5; 6–10; 11–20; 21+.

Source: From Polivy Herman, & Howard (1980).

Psychodynamic Views

Many psychodynamic theories of eating disorders propose that the core cause is to be found in disturbed parent–child relationships and that certain core personality traits, such as low self-esteem and perfectionism, are found among people with eating disorders. As we will see in a

later section, these personality characteristics are not solely the province of psychodynamic theorists. Psychodynamic theories also propose that the symptoms of an eating disorder fulfill some need, such as increasing one's sense of personal effectiveness by being successful in maintaining a strict diet or avoiding growing up sexually by being very thin and thus not achieving the usual female shape (Goodsitt, 1997).

One view holds that anorexia nervosa is an attempt by children who have been raised to feel ineffectual to gain competence and respect and to ward off feelings of helplessness, ineffectiveness, and powerlessness (Bruch, 1980). This sense of ineffectiveness is believed to be created by a parenting style in which the parents' wishes are imposed on the child without consideration of the child's needs or wishes. For example, parents may arbitrarily decide when the child is hungry or tired, failing to perceive the child's actual state. Children reared in this way do not learn to identify their own internal states and do not become self-reliant. Facing the demands of adolescence, the child seizes on the societal emphasis on thinness and turns dieting into a means of acquiring control and identity. Moreover, negative self-perceptions about weight become the broad lens through which the child sees other aspects of the self, thus contributing to an overall poor self-evaluation.

Another psychodynamic theory, described by Goodsitt (1997), proposes that bulimia nervosa in women stems from a failure to develop an adequate sense of self because of a conflictual mother–daughter relationship. Food becomes a symbol of this failed relationship. The daughter's bingeing and purging represent the conflict between the need for the mother and the desire to reject her.

Although interesting theories, the evidence in favor of these psychodynamic views is limited. Independent of these psychodynamic theories, studies of personality characteristics of people with eating disorders and studies of the characteristics of their families have revealed interesting relationships between these variables and eating disorders. But it is difficult to reach definitive conclusions in either area of research because the disorder itself may have resulted in changes in personality or in the patient's family.

Cognitive Behavioral Views

Anorexia Nervosa Cognitive behavioral theories of anorexia nervosa emphasize fear of fatness and body-image disturbance as the motivating factors that powerfully reinforce weight loss. Many who develop anorexia symptoms report that the onset followed a period of weight loss and dieting. Behaviors that achieve or maintain thinness are negatively reinforced by the reduction of anxiety about becoming fat. Furthermore, dieting and weight loss may be positively reinforced by the sense of mastery or self-control they create (Fairburn, Shafran, & Cooper, 1999; Garner, Vitousek, & Pike, 1997). Some theories also include personality and sociocultural variables in an attempt to explain how fear of fatness and body-image disturbances develop. For example, perfectionism and a sense of personal inadequacy may lead a person to become especially concerned with his or her appearance, making dieting a potent reinforcer. Similarly, seeing portrayals in the media of thinness as an ideal, being overweight, and tending to compare oneself with especially attractive others all contribute to dissatisfaction with one's body (Stormer & Thompson, 1996).

The fear of being fat, which is so important in eating disorders, is partly based on society's negative stereotypes about overweight people. (The Copyright Group/SUPERSTOCK.)

Another important factor in producing a strong drive for thinness and a disturbed body image is criticism from peers and parents about being overweight (Paxton et al., 1999). In one study supporting this conclusion, adolescent girls aged 10 to 15 were evaluated twice, with a 3-year interval between assessments. Obesity at the first assessment was related to being teased by peers and at the second assessment was linked to dissatisfaction with their bodies. Dissatisfaction was in turn related to symptoms of eating disorder.

It is known that bingeing frequently results when diets are broken (Polivy & Herman, 1985). Thus, when a lapse occurs in the strict dieting of a person with anorexia nervosa, the lapse is likely to escalate into a binge. The purging after an episode of binge eating can

again be seen as motivated by the fear of weight gain that the binge elicited. People with anorexia who do not have episodes of bingeing and purging may have a more intense preoccupation with and fear of weight gain (Schlundt & Johnson, 1990) or may be more able to exercise self-control.

Bulimia Nervosa People with bulimia nervosa are also thought to be overconcerned with weight gain and body appearance; indeed, they judge their self-worth mainly by their weight and shape. They also have low self-esteem, and because weight and shape are somewhat more controllable than are other features of the self, they tend to focus on weight and shape, hoping their efforts in this area will make them feel better generally. They try to follow a pattern of restrictive eating that is very rigid, with strict rules regarding how much to eat, what kinds of food to eat, and when to eat. These strict rules inevitably are broken, and the lapse escalates into a binge. After the binge, feelings of disgust and fear of becoming fat build up, leading to compensatory actions such as vomiting (Fairburn, 1997). Although purging temporarily reduces the anxiety from having eaten too much, this cycle lowers the person's self-esteem, which triggers still more bingeing and purging, a vicious circle that maintains desired body weight but has serious medical consequences (see Figure 9.3 for a summary of this theory).

One group of researchers developed the Restraint Scale (see Table 9.3), a questionnaire measure of concerns about dieting and overeating (Polivy et al., 1980). These researchers have conducted a series of laboratory studies on people with high scores on this measure. These studies are generally conducted under the guise of being taste tests. One such study was described as an assessment of the effects of temperature on taste (Polivy, Heatherton, & Herman, 1988). To achieve a "cold" condition, some participants first drank a 15-ounce chocolate milk shake (termed a *preload* by the investigators) and were then given three bowls of ice cream to taste and rate for flavor. Participants were told that once they had completed their ratings, they could eat as much of the ice cream as they wanted. The researchers then measured the amount of ice cream eaten.

In laboratory studies following this general design, people who scored high on the Restraint Scale ate more than nondieters after a fattening preload, even when the preload was perceived as fattening but was actually low in calories (e.g., Polivy, 1976) and even when the food was relatively unpalatable (Polivy, Herman, & McFarlane, 1994). Thus, people who score high on the Restraint Scale show a pattern similar to that of people with bulimia nervosa, albeit at a much less intense level.

Several additional conditions have been found to further increase the eating of restrained eaters after a preload, most notably various negative mood states, such as anxiety and depression (e.g., Herman et al., 1987). The increased consumption of restrained eaters is especially pronounced when their self-image is threatened (Heatherton, Herman, & Polivy, 1991) and if they have low self-esteem (Polivy et al., 1988). Finally, when restrained eaters are given false feedback indicating that their weight is high, they respond with increases in negative emotion and increased food consumption (McFarlane, Polivy, & Herman, 1998).

The eating pattern of people with bulimia is similar to, but more extreme than, the behavior highlighted in the studies of restrained eaters. People with bulimia nervosa typically binge when they encounter stress and experience negative affect, and this has been shown in several studies. In experience sampling studies, the investigators were able to show how specific binge-and-purge events were linked to changes in emotions and stress in the course of daily life (Smyth et al, 2007).

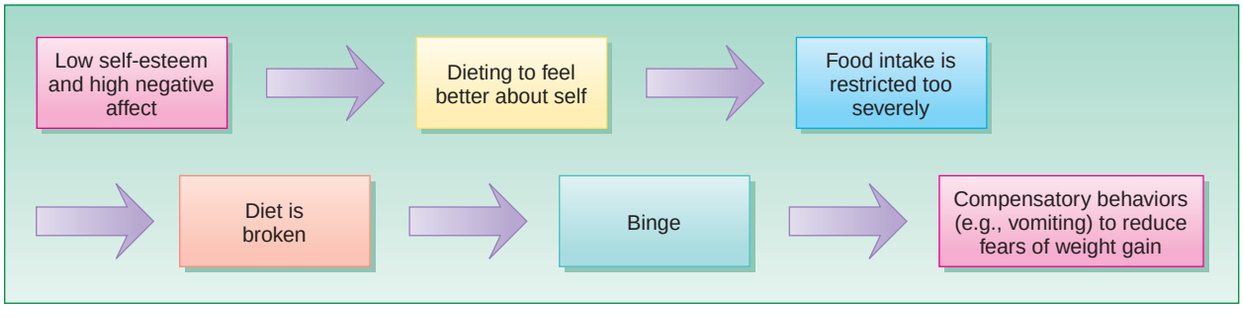


Figure 9.3 Schematic of cognitive behavioral theory of bulimia nervosa.

They found that high negative affect and stress alongside low positive affect predicted later bingeing. The binge may therefore function as a means of regulating negative affect (Stice & Agras, 1999; Smyth et al., 2007). Evidence also supports the idea that stress and negative affect are relieved by purging. People with bulimia report increased anxiety and show heightened skin conductance when they eat a meal and are not allowed to purge (Leitenberg et al., 1984; Williamson et al., 1988). In addition, negative affect levels decline and positive affect levels increase after a purge event, supporting the idea that purging is reinforced by negative affect reduction (Jarrell, Johnson, & Williamson, 1986; Smyth et al., 2007). Given the similarities between people who score high on the Restraint Scale and people with bulimia nervosa, we might expect that restrained eating would play a central role in bulimia. In fact, a study of the naturalistic course of bulimia (i.e., the course of bulimia left untreated) has found that the relationship between concern over shape and weight and binge eating was partially mediated by restrained eating (Fairburn et al., 2003). In other words, concerns about body shape and weight predicted restrained eating, which in turn predicted an increase in binge eating across 5 years of follow-up assessments. Other studies have failed to find this relationship (Burne & McLean, 2002), however, and thus additional research will need to sort out the ways in which restraint is linked with the symptoms of bulimia.

Sociocultural factors appear to play a role in the faulty perceptions and eating habits of those with eating disorders. We turn to these influences next.

Quick Summary

Genetic factors appear to play a role in both anorexia and bulimia. Both disorders tend to run in families, and twin studies support the role of genetics in the actual disorders and particular characteristics of the disorders, such as body dissatisfaction, preoccupation with thinness, and binge eating. The hypothalamus does not appear to be directly involved in eating disorders, and low levels of endogenous opioids are seen concurrently with bulimia, but not before the onset of the disorder. Thus, changes in food intake could affect the opioid system instead of changes in the opioid system affecting food intake. Research findings on the role of serotonin in anorexia are mixed. Serotonin may play a role in bulimia, with studies finding a decrease in serotonin metabolites, smaller responses to serotonin agonists, and an increase in cognitions related to eating disorders, such as feeling fat, among people formerly diagnosed with bulimia who had their serotonin levels reduced. Newer research suggests dopamine may play a role in restrained eating, a characteristic that is found in people with eating disorders. The neurobiological factors do not do a particularly good job of accounting for some key features of anorexia and bulimia, in particular the intense fear of becoming fat.

Psychodynamic theories focus on disturbed parent–child relationships and personality characteristics. There is not much research support for these views.

Cognitive behavioral theories focus on body dissatisfaction and preoccupation with thinness. The Restraint Scale measures concerns about dieting and overeating, and high scores are linked to binge eating among people without eating disorders. The eating pattern of people with bulimia is similar to, but more extreme than, the behavior highlighted in the studies of restrained eaters. Studies have found that concerns about body shape and weight predicted restrained eating, which in turn predicted an increase in binge eating.

Sociocultural Factors

Throughout history, the standards societies have set for the ideal body—especially the ideal female body—have varied greatly. Think of the famous nudes painted by Rubens in the seventeenth century: according to modern standards, these women are chubby. Over the past 50 years, the American cultural ideal has progressed steadily toward increasing thinness. *Playboy* centerfolds became thinner between 1959 and 1978, for example (Garner et al., 1980), and beauty pageant contestants also became thinner through 1988. A study that calculated the body mass index (BMI) of *Playboy* centerfolds from 1985 to 1997 (Owen & Laurel-Seller, 2000) found that all but one of the *Playboy* centerfolds had a BMI of less than 20, which is considered to be a low weight, and almost half of the centerfolds had a BMI of less than 18, which is considered to be underweight.

For men, the situation appears somewhat different. In a study parallel to the studies examining *Playboy* centerfolds, researchers analyzed the BMI of *Playgirl* male centerfolds from 1973 to 1997 (Leit, Pope, & Gray, 2001). They found that the centerfolds' BMI *increased* over the period and that their muscularity, assessed using a fat-to-muscle estimate, increased even more. Thus for men, magazines focus attention on the masculine ideal of normal body weight or on increased muscle mass (Mishkind et al., 1986).

Somewhat paradoxically, as cultural standards were moving in the direction of thinness over the later part of the twentieth century, more and more people were becoming overweight. The prevalence of obesity has doubled since 1900 (see Focus on Discovery 9.1). Currently, nearly 30 percent of Americans are obese (BMI greater than 30), setting the stage for greater conflict between the cultural ideal and reality.

As society has become more health and fat conscious, dieting to lose weight has become more common; the number of dieters increased from 7 percent of men and 14 percent of women in 1950 to 29 percent of men and 44 percent of women in 1999 (Serdula et al., 1999). The focus on cutting carbohydrates, so widespread during the past few years, added yet another craze to dieting. For example, the sale of low-carb foods yielded nearly $30 billion in 2004; more than 1,500 new low-carb foods were introduced in a 2-year period; the number of low-carb diet books increased from 15 to 194 between 1999 and 2004; and 26 million people in the United States were on a diet that severely limited carbohydrate consumption in 2004 (*Time*, May 3, 2004). Like many diet fads, the low-carb craze has quieted a bit since 2004. Finally, surgeries such as liposuction (vacuuming out fat deposits just under the skin) and gastroplasty (surgically changing the stomach so it cannot digest as much food) are becoming more common despite their risk (Brownell & Horgen, 2003).

The percentages above indicate that women are more likely than men to be dieters. The onset of eating disorders is typically preceded by dieting and other concerns about weight, supporting the idea that social standards stressing the importance of thinness play a role in the development of these disorders (Killen et al., 1994; Stice, 2001).

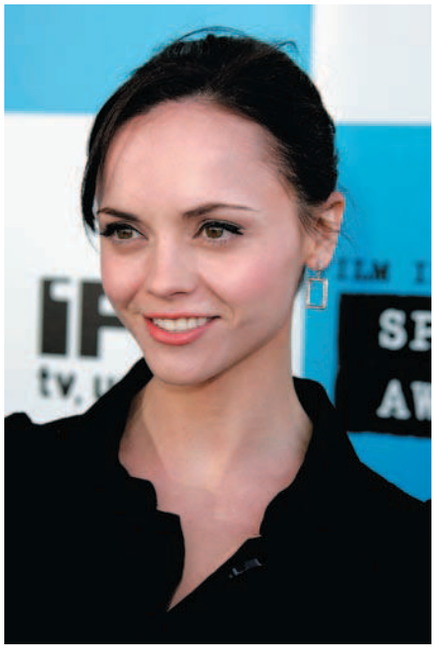
It is likely that women who either are actually overweight or fear being fat are also dissatisfied with their bodies. Not surprisingly, studies have found people with both a high BMI and body dissatisfaction are at higher risk for developing eating disorders (Fairburn et al., 1997; Killen et al., 1996). Body dissatisfaction is also a robust predictor of the development of eating disorders among adolescent girls (Killen et al., 1996). In addition, preoccupation with being thin or feeling pressure to be thin predicts an increase in body dissatisfaction among adolescent girls, which in turn predicts more dieting and negative emotions. Preoccupation with thinness and body dissatisfaction both predict greater eating disorder pathology (Stice, 2001), and these factors were operating in the case of Jill, presented earlier. Finally, exposure to media portrayals of unrealistically thin models can influence reports of body dissatisfaction. One study reviewed results from 25 experiments that presented images of thin models to women and then asked the women to report on their body satisfaction. Perhaps not surprisingly, results from these studies showed that women reported a decline in body satisfaction after viewing these images (Groesz, Levine, & Murnen, 2002). Another study found that men's body dissatisfaction, as indexed by a greater discrepancy between the muscularity of the actual and ideal self, increased after viewing images of muscular men (Leit, Gray, & Pope, 2002).

The sociocultural ideal of thinness is a likely vehicle through which people learn to fear being or even feeling fat, and this was probably influential in the cases of both Lynne and Jill. In addition to creating an undesired physical shape, fat has negative connotations, such as being unsuccessful and having little self-control. Obese people are viewed by others as less smart and are stereotyped as lonely, shy, and greedy for the affection of others (DeJong & Kleck, 1986). Even more disturbing, health professionals who specialize in obesity have also exhibited beliefs that obese people are lazy, stupid, or worthless (Schwartz et al., 2003). Reducing the stigma associated with being overweight will be beneficial to those with eating disorders as well as obesity.

Not only does the fear of being fat contribute to eating pathology, but more recently the celebration of extreme thinness via websites, blogs, and magazines may also play a role. Websites that are "pro-ana" (short for anorexia) or "pro-mia" (short for bulimia) and other "thinsperation" websites and blogs have developed a following of women who seek support and encouragement for losing weight, often to a dangerously low level. These sites often post photos of female celebrities who are extremely thin as inspiration (hence, the term *thinsperation*). Some of these women have publicly discussed their struggles with eating disorders (e.g., the actress Christina Ricci), but others have not.

Gender Influences We have discussed the fact that eating disorders are more common in women than in men. One primary reason for the greater prevalence of eating disorders among women is likely due to the fact that Western cultural standards about thinness have changed over the past 50 years, today reinforcing the desirability of being thin for women more than for men.

Al Roker, the weatherperson on NBC's *Today Show*, had Gastric Bypass surgery to accomplish weight loss. (Evan Agostini/Getty Images News and Sport Services.)



Celebrities such as Christina Ricci have publicly discussed their struggles with eating disorders. (Allstar Picture Library/Alamy.)

Cultural standards regarding the ideal feminine shape have changed over time. Even in the 1950s and 1960s, the feminine ideal was considerably heavier than what it became in the 1970s, 1980s, 1990s, and today. (Top: Corbis Images; center: Eve Arnold/Magnum Photos, Inc.; bottom: Maria C. Valentino/Corbis Sygma.)

Another sociocultural factor, though, has remained remarkably resilient to change—namely, the objectification of women's bodies. Women's bodies are often viewed through a sexual lens; in effect, women are defined by their bodies, whereas men are esteemed more for their accomplishments. According to objectification theory (Fredrickson & Roberts, 1997), the prevalence of objectification messages in Western culture (in television, advertisements, and so forth) has led some women to "self-objectify," which means that they see their own bodies through the eyes of others. Research has shown that self-objectification causes women to feel more shame about their bodies. The emotion of shame is most often elicited in situations where an individual's ideal falls short of a cultural ideal or standard. Thus, women are likely experiencing body shame when they see a mismatch between their ideal self and the cultural (objectified) view of a woman. Research has also shown that both self-objectification and body shame are associated with disordered eating (Fredrickson et al., 1998; McKinley & Hyde, 1996; Noll & Fredrickson, 1998). The risk for eating disorders among groups of women who might be expected to be particularly concerned with their weight—for example, models, dancers, and gymnasts, as in the case of Jill—appears to be especially high (Garner et al., 1980).

Do eating disorders and weight concerns go away as women get older? A large, 20-year prospective study of over 600 men and women recently reported important differences in dieting and other eating disorder risk factors for men and women (Keel et al., 2007). The men and women were first surveyed about dieting, BMI, weight, body image, and eating disorder symptoms when they were in college. Follow-up surveys were completed 10 and 20 years after college. Thus, the men and women were around age 40 at the 20-year follow-up assessment. The researchers found that after 20 years, women dieted less and were less concerned about their weight and body image compared to when they were in college, even though they actually weighed more. In addition, eating disorder symptoms decreased over the 20 years for women, as did the risk factors for eating disorders (body-image perception, frequency of dieting). Changes in life roles—having a life partner, having a child—were also associated with decreases in eating disorder symptoms for women. By contrast, men were more concerned about their weight and were dieting more. Like women, they weighed more in their early forties than when they were in college. Decreases in risk factors such as body image and dieting frequency were also associated with decreases in eating disorder symptoms for men.

Cross-Cultural Studies Evidence for eating disorders across cultures depends on the disorder. Anorexia has been observed across a number of cultures and countries besides the United States; for example, in Hong Kong, China, Taiwan, England, Korea, Japan, Denmark, Nigeria, South Africa, Zimbabwe, Ethiopia, Iran, Malaysia, India, Pakistan, Australia, the Netherlands, and Egypt (Keel & Klump, 2003). Furthermore, cases of anorexia have been documented in cultures with very little Western culture influence. An important caveat must be made, however. The anorexia observed in these diverse cultures does not always include the intense fear of gaining weight or being fat that is part of the DSM-IV-TR criteria. Thus, intense fear of fat likely reflects an ideal more widely espoused in more Westernized cultures. For example, Lee (1994) has described a disorder similar to anorexia nervosa that exists in several nonindustrialized Asian countries (India, Malaysia, the Philippines). This disorder involves severe emaciation, food refusal, and amenorrhea, but not a fear of becoming fat. Is this a cultural variant of anorexia or a different disorder, such as depression? This question is but one of the challenges that face cross-cultural researchers (Lee et al., 2001). Indeed, in some other cultures, higher weight among women is especially valued and considered a sign of fertility and healthiness (Nasser, 1988). The wide variation in the clinical presentation of anorexia across cultures provides a window into the importance of culture in establishing realistic versus potentially disordered views of one's body.

Another feature of eating disorders that may be heavily influenced by Western ideals of beauty and thinness is body image. In a study supporting the notion of cross-cultural differences in body-image perception, Ugandan and British college students rated the attractiveness of drawings of nudes ranging from very emaciated to very obese (Furnham & Baguma, 1994). Ugandan students rated the obese females as more attractive than did the British students.

Bulimia nervosa appears to be more common in industrialized societies, such as the United States, Canada, Japan, Australia, and Europe, than in nonindustrialized nations. In addition, as cultures undergo social changes associated with adopting the practices of more Westernized cultures, the incidence of bulimia appears to increase (Abou-Saleh, Younis, & Karim, 1998; Nasser, 1997). Indeed, a comprehensive review of research on culture and eating disorders could not find evidence of bulimia outside of a Westernized culture (Keel & Klump, 2003).

Another indication of our society's preoccupation with thinness is what happened with Miss Universe of 1996, Alicia Machado. When, after winning the title, she gained a few pounds, some people became outraged and suggested she give up her crown. (AP/Wide World Photos.)

Ethnic Differences In the United States, it was reported at one time that the incidence of anorexia was eight times greater in white women than in women of color (Dolan, 1991). More recent studies confirm somewhat greater eating disturbances and body dissatisfaction among white women than black women (Grabe & Hyde, 2006; Perez & Joiner, 2003), but differences in actual eating disorders, particularly bulimia, do not appear to be as great (Wildes, Emery, & Simons, 2001). In addition, the greatest differences between white and black women in eating disorder pathology appear to be most pronounced in college student samples; fewer differences are observed in either high school or nonclinical community samples (Wildes et al., 2001). Finally, a recent meta-analysis found more similarities than differences in body dissatisfaction between ethnic groups in the United States (Grabe & Hyde, 2006). White women and Hispanic women reported greater body dissatisfaction than African American women, but no other ethnic differences were reliably found.

Differences have been observed in the United States in some areas, however. Studies show that white teenage girls diet more frequently than do African American teenage girls and are more likely to be dissatisfied with their bodies (Fitzgibbons et al., 1998; Striegel-Moore et al., 2000). The relationship between BMI and body dissatisfaction also differs by ethnicity. Compared with African American adolescents, white adolescents become more dissatisfied with their bodies as their BMI rises (Striegel-Moore et al., 2000). As already noted, both dieting and body dissatisfaction are related to an increased risk for developing an eating disorder. Indeed, one study found that white women with binge eating disorder were more dissatisfied with their bodies than African American women with binge eating disorder, and the white women were more likely to have a history of bulimia nervosa than the African American women (Pike et al., 2001).

Ethnic group membership is not the only critical variable in observed differences. Socioeconomic status may also be important (Caldwell, Brownell, & Wilfley, 1997; French et al., 1997). The emphasis on thinness and dieting has spread beyond white women of upper and middle socioeconomic status to women of lower socioeconomic status, as has the prevalence of eating disorder pathology (e.g., Story et al., 1995; Striegel-Moore et al., 2000). In addition, acculturation, the extent to which someone assimilates their own culture with a new culture, may be another important variable to consider. This process can at times be quite stressful. A recent study found that the relationship between body dissatisfaction and bulimia symptoms was stronger for African American and Hispanic college students who reported higher levels of acculturative stress compared to those students who reported lower levels of this type of stress (Perez et al., 2002).

Standards of beauty vary vary cross-culturally as shown by Gaugain's painting of Tahitian women. (Musee d'Orsay, Paris/Lauris-Giraudon, Paris/SUPERSTOCK.)

Finally, very little is known about the prevalence of eating disorders among Latina or Native American women, and this remains a much-needed research focus. Data from a recent epidemiological study of Latina women age 18 or older found that binge eating disorder was more prevalent than bulimia nervosa but that the prevalence rates for both disorders were comparable to prevalence rates in Caucasian women (Alegria et al., 2007). The diagnosis of bulimia was more likely for women who had lived in the United States for several years than for women who had recently immigrated, indicating that acculturation may play a role. In contrast to other eating disorders, anorexia nervosa was very rare among Latina women (only 2 out of over 2,500 women had a lifetime history of anorexia).

Beyond the study of racial or ethnic differences in eating disorders, attention should also be paid to stereotyped beliefs about race and eating disorders.

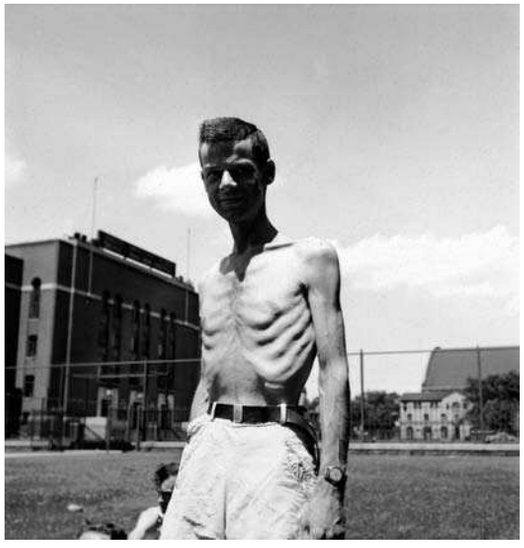
One study found that college students who read a fictional case study about a woman with eating disorder symptoms were more likely to ascribe an eating disorder to the woman if her race was presented as Caucasian rather than African American or Hispanic (Gordon, Perez, & Joiner, 2002). In other words, the symptoms were only rated as clinically significant for the white case presentation, not for the African American or Hispanic ones, even though the details were identical. Although it remains to be seen if mental health professionals would also exhibit the same stereotypes when making clinical judgments, it suggests that symptoms may be more easily overlooked among non-Caucasian women.

Other Factors Contributing to the Etiology of Eating Disorders

Personality Influences We have already seen that neurobiological changes occur as a result of an eating disorder. It is also important to keep in mind that an eating disorder itself can affect personality. A study of semistarvation in male conscientious objectors conducted in the late 1940s supports the idea that the personality of people with eating disorders, particularly those with anorexia, is affected by their weight loss (Keys et al., 1950). For a period of 6 weeks, the men were given two meals a day, totaling 1,500 calories, to simulate the meals in a concentration camp. On average, the men lost 25 percent of their body weight. They all soon became preoccupied with food; they also reported increased fatigue, poor concentration, lack of sexual interest, irritability, moodiness, and insomnia. Four became depressed, and one developed bipolar disorder. This research shows vividly how severe restriction of food intake can have powerful effects on personality and behavior, which we need to consider when evaluating the personalities of people with anorexia and bulimia.

In part as a response to the findings just mentioned, some researchers have collected retrospective reports of personality before the onset of an eating disorder. This research describes people with anorexia as having been perfectionistic, shy, and compliant before the onset of the disorder. The description of people with bulimia includes the additional characteristics of histrionic features, affective instability, and an outgoing social disposition (Vitousek & Manke, 1994). It is important to remember, however, that retrospective reports in which people with an eating disorder and their families recall what the person was like before diagnosis can be inaccurate and biased by awareness of the patient's current problem.

Prospective studies examine personality characteristics before an eating disorder is present. In one study, more than 2,000 students in a suburban Minneapolis school district completed a variety of tests for three consecutive years. Among the measures were assessments of personality characteristics as well as an index of the risk for developing an eating disorder based on the Eating Disorders Inventory. During year 1 of the study, cross-sectional predictors of disordered eating included body dissatisfaction; a measure of interoceptive awareness, which is the extent to which people can distinguish different biological states of their bodies (see Table 9.1 for items that assess interoceptive awareness); and a propensity to experience negative emotions (Leon et al., 1995). At year 3, these same variables were found to have prospectively predicted disordered eating (Leon et al., 1999). An additional study found that perfectionism prospectively predicted the onset of anorexia in young adult women (Tyrka et al., 2002).



Severe food restriction can have profound effects on behavior and personality, as illustrated by the Keyes study. (Wallace Kirkland/Time & Life Pictures/Getty Images, Inc.)

Additional research has taken a closer look at the link between perfectionism and anorexia. Perfectionism is multifaceted and may be self-oriented (setting high standards for oneself), other-oriented (setting high standards for others), or socially oriented (trying to conform to the high standards imposed by others). A recent review of many studies concludes that perfectionism, no matter how it is measured, is higher among girls with anorexia than girls without anorexia and that perfectionism remains high even after successful treatment for anorexia (Bardone-Cone et al, 2007). A multinational study found that people with anorexia scored higher on self and other-oriented types of perfectionism than people without anorexia (Halmi et al., 2000). Finally, mothers of girls with anorexia scored higher on perfectionism than mothers of girls without anorexia (Woodside et al., 2002). This intriguing finding needs to be replicated, but it suggests that what is genetically transmitted in anorexia could be a personality characteristic, such as perfectionism, that increases the vulnerability for the disorder rather than the disorder per se.

Characteristics of Families Studies of the characteristics of families of people with eating disorders have yielded variable results. Some of the variation stems, in part, from the different methods used to collect the data and from the sources of the information. For example, self-reports of people with eating disorders consistently reveal high levels of conflict in the family (e.g., Bulik, Sullivan, et al., 2000; Hodges, Cochrane, & Brewerton, 1998). Reports of parents, however, do not necessarily indicate high levels of family problems.

Family characteristics may contribute to the risk for developing an eating disorder; however, eating disorders also likely have an impact on family functioning. One study assessed both people with eating disorders and their parents on tests designed to measure rigidity, closeness, emotional overinvolvement, critical comments, and hostility (Dare et al., 1994). The families showed considerable variation in whether parents were overinvolved with their children; the families were also quite low in conflict (low levels of criticism and hostility). A family study in which assessments were conducted before and after treatment of the patient found that ratings of family functioning improved after treatment (Woodside et al., 1995). Finally, one study examined identical twins discordant for bulimia (i.e., one twin had the disorder; the other didn't). The twin who developed bulimia reported greater family discord than the twin who did not develop the disorder. Because these studies rely on retrospective self-report, it remains unclear whether the family discord was a contributory factor or consequence of the eating disorder.

People with eating disorders consistently report that their family life was high in conflict. (Penny Tweedie/Stone/Getty Images.)

To better understand the role of family functioning, it will be necessary to study these families directly by observational measures rather than by self-reports alone. Although an adolescent's perception of his or her family's characteristics is important, we also need to know how much of reported family discord is perceived and how much is consistent with others' perceptions. In one of the few observational studies conducted thus far, parents of children with eating disorders did not appear to be very different from control parents. The two groups did not differ in the frequency of positive and negative messages given to their children, and the parents of children with eating disorders were more self-disclosing than were the controls. The parents of children with eating disorders did lack some communication skills, however, such as the ability to request clarification of vague statements (van den Broucke, Vandereycken, & Vertommen, 1995). Observational studies such as this, coupled with data on perceived family characteristics, would help determine whether actual or perceived family characteristics are related to eating disorders.

Child Abuse and Eating Disorders Some studies have indicated that self-reports of childhood sexual abuse are higher among people with eating disorders than among people without eating disorders, especially those with bulimia nervosa (Deep et al., 1999; Webster & Palmer, 2000). Since, as discussed in Chapter 6, some research indicates that reports of abuse may be created in therapy, it is notable that high rates of sexual abuse have been found among people with eating disorders who have not been in treatment as well as those who have (Romans et al., 2001; Wonderlich et al., 1996, 2001). Still, the role of childhood sexual abuse in the etiology of eating disorders remains uncertain. Furthermore, high rates of childhood sexual abuse are found among people with different diagnoses so if it plays some role, it may not be highly specific to eating disorders (Fairburn et al., 1999; Romans et al., 2001).

Research has also found higher rates of childhood physical abuse among people with eating disorders. These data suggest that future studies should focus on a broad range of abusive experiences. Furthermore, it has been suggested that the presence or absence of abuse may be too general a variable. Abuse at a very early age, involving force and by a family member, may bear a stronger relationship to eating disorders than abuse of any other type (Everill & Waller, 1995).

Quick Summary

Sociocultural factors, including society's preoccupation with thinness, may play a role in eating disorders. This preoccupation is linked to dieting efforts, and dieting precedes the development of eating disorders among many people. In addition, the preoccupation with thinness, as well as media portrayals of thin models, predicts an increase in body dissatisfaction, which also precedes the development of eating disorders. Stigma associated with being overweight also contributes. Women are more likely to have eating disorders than men, and the ways in which women's bodies are objectified may lead some women to see their bodies as others do (self-objectify), which in turn may increase body dissatisfaction and eating pathology. Anorexia appears to occur in many cultures; bulimia appears to be more common in industrialized and Westernized societies. Eating disorders are slightly more common among white women than women of color, with the difference being most pronounced in college student samples. Eating disorders used to be more common among women of higher socioeconomic status, but this is less true today.

Research on personality characteristics finds that perfectionism may play a role. Other personality characteristics that predicted disordered eating across 3 years include body dissatisfaction, the extent to which people can distinguish different biological states of their bodies, and a propensity to experience negative emotions. Troubled family relationships are fairly common among people with eating disorders, but this could be a result of the eating disorder and not necessarily a cause of it. High rates of sexual and physical abuse are found among people with eating disorders, but these are not risk factors specific to the development of eating disorders.

Check Your Knowledge 9.2

True or false?

1. The brain structure linked to the cause of eating disorders is the hypothalamus.
2. Prospective studies of personality and eating disorders indicate that the tendency to experience negative emotions is related to disordered eating.
3. Anorexia appears to be specific to Western culture; bulimia is seen all over the world and is thus not culture specific.
4. Child abuse appears to be a specific causal factor for eating disorders.
5. Cognitive behavioral views of bulimia suggest that women judge their self-worth by their weight and shape.

Treatment of Eating Disorders

Hospitalization is frequently required to treat people with anorexia so that their ingestion of food can be gradually increased and carefully monitored. This was necessary for Lynne. Weight loss can be so severe that intravenous feeding is necessary to save the patient's life. The medical complications of anorexia, such as electrolyte imbalances, also require treatment. For both anorexia and bulimia, both medications and psychological treatments have been used.

Medications

Because bulimia nervosa is often comorbid with depression, it has been treated with various antidepressants, such as fluoxetine (Prozac). In one multicenter study, 387 women with bulimia were treated as outpatients for 8 weeks. Fluoxetine was shown to be superior to a placebo in reducing binge eating and vomiting; it also decreased depression and lessened distorted attitudes toward food and eating. Findings from most studies, including double-blind studies with placebo controls, confirm the efficacy of a variety of antidepressants in reducing purging and binge eating, even among people who had not responded to prior psychological treatment (Walsh et al., 2000; Wilson & Fairburn, 1998; Wilson & Pike, 2001).

On the negative side, many people with bulimia drop out of drug treatment (Fairburn, Agras, & Wilson, 1992). In the multicenter fluoxetine study cited, almost one-third of the women dropped out before the end of the 8-week treatment, primarily because of the side effects of the medication. In contrast, fewer than 5 percent of women dropped out of cognitive behav-

ioral therapy (Agras et al., 1992). Moreover, most people relapse when various kinds of antidepressant medication are withdrawn (Wilson & Pike, 2001), as is the case with most psychoactive drugs. There is some evidence that this tendency to relapse is reduced if antidepressants are given in the context of cognitive behavior therapy (Agras et al., 1994).

Medications have also been used to treat anorexia nervosa. Unfortunately, they have not been very successful in improving weight or other core features of anorexia (Attia et al., 1998; Johnson, Tsoh, & Varnado, 1996). Medication treatment for binge eating disorder has not been as well studied. Limited evidence suggests that antidepressant medications are not effective in reducing binges or weight loss (Grilo, 2007). Recent trials of antiobesity drugs, such as sibutramine and atomoxetine, show some promise in binge eating disorder, but additional clinical trials are needed.

Psychological Treatment of Anorexia Nervosa

Little in the way of controlled research exists on psychological treatments for anorexia nervosa, but we will present what appear to be the most promising of the psychotherapeutic approaches to this life-threatening disorder.

Therapy for anorexia is generally believed to be a two-tiered process. The immediate goal is to help the patient gain weight in order to avoid medical complications and the possibility of death. The patient is often so weak and physiological functioning so disturbed that hospital treatment is medically imperative (in addition to being needed to ensure that the patient ingests some food). Operant conditioning behavior therapy programs (e.g. providing reinforcers for weight gain) have been somewhat successful in achieving weight gain in the short term (Hsu, 1990). However, the second goal of treatment—long-term maintenance of weight gain—remains a challenge for the field.

Beyond immediate weight gain, psychological treatment for anorexia can also involve cognitive behavior therapy (CBT). One study that combined hospital treatment with CBT found that reductions in many anorexia symptoms persisted up to 1 year after treatment (Bowers & Ansher, 2008).

Family therapy is the principal form of psychological treatment for anorexia, based on the notion that interactions among members of the patient's family can play a role in the disorder (le Grange & Lock, 2005). In one kind of family therapy, anorexia is cast as an interpersonal rather than individual issue and attempts to bring the family conflict to the fore. How is this accomplished? The therapist holds family lunch sessions, since conflicts related to anorexia are believed to be most evident at mealtime. These lunch sessions have three major goals:

Family therapy is a main form of treatment for anorexia nervosa. (Michael Newman/PhotoEdit.)

1. Changing the patient role of the person with anorexia
2. Redefining the eating problem as an interpersonal problem
3. Preventing the parents from using their child's anorexia as a means of avoiding conflict

One strategy is to instruct each parent to try individually to force the child to eat. The other parent may leave the room. The individual efforts are expected to fail. But through this failure and frustration, the mother and father may now work together to persuade the child to eat. Thus, rather than being a focus of conflict, the child's eating will produce cooperation and increase parental effectiveness in dealing with the child (Rosman, Minuchin, & Liebman, 1975).

Family therapy has not yet been sufficiently studied for its long-term effects. One study of 50 girls being treated for anorexia with family therapy suggested that as many as 86 percent of the girls were still functioning well when assessed at times ranging from 3 months to 4 years after treatment (Rosman, Minuchin, & Liebman, 1976). A newer family-based therapy (FBT) was developed in England, and preliminary evidence suggests that it is effective (Lock & le Grange, 2001; Lock et al., 2001; Loeb et al., 2007).

Actress Mary-Kate Olsen has been treated for an eating disorder. (Peter Kramer/Getty Images News and Sport Services.)

Psychological Treatment of Bulimia Nervosa

Cognitive behavior therapy (CBT) is the best-validated and most current standard for the treatment of bulimia (Fairburn, 1985; Fairburn, Marcus, & Wilson, 1993). In CBT, people with bulimia are encouraged to question society's standards for physical attractiveness. People with bulimia must also uncover and then change beliefs that encourage them to starve themselves to avoid becoming overweight. They must be helped to see that normal body weight can be maintained without severe dieting and that unrealistic restriction of food intake can often trigger a binge. They are taught that all is not lost with just one bite of high-calorie food and that snacking need not trigger a binge, which will be followed by induced vomiting or taking laxatives that will lead to still lower self-esteem and depression. Altering this all-or-nothing thinking can help people begin to eat more moderately. They are also taught assertiveness skills to help them cope with unreasonable demands placed on them by others, and they also learn more satisfying ways of relating to people.

The overall goal of treatment in bulimia nervosa is to develop normal eating patterns. People with bulimia need to learn to eat three meals a day and even some snacks between meals without sliding back into bingeing and purging. Regular meals control hunger and thereby, it is hoped, the urge to eat enormous amounts of food, the effects of which are counteracted by purging. To help people with bulimia develop less extreme beliefs about themselves, the cognitive behavior therapist gently but firmly challenges such irrational beliefs as "No one will respect me if I am a few pounds heavier than I am now" or "Eric loves me only because I weigh 112 pounds and would surely reject me if I ballooned to 120 pounds." A generalized assumption underlying these and related cognitions for women might be that a woman has value only if she is a few pounds underweight—a belief that is put forth in the media and advertisements.

One intervention that is sometimes used in the cognitive behavioral treatment approach has the patient bring small amounts of forbidden food to eat in the session. Relaxation is employed to control the urge to induce vomiting. Unrealistic demands and other cognitive distortions—such as the belief that eating a small amount of high-calorie food means that the patient is an utter failure and doomed never to improve—are continually challenged. The therapist and patient work together to determine events, thoughts, and feelings that trigger an urge to binge and then to learn more adaptive ways to cope with these situations. In the case of Jill, she and her therapist discovered that bingeing often took place after she was criticized by her coach. Therapy included the following:

- Encouraging Jill to assert herself if the criticism is unwarranted
- Desensitizing her to social evaluation and encouraging her to question society's standards for ideal weight and the pressures on women to be thin—not an easy task by any means
- Teaching her that it is not a catastrophe to make a mistake and it is not necessary to be perfect, even if the coach's criticism is valid

The outcomes of cognitive behavioral therapies are rather promising, both in the short term and over time. A meta-analysis showed that CBT yielded better results than antidepressant drug treatments (Whittal, Agras, & Gould, 1999), and therapeutic gains were maintained at 1-year follow-up (Agras et al., 2000), nearly 6 years later (Fairburn et al., 1995), and 10 years later (Keel et al., 2002). But there are limitations to these positive outcomes, as we will see.

Findings from a number of studies indicate that CBT often results in less frequent bingeing and purging, with reductions ranging from 70 to more than 90 percent. Extreme dietary restraint is also reduced significantly, and there is improvement in attitudes toward body shape and weight (Compas et al., 1998; Richards et al., 2000). However, if we focus on the people themselves rather than on numbers of binges and purges across people, we find that at least half of those treated with CBT improve very little (Wilson, 1995; Wilson & Pike, 1993). Clearly, while CBT may be the most effective treatment available for bulimia, it still has room for improvement!

To better understand how it works and how it might be improved, some investigators are conducting what are called component analyses of the CBT therapy package just described. One important aspect that has been examined is the exposure and ritual prevention (ERP) component (recall this aspect of the cognitive behavioral treatment of obsessive-compulsive disorder

in Chapter 5). This ERP component involves discouraging the patient from purging after eating foods that usually elicit an urge to vomit. Indications are that this is an important component, because ERP and CBT combined appear to be more effective than CBT without ERP, at least in the short term (e.g., Fairburn et al., 1995). The ERP component may have its strongest effect early in treatment. A research review found that about 70 percent of the total improvement in frequency of bingeing and vomiting is evident by the third week of treatment (Wilson & Pike, 2001). ERP may not continue to be an advantage over the long term, however. One study examined outcome 3 years after treatment for people with bulimia who had received CBT either with or without ERP. They found similar outcomes for the two groups (Carter et al., 2003). That is, 85 percent of people with bulimia did not meet criteria for bulimia 3 years after treatment, regardless of which treatment they received.

People with bulimia who are successful in overcoming their urge to binge and purge also improve in associated problems such as depression and low self-esteem. This result is not surprising. If a person is able to achieve normal eating patterns after viewing bulimia as an uncontrollable problem, she can be expected to feel less depressed and to feel generally better about herself.

CBT alone is more effective than any available drug treatment (Compas et al., 1998; Walsh et al., 1997). But are outcomes better when antidepressant medication is added to CBT? Evidence on this front is mixed. Adding antidepressant drugs, however, may be useful in alleviating the depression that often occurs with bulimia (Keel et al., 2002; Wilson & Fairburn, 1998).

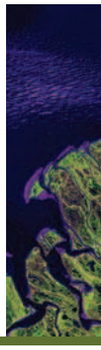
In several other studies (Fairburn et al., 1991, 1993), interpersonal therapy (IPT) fared well in comparisons with CBT, though it did not produce results as quickly. The two modes of intervention were equivalent at 1-year follow-up in effecting change across all four of the specific aspects of bulimia: binge eating, purging, dietary restraint, and maladaptive attitudes about body shape and weight (Wilson, 1995). This pattern—CBT superior to IPT immediately after treatment but IPT catching up at follow-up—was replicated in a later study (Agras et al., 2000).

Family therapy is also effective for bulimia, though it has been studied less frequently than either CBT or IPT. A recent randomized clinical trial demonstrated that family-based therapy was superior to supportive psychotherapy for adolescents with bulimia with respect to decreasing bingeing and purging up to 6 months after treatment was completed (le Grange et al., 2007).

Psychological Treatment of Binge Eating Disorder

Although not as extensively studied as with bulimia nervosa, cognitive behavior therapy has been shown to be effective for binge eating disorder in several studies (Grilo, 2007). CBT for binge eating disorder targets binges as well as restrained eating and other associated features (e.g., depression). Gains from CBT appear to last for up to 1 year after treatment. CBT also appears to be more effective than treatment with fluoxetine (Grilo, 2007). To date, one randomized controlled clinical trial has shown that interpersonal therapy (IPT) is equally effective as CBT for binge eating disorder (Wilfley et al., 2002).

Quick Summary



Antidepressant medications have shown some benefit for the treatment of bulimia, but not anorexia. However, people with bulimia are more likely to discontinue the medication than discontinue therapy. Psychological treatment of anorexia must first focus on weight gain. Family therapy is common for anorexia, but studies are needed to demonstrate whether this is effective. The most effective psychological treatment for bulimia is cognitive behavior therapy. CBT involves changing a patient's beliefs and thinking about thinness, being overweight, dieting, and restriction of food, with the overall goal being to reestablish normal eating patterns. Exposure plus ritual prevention is one CBT component that is effective early in treatment. CBT alone is more effective than medication treatment, though antidepressants can help lessen comorbid depression. CBT is also effective for binge eating disorder.

Preventive Interventions for Eating Disorders

A different approach to treating eating disorders involves prevention. Intervening with children or adolescents before the onset of eating disorders may help to prevent these disorders from ever developing. Broadly speaking, three different types of preventive interventions have been developed and implemented:

1. *Psychoeducational approaches*. The focus is on educating children and adolescents about eating disorders in order to prevent them from developing the symptoms.
2. *Deemphasizing sociocultural influences*. The focus here is on helping children and adolescents resist or reject sociocultural pressures to be thin.
3. *Risk factor approach*. The focus here is on identifying people with known risk factors for developing eating disorders (e.g., weight and body concern, dietary restraint) and intervening to alter these factors.

Stice, Shaw, and Marti (2007) conducted a meta-analysis of all such prevention studies conducted between 1980 and 2006, and they found modest support for some of these prevention approaches. The most effective prevention programs are those that are interactive rather than didactic, include adolescents age 15 or older, include girls only, and involve multiple sessions rather than just one session. Some effects appear to last as long as two years.

Prevention programs that are interactive have been effective for girls with eating disorders. (Tony Freeman/PhotoEdit.)

One recent randomized trial found that two types of preventive interventions show promise for reducing eating disorder symptoms among adolescent girls (average age of 17). One program, called the dissonance reduction intervention, focused on deemphasizing sociocultural influences; the other, called the healthy weight intervention, targeted risk factors (Stice et al., 2008). Both programs included just one 3-hour session. Specifically, girls in the dissonance reduction intervention talked, wrote, and role-played with one another to challenge the society's notions of beauty (i.e., the thin-ideal). Girls in the healthy weight intervention worked together on developing healthy weight and exercise programs for themselves. Participation in either program was associated with less negative affect, less body dissatisfaction, lower thin-ideal internalization, and lower risk of developing eating disorder symptoms 2 to 3 years after the session compared to girls who did not participate in a session. These findings point to the importance of continuing to develop and implement prevention programs.

Check Your Knowledge 9.3

Fill in the blanks.

1. Research suggests that _____ therapy is an effective treatment for bulimia, both in the short and long term.
2. For anorexia, _____ may be required to get the patient to gain weight. There are not many _____ that have been shown to be effective. The most common type of therapy used to treat anorexia is _____.
3. Research on prevention programs has shown that two programs show promise up to 3 years after the intervention: A _____ intervention and a _____ intervention.